

20-1025 (Lead); 20-1138 (Consolidated)

**UNITED STATES COURT OF APPEALS
FOR THE DISTRICT OF COLUMBIA CIRCUIT**

ENVIRONMENTAL HEALTH TRUST; CONSUMERS FOR SAFE CELL
PHONES; ELIZABETH BARRIS; THEODORA SCARATO

CHILDREN'S HEALTH DEFENSE; MICHELE HERTZ; PETRA BROKKEN;
DR. DAVID O. CARPENTER; DR. PAUL DART; DR. TORIL H. JELTER; DR.
ANN LEE; VIRGINIA FARVER, JENNIFER BARAN; PAUL STANLEY, M.Ed.

Petitioners

v.

FEDERAL COMMUNICATIONS COMMISSION;
UNITED STATES OF AMERICA

Respondents

Petition for Review of Order Issued by the
Federal Communications Commission

DEFERRED JOINT APPENDIX**VOLUME 7**

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341	9410-9411	Jul. 12, 2016	Dianne Black	Radiation Sickness & ADA/FHA; Dianne Black Comments
342	9412-9415	Jul. 13, 2016	Derek C. Bishop	Radiation Sickness & ADA/FHA; Derek C. Bishop Comments
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385	10013-10015	Aug. 13, 2013	John Puccetti	ADA/FHA; Organizations; American Academy of Environmental Medicine, Letter to the FCC
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US Exposures Limits - A History of Their Creation, Comments and
Explanations; Eng. Lloyd Morgan

US Exposures Limits

A History of Their Creation

Comments and Explanations

Historical: [ANSI C95.1-1982 Exposure-Limit Standard](#)

Existing: [IEEE C95.1-1991 Exposure-Limit Standard](#)

Introduction

More than four years ago the World Health Organization (WHO) declared cellphone radiation is a Group 2B (possible) Human Carcinogen substantially based on scientific studies which found statistically significant risks of brain cancer and a hearing nerve tumor called an acoustic neuroma.

An industry consultant's response: "Dr. Meir J. Stampfer, a professor at the Harvard School of Public Health and a consultant to the cellphone industry. 'In science, unlike math, we can't have absolute certainty, but in the scheme of things, this is not a health risk I would be concerned about at all.'" [NY Times, June 6, 2011]

In 1985 the United States government adopted an exposure limit from the American National Standards Institute (ANSI-an industry & military organization), ANSI C95.1-1982 Exposure Limit Standard. In 1996 the U.S. government adopted (updated from the ANSI Standard) the IEEE C95.1-1991 Exposure Limit Standard. IEEE, the Institute of Electrical and Electronic Engineers is substantially an industry and military organization.

What follows is the history of how these two standards were created, based on the actual text of these two standards

The History

This page highlights important understandings gleaned from a review of the the [ANSI C95.1-1982 Standard](#) and [the IEEE C95.1-1991 Standard](#).

Summary of Key Findings (within ANSI and IEEE Standards):

- **The authors understood that the mean (average) SAR value ignored "hotspots"** which can be many-fold higher than the mean SAR.
- **The authors were aware of the existence of "modulation-specific effects,** such as efflux of calcium ions" (resulting in harmful effects on cells), but because the authors were mostly military and/or associated with corporations with a vested interest in promoting microwave radiation, they *chose*, in effect, to ignore these results.

- **The authors were aware that other characteristics of the exposure** such as modulation frequency and peak intensity may pose a risk to health.
- **There is no 5-fold “safety level” for the general public.** With the IEEE Standards, the general public’s exposure was reduced 5-fold compared to electrical workers. However, the allowed averaging time was increased 5-fold for the general public compared to electrical workers and the end result was that the *the total allowed absorbed radiation is identical*. In effect this change *negates* the so-called 5-fold “safety level” for the general public.

Part 1: Highlights from the [ANSI C95.1-1982 Exposure Limit Standard](#)

Note: Pdf Pages 2-8 are missing in the OCR (Optical Character Recognition) version.

[Pdf page 3](#) lists the “Co-Secretariat: Institute of Electrical and Electronic Engineers, US Department of the Navy (Naval Electronic Systems Command);

Pdf page 5 is a Forward. However it states: “This Forward is not part of ANSI C95.1-1982, American National Standard Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 300 kHz to 100 GHz.”

It should be noted, in spite of its official sounding name, ANSI is not an a governmental entity.

1. **1. No medical organizations were included (beyond the military) nor any public health agency.** Please note these Members of the Committee (a partial listing) which reviewed and approved this standard. (pdf page 6) :

- American Petroleum Institute
- DoD (Defense Nuclear Agency)
- International Microwave Power Institute
- Office of the Assistant Secretary of Defense
- US Department of Commerce
- US Department of the Air Force School of Aerospace Medicine
- US Department of the Air Force Office of the Surgeon General
- US Department of the Army, Army Material Command’
- US Department of the Army, Army Environmental Hygiene Agency
- US Department of the Navy Bureau of Medicine and Surgery
- US Department of the Navy, Naval Sea System Command;

2. **Many Committee members were members of corporations and/or consultants to these corporations.** ([See ANSI pdf page 6](#) to read the full list of organizations and the name of person representing these organizations who reviewed and approved the standard. ANSI [Pdf page 7](#) names the members of the committee at the time the standard was approved.
3. **The mean (average) SAR value ignored “hotspots”** which can be many-fold higher than the mean SAR as noted by this statement.
 - “It is recognized, however, that a whole-body-averaged SAR is the mean of a distribution, the high side of which is an envelope of electrical hotspots.” (page 12)
4. **The exposure limit is based on an immediate (AKA acute) effect** where hungry rats trained to find food ceased looking for food. See the highlight below saying that a long-time (AKA chronic) exposure should be considered “an irreversible injury”.
 - “The assumption is that reversible disruption during an acute exposure is tantamount to irreversible injury during chronic exposure.” (Page 13)
5. **The authors were aware of “modulation-specific effects, such as efflux of calcium ions”** (resulting in harmful effects on cells), but because the authors were mostly military and/or associated with corporations with a vested interest in promoting microwave radiation, they *chose*, in effect, to ignore these results. Nevertheless, they felt obligated to report that *there were effects*. This is extraordinarily important.
 - “In addition, modulation-specific effects, such as efflux of calcium ions from brain materials were not considered adverse because of the inability of the subcommittee's members to relate them to human health. The narrow ranges of power density and the low and narrow range of modulation frequencies associated with field-induced efflux of calcium ions, and the authors' findings that the phenomenon is reversible, are factors that entered into the subcommittee's deliberations.” (Page 13, column 2)
6. **At a whole-body SAR_{WB}=4 W/kg, a potentially deadly, short-term (AKA acute) effect was found:** hungry rats trained to find food ceased eating. As noted above, a long-term (AKA chronic) exposure is “tantamount to irreversible injury.”
 - “**6.6 Safety Factor.** To ensure a wide margin of safety, an order-of-magnitude reduction in the permissible whole-body-averaged specific absorption rate (SAR) to 0.4 W /kg was invoked. This decision was nearly unanimous; one biological scientist dissented on the grounds that a specific justification should be given for the power-of-ten reduction. Different biological scientists offered different reasons, but beyond the need for a wide margin of safety, no quantitative justification was advanced. None of the members of the subcommittee offered an argument to widen the margin of safety [emphasis in original Pages 13-14].”

7. While it may appear that a 10-fold “Safety Factor” should be a sufficient reduction to provide protection against harm, Fig. A3 (page 24) contradicts this appearance.

See below:

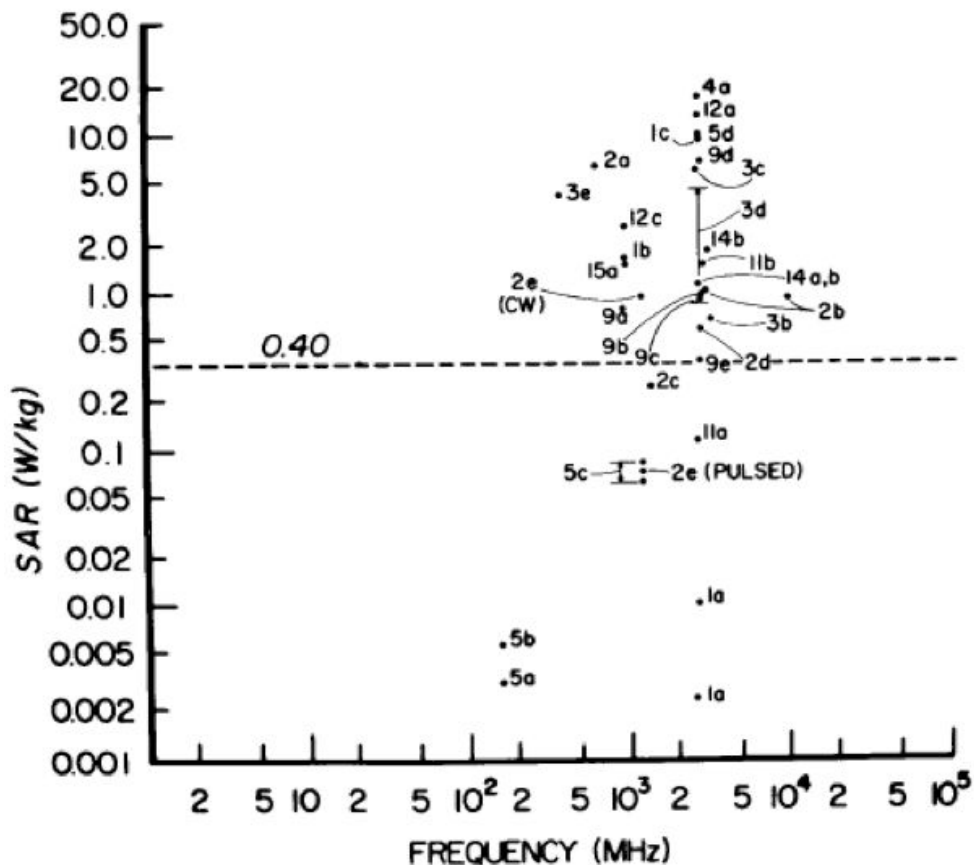


Fig A3
Whole-Body-Averaged SAR
Corresponding to Biological Effects Reported in
Various References of Appendix

- Fig A3 Explained (Page 24)
- The dashed line represents the 10-fold “safety” factor exposure level ($SAR=0.40$ W/kg) from the $SAR=4$ W/kg whose long-term exposure is tantamount to an irreversible injury.
- The references in this figure refer to studies found in the Appendix (pages 17-19). For example: “4a” refers to “A4. Teratology BERMAN, E., KIN, J. B. and CARTER, H. B. Observations of Mouse Fetuses After Irradiation with 2.45 GHz Microwaves. Health Physics, 35, 1978, pp 791-801 on page 18; “9d” refers to the 4th study in the section “A9. Thermoregulation and Metabolism,” “LU, S. and T., LEBDA, N., MICHAELSON, S. M., PETTIT, S. and RIVERA, D. Thermal and

Endocrinological Effects of Protracted Irradiation of Rats by 2450 MHz Microwaves. Radio Science, 12(6S), 1977, pp 147-156.” Found on page 18.

- **Note that there are 8 references far below the “safety” factor, some of these studies had exposures 100 times lower than the “safety factor.** Reference 5 is one of these studies. Its title is: “Induction of Calcium-Ion Efflux from Brain Tissue by Radio-Frequency Radiation: Effects of Modulation Frequency and Field Strength.”
 - Calcium levels are critically important for the viability of human cells. Efflux is defined as “the flowing out of a particular substance or particle.”
 - The exposure level is on the order of a SAR=0.005 W/kg--8-fold lower than the “safety” factor (AKA limit). Why was the “safety” factor not set 10-times lower than the level where calcium flowed out of brain cells? That is, SAR=0.0005 W/kg?
8. **Figure A3 shows that we knew *more than 34 years ago* that the “safety factor” does not protect us from biological effects!**
- As we see in Fig. A3, a SAR_{WB}=0.4 W/kg “safety” factor ignored studies which found adverse effects from below the “safety” factor established by ANSI-C95.1-1982.
9. **The authors were aware that other characteristics of the exposure could be a health risk.**
- It is clearly stated that modulation frequency and peak intensity may pose a risk to health. See the quote below:
 - **Other Factors.** It was recognized by the subcommittee that the specific absorption rate (SAR), which provides the basis for limiting power densities, does not contain all of the factors that could be of importance in establishing safe limits of exposure. First, *other characteristics of an incident field such as modulation frequency and peak intensity may pose a risk to health* [emphasis in the original; emphasis added].” (Page 14, column 2)
10. **It was well understood prior to 1982 that peak SARs can be far higher than averaged SARs.**
- “By implication and demonstration, peak SARs in a biological body can range *more than an order of magnitude above the average SAR* over a limited mass of the exposed tissue.” (Page 15, column 2, *emphasis added*)
11. **The Committee retained 6 minutes of averaged exposure relying on the false assumption that the only adverse biological effects from exposure to non-ionizing radiation (NIR) is heating.**
- “**6.12 Time Averaging.** The subcommittee retained 0.1 h (6 min) as the significant period of time over which exposures, the values of specific absorption

rates (SAR) and input power are to be averaged.” (Page 16, column 2. **emphasis** in the original)

Part 2: Highlights From The IEEE C95.1-1991 Standard^{III}

1. **Language in the second paragraph found on [pdf page 2](#) is of interest as the “wholly voluntary” wording appears to absolve the IEEE from any consequences.**
“Use of an IEEE Standard is *wholly voluntary*. The existence of an IEEE Standard does not imply that there are no other ways to produce, test, measure, purchase, market, or provide other goods and services related to the scope of the IEEE Standard. Furthermore, the viewpoint expressed at the time a standard is approved and issued [*emphasis added*].”
2. In essence IEEE took over from ANSI but the membership was nearly identical. See the Forward ([pdf page 3](#)) which states
 - Prior to 1988, C95 standards were developed by an accredited standards committee C95, and submitted to ANSI for approval and issuance as ANSI C95 standards. Between 1988 and 1990, the committee was converted to Standards Coordinating Committee 28 under the sponsorship of the IEEE Standards Board. In accordance with policies of the IEEE, C95 standards will be issued and developed as IEEE standards, as well as being submitted to ANSI for recognition.

Major Changes From the ANSI c95.1-182 Standard

A review of this document shows the following major changes (or lack of changes) from the previous ANSI C95.1-1982 standard were the following:

1. **The supposed creation of *two exposure limit values* has the end result of identical exposure limits in that the allowed Specific Absorption (SA)—in other words, despite the two types of limits- the total allowed radiation absorption—*is identical* between electrical workers and the general public.**

How did this happen? Please note the following:

- As will be seen below, the general public’s exposure was reduced 5-fold compared to electrical workers. However, the allowed averaging time has increased 5-fold for the general public compared to electrical workers.
- The introduction of “spatial-peak SAR” (SAR_{sp}) was added in addition to the previous whole-body SAR (SAR_{wb}). The former occurs when the radiation source is close to the body such that only a portion of the body absorbed the radiation (e.g., holding a cellphone to the ear); the latter is when the radiation is far from the body such that the whole body absorbed the radiation (e.g., a cellphone tower).

- For both electrical workers and the general public the SAR_{ps} is 5-fold higher than the SAR_{wb} .
- For the general public the allowed $SAR_{wb}=0.08$ W/kg and the allowed $SAR_{sp}=1.6$ W/kg.

In other words, the brain (and other similarly sensitive tissues) is allowed to absorb 20 times more radiation than was allowed by the ANSI C 95.1-1982 Standard. In addition a new exposure limit for the hands, wrists, feet and ankles was defined.^[2]

Unlike the 1982 ANSI standard, this IEEE standard introduced the concept of Maximum Permissible Exposure (MPE). Because the MPEs do not directly refer to the Specific Absorption Rate (SAR), but list values for the electric field, magnetic field, and power density, we will only focus on the SAR values along with the two exposure limits.

Please note the following highlights from the text of the document.

“Abstract: IEEE C95.1-1991 gives recommendations to prevent harmful effects in human beings exposed to electromagnetic fields in the frequency range from 3 kHz to 300 GHz.^[3] The recommendations are intended to apply to exposures in controlled, as well as uncontrolled, environments.” (Page 1)

“Also, a distinction is made between controlled and uncontrolled environments relative to safe exposure limits [emphasis added].” (Page 4)

From the “Definitions” section:

“continuous exposure. Exposure for durations exceeding the corresponding averaging time. Exposure for less than the averaging time is called short-term exposure [emphasis in the original].”

“controlled environment. Controlled environments are locations where there is exposure that may be incurred by persons who are aware of the potential for exposure as a *concomitant of employment* ... [emphasis in the original; emphasis added].”

Concomitant of employment is that worker must accept higher exposure if they want to work.

“RF ‘hot spot’. A highly localized area of relatively more intense radio-frequency radiation that manifests itself in two principal ways [emphasis in the original]:

- (1) The presence of intense electric or magnetic fields immediately adjacent to conductive objects that are immersed in lower intensity ambient fields (often referred to as re-radiation), and
- (2) Localized areas, not necessarily immediately close to conductive objects, in which there exists a concentration of radio-frequency fields caused by reflections and/or narrow beams produced by high-gain radiating antennas or other highly directional sources. In both cases, the fields are characterized by very rapid changes in field strength with distance. RF hot spots are normally associated with very nonuniform exposure of the body (partial body exposure). This is not to be confused with an actual thermal hot spot within the absorbing body.”

The reality that “hot spots” will exist is noted but it is interesting to note that no statement is made about exposure limits being applicable to “hot spot” regions.

“**4.2.1 Controlled Environment.** At frequencies between 100 kHz and 6 GHz, the MPE in controlled environments for electromagnetic field strengths *may be exceeded if*:

- (a) the exposure conditions can be shown by appropriate techniques to produce SARs below 0.4 W/kg as averaged over the whole-body and spatial peak SAR, not exceeding 8 W/kg as averaged over any 1 g of tissue (defined as a tissue volume in the shape of a cube), except for the hands, wrists, feet and ankles where the spatial peak SAR shall not exceed 20 W/kg, as averaged over any 10 g of tissue (defined as a tissue volume in the shape of a cube), ...

The SARs are averaged over any 6-min interval [emphasis in the original].” (Page 17)

The end result of these changes is that:

1. The spatial-peak SAR hands, wrists, feet and ankles^[4] was increased by 250% from $SAR_{sp}=8$ W/kg to 20 W/kg, and the tissue volume was increased 10-fold from 1 gram to 10 grams of tissue. Because a 10-gram SAR can be up to 3-times higher within any 1 gram of tissue, the effect was to *increase the spatial-peak SAR 7.5-fold* ($2.5 \times 3=7.5$). For electrical workers who concomitant to their employment must accept the 5-fold higher exposure.
2. Uncontrolled environments allow a 5-fold lower exposure for the general public.

- “**4.2.2 Uncontrolled Environments** At frequencies between 100 kHz and 6 GHz, the MPE in uncontrolled environments for electromagnetic field strengths *may be exceeded if*:
 - The exposure conditions can be shown by appropriate techniques to produce SARs below 0.08 W/kg, as averaged over the whole body, and spatial peak SAR values not exceeding 1.6 W/kg, as averaged over any 1 g of tissue (defined as a tissue volume in the shape of a cube), except for the hands, wrists, feet and ankles where the spatial peak SAR shall not exceed 4 W/kg, as averaged over any 10 g of tissue (defined as a tissue volume in the shape of a cube) ... [**emphasis** in the original; *emphasis* added].” (Page 17)
- However, like for electrical workers, the spatial-peak SAR for hands, wrists, feet and ankles^[5] was increased by 250% from $SAR_{sp}=1.6\text{W/kg}$ to 4 W/kg, and the tissue volume was increased 10-fold from 1 gram to 10 grams of tissue. Because a 10-gram SAR can be up to 3-times higher within any 1 gram of tissue, the effect was to increase the spatial-peak SAR 7.5-fold.
 - The above limits are also applicable for the general public.
 - “For both pulsed and non-pulsed fields ... the SARs, as applicable, are averaged over any 6-min or 30-min period.” (Page 18)
 - As noted in the ANSI section above, because for electrical workers the 5-fold higher SARs are averaged over 6 minutes and for the general public the 5-fold lower SARs are averaged over a 5-fold longer time (30 minutes), *the total allowed absorbed radiation is identical*.
- 3. **The document states that *Non-thermal effects*, such as efflux of calcium ions from brain tissues, are mentioned as potential health hazards:**
 - “To some, it would *appear attractive and logical* to apply a larger, or different, safety factor to arrive at the guide for the general public. Supportive arguments claim subgroups of greater sensitivity (*infants, the aged, the ill and disabled*), *potentially greater exposure durations* (24-hr/day vs. 8-hr/day), *adverse environmental conditions* (excessive heat and/or humidity), *voluntary vs. involuntary exposure*, and psychological/emotional factors that can range from anxiety to ignorance. *Non-thermal effects*, such as efflux of calcium ions from brain tissues, are also mentioned as potential health hazards [*emphasis* added].” (Page 23)
- 4. **The document states that spatial peak SAR values can exceed a whole-body average value.**
 - “It has been established through *thermographic* analyses of models of rats and man, and cadavers of rabbits, that spatial peak SAR values can exceed a whole-body average value by more than a factor of 20. Comparable findings have

been reported. However, several factors preclude explicit use of peak SAR, such as the following:

- (1) The availability of data on distributive SARs is limited ... [*emphasis added*]” (Pages 24-25)
- “[T]hermographic” (i.e., temperature change) analyses show a 20-fold higher SAR underestimates the instantaneous SAR value. This is of critical importance, because virtually all adverse biological effects reported since 1991 are non-thermal effects [e.g., various human cancers, deleterious effects on sperm (in vitro, in vivo, and human epidemiological studies), double-strand DNA breaks (in vivo studies)].

L. Lloyd Morgan
Senior Research Fellow
Environmental Health Trust

More than four years ago the World Health Organization (WHO) declared cellphone radiation is a Group 2B (possible) Human Carcinogen substantially based on scientific studies which found statistically significant risks of brain cancer and a hearing nerve tumor called an acoustic neuroma.

An industry consultant’s response: “Dr. Meir J. Stampfer, a professor at the Harvard School of Public Health *and a consultant to the cellphone industry*. ‘In science, unlike math, we can’t have absolute certainty, but in the scheme of things, this is not a health risk I would be concerned about at all.’” [NY Times, June 6, 2011]

^[1] This became ANSI/IEEE C95.1-1992. The only change was the American National Standards Institute (ANSI) ratified the IEEE C95.1-1991 Standard. In 1996, the Federal Communications Commission (FCC) substantially adopted the ANSI/IEEE C95.1-1992 as the U.S. Government’s exposure limit regulation.

^[2] Recently the FCC defined the ear (AKA pinna) as an extremity like hands, wrists, feet and ankles.

^[3] 3 kHz to 300 GHz is the IEEE definition of the radio frequency (RF) spectrum range. It is only a convenient definition for engineers. Per se, there is no biological meaning.

4 Recently the FCC defined the ear (AKA pinna) as an extremity like hands, wrists, feet and ankles.

4 Recently the FCC defined the ear (AKA pinna) as an extremity like hands, wrists, feet and ankles.

Biosystem & Ecosystem; Birds, Bees and Mankind: Destroying Nature
by 'Electrosmog': Effects of Mobile Radio and Wireless Communication.
Dr. Ulrich Warnke, PhD., 2007



BEEES, BIRDS AND MANKIND

Destroying nature by “electrosmog”

Ulrich Warnke

Effects of Mobile Radio and Wireless Communication

A series of papers by the
Competence Initiative for the Protection of Mankind,
Environment and Democracy

Brochure 1

Effects of mobile radio and wireless communication

A series of papers by the Competence Initiative for the Protection of Mankind,
Environment and Democracy

Published by Prof. Dr. med. Karl Hecht, Dr. med. Markus Kern,
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Brochure 1

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Kempton, November 2007

Competence Initiative

for the Protection of Mankind, Environment and Democracy

The founding programme in brief

The Competence Initiative for the Protection of Mankind, Environment and Democracy was founded in the beginning of May 2007. It is an association of independent scientists, doctors, technicians and environmentally committed organisations. It operates interdisciplinary, throughout Germany and internationally.

Its foundation was driven by the observed culture deficits in the health, political, environmental and democratic fields.

It criticises:

- the irresponsible electromagnetic overloading of our living space;
- inadequate protection of consumers and environment;
- unconstitutional alliances of state and industry, forcing hazards to health, loss of values and displacement upon more and more people.

It demands:

- acknowledgement and promotion of industry-independent research by those politically responsible;
- sustainable consumer and environmental protection, possible only on this basis;
- a democratic culture ensuring adherence to laws for the protection of citizens under the constitution and the European Commission for Human Rights.

If you wish to support this programme, you have three options:

- to join the Competence Initiative and actively work together with us;
- to let us know that you agree with the values and targets in the founding statement;
- to support our work, which costs a considerable amount in terms of information material, legal actions, organisation of conferences, etc., with a contribution.

Our banking details:

Raiffeisenbank Kempten
Acc. no. 1020 -102, Bank code 733 699 02

Keyword: "Kompetenzinitiative"

The full text of the *Competence Initiative for the Protection of Mankind, Environment and Democracy* programme can be viewed at www.kompetenzinitiative.de, where we also inform you of important results of our work and suggest options for cooperation. The programme is also available as a reprint (see left-hand side).

The launch of the new series follows the publication of two scientific papers to which the Competence Initiative has made contributions:

Dr. med. Wolf Bergmann
and Dr. med. Horst Eger:

Mobile radio effects on human health.
Conclusions and demands from a medical point of view.

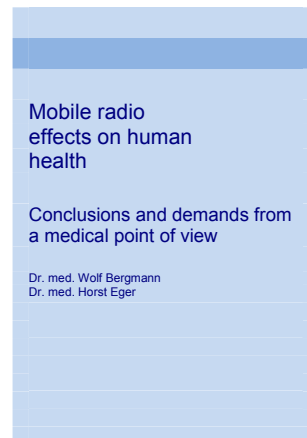
Published by the *Association for the Protection of the Population against Electrosmog* and by the *Competence Initiative for the Protection of Mankind, Environment and Democracy*, Stuttgart, 2007.

In this paper, the two renowned physicians show how the technical information in mobile radio and wireless communication is biologically false information to living organisms – based on the state of the information of independent science and medicine.

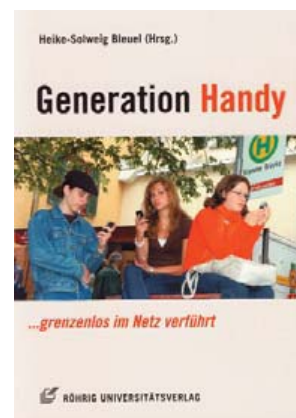
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Heike-Solweig Bleuel (Publisher):
Generation Handy
... grenzenlos im Netz verführt
St. Ingbert (Röhrig Universitätsverlag)
2007



This publication, substantially supported by members of the Competence Initiative, highlights the seduction, endangerment and impairment of children and the youth from a number of perspectives. It intends to give a voice to an endangered generation that believes in the state's duty to protect and is therefore falling victim to advertising and abuse.

More: www.generation-handy.de

Price: € 19.80

ISBN 978-3-86110-432-2

BEES, BIRDS AND MANKIND

Destroying nature by “electrosmog”

Ulrich Warnke

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For different protection of mankind, environment and democracy

Preamble by the publishers at the launch of the series of papers: *Effects of Mobile Radio and Wireless Communication*

The bio-scientist Ulrich Warnke is more familiar with nature's electromagnetic housekeeping than most. In this paper, he shows how wise and sensitive nature was about using electrical and magnetic fields in the creation of life. But he can for this reason also convincingly criticise the present foolish and irresponsible interference in nature's housekeeping. It is clear from his paper that the powers that be in politics, the economy and science are in the process of destroying what nature has built up over millions of years. The traces of this destruction have long been evident in our living environment. The paper shows, however, how short-sightedly we are treating not only our health and the economy, but especially also future generations' right to life.¹ All of the above is documented not as probabilities but based on reproducible effects. This should give pause also to those who regularly justify their actions with the argument that they are unaware of any proof of damage.

Under the term "radio communication", we combine all wireless communication technology, increasingly flooding our residential zones and the environment with electromagnetic fields. A recent, comprehensive research report by the *BioInitiative Working Group*, a consortium of renowned international

scientists, shows how many of the damaging effects of such fields have already been proven (www.bioinitiative.org). The report evaluates the present limiting values as a useless edifice, protecting nobody. Based on this, the European Environment Agency (EEA), the top scientific environment authority of the EU, has warned of the possibility of looming environmental disasters following the increasing density of electromagnetic fields. And the coordinator of the European Reflex project, Prof. Franz Adlkofer, has informed the public on the new research results, proving the high degree of gene-toxicity of UMTS radiation.

The public is little aware of these risks because they are hardly addressed in the "enlightenment" provided by officialdom and industry. The public is given the assurance that they are well-protected by the limits and the compliance-assuring measurements and that UMTS radiation is as harmless as GSM radiation – more antennae in residential areas are recommended in principle.² And whilst Ulrich Warnke demonstrates how vulnerable man and environment are, we are told that we are more robustly organised than our machines.³ The original "radiation protection" has deteriorated to the protection of commercial interests.

The involvement of government in industry and the high percentage of industry-financed research and industry-beholden panels and consultants, have spawned a questionable system of environment and consumer protection. Only that which does not seriously endanger common commercial interests is noted and supported. The rights of the citizen to protection and the suffering of the people are flatly ignored. Those with political responsibility have apparently still not realised that their negligent handling of the obligation to take precautions has long since been proven to be one of the main causes of past environmental disasters and scandals.⁴

As a result of their quarrel with politics of carelessness, an interdisciplinary association of scientists and physicians founded the *Competence Initiative for the Protection of Mankind, Environment and Democracy* in May 2007 (www.kompetenzinitiative.de). This paper is the first in a new scientific series. The reported results are intended as a correction to trivialising "enlightenment" that does not protect, but endangers. The series intends to maintain a high level of technical information, without being unreadable to the interested layman.

¹ On injury to the health of children and the youth refer also to the collection published by Heike-Solweig Bleuel "Generation Handy... grenzenlos im Netz verführt", St. Ingbert 2007.

² Quoting scientists of the Jacobs University Bremen-Grohn under Prof. Alexander Lerchl: "UMTS doch nicht schädlicher als GSM", www.pcmagazin.de, 2.7.2007, and A. Lerchl at a presentation in Ritterhude acc. to a newspaper report of the Osterholzer Kreisblatt dated 16.6.2007: "More radio masts in the centre of town". Professor Lerchl appeals to all communities not to spend further tax money on mobile radio studies

³ Statement at the end of a brochure: *Mobilfunk und Funkwellen: Information, Fakten, Antworten*; published by the Saarland Department of Justice, Health and Social matters, Saarbrücken 2005 (copy of a brochure of the LfU Baden-Württemberg).

⁴ Compare the paper published by the European Environmental Agency and its German translation published by the Federal environmental office: "Späte Lehren aus frühen Warnungen: Das Vorsorgeprinzip 1896-2000", Copenhagen and Berlin 2004.

⁵ "Die große Gier. Korruption, Kartelle, Lustreisen: Warum unsere Wirtschaft eine neue Moral braucht", Berlin 2007.

Placing economic interests above culture and morality has contributed significantly to turning Germany into a country of declining education. As the journalist Hans Leyendecker so tellingly describes in his book *Die große Gier*⁵, it started Germany too on a new career on the ladder of corruption. There is nothing that the business location Germany needs more, he concludes, than “new ethics”. But this also requires a different perception of progress. Whether we can watch TV via our mobile telephone, is irrelevant to our future. Our future will depend on whether we can return to more human, social and ethical values again in the shaping of our lives and our relationship with nature.

Everyone who thinks beyond today and who inquires about what it means to be human is, in our opinion, called upon to contribute to this future: politicians guided by values rather than economical and tactical election issues; scientists and doctors more often remembering their obligation to the wellbeing of society and mankind; companies understanding, also in Germany, that profit and morality must be in harmony if they wish to remain successful in the long term.

But what we need above all is critical citizens, who can spot the difference between technical progress and consumer foolishness: Citizens who, in both their roles as voters and consumers, remember that democracy once meant rule of the people, not ruling the people.

The dramatic escalation of recorded degradation challenges those with political responsibility to take to heart the protection directives of the constitution and the European Convention on Human Rights. To base your actions affecting millions of your protégés on a half truth, at best, appears to us a political crime affecting health and the future – considering the state of our knowledge.

Religious and ethical cultures still profess to the mandate of conserving creation. But its actual treatment is guided by the pseudo culture of a new class of masters who ruthlessly exploit and manipulate the organisation, finally destroying it.

Prof. Dr. Karl Hecht

Dr. med. Markus Kern

Prof. Dr. Karl Richter

Dr. med. Hans-Christoph Scheiner

Electromagnetic fields as prerequisite and hazard to life

Author's introduction to this paper

The question of causal effects and biological relevance of electrical and magnetic parameters is generally posed without simultaneous reference to their relevance to life's organisation. These questions cannot, however, be considered in isolation of each other. What role have the electrical and magnetic fields played in the evolution of life on earth? What role are they playing in the individual development and physiological capacities of an organism? Whoever investigates these questions must sooner or later conclude: Not only did the electrical and magnetic fields of our planet exist before all life, but they have had a decisive hand in the evolution of the species – in water, on land and in the near-earth atmosphere. Living creatures adapted to it in the development of their kind.

Biological experience teaches us that life will use the energy pool in which it finds itself to its best advantage. Advantageous not only because the absorbed energy is a carrier of information, useful for orientation in the environment (see glossary; hereinafter GL). But advantageous also because the organism developed to make use of gravitational and electromagnetic interactions, creating decisive functionalities of life. The biological system expresses itself just as the environment does and unity and coordination with its environment is its guiding principle.

But if bees and other insects disappear, if birds are no longer present in their traditional territories and humans suffer from inexplicable functional deficiencies, then each on its own may appear puzzling at first. The apparently unrelated and puzzling phenomena actually have a common trigger, however. Man-made technology created magnetic, electrical and electromagnetic transmitters which fundamentally changed the natural electromagnetic energies and forces on earth's surface – radically changing million-year-old pivotal controlling factors in biological evolution.

This destruction of the foundations of life has already wiped out many species for ever. Since this extinction of species mostly affected ecological niches and hardly ever own life, most of us were not interested. But now, the endangerment of animals is also threatening the survival of man in a new and unexpected way.

Animals that depend on the natural electrical, magnetic and electromagnetic fields for their orientation and navigation through earth's atmosphere are confused by the much stronger and constantly changing artificial fields created by technology and fail to navigate back to their home environments. Most people would probably shrug this off, but it affects among other one of the most important insect species: the honeybee.

Because the bee happens to be the indispensable prerequisite for fructification: without bees, the fruit, vegetable and agricultural crops will fall short.

We are, however, not only affected by the economic consequences of our actions. It can also be proven that the mechanisms evidently affecting birds and bees are also affecting the human organism. An all-round unnatural radiation with an unprecedented power density (GL) is also harming human health in a novel way.

But, unless mankind reminds itself of the basics of its existence and unless the politicians in charge put a stop to the present development, the damage to health and economic fundamentals is predictable and will fully manifest itself not now, but in the next generation.

The reasons for this are explained in this paper. It endeavours to quantify natural electrical and magnetic signals provided to men and animals as guiding signals throughout evolution. The paper, however, places particular emphasis on what happens when these natural signal amplitudes are suppressed, changed and distorted on an unprecedented scale by technically generated artificial fields. Mankind can only take successful countermeasures if the damage mechanisms are understood.

The following analyses are intended to remain readable also for interested laymen. This approach has its limits where experimental fundamentals or specific technical descriptions must be included. The following text therefore offers three options for reading. In its totality, it is intended for readers with a scientific background. It has, however, also the interested layman in mind, by allowing him to skip identified parts containing specific technical justification and arguments. And the parts against a coloured background are intended as a first overview.

I thank Prof. Dr. Karl Richter for the editorial supervision of the paper and Dipl.-Met. Walter Sönning, medical meteorologist, for his technical comments on the sferics question and the compilation of a glossary for the interested layman.



If all the functions bees perform for natural life and its preservation are observed holistically, their importance cannot be overstated. Without the bees, we humans will also suffer major deficiencies.

1. The organisation of life underlying its vulnerability

1.1 We should have known long ago

The relationship between life and the physical parameters of earth's surface and atmosphere have been known for many decades. Those responsible therefore had the opportunity long ago to question to what extent the excesses of technically created electrical and magnetic fields might have the potential to destroy nature's housekeeping.

There are only two types of energy capable of transmitting information over great distances: electromagnetic and gravitational energy. Any forces acting beyond the boundaries of an atom can be traced back to these two energies; ultimately they have an infinite reach. Both energies are universally present and can be modulated in many ways (GL). This is true, for instance for light, the earth's magnetic field, cloud charges, atmospheric electric fields and changes in atmospheric pressure. Together with atmospheric moisture and olfactory particles, they are recognised as orientation aids to mobile organisms.

In the natural environment, there are "oscillating" electromagnetic fields of many orders of magnitude and with frequencies ranging over a virtually unlimited spectrum covering many frequency decades. They manifest themselves as a continuous and enormous "hiss" – like an unlimited ocean, the surface of which is agitated by waves of any imaginable amplitude and extent. Nature has created senses that filter out very specific frequencies and intensities from this ocean of waves, analyse them and convert them to forces.

These filtered frequencies identify a specific sphere of life for specific life forms.

Only those energies that are important to the life of an animal are transformed. The forces generated from these energies control nerve cell membranes and protein structures such as enzymes – creating patterns, images and impressions that we call experience. Sensory organs are organs functioning as frequency analysers (GL), information amplifiers (GL) with gains up to a million, sometimes including contrast enhancement and noise suppression. Eyes, ears, sense of smell, taste, sensitivity of touch, light, warmth, chemical, electrical, magnetic and pain receptors. The living world perceives stimuli such as light (including ultraviolet and infrared), sound (including ultrasound and infrasound), electrical fields and currents, magnetic fields and also smells and water currents. And the sensory performance of animals is often comparable to our technical measurement apparatus, sometimes even far superior. Physiologists can prove this by some astounding numbers: Snakes, for instance, can sense temperature variations of a thousandth of a degree centigrade; long-horned grasshoppers and cockroaches can register mechanical surface vibrations with amplitudes (GL) down to $1/25^{\text{th}}$ of the diameter of a hydrogen atom.

The high "intelligence of the systems" is particularly obvious, however, with orientation, navigation and early warning systems. In this regard, the earth's magnetic field has an important role to play. The local geographic position and time of day can be established from the density,

direction and inclination of the field lines and their temporal variation.

Every location, together with other physical information, has a specific identifiable pattern. The sensitive reception apparatus of animals use the magnetic field information for orientation and navigation, among other (WARNKE, 2006).

1.1.1 Magnetic fields as global parameter for space and time orientation of all life

To the best of our present knowledge, biological organisms depend less on static magnetic fields than on the very important intensity variations of sufficiently high frequency. If we take a closer look at such variations, the earth's magnetic field cannot be considered in isolation. Other magnetic fields must also be included in the analysis: such as the ionospheric field, for instance, and the field of the Van Allen belt – a radiation belt of very high intensity with rotational symmetry around the magnetic axis and mirror symmetry around the magnetic equator around the earth. Both the ionosphere and the Van Allen belt are held together by earth's magnetic field. The protons and electrons captured from the cosmic radiation or the solar wind (= stream of ionised particles emanating from the sun) by earth's magnetic field, create a protective shield for all life on earth – the Van Allen radiation belt.

The external magnetic fields act as moderators (GL) on the earth's magnetic field. They exhibit both a pronounced solar and also a lunar (moon-dependent) diurnal variation. The reason for the solar-induced variation lies in the diurnal warming of the atmosphere through solar radiation. This is accompanied by horizontal eddy currents with amplitudes up to 90 000 Ampere in the ionosphere, that generate magnetic fields again. These daily variations also have a pronounced annual cycle.

The moon-dependent variations are furthermore only evident during the day. These are also generated by electrical currents at about 100 km altitude, but they have current amplitudes of "only" 10 000 Ampere. These eddy currents cannot be explained by temperature gradients as with the solar effects, but they are influenced by the gravitational remote action of the moon. The earth's atmosphere is rocked to and fro inside the earth's magnetic field in the rhythm of the tides, inducing electrical currents in the ionised layers of the upper atmosphere where the conductivity is high through the presence of the negatively and positively charged particles (ions). It appears that the conductivity of the ionosphere is too low at night to maintain induction processes (GL) – due to reduced ion densities (WARNKE 1993).

Within the realm of the conventional variations of the magnetic field that have been explained so far, electromagnetic oscillations that occur in mainly two frequency bands also deserve to be mentioned: 10 Hz and 10-25 kHz. On the one hand, there is a resonating electromagnetic oscillation between earth and ionosphere in the 10 Hz region (Schumann resonance, 7.83 Hz) and on the other, thunderstorm activities on earth constantly reinforce certain electromagnetic oscillations. The dominant frequency of approximately 10 kHz generated by vertical lightning flashes corresponds to a transmitter dipole of cloud-to-earth length, whilst horizontal cloud-to-cloud lighting generates about 20 kHz.

These characteristics may be exploited for the design of thunderstorm warning apparatus. Our device measures the thunderstorm activity in a range of at least 800 km and simultaneously also the activity within a range of 200 km. Under favourable conditions, we can therefore register thunderstorms over the Mediterranean from our location in Saarbrücken.

Lightning also simultaneously generates very low frequency electromagnetic oscillations. Under certain conditions, these oscillations are all guided through the ionosphere along the magnetic field lines, travel far into space and return to earth along the opposite magnetic field lines. They are reflected at the ground and the waves travel the same way again and again until their energy is dissipated. The higher frequencies are propagated somewhat faster than the lower ones. If this process is made audible through an amplifier, a whistling noise is heard, continuously decreasing in frequency down to a hum, as in a switched-off siren, but much faster, typically for approx. 1/3 of a second. This phenomenon was therefore called "Whistler".

The so-called earth-magnetic storms (magnetic induction $B \sim 1 \mu T$) are triggered by the magnetic shock waves escaping from the solar flare region at 2000 km/sec and still have a speed of 100 km/sec as they reach earth. This induces unusually high currents in the earth's magnetic field, which in turn change the earth's magnetic field and generate secondary currents. Such currents manifest themselves in long conductive paths such as pipelines, transmission lines etc. and routinely cause technical headaches.

The most important parameters, constant over millions of years, are: earth's static magnetic field: $31 \mu T$ (geomagnetic equator); resultant diurnal variation of the earth's field: 60 nT; magnetic storms: 500 nT; sferics field strengths: 0.25 – 3.6 pT per \sqrt{Hz} .

The natural high frequency radiation sources have far less energy than the technically generated transmission powers and energies. This is a precondition for transmission of news and communication.

The integrated power density over all frequencies up to 300 GHz is 600-800 pW/m² at the earth's surface. The power density of the microwave solar radiation is about $0.1 \mu W/m^2$, escalating to several $100 \mu W/m^2$ during solar flares.

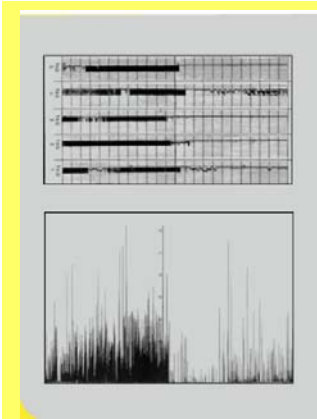


Fig. 1 Top: The "midnight phenomenon". The activity of the electromagnetic impulses (shown on 5 different days) abruptly ends at midnight.

Ref. Hans Baumer: (1987) Sferics. Die Entdeckung der Wetterstrahlung. Rowohlt, Hamburg

Bottom: Our original recorded activity cycles of 20 caged bees in a laboratory experiment. The vertical axis (ordinate) shows the total electrical field arising from the electrostatic charging of the wings. It is clear that the bees suddenly all come to rest at midnight.

Acc. to Warnke (1982), published in Baumer's book (1987)

1.1.2 Examples of the utilisation of earth's magnetic field parameters

For a period of millions to a billion years, life on earth had the time in the evolution of the species to adapt to the magnetic and electromagnetic conditions of their environment. They learned to use the natural magnetic field parameters also as conveyors or carriers of a diversity of information:

- The geographic location can be established by the density of the field lines, their direction and variation in time.
- Time of day and annual seasons can be deciphered in the daily, lunar and solar periodic magnetic signals.
- Frontal weather systems and air mass movements transmit characteristic electromagnetic signals, the so-called sferics. These are short oscillations comprising just a few cycles (= impulses) in the range between approx. 3 kHz and 60 kHz (= very low frequency) with a repetition frequency of up to 100/sec or more, depending on the intensity and type of atmospheric processes.

The biosphere at the earth's surface is in contact with the electromagnetic fields of the universe via two narrow frequency windows through the atmosphere. One of these windows is in the narrow medium to long wavelength UV radiation region, including the visible light spectrum and the near (short wave) infrared radiation (average 1milliwatt/m²); another window is in the high frequency radiation region at wavelengths of 0.1 to 100 m (average 1 nanowatt/m² up to 1 milliwatt/m² (GL) during solar flares).

Effects of the earth's field and of its compensation or effects of weak artificial fields have been detected in life at all levels of development: with bacteria, single and multi-cellular algae, higher plants, protozoa, flatworms, insects, snails and vertebrates:

- Magneto bacteria (Aquaspirillum magnetotacticum) in the bottom sludge of the oceans utilise the intensity of the earth's magnetic field for orientation. Magnetite crystals (Fe₃O₄) in their bodies form a chain of "compass needles" creating a magnetic moment that the bacteria align against the thermal movement of the water molecules. (The earth's magnetic field applies an energy of 1.4×10^{-18} J (GL) to the bacteria – 200 times greater than the energy of the thermal movement at 22°C).
- Fish navigate in the earth's magnetic field. When sharks and stingrays, for instance, move in earth's magnetic field, they experience induced electrical fields of varying strength. The field strength is a function of the direction of movement relative to the direction of the magnetic field. Local physical water currents also generate direction-dependent electrical fields that can be detected. The sensory organ for electrical fields is highly sensitive. (So-called Lorenzian ampoules responding to voltage gradients of less than 0.1 microvolt/m).
- Compass termites (Amitermes) build their metre-high mounds in a north-south direction. With other termites and the woodlouse, the feeding activity is subject to natural magnetic alternating fields (sferics) and the earth's magnetic field.
- Bees make use of the earth's magnetic field and its daily fluctuations for their orientation and communication. They also gain information on weather developments through the natural impulse signals in the atmosphere, i.e. the sferics already mentioned above.
- Whales can sense the magnetic field of the earth.
- Carrier pigeons are affected by variations in the earth's magnetic field down to flux densities in the nano-Tesla region.
- Migratory birds have a mechanism acting like a compass.

- Humans react to atmospheric alternating electromagnetic fields between 10 and 50 kHz through various symptoms of the central nervous system. There are also correlations between activities in earth's magnetic field and sleep-affecting factors, circadian rhythms (HECHT 2005, 2006, 2007), enzyme conversion and hormone production in the central nervous system, the vitamin level in the blood serum, the average skin temperature, vision in half-light and iron content in the blood serum.

All the examples support the existence and the vital control functions of biologically active magnetic and electromagnetic fields with a specific frequency structure and corresponding information content, "arranged" to suit biological systems.

They are characterised by, among other:

- specific flux densities and gradients ("amplitude windows"), i.e. weak fields may have a greater effect than strong fields,
- specific impulse frequencies and impulse sequences ("frequency window"),
- specific impulse shapes and a certain complexity of the impulse spectrum,
- specific vector characteristics with respect to the body,
- minimum effective duration of coherency and specific co-factors, e.g. light.

Life forms, even of the same species, may be quite differently organised, but coordinated in a collective or social group (fish shoals and flocks of birds). In an isolated form of life, the instantaneous interaction with its environment is therefore exceedingly varied. Reproducible magneto experiments in or between individuals are therefore unlikely in the case of complex organisms, including humans; the specific metabolism parameters are, for instance, also too varied. None of these parameters can be kept as constant as required for reproducibility. "Proof", in the sense of classical scientific criteria, is therefore illusory.

1.1.3 Technical wireless communication is only possible because the transmission is stronger than the natural high-frequency radiation

Technical wireless communication such as mobile radio, radio, TV and satellite communication is only possible because the power density of the utilised technical high frequency spectrum far exceeds that of natural radiation. Natural radiation at the surface of the earth in the 300 MHz to 300 GHz range is approximately $0.001 \mu\text{W}/\text{m}^2$ ($=0.001 \mu\text{W}/\text{m}^2$); today's typical technically created radiation level in cities is $10\,000 \mu\text{W}/\text{m}^2$. And the legal German limits even allow values up to $4.5 \text{ million } \mu\text{W}/\text{m}^2$ for the D-grid, up to $9 \text{ million } \mu\text{W}/\text{m}^2$ for the E-grid and up to $9.8 \text{ million } \mu\text{W}/\text{m}^2$ for UMTS.

As we evolved, we were of course also exposed at times to strong static and low-frequency electrical fields (typical voltages: cloud electricity up to $10\,000 \text{ V}$, volcano electricity up to $20\,000 \text{ V}$, lightning $500\,000 \text{ V}$, spheres 10 V), in addition to constant static and low-frequency magnetic fields (earth's field, ionospheric field, cosmic field, lightning). But there were never fields as constant and with as many superpositions of different frequencies from different sources as we are now generating with our technology.

1.1.4 Radiation by organisms themselves could be established in the evolution, because there was no interference by continuously changing external radiation

The same high frequency radiation that technology utilises for communication is also copiously generated inside our bodies. The body also requires it for communication purposes: for biological communication through functional oscillation of our molecules.

Provided there is no interfering external radiation, the body can utilise its built-in frequencies for its internal organisation.

The body internally radiates frequencies in the 1 to $1\,000$ gigahertz (GHz) range at power densities of about $0.1 \mu\text{W}/\text{m}^2$, i.e. lower than those of average solar radiation. If we add up the total range of high frequencies (HF and VHF) present within our organism, we arrive at natural power densities of about $10\,000 \mu\text{W}/\text{m}^2$. The power generated by our internal electromagnetic oscillations, that we describe as heat (wavelengths around $3 - 10 \mu\text{m}$), corresponds approximately to that of a 100 Watt globe.

To understand the natural oscillation of our functional molecules (enzymes and other proteins, nucleic acids, hormones and many more) it is important to realise that what we generally describe as "chemistry" is actually pure physics. All the bonds and their modulations (changes) between atoms on the one hand and molecules on the other are based on physical phenomena. In this context, the electrostatic Coulomb forces (= force between different electrical charges) and the electromagnetic force (e.g. van der Waal force = force between dipoles with different moments and fast oscillations) are prominent. DNA and all the enzymes, for instance, can only carry out their functions through their natural electromagnetic oscillations.

Resonances are of particular importance here. Chain molecules, for instance, can be excited to so-called wring-resonances by high-frequency electromagnetic fields. Proteins exhibit such natural resonances in the range of $1 - 10 \text{ GHz}$; DNA resonates at 10 MHz to 10 GHz . Both of these therefore fall into the spectrum of common mobile radio frequencies.

Wring frequencies (modes) cause wringing of the molecular chains that directly affect the structure of the individual molecules. The structure of the molecules (conformance and configuration) is essential, however, for their specific functionality. Even minor displacements render the molecule useless. The chains may even break apart under energetic external influences.

Biological systems are obviously very sensitive in their reaction to microwave fields. For instance, Belyaev et al, 1996, reported resonance effects on the DNA structure at extremely low power densities of $0.000001 \mu\text{W}/\text{m}^2$ in the $40 - 50 \text{ GHz}$ frequency range. This surprising result must still be confirmed by other working groups. Nevertheless, it must be stated that: The ultra-weak, but biologically very effective natural electromagnetic fields are contrasting strangely with the technical radiation fields permitted in Germany. On recommendation by the ICNIRP association (Munich), technical radiation fields up to power densities of $10\,000\,000 \mu\text{W}/\text{m}^2$ were legalised – still considered as harmless by the experts. The population, animals and plants may therefore be legally subjected to radiation in the critical frequency spectrum that is more than 10 orders of magnitude higher than the natural fields.

But organisms are not only sensitive to high frequencies; the following examples show that very high sensitivities evolved also in the low frequency ranges.

2. About the disappearing bees and birds

2.1 The bees as evolutionary force and indispensable economic factor

Honeybees existed on earth from about 40 million years ago; a "primal" honeybee encased in amber was found on the coast of the Baltic Sea. Man soon realised the usefulness of animals. And we know today that the enormous development of earth's vegetation, comprising about 200 000 species of a variety of flowering plants, is based on animals. Because about 85% of these flowers are pollinated mainly by bees and propagate through the formation of fruit and seeds.

Since also fruit trees (such as cherry, apple, pear and plum) and agricultural crops (such as rap, sunflower, red clover, lucerne, horse bean as well as vegetables such as tomato, cucumber, pumpkin) fall under these, it is not difficult to understand that bees are one of mankind's most important production animals.

In central Europe the commercial benefit of bees is estimated at 4 billion euros per annum, in the US it is estimated at over 15 billion dollars. These figures are from the *New York Times*. It quotes estimates by Cornell University, New York State. This included the pollination of fruit and vegetable plants, almond trees and fodder such as clover. That said, however, even the global honey production of 25 000 tons per annum is an important economic factor already.

But if we aggregate all the functions the bees are performing for nature's life and the preservation thereof, their significance can hardly be overestimated. Their industry cannot be substituted either by other insects or by technical measures. If the bees should disappear, we humans will also suffer major deficiencies.

2.2 No chance of survival: Colony Collapse Disorder (CCD)

In some countries there are reports of mysteriously dying bees. It appears as if the losses are at their worst in the northern American states and in neighbouring Canada. 25% to 50% of the American bee-keepers report losses through "Colony Collapse Disorder" (New Scientist, 2007). They reported 50% to 90% of their bees to have disappeared within the previous 6 months, and the remaining bee colonies were said to be so weak they can produce hardly any honey (CNN, 2007).

But unusual losses are also reported in Germany, Switzerland, Austria, South Tyrol, Spain, Poland and New Zealand. In Germany, for instance, the beekeeper associations last winter recorded a loss of about 13% in over 7 000 bee colonies - double the previous year's figure (<http://orf.at/070416-11296/-index.html>). In accordance with a report in the *Stern* magazine edition 34/2007, German bee monitoring did not confirm this number, only acknowledging an average loss of just on 8%. A 10% loss over the winter months is not regarded as unusual.

What is completely unusual, however, is the statement by the president of the DBIB (*German Federation of Occupational and Purchasing Beekeepers*), Manfred Hederer, in the *Deutschlandradio Kultur*, on the Federal territory: "The beehives are empty." He paints a picture of bee colonies reduced by 25% - in some cases even 80% (Spiegel 12/2007).

In 2006, the Swiss federal research institute for production animals and dairy farming, Agroscope, (Federal office for agriculture), reported that also all of Switzerland was affected by bee deaths, to a regionally more or less severe degree (*Zürichseezeitung*, 5. May 2006). Roughly 30 percent of the bees were lost without a trace after winter - about half a billion animals in this year alone (<http://www.heute-online.ch/wissen/play/artikel/60601>).

Beekeepers from Styria are also reporting a mysterious disappearance of bees. Beekeepers in Vienna estimate a 30% loss. They agree on the following: "*The bees are not developing properly anymore. They do survive the winter, but in spring they disappear as if by magic. The hives are simply empty.*" (This according to beekeeper Hermann Elsasser of Fladnitz in the Raab valley; <http://oesterreich.orf.at/steiermark/stories/184609/>). Only the brood remains in the hives, and without the care of the older bees, they will die.

Ferdinand Ruzicka, scientist and beekeeper himself, reports: "*I observed a pronounced restlessness in my bee colonies (initially about 40) and a greatly increased urge to swarm. As a frame-hive beekeeper, I use a so-called high floor, the bees*

did not build their combs in this space in the manner prescribed by the frames, but in random fashion. In the summer, bee colonies collapsed without obvious cause. In the winter, I observed that the bees went foraging despite snow and temperatures below zero and died of cold next to the hive. Colonies that exhibited this behaviour collapsed, even though they were strong, healthy colonies with active queens before winter. They were provided with adequate additional food and the available pollen was more than adequate in autumn. The problems only materialised from the time that several transmitters were erected in the immediate vicinity of my beehives" (RUZICKA, 2003).



About 85% of these flowers are pollinated mainly by bees and propagate through the formation of fruit and seeds. We have the utility of animals to thank for the enormous development of earth's vegetation, comprising about 200 000 species of a variety of flowering plants.

Ruzicka organised a survey through the magazine *Der Bienenvater* (2003/9):

- Is there a mobile radio antenna within 300 m of your beehives? - This was confirmed in 20 replies (100%).
- Are you observing increased aggressiveness of the bees compared to the time before the transmitters were in operation? - 37.5% confirmed this.
- Is there a greater tendency to swarm? - 25% confirmed.
- Are colonies inexplicably collapsing? - 65% confirmed.

Such colony collapses, heralded by "angry" swarming of the bees, were also reported in New Zealand (FIR-STENBERG, 2007).

Other reasons possibly explaining the disappearance of the bees are also under discussion: Monocultures, pesticides, the Varroa mite, migratory beekeeping, dressed seed, winters too severe, genetically modified plants. There is no doubt that some problems can be attributed to this. But the fairly sudden and country-spanning appearance two to three years ago of the dying bees

phenomenon cannot be convincingly explained by any of the aforementioned causes. Should the bees simply be too weak or ill, they should also die in or near the hive. But no ill bees were found in the research into this phenomenon.

2.3 Some bird species are disappearing

But not only are bees and other insects disappearing – birds as well. The house sparrow, for instance, has become clearly scarcer in England and some western European countries. An investigation carried out between October 2002 and May 2006 in Valladolid in Spain, was launched to examine whether this decline in the sparrow population was related to electromagnetic radiation by mobile base stations. The result showed with a high degree of statistical confidence that the number of sparrows was reduced when the electrical field strengths of the antennae exceeded certain values. (BALMORI, HALLBERG, 2007).

A similar investigation was carried out in Belgium. The numbers of house sparrows were counted in the vicinity of several mobile radio base stations, during their breeding season. This confirmed a significant relationship between the electrical field strength in the 900 and 1 800 MHz bands and the diminishing numbers of birds (EVERAERT et al. 2007).

It was noticed even earlier that storks that built their nests within a 200 metre radius of base stations could not rear any chicks, remaining without offspring. The results improved at distances of 200 to 300 metres. From a distance exceeding 300 m, the storks bred with a success rate of 96.7%. The electrical field strength at a distance of 200 metres averaged 2.36 ± 0.82 V/m, and only 0.53 ± 0.82 V/m at 300 metres. From their results, the authors concluded that the electrical fields of base stations are damaging to the reproduction of the white stork (BALMORI, 2005).

3. Mechanisms of disorientation and damage

3.1 Magnetic field sensitivity in the animal world

Birds, insects, fish and snails are assumed to have a specific organ for sensing magnetic forces. It is questionable, however, whether it is always necessary to assume such a specific magnetic sense. Electrical fields do not penetrate deep into living organisms and currents follow only certain paths. A magnetic field, however, fully penetrates the organism, without major changes. It is too short-sighted to conclude from this that such fields have no effect because they are not absorbed. To start off with, even weak magnetic fields in the body are more energetic than strong electrical fields. As such, the energy in earth's magnetic field inside us is 10 000 times stronger than the strongest possible electrical field in the atmosphere (3 Megavolt/m; WEISS, 1991). Such penetrating forces as the quasi-static magnetic field and the low-frequency electromagnetic field do not theoretically need an own amplifying reception organ. Inside the organism, they can also directly couple into aggregates of orderly paramagnetic molecules or into the electro-mechanical (photon-phonon) code of the endogenous information transmission and storage.

Magnetite was found in all animals that can navigate using their own compass, sometimes in the form of ferritin-proteins (KIRSCHVINK et al. 1981). But it is also present in our brains (KIRSCHVINK et al. 1992). And it reinforces the external magnetic fields in both cases. In the tissue of birds, bees, fish and whales (WALKER et al. 1992), the magnetite concentration exceeds that in the human brain. Most areas of our brain nevertheless contain about 5 million magnetite crystals per gram

and even 100 million in the brain membrane.

Because magnetite reacts about 10 000 000 times stronger to external magnetic fields than normal dia- and para-magnetic tissue, the transmission of information separate from the neurons must be considered. Oscillating magnetite excited by ELF fields could, for instance, play a role in transport channels or cell-interconnection channels, raising the possibility of interference by communication and other negative effects of technically created electro-/magneto-smog.

It is easy to prove mechanically acting forces in insects subjected to relatively strong magnets. Own experiments with bees and flies yielded the following results (WARNKE, not published):

- A newly captured swarm of bees is exceptionally sensitive to magnetic forces. If a magnet with only a few mT field strengths is brought close to the swarm in a dark wooden hive, the entire swarm becomes excited.
- Captive bees assume a horizontal rest position at night, aligned to an artificial magnetic field of several mT in the environment.
- Dead bees, flies and a range of other insects can be made to float on an electrostatically neutral water surface and in this condition an electrostatically neutral strong magnet can be used to attract them, drag them across the surface and in some cases repel them.

In the laboratory, bees can sense not only the compass direction but also the intensity and the gradient of this magnetic field (SCHMITT et al. 1993). It was found in a 1982 publication (KUTERBACH et al. 1982) already that the magnetite found in bees is the source of this sensitivity to magnetic fields, and this theory was recently checked and finally confirmed (HSU et al. 2007).

We also found ferrite particles together with pollen lodged in the bristles of the body surface; these might also be responsible for the abovementioned magnetic moment.

It has been demonstrated that the birds' magnetic compass only functions in a certain range of intensities between 43 μ T and 56 μ T – precisely in the range of the earth's magnetic field intensity. After a three-day adaptation

period the animals could, however, also orientate themselves in fields of 16 μ T and 150 μ T (SCHNEIDER et al. 1992) – interpreted as an adaptation to the environment.

The platypus of Australia (Ornithorhynchus anatinus) has the electrical receptors for detecting its prey, in its bill. The receptors can sense direct and alternating voltages in the range of 20 mV and have a connection to the Trigeminal nerve. Fish with similar receptors use the acoustical nerve for transmission of the electrical stimulus. This shows that evolution exploited the electrical and magnetic environment in different ways. The Lorenzian ampoules of fish are capable of distinguishing between stimuli of magnetical or electrical origin (BROWN et al. 1978). It has not been established whether the receptors of platypus also have this capability. This question is of interest because ducks also have bill receptors. Although these are specialised to react to mechanical stimuli, they are so sensitive that the mechanical Coulomb forces accompanying electrical fields might well be detectable as well.

When magnetic fields penetrate an organism, two fundamentally different aspects must be clarified:

1. Is the organism merely subjected to a large increase in energy - or
2. does the organism gain information?

In various insect species we are aware of a time function based on magnetic field variations. In particular, the feeding habit of termites is correlated with the 27-day solar cycle (BECKER, 1973), and there is also an increased building activity for a few days around new and full moon in laboratory experiments – as it is with bees. It is also known that termites show directional behaviour under the influence of extremely low field strengths (BECKER 1976, 1979). Time triggers via similar channels sensing sun and moon appears a reasonable assumption.

The changes to the circadian activity rhythm of the house sparrow (Passer domesticus) can be correlated with cyclical changes of the earth's magnetic field. The sparrow reacts down to 200 nT in laboratory experiments.

Without doubt, light is the dominant timing mechanism of life. But also the earth's magnetic field is meanwhile recognised as a timing mechanism.

3.2 Bees and other small life forms under investigation

Insects have many aids for their navigation and orientation in space: sunlight, also polarised (WARNKE, 1975), gravity, aromatic molecules, colour as electromagnetic oscillation in a specific frequency range, variations in air pressure, occasionally also the degree of ionisation of the air (ALTMANN et al. 1971, WARNKE, 1976). Many species, however, cannot do without the magnetic field.

In this respect, bees are welcome objects for experimentation. Because different modalities of their orientation are inseparably linked to the magnetic field of the earth and to electromagnetic oscillations (LINDAUER and MARTIN 1968; HÜSING et al. 1959, SCHUA 1952, WARNKE, 1976).

In our working group, we recorded the directional behaviour of captive bees in an artificial field and during the night. A preference to assume rest positions with the body either parallel or orthogonal to the field lines was evident.

They share this alignment reaction with other insects such as various termites (BECKER, 1963), diptera (BECKER et al. 1964) and Drosophila (WEHNER et al. 1970).

The behaviour of termites (BEKKER, 1963) was studied particularly intensively in Germany, that of the Christmas beetle (SCHNEIDER, 1961, 1963) in Switzerland and that of insects, worms, snails, snakes and other small creatures in the USA. The

investigations concentrated on the influence of cosmic physical fields in which the magnetic field time and again played a pivotal role. All experiments confirmed the existing relationships. They also all showed, however, that constant laboratory conditions are impossible in practise, because cosmic influences change the magnetic component in any normal room and cage, thereby affecting the orientation behaviour of the animals.

The experiments with Christmas beetles and termites may be termed spectacular. In accordance with the above literature, Christmas beetles not only determine their rest position by magnetic and electrostatic fields, but also by interference patterns of gravitational waves of terrestrial and cosmic matter. In the final analysis, the evidence points to the influence of a physical field or radiation, varying in space and time in accordance with an unknown programme, that is registered through an unknown organ in the Christmas beetle for an unknown purpose, but the existence of which physicists doubt because it cannot be measured by any instrument. The Christmas beetle therefore becomes the instrument for measuring this unknown agent. The effect is often intimately coupled to that of the magnetic fields (SCHNEIDER, 1974). The orientation at rest is based on the Christmas beetle choosing the position of least or most symmetrical stimuli when awakening from the rigor of cold. Using interference patterns and models resonating with gravitational Earth-Sun waves, complex combinations of dynamic stimuli were constructed, to which the Christmas beetle responded by changing its position (SCHNEIDER, 1972).

Also termites (Isoptera), whose feeding activity and O₂ consumption are important indicators, react to more than just magnetic components. Their communication modes also include natural electromagnetic sferics impulse patterns, gravitational influences and electrical fields. The statistical correlation between the feeding activity of termites in the laboratory and the number of deaths in Berlin is described in detail; the consequences of this are as yet unfathomable. There is an increased frequency of human deaths on the days on which termites feed less.

The authors point to the magnetic field of the earth and its variation with solar influences as the common factor linking the apparently unconnected facts. Further down, other and earlier literature is cited in which an increased incidence of human death is described during unusual variations in the magnetic field.

3.3 Birds as prototypes of magnetic field orientation

This research shows that birds' orientation by magnetic fields has been a frequently discussed topic for decades. Thanks to the thorough and meticulous work of a number of researchers (WILTSCHKO, WALCOTT, MERBEL), it is today beyond doubt that several species of birds sense the earth's magnetic field and use it to establish their position during migration. As described for insects and snails, some species of birds are also particularly sensitive to a range of magnetic field strengths corresponding exactly to the earth's magnetic field – the robin, for instance. When the field is attenuated or amplified, the birds become disoriented. Setting on a certain field range could, however, change through adaptation.

The mechanism by which birds sense magnetic fields has meanwhile been largely explained. An area with iron-containing tissue was discovered in the skull of pigeons. Strangely, only one half of the skull contains material that is permanently magnetic. But opposite to this, material was found that is only very weakly permanently magnetic. Measurements indicate magnetite inclusions - the same crystal that was found in bees, bacteria, snails, whales and humans. The magnetite-containing tissue of the pigeons is even supplied with nerve ends that can sense the orientation changes signalled by the crystals (WARNKE, 1993).

It could be demonstrated at the Zoological institute of the University of Frankfurt/Main that the top half of the pigeon's bill has three magnetite-containing bodies, with a neuron ending at each of these. They constitute a three-channel system enabling the brain to construct a spatial picture of the surrounding magnetic field the pigeon can use to orientate itself in flight (source: TV programme *Planet Wissen* in BR on 18.09.2007 at 16.15 on carrier pigeons. Reference by W. Sönning).

Birds also have magnetite in the edge of the bill. In addition, light and magnetic fields result in an increase of certain free radicals in the eye, the concentration of which can obviously be accurately registered by the animals (WARNKE, 1995). This relationship will be dealt with again further on.

3.4 Animals with a navigation system are extremely sensitive to electrical and magnetic fields

Birds having a navigation system are extremely sensitive to the weather. A thunderstorm changes the magnetic field, light and many other characteristics – potentially causing the orientation to collapse. Birds and other animals are particularly sensitive to a solar eclipse. They exhibit an abnormally changed behaviour: sometimes lethargic, sometimes restless. Research attributes the reactions to the suddenly occurring electromagnetic long and medium wave radiation, typical for night time, but surprising with the suddenly occurring darkness of a solar eclipse. The lack of ionisation in the ionosphere by light has the effect of many oscillating impulses propagating 100 times better on the surface of the earth.

This unexpected electromagnetic impulse effect may also, in principle, explain the early warning system animals have for earthquakes.

The so-called sensitivity to weather or inclement weather, traceable to short electromagnetic impulses with a certain frequency content and rapidly decreasing amplitudes, has also been known for a long time.

These impulses originate at frontal weather systems, where colder air from sub-polar regions undercuts warm subtropical air masses. In the regions where warm or cold fronts mix, thermodynamically driven turbulent air currents with vertical and horizontal components are created. This is in essence where the abovementioned natural electromagnetic impulse radiation of the atmosphere, also known as sferics, is created. Many life forms such as insects, frogs, birds and various mammals react to this meteorologically based impulse activity in the atmosphere. By receiving and frequency-analysing these "weather code" signals of changes in the weather or approaching thunder-storms, they can then dive for cover or fly around these thunderstorm regions (WARNKE, 2006).

Walter Sönning: "These weather signals or sferics are indicators of unstable processes in the troposphere – the weather-creating layer of the atmosphere –, since their source is in the weather centre. They originate in invisible discharges between positive and negative space charge clouds, created and maintained by different processes of ionisation such as cosmic radiation, UV radiation, natural radioactivity or the Lenard effect (= spray electrification or break-up of droplets or ice crystals with opposite charges). In terms of physics, our air could therefore also be described as a "plasma" gas. When differing space charges of possibly predetermined magnitude are electrically equalised, the ion-front of this basic plasma or gas discharge propagates at velocities of about 200 km/s along a tubular channel of about 40 cm diameter in the direction of the maximum potential difference, covering distances between 40 and 100 metres, until the electrical potentials are equalised. If the ion density in the air is sufficiently high, the following discharge impulse follows immediately. Each of these invisible and "quiet" discharges that occur at varying intensities in all weather conditions, is the source of an electromagnetic, three-dimensional impulse or space wave, a so-called EMP or primal impulse, similar in its waveform to impulses from other sources (nerves, atmospheric nuclear

explosions etc.). This 3-dimensional wave propagates at the speed of light. When recorded on an oscilloscope, for instance, it vaguely resembles a sinusoidal half-wave, but has a steeper rise time and an exponential decay of the amplitude. In a Fourier analysis, it is therefore not equivalent to a sine wave of a certain frequency.

Depending on the meteorological and atmospheric electrical propagation conditions, these EMP's are dampened at a distance of 60 to 100 km from the source to lower frequency sinusoidal Fourier components with a continuous spectrum between approx. 3 kHz and 60 kHz. True to their origin in an impulse discharge, these "impulses" have waveshapes of a few oscillations with rapidly decaying amplitudes from a maximum down to zero. Particularly well-defined impulse shapes in the total collection of atmospheric impulses convey, through their resonating sinusoidal oscillations at certain frequencies and also in the subsequent impulse frequencies up to 100 Hz, the meteorological information on their origin and propagation conditions – like a kind of code. These impulses can be displayed after suitable electronic filtering and are known in the technical literature as CD sferics a.t.B. (CD = convective discharge, i.e. created in atmospheric convection or turbulence without luminosity; a.t.B. = according to BAUMER). They gained special significance, however, at the beginning of the eighties in the context of industrial four-colour copper gravure printing. In this context, their highly differentiated effectiveness in the diffusion capability of biochemical biological membrane systems, in dependence on characteristic weather processes, was also shown.

The signals of visible lightning, occurring over the period of the main lightning discharge comprising of virtually uninterrupted sequences of such EMP's, together showing impulse periods up to tenths of seconds with a continuous spectrum into the MHz range, are a strictly different phenomenon.

As such, however, they are suitable as a special weather or thunderstorm signal for the animal and possibly the plant world, i.e. for any organism fitted with the corresponding reception sensors.

Sferics or atmospherics of the various kinds could therefore deliver an almost complete picture of the weather of the day, including prognostic clues, for a biological strategy, considering that the sferics propagate from a weather front at the speed of light and are travelling for hundreds of kilometres, always clearly recognisable as encoded weather to those who have the reception sensors. This can be proven by the example of the reactions of the biochemical membrane system of dichromate gelatine. Also: throughout evolutionary time, both the constant impulse frequency spectrum of the CD sferics a.t.B. and the daily excursions, constant in their climatological average, have provided a wealth of precision information on the meteorological and geophysical environment to those equipped with the receptors for the signals and the experience; which is more than today's weather services can achieve with the most modern high-tech equipment." (End of the contribution by Walter Sönnig).

Animals have a typical electrical charge pattern for each weather phase. Since all movement of charge is associated with forces, animals can analyse the approaching weather via the electrical quantities, even long before the arrival of a thunderstorm.

As a function of special electrical weather events, the bodies of the animals are therefore affected through a complicated interaction of different components: charged, reverse charged, discharged, dielectrically polarised. Polarisation is by a natural electrical DC field. It can be shown that animals are slowly electrically charged in good weather, whilst approaching thunderstorms cause a rapid discharge due to a high concentration of small ions in the atmosphere and charging changes rapidly between positive and negative as the thunderstorm approaches.

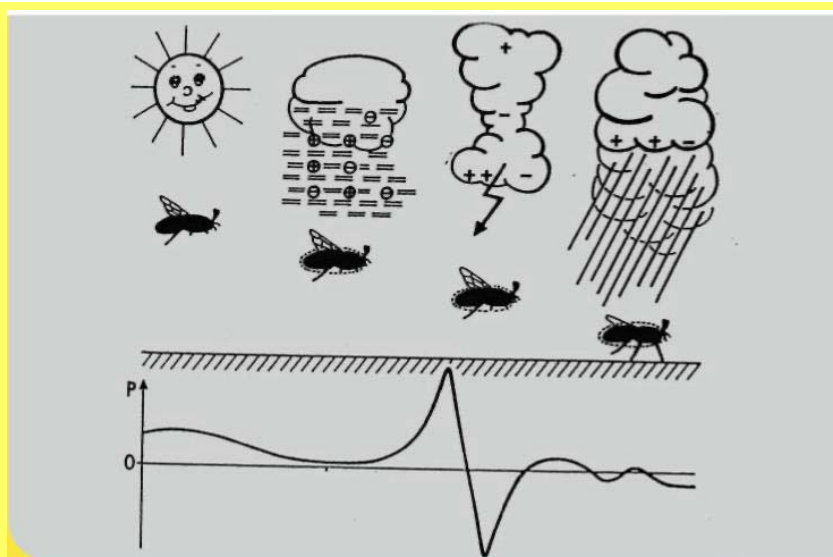


Fig. 2: Top sketch: The electrical charging of the insects changes typically as the weather parameters change. The bottom curve shows the changes in the electrical field of a freely flying bee as a function of the weather condition.

Warnke 1989, Copyright Ulrich Warnke

Insects such as bees receive these oscillations and recognise them as storm warnings. We were able to show that bees return in great numbers when these oscillations are simulated and transmitted, using a highly amplified signal generator signal. If the amplitudes of the artificial oscillations overlap with the natural signals, however, the return rate rapidly decreases. The bees fail to find their way home.

The sensitivity of the honeybee to weather is based mainly on electromagnetic information. When an approaching thunderstorm threatens the bees, flying bees return en masse when the natural 10 – 20 kHz component of the sferics activity increases within a radius of approx. 200 km (WARNKE 1973). The suction performance of the bees also correlates with the approach of the front and the associated sferics (SCHUA, 1952).

And ultimately, bees even use the receptor channel for electromagnetic waves for communication. Russian researchers found in 1975 already that bees generate electromagnetic signals with a modulation frequency between 180 and 250 Hz as they perform their communication dance. Hungry bees react to the frequencies by holding their antennae erect (ESKOV et al. 1976).

Such electromagnetic communication impulses of the antennae when touching another bee can be measured with an oscilloscope (WARNKE, 1989).

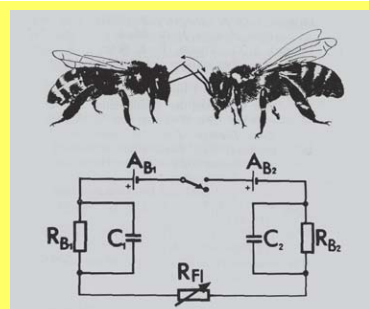


Fig. 3: Bees communicate via electrical "switching" when their antennae touch. Warnke 1989, Copyright Ulrich Warnke

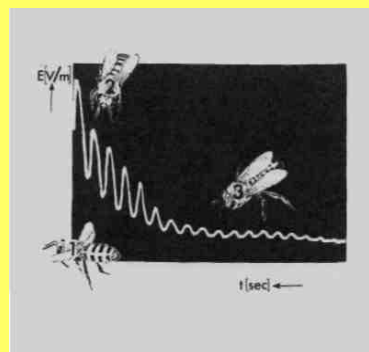


Fig. 4: Oscillogram of the electrical field of a bee flying past (1). The field strength rises as it approaches a receiver (2) and drops again at a distance from the receiver (3). König, H. Unsichtbare Umwelt. Heinz Moos Publishers, Munich 1973. Copyright Ulrich Warnke

Some bird species, such as carrier pigeons, are sensitive to exactly the same electromagnetic oscillation amplitudes as the bees. Birds, particularly species of duck, also communicate by means of electrical fields (WARNKE, 1989). This interesting aspect will be dealt with in more detail below.

3.5 Humans are also sensitive to weather through electro-magnetic pulses

The interest in sferics and their effect was greater in the sixties than it is today. In those days, a number of valuable overviews were compiled of their effects on the organism (REITER, 1960; ASSMANN, 1963).

Mammals and man are also influenced by sferics.

Sferics impulses change the tissue pH independent of the amplitude of the field. This is true in the minimal field strengths occurring in nature and also in the laboratory with simulated impulses and increased field strengths. Especially in the frequency band between 2 and 20 kHz, in which the energy of the atmospheric-electrical waves is at a peak, the effect is the strongest. Pain associated with amputations and with brain injury also correlates with the presence of sferics both in the laboratory and in nature (REITER, 1960). The paper by Reiter also contains notes on the triggering of bronchial asthma, heart and circulatory disorders, insomnia, headaches, glaucoma, gall and urinary convulsions, heart attacks and strokes – among other by sferics.

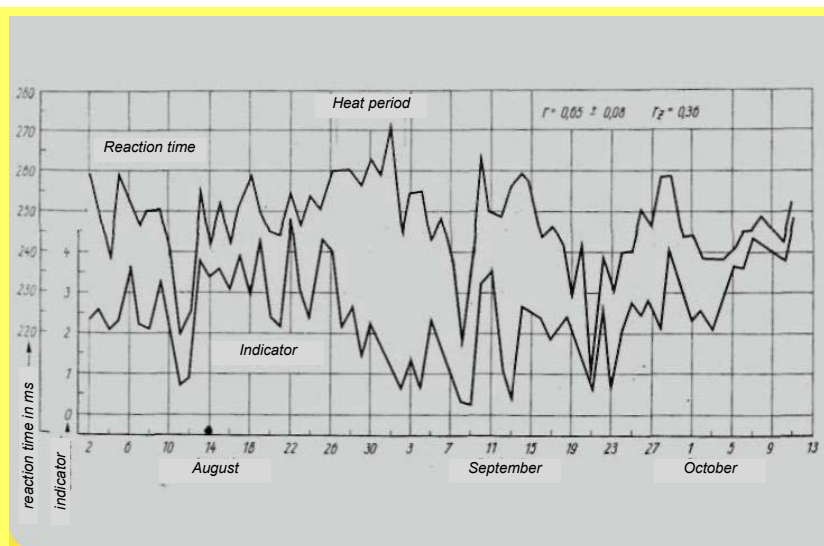


Fig. 5: Significant linear correlation between electromagnetic very long wave activity and average reaction times of exhibition visitors. Reiter, R. 1960 Meteorobiologie und Elektrizität der Atmosphäre. Akademische Verlagsges. Geest & Portig, Leipzig

It has been known for a long time that certain weather conditions give rise to thromboses, heart attacks and embolisms; the correlation is statistically significant (ARNOLD, 1969; BREZOWSKY, 1965). A significant increase in platelet adhesion could be shown with certain electromagnetic oscillations such as those generated by exchange of electrical charge in the frontal regions of the atmosphere. These long-wave sferics easily penetrate into buildings. The mean impulse repetition frequency is in the region of 5-15 imp/sec, i.e. in the biologically active window. Thrombocyte adhesion was measured in subjects in a controlled laboratory study using a sferics stimulator (JACOBI et al. 1975). The result was a highly significant ($p < 0.0005$) increase in adhesive property at a carrier frequency of 10 kHz and an impulse repetition frequency of 10 Hz. The thrombocyte adhesiveness was reduced at repetition frequencies of 2.5 and 20 Hz and with no electrical signals. Pharmaka (75 mg Dipyridamol plus 300 mg Acetyl salicylic acid) prevents sferics-related thrombocyte adhesiveness. Mentally unstable subjects were more affected by the change in adhesiveness than stable ones.

The daily work performance is also correlated with diurnal sferics activity (RANTSCHT-FROEMSDORF, 1962).

After further investigation by Jacobi (1977), the physiologic detector location was found to be in the head. If the head is largely screened from sferics, the thrombocyte adhesiveness disappears under otherwise equal experimental conditions – a result that is not in agreement with the effects of screening found by other researchers.

The fundamental sferics frequency is 7.5 Hz, considering the speed of propagation of the electromagnetic oscillations generated by the lightning discharge and the resonant path given by the circumference of the earth between the earth's surface and the ionosphere. The bandwidth of the fields is several kHz.

The correlation between heart attacks and weak magnetic field variations has been described in 1979 in *Nature*, one of the foremost scientific magazines.

This result is not an isolated case. Other experiments even found a correlation between the average number of deaths and earth's magnetic activity.

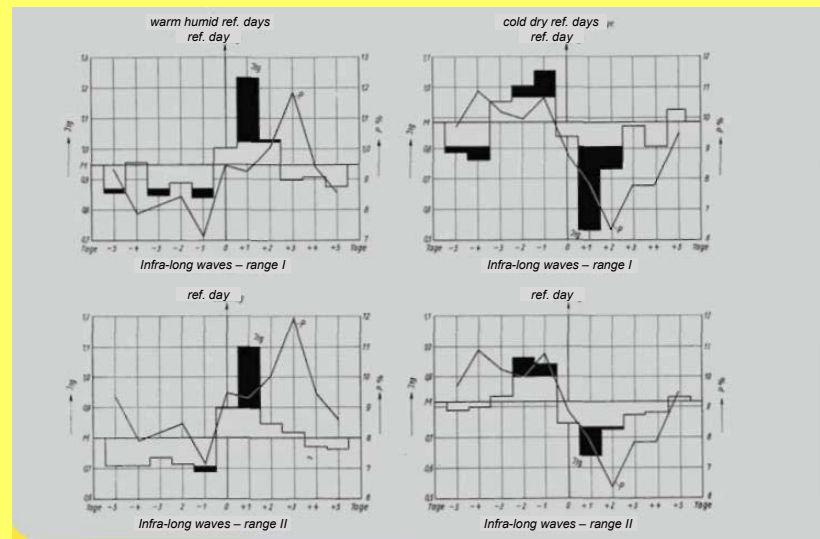


Fig. 6: Significant synchronicity of very long waves and contracting poliomyelitis. The black bars show days of low (bars downward) or high (bars upward) sferics activity and the superimposed curves show the correlated levels of poliomyelitis contraction in the fifties. Reiter, R. 1960 *Meteorobiologie und Elektrizität der Atmosphäre*. Akademische Verlagsges. Geest & Portig, Leipzig

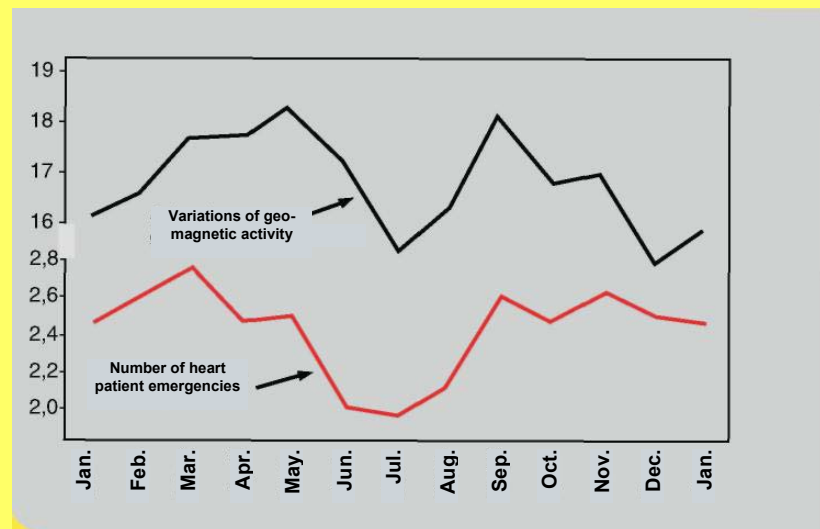


Fig. 7: Daily emergency hospital admissions for heart attacks as monthly average (bottom curve) and geomagnetic activity (top curve) Malin SRC, Srivastava BJ. Correlation between heart attacks and magnetic activity. *Nature* 1979;277:646-648

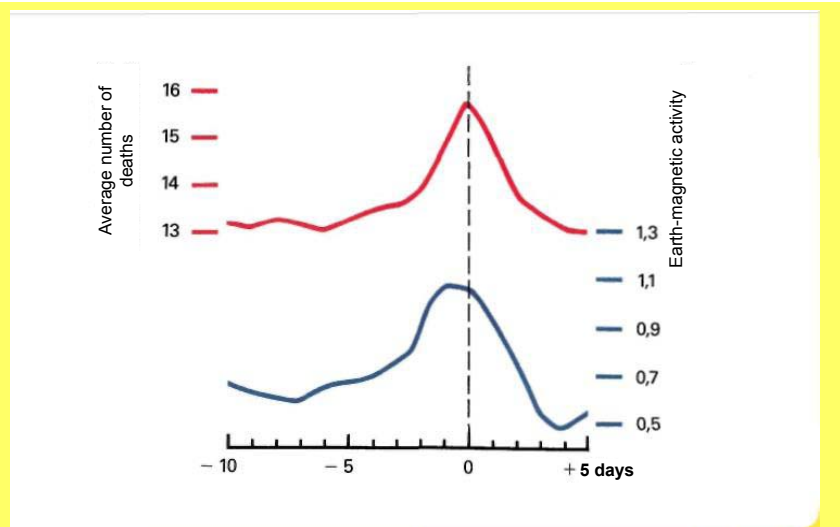


Fig. 8: Magnetic storms (bottom) and fatalities from nervous and cardiovascular illnesses. Weiß 1991

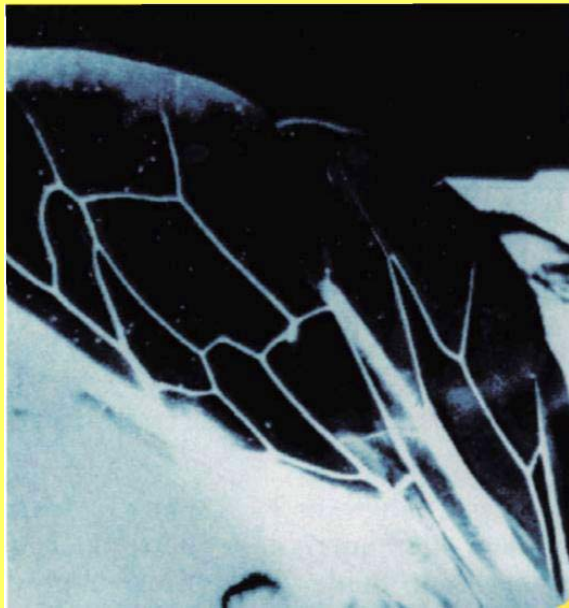


Fig. 9: Wing of a bee under a scanning electron microscope. The electrical current pattern was recorded. All the white regions have high electron mobility, whilst the darker areas are highly electrostatically charged due to low electron mobility. Discharge is very difficult. Wamke 1989, Copyright Ulrich Wamke

3.6 Bees transmit electrical fields

Electrical fields with high amplitudes are always in evidence when the unipolar charge accumulations creating the fields cannot be repeatedly neutralised. Charges are easily neutralised when they are highly mobile.

All land-based insects with rigid body shells (cuticula) and also animals with scales, shields, feathers and hair have used these structures to form surfaces that have excellent electrical insulating properties. These body parts have semi-conducting properties and are piezo-electric and pyro-electric – distortion and temperature changes therefore both create electrical effects. The conductivities are therefore subject to the well-known laws of semiconductor theory: temperature changes, light effects, microwave effects, changes to atmospheric ion concentration – all these parameters change the conductivity pattern.

The areas of different conductivity can be shown in a visually impressive way – demonstrated here on a bee's wing - by using a scanning electron microscope with sample current imaging.

In terms of electrostatic charging, it is also important whether the animals are in flight or on the ground. Animals having sweat, scent and adhesion glands afford excellent galvanic contact. Animals walking on hoofs, toes or claws are largely isolated from earth, however.

There is a salient point about different insects. Flies, bees and others have a glandular adhesive pad (arolium) between two toes on their feet. This adhesive pad can be folded in or folded out when walking.

When the arolium is folded in, the animals walk on their claws, insulating them electrically from the environment allowing them to get statically highly charged up. If the arolium is folded out and touches the surface on which it is walking, however, the insect is instantly discharged, assuming the electrical potential of the surface. In bees, this happens just prior to taking off from a flower, in which case certain parts of the animal are discharged or obtain a different charge or sometimes even reverse polarity. Since flowers normally are at earth potential, the "arolium switch" effectively standardises the insect's potential to zero. When the bees arrive at the hive, they carry different charges that they picked up in flight and that cannot dissipate that fast (WARNKE, 1977).

When two surfaces make contact on the molecular level (10 to the power of minus 10 m), positive and negative charges are separated at the point of contact through charge transfer. Many such points are activated in a short space of time by friction. Frictional electricity is one of man's oldest observations and has lent its name to the entire electrical discipline (electron: Greek for amber). It is therefore surprising that we thus far hardly spared a thought for the significance of electricity in animals.

Especially in flight, animals could become statically highly charged through friction between air molecules and body tissue – up to electrical field strengths in excess of 1 000 V/cm.

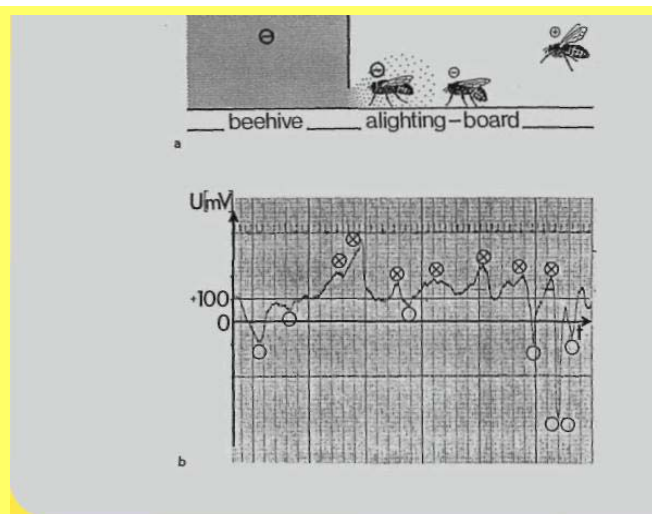


Fig. 10: Every bee landing at the hive carries a specific charge (circle with cross) thereby changing its pattern of charge at the hive entrance, determined by the total electrical charge of the colony. Every departing bee carries with it electrical charge from the hive (circle). Warnke 1989, Copyright Ulrich Warnke

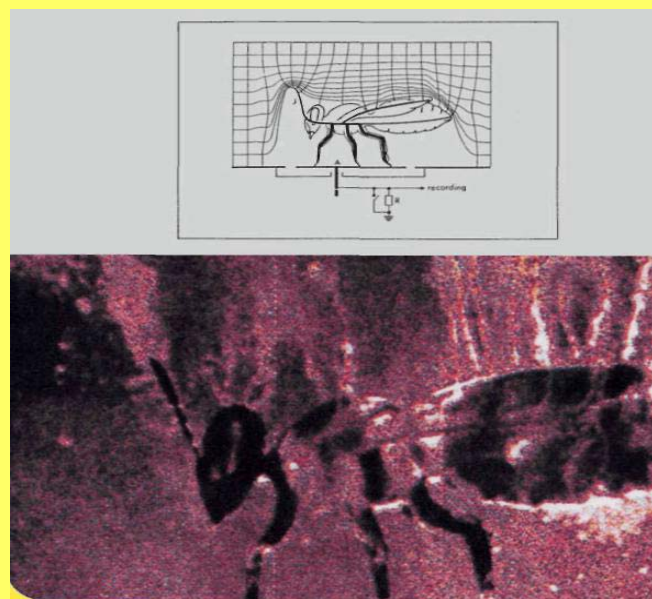


Fig. 11: A bee in an electrical field; top: a construction, bottom: an experiment. It is shown how the field strength increases around certain surface structures. Warnke 1989, Copyright Ulrich Warnke



Fig. 12: Bee in flight in an electric field. The fields around the antennae are particularly strong.
Warnke 1986, Copyright Ulrich Warnke

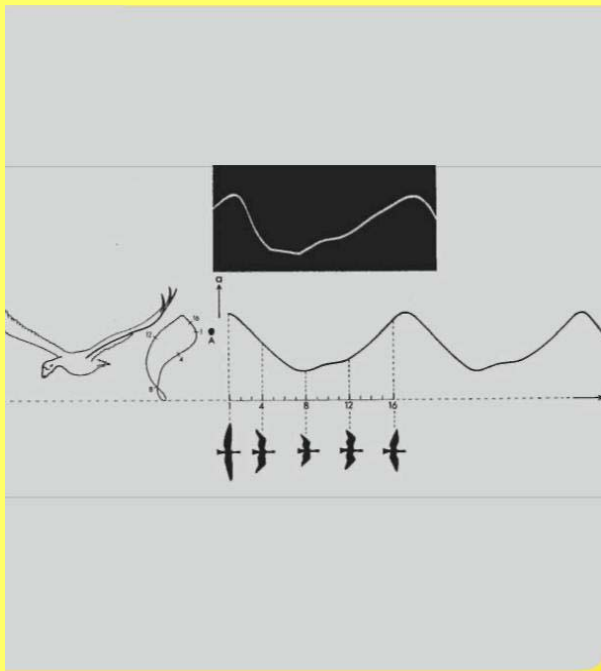


Fig. 13: Wing movement and the electrical field – with reference to the wing – are in phase.
Warnke 1989, Copyright Ulrich Warnke

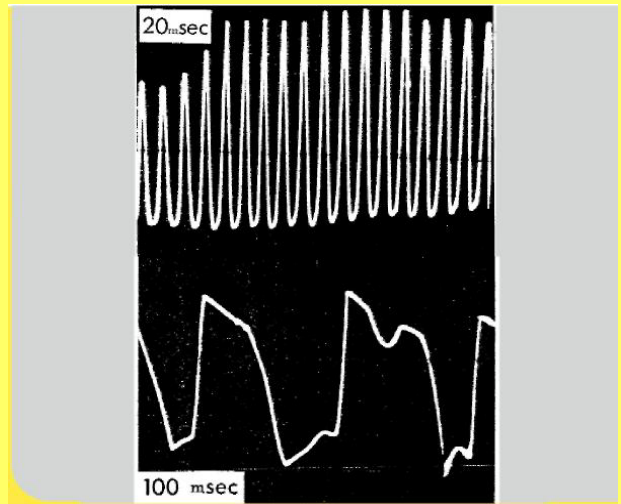


Fig. 14: Oscillogram of the alternating electrical field around bees (top) and pigeons (bottom) in a wind tunnel.
Warnke 1989, Copyright Ulrich Warnke

To increase these field strengths, animals have various aids such as protruding spikes on insect wings, but especially the field focussing effect of insects' antennae is measurable, developing appreciable Coulomb forces.

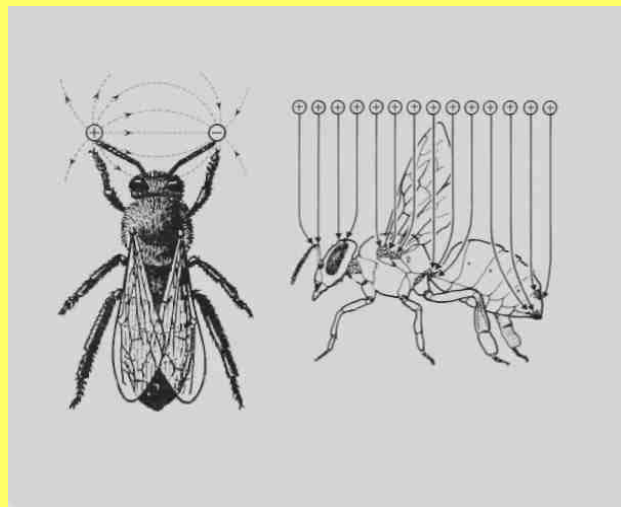


Fig. 14.1: Representation of a measurable "dipole effect" on the antennae of the honeybee. Bees are able to change the polarity of their antennae at will (e.g. from positive to negative charge) – within a second. The dotted lines are an indication of the forces in the field.
Warnke 1989, Copyright Ulrich Warnke

3.7 Effects of technically generated fields on bees

We investigated the reaction of bees to artificially created electrical fields in the laboratory (WARNKE 1975, 1976, WARNKE et al. 1976) and found the following: 50 Hz AC fields with field strengths of 110 V/cm cause significant restlessness of the bees in their enclosure. The colony temperature increases greatly. The defence of the social territory is uncontrollably increased to the point where individuals in a colony stab one other to death. They no longer recognise one other.

After a few days in the field, the bees tear their brood from the cells; no new brood is reared. Honey and pollen are also depleted and then no longer collected. Bees that were newly established in their hives shortly before the start of the experiment always abandon the hive again and disappear when the electrical field is switched on. Bees that have lived in their hive for a long time, plug all the cracks and holes with propolis, including the entrance. This otherwise only happens in winter in a cold draught.

Since an acute lack of oxygen develops when the cracks and the entrance are plugged, the bees attempt to introduce air by intensive fanning. In this process, the wing muscles generate temperatures high enough to melt the wax. The animals attempt to fight the temperature increase by more fanning. In the end, the colony burns itself out. This implies the death of all members of the colony – which we could obviously prevent in future.

With very sensitive colonies, the reaction signal was measurable from field strengths of 1 V/cm and frequencies between 30 Hz and 40 kHz. When the field is switched on, the animals suddenly move their wings and buzz at frequencies of 100–150 Hz (WARNKE 1973, 1976, WARNKE et al. 1976).



Fig. 15: Wing section of bees enlarged with scanning electron microscope. Observe the special structures serving to focus the electrical field.
Warnke 1989, Copyright Ulrich Warnke

With signals in the frequency range of 10 to 20 kHz, the aggressiveness was increased and the homing ability much reduced even though the natural meteorological and electromagnetic environment was intact in the flight space (WARNKE, 1973).

Scientists from the University of Koblenz-Landau conducted several experiments, looking at different aspects and questions, to measure the homing behaviour of bees (*Apis mellifera carnica*) as well as the development of mass and area of the combs under the influence of electromagnetic radiation (KUHN et al. 2001, 2002, STEVER et al. 2003, 2005, HARST et al. 2006).

They recorded an increase in agility, an increased swarming drive and no winter clustering when under the influence of EM radiation of cordless telephones.

In other experiments with base station fields of the DECT cordless telephones (1 880-1 900 MHz, 250 mW EIRP, 100 Hz pulsed, 50 m range, permanent exposure), the weight and area development of the colonies was slower compared to the colonies that were not exposed to a field.

The homing ability of the bees was tested from five days after the DECT telephones were introduced. There were significant differences in the return times of the colonies that were in the field and those that were not. No more than six of the bees exposed to the field ever returned – sometimes none returned. With the bees not exposed to a field, there were returning bees at any point in time of the experiment.

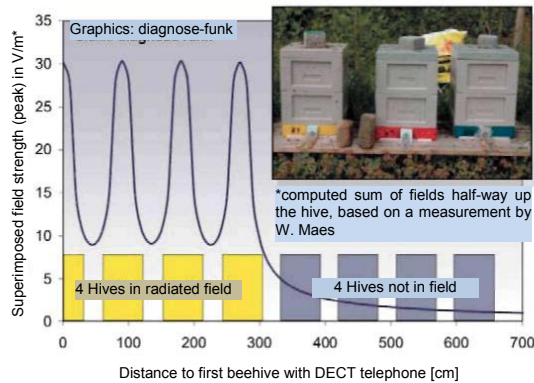


Fig. 16: Estimated diagnosis radio field strength in the four beehives with and four without DECT telephone installations at the University Koblenz-Landau. The beehives were not electromagnetically screened, implying that the control colonies were also subjected to some field strength.
Diagnosefunk, <http://www.diagnose-funk.ch/impressum.php>

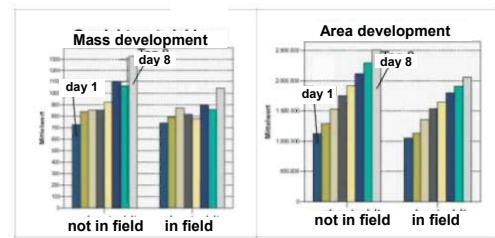


Fig. 18: Mass and surface development of combs of bees in and outside of a field.
Harst et al. 2006

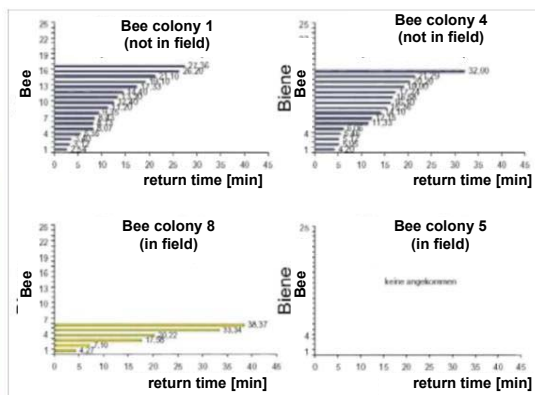


Fig. 17: Top left and right: return times of bees not subjected to a field; bottom: return times and non-return when subjected to a field. Of the bees from hives not exposed to a field, 40% returned in total, of those subjected to a field only 7% returned.

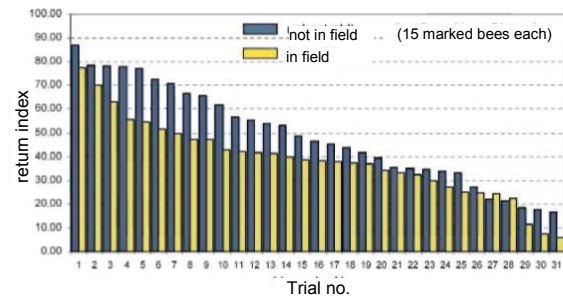


Fig. 19: Significant difference in the homing behaviour of bees subjected to a field and others. A higher index indicates more returned bees and/or shorter return times.
Harst et al. 2006

Two earlier NASA financed studies by one of their working groups found neither an increased fatality rate of bees in high frequency fields (2.45 GHz, CW) nor diminished orientation (WESTERDAHL et al. 1981a/b).

3.8 The highly sensitive region for interference of bees

If a new food source is discovered within 80-100 m, the bee performs a circular dance on the comb in the hive. If the food source is further away, communication is by means of a waggle dance. This waggle dance of the honeybee communicates information on the direction and distance of the new food source relative to the hive. In this dance, the returning worker bee initially traces a straight line and then dances sideways and down in a semi-circle. She then again starts along the straight and dances a semi-circle downwards again, but to the opposite side. The distance to the food source is given by the number of deflections of the abdomen on the straight (wagging). These waggles can also be measured in the form of electrical and magnetic alternating fields.

The distance to the food source is registered by reference to optical features of the landscape over which the bees are flying. The information on the direction to the food source is given by the angle between the straight line to the food source and the azimuth of the sun in each case. This angle is conveyed in the darkness of the hive via the direction of the dance with respect to the vertical (gravitational vector).

All this can be proven. The credit for discovering this evolved strategy of bee communication goes to the Austrian Karl von Frisch (FRISCH von, 1967). But we know in the meantime that the communication processes are associated with far more complicated mechanisms.

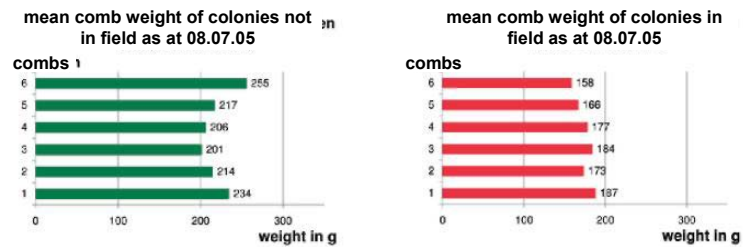


Fig. 20: Starting with the same comb mass, the average values of the total mass of the colonies subjected to a field and those that were not, were 1326g and 1045g at the end of the test. The difference is therefore 281g (21.1%)
Harst et al. 2006

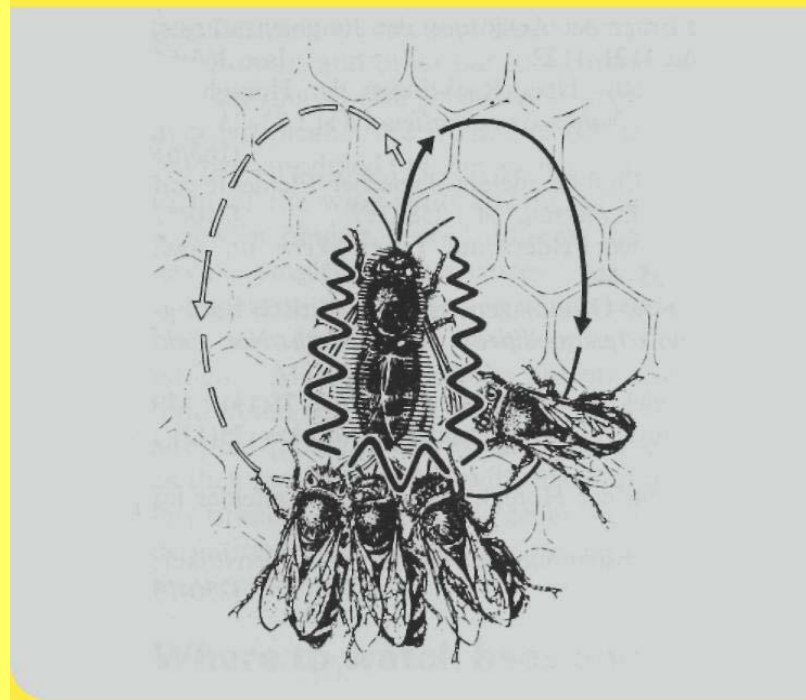


Fig. 21: The waggle dance of the bees generates electrical oscillating fields.
Warnke 1989, Copyright Ulrich Warnke

Apart from the position of the sun, the bees can also identify polarisation of the light. And in case of overcast skies, the positions of permanent landmarks are memorised (DYER, 1981).

Navigation to the food sources and back to the hive makes use of other physical quantities, however: these are exactly those quantities that have existed on the surface of the earth for millions of years –taking us back to our subject. How does the bee know the azimuth of the sun at any given moment?

She needs this information to recognise the time of day. And she needs to know about time because many flowers only open at a particular time of day and because navigation is coded via the position of the sun.

The answer to this question illustrates how finely nature has analysed the naturally occurring energies and forces, making these available to the organism. The higher the sun in the sky, the more the atmosphere heats up. The higher the atmospheric temperature, the faster the atmospheric molecules move. The faster the molecules, the more energetic the collisions between them. The larger the collision forces, the larger the air volume and the more intense the turbulences manifesting themselves also as eddies. These eddies ultimately also affect the ionosphere. The increased movement of ions in the ionosphere generates huge electrical currents. These directional electrical mass-flow currents in turn generate strong magnetic fields.

These magnetic fields reach the earth's surface and have a typical diurnal pattern – analogous to the described effect of solar radiation. They are characteristic diurnal magnetic field variations, superimposed on the largely uniform magnetic field of the earth. Exactly these variations can be measured by the bees. And they use these measurements to calculate the azimuth of the sun and the time of day.

In the experiments on navigation and orientation, this magnetic field component is recognised as a so-called "precision error" in the performance of the waggle dance. The expression "precision error" was created when a deviation of the dance direction from the principle described above was noticed, but the influence of the magnetic field was as yet unknown. Since a few decades we now know: the reason lies in the consideration of the magnetic field variation which modulates the direction angle of the waggle dance (KIRSCHVINK, 1981). The "remaining precision error" in the dance disappears if the entire magnetic field is compensated to 0-4%.

The maximum sensitivity of the bee to earth's magnetic variations is around 26 nT. It must be emphasised here already that the system is particularly sensitive in the naturally existing physical range. Significantly amplifying the magnetic field compared to the normal biological range, causes a stronger variation in direction communication. If the field is amplified to 10-times that of the earth's magnetic field, the colony swarms, away from its hive.

The question of how bees sense these magnetic field variations has been investigated in a number of studies (GOULD et al. 1978, 1980, GOULD 1986, FRIER et al. 1996, HSU et al. 1994, KALMIJN et al. 1978, KIRSCHVINK 1992, KIRSCHVINK et al. 1981, 1991, 1997, WALKER et al. 1985, 1989 a/b/c, COLLETT et al. 1994).

To summarise, it may be said that (HSU et al. 2007): The construction of combs and the homing capability of bees change if the bees are subjected to magnetic fields superimposed on the earth's magnetic field. Bees in free flight can sense extremely slight variations of the magnetic field intensity - in the range of 26 nT. They can be trained to magnetic anomalies, but only provided the changes remain stable for a longer period.

Many experiments proved that an accumulation of bio-magnetite particles (Fe_3O_4) serves as receptor of the magnetic field. These iron granules are arranged in a band in the abdomen of the bee. They have a diameter of only about $0.5\text{ }\mu\text{m}$ and are located in special cells, the trophocytes. Magnetite has the effect of amplifying the magnetic variations. If 30% of the intensity of the horizontal earth field component is modulated, the activity of the neurons in the ganglion of the abdomen changes (SCHIFF, 1991).

Apart from super-paramagnetic magnetite, FeOOH was also found in the abdomen. Magnetic material was also shown to be present in the antennae, head and claws of stingless bees.

The iron granules are enclosed in small vesicles, touched by a cell framework. As in higher organisms, microscopic filaments (micro-tubules) serve as the cell framework. The vesicles also contain some phosphorus and calcium together with the iron. The density of the iron granules is 1.25g/cm^3 , that of the Fe_3O_4 magnetite is 5.24g/cm^3 .

Where does the magnetic material come from? Most of the iron originates from pollen (approx. $0.16\text{ }\mu\text{g/mg}$) (BOYAIN-GOITIA et al. 2003). If an additional magnetic field is applied to the bee, the size and shape of the biomagnetic granula changes (HSU et al. 2007). These changes are detected by the micro-tubules and micro filaments, and the trophocytes thereafter secrete more Ca^{2+} . The fat cells of the bee also show this effect, but much less so than the trophocytes. It has been known for a long time that cells secrete Ca^{2+} under the influence of a weak magnetic field; macrophages, for instance (FLIPO et al. 1998), astrocytoma cells (PESSINA et al. 2001, ALDI-NUCCI 2000) and chrome-affine cells (MORGADO-VALLE et al. 1998).

It is also known that the Ca^{2+} secretion can be triggered by many different cell changes such as changes to the structure of membranes, changes to the electrical membrane and cell surface potential and changes to the structure and distribution of protein within the membrane. The magnetic field can stimulate two mechanisms for increasing the Ca^{2+} content in the cells: firstly by opening Ca^{2+} channels and by increased flow of external molecules into the cell; secondly by an increased release of Ca^{2+} from storage inside the cell (IKEHARAA et al. 2005, PETERSEN 1996). This explains the increased accumulation of Ca^{2+} in fat cells.

The magnetite mechanism amplifies the effects tremendously (SCHIFF, 1991). The property of the granules, to expand in an external magnetic field, turns them into magnetic field sensors (TOWNE et al. 1985). The affected micro filaments make contact with the cell membrane (HSU et al. 1993, 1994), affecting signal transfer into the cell.

If the colchicine and latrunculin B toxic substances, known to shut down the micro-tubules and micro filaments, are administered, then an additionally applied magnetic field will not increase the Ca^{2+} in the cells.

A model of the magnetic field orientation is therefore as follows: If the bee flies parallel to the magnetic field lines, the magnetic granule vesicles will expand; if she flies vertically to the field lines, the granules contract. This change of shape is sensed by the cyto-framework and communicated to the membrane.

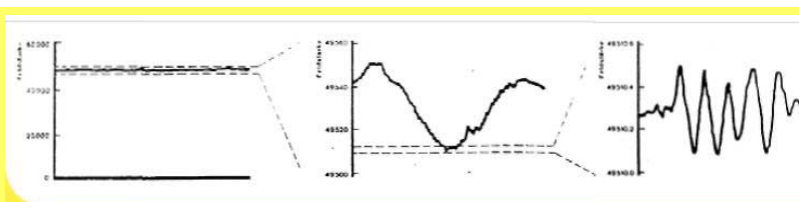


Fig. 22: Variations in earth's magnetic field: Sensitivity of the measurements increased a 1000 times in each case. Diurnal rhythms and micro-pulsations are visible, utilised by bees and other organisms for orientation in space and time. Wamke 1978

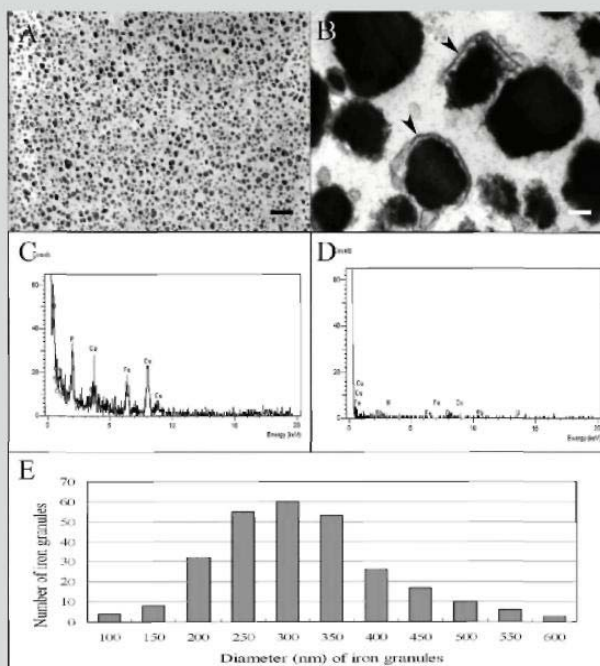


Fig. 23:
A) Iron granules in the trophocytes of the honeybee (bar: $1\text{ }\mu\text{m}$)
B) Iron granules enclosed in lipid membranes (bar: 100 nm)
C) and D) Energy dispersing radiation analysis of the granules; they contain calcium, phosphorus and iron.
E) Histogram of the granule sizes.
Copyright by: HSU, C., KO, F., LI, C., LUE, 1 Magnetoreception System in Honeybees (*Apis mellifera*) PLoS ONE 2007;2(4): e395

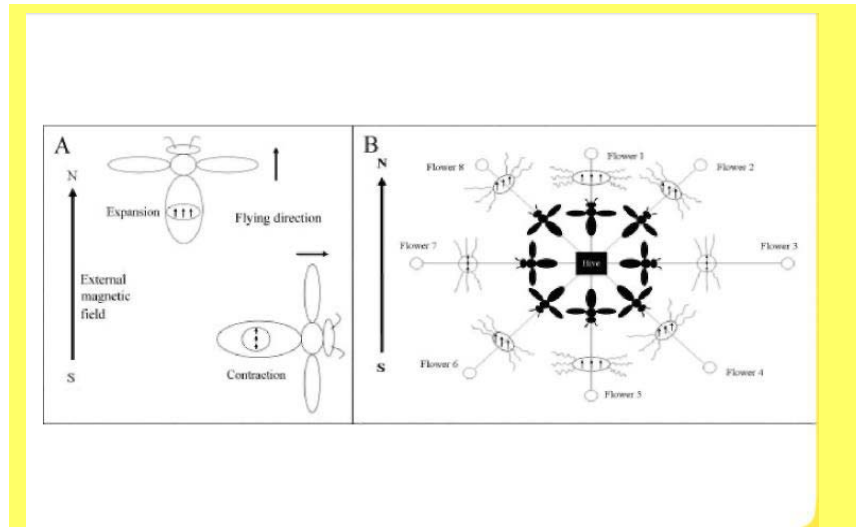


Fig. 24: Schematic of the magnetic field orientation of a bee through use of the magnetic granules.

Copyright by: HSU, C, K0, F., LI, C, LUE, J. Magnetoreception System in Honeybees (*Apis mellifera*) PLoS ONE 2007;2(4): e395

That is where Ca^{2+} channels are correspondingly opened or closed. This signal transfer results in a magnetic field map for the duration of a flight, that can be used for orientation – particularly also for returning home, by reversing the time sequence of the magnetic field (RILEY et al. 2005, MENZEL et al. 2005). It is extraordinary that, in this process, variations of 26 nT can be sensed against the background of earth's 45 000 nT.

This model explains:

1. The flight from the hive to the food source must be in a straight line. Bees navigate by means of a memory map (RILEY et al. 2005, MENZEL et al. 2005).
2. During the known circular orientation flights, the magnetic field is mapped over 360°. It is known that the orientation flight is indispensable for successful return to the hive (BECKER 1958, CAPALDI et al. 2000, WINSTON 1987). Nature arranged this similarly to the pigeons, who also circle several times before flying towards their goal.

3.9 Constant change in the magnetic environment makes learning impossible for the bees

Bees learn the patterns of the landscape they fly over and also use the magnetic field to differentiate. This is always the case when other orientation aids such as sunlight are covered by clouds. The optical patterns are therefore also associated with a magnetic coordinate (FRIER et al. 1996).

Bees may be conditioned to magnetic deviations from the normal earth magnetic field (WALKER et al. 1989a); they can also be trained to recognise small changes in the earth's magnetic field (WALKER et al. 1989b). It is a prerequisite that the change in the magnetic field remains constant over the learning period. If the field varies continuously, learning becomes impossible.

But this is exactly the situation the bees find themselves in, with wireless communication fields. The magnetic component is continuously changing – during the day and at night.

3.9.1 HAARP changes the natural diurnal variation of the magnetic fields

The information on the HAARP project is thanks to Guy Cramer (USA); it was made available to me by Joris Everaert (Belgium).

HAARP (High-frequency Active Auroral Research Project) is the abbreviation for a military project of the US Air Force and Navy. 180 towers have been erected in an uninhabited area near the city of Gakona in Alaska, together constituting an antenna complex. The frequency is around 2.5-10 MHz and the power is extremely high at 3 million Watt ("high power, high frequency phased array radio transmitter"). This is the strongest technical transmitter on earth. Its effectiveness is increased by linking the antenna array with another antenna array in Alaska, via HIPAS (High Power Auroral Stimulation). The transmitters communicate with submarines deep in the ocean and scan the horizon as a type of deep earth radar.

But the frequencies are also absorbed by the ionosphere. They heat up certain layers, creating ion turbulences by day, that are modulated onto the earth magnetic field as unnatural magnetic fields. This masks the regular effects the sun has on the ionosphere. As such, the bees lose an orientation that served them for millions of years as a reliable indicator of the time of day – encoded in the regular variations of the magnetic field changes as the sun rises and the ionosphere temperature rises.

The effects of the HAARP transmitter activity should be further investigated especially in Canada, the USA and Europe. Since the disappearance of the bees was first documented in precisely these countries, a causal relationship can no longer be excluded. The following simultaneous events tend to confirm this: In 2006, the increase of the transmitting power from 960 000 Watt to four times that power (3 600 000 Watt), was approved for the first time. Exactly in this year, reports originated in all the "scanned" transmission regions of the disrupted homing ability of bees.

Another disruptive effect may play a role. Through the irregular heating of the ionosphere, the air at great heights begins to "glow", with visible frequencies in the near infrared region (630 nm) and the associated magnetic field can be detected at the earth's surface (PEDERSEN et al. 2003, RODRIGUEZ et al. 1998).

Since the bees use not only the UV component of sunlight for orientation, but also the longer infrared wavelengths (EDRICH et al. 1979, VAN DER GLAS 1977) the new light in the sky may also be a new disrupting stimulus to them.

3.10 Disrupted NO system damages learning ability, olfactory orientation and the immune system

In following, we will further detail the significance of the (NO) system and the consequences of its disruption – in other experimental animals and particularly also in humans. The salient fact is that the NO system is affected by magnetic and



Fig. 25: HAARP location and construction.

electromagnetic oscillations and may in the worst case become totally disrupted - finally destroying molecular functions.

As in mammals, nitric oxide (NO) normally acts as a carrier of information in insects as well. The synthesis and excretion of NO is particularly high in the insect brain. In bees, NO plays a role in the ability to smell and in learning processes (MÜLLER, 1997).

As proven in humans, if the NO system of bees is disrupted through the effect of technical magnetic fields, they lose the ability to orientate themselves by smell and the vital learning programme also becomes defunct. But since NO also materially controls the immune system, disruptions to the NO household always affect the immune defences of the organism as well.

Dennis van Engelsdorp of the American Association of Professional Apiculturists (University of Pennsylvania), in his report on the investigation into the disappearance of bees, says:

"We have never seen so many different viruses together. We also found fungi, flagellates and other micro-organisms. This multiplicity of pathogens is confusing." It is also striking that the excretion organs of the bees are affected. Dennis van Engelsdorp suspects that a weakened immune system may be behind the mysterious phenomena (VAN ENGELSDORP 2007). But he rightly asks: "Are these agents the causative stress factor or the consequences of a totally different stress?"

Diana Cox-Foster, a member of the CCD working group, says: *"It is very alarming that the deaths are associated with symptoms that have never been described before". It appeared that the immune system of the animals had collapsed and some bees suffered from five or six infections simultaneously. But dead bees are nowhere to be found (Spiegel 12/2007).*

3.11 Birds sense high frequency transmitters

Birds also sense high frequency transmitters very clearly and belong to the group of animal species that reacts very sensitively to electromagnetic fields. They absorb the impinging energy particularly intensively via the feathers of their wings (CHOU et al. 1985, VAN DAM et al. 1970, BIGU-DEL-BLANCO et al. 1975 a/b).

How sensitively and promptly they react can be demonstrated by an example. Chicks exposed to a high power microwave field flee within seconds (TANNER, 1966). Investigations have, in particular, also shown how strongly microwave radiated fields affect the behaviour within a flock (WASSERMANN et al. 1984). It has repeatedly been observed that flocks of migratory birds split up when nearing a power station, to circumvent the station as if avoiding an invisible obstacle, only to re-unite again in flight afterwards. Technical disturbances in the frequency range of natural sferics, but with higher amplitudes, cause massive loss of orientation in migrating birds. The V-formation of cranes, for instance, is disrupted as they fly over transmitter stations. This phenomenon is particularly pronounced over water surfaces parallel to the flight path, which reflect electromagnetic waves.

Researchers have for a long time been pondering on how flocks of birds and also insect swarms and schools of fish stay together. It is surprising, for instance, that large flocks of starlings in an area of an estimated 500 m² or more, packed with birds, can perform complex flying manoeuvres within 5 milliseconds. But how can the animals, each at a different location in the flock, receive and react to signals in a short time? Transmission by sound would require more time and visual observation of a lead animal is blocked by other animals.

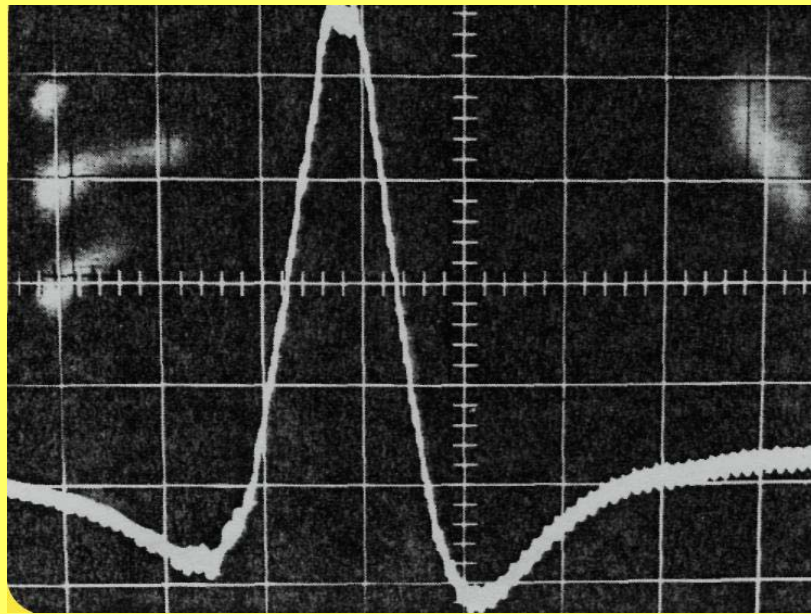


Fig. 26: Electrical field of a passing flock of birds. The small superimposed oscillation is interpreted as interference by the beat of individual wings.
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A hypothesis that the flying manoeuvres were coordinated by electromagnetic signals therefore appeared reasonable. Such a signal, propagating at approximately the speed of light, could reach all individuals at the same time and independent of their position. This hypothesis appears more plausible when taking into consideration that the flying animals are highly electrostatically charged.

We were able to record by oscilloscope that the electrical field caused by the aggregation of animals resulted in a predominantly positive overall electrostatic field. The figure also shows the very small wing beat modulation compared to the total electrical field. This modulation can be explained as a "beat" resulting from all the individual wing beats.

This beat frequency is always smaller than the wing beat frequency of an individual. The maximum beat amplitude is always much larger than the individual wing beat amplitude, however. The measured values are dependent on meteorological conditions and the geometry around the measurement.

These data allow us to conclude that flocks of small birds flying at a height of about 40 metres are electrically charged to more than 6 000 Volt. We can only speculate about the type of coded signals given for direction changing manoeuvres. It appears that each individual bird has a set beat frequency and amplitude that is corrected immediately it weakens, by changing the direction of flight.

There are presently two theories explaining the typical wedge-shaped flight formation of larger birds:

One of these assumes unhindered contact and simultaneously minimum danger of collision. The other relates aerodynamic advantage to energy economy. The first theory is based on experience with formation flying of military aircraft; the second is based mainly on calculations.

Both theories leave a couple of questions unanswered, however. How sensitive is the system to crosswinds? When a critical wind speed is exceeded – should turbulences behind the wings not distort the formation or even break up the flock? Why do the members of the flock not permanently remain in the minimum energy zone? And why is there never a formation in reverse – open to the front? Can the recognisable geometric particularities of the total formation, considering species-specific bird size and typical distances, be explained by wing-induced updrafts?

In the following, our theory of biologically sensible formations, published 25 years ago, is repeated. It describes a functional system of nature that is largely immune to meteorological interference parameters. Electrical and magnetic external fields can, however, completely destroy the formation by superposition on the biological system's own fields.

The system we are discussing allocates a position to each animal, but also considers all the flying members of the formation. It could also be identified by the analysis of formations filmed in nature. Let us inspect some facts in more detail.

Bird species flying in formation generally maintain a typical order, even in the case of only two birds flying:

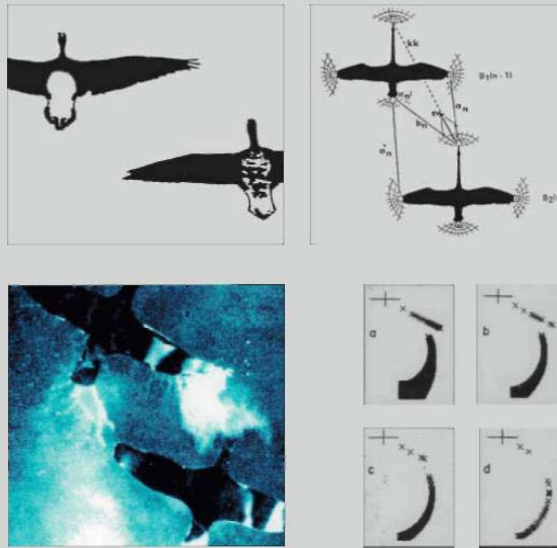


Fig. 27: Birds use electrical fields for formation flying. Top left: Two geese in formation flight. Bottom left: Experimental model to visualise the electrical field forced between these birds. Top right: Vector diagram of the field distribution. Bottom right: Calculation of the position of the bird behind with reducing degrees of freedom a-d.

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The second bird flies laterally displaced behind the first. The electrical force relationships in space are in agreement with the electrical forces determined experimentally and depicted schematically in Fig 27. The highest field strengths are at the bill, the tail and the wing tips. Referring to the bird at the back, the bill-head region is charged in the alternating field of the wings of the first bird, creating an increased force field. Simultaneously, however, the wingbeat of the bird at the back induces charges in the tail region or the extremities facing backwards and resting against the body of the first bird. There is therefore a force field between these body parts as well. The respective induced charges are coupled - as shown in the model - by the electrical field. The balancing

charges of opposite polarity, released from the former equilibrium, are free to move. They generate an effective new and measurable field. The bird under consideration, i.e. the second one, therefore not only received induced charges from the first bird but also indirectly - i.e. via the tail end of the first bird - originating from itself.

The field strength diminishes approximately as the square of the distance from the inducing charges. The magnitude of the active forces is therefore a function of the distances. Each bird is connected to every other bird via electrical fields of a certain amplitude and direction. These fields can be calculated for each species of bird - yielding the typical formation.

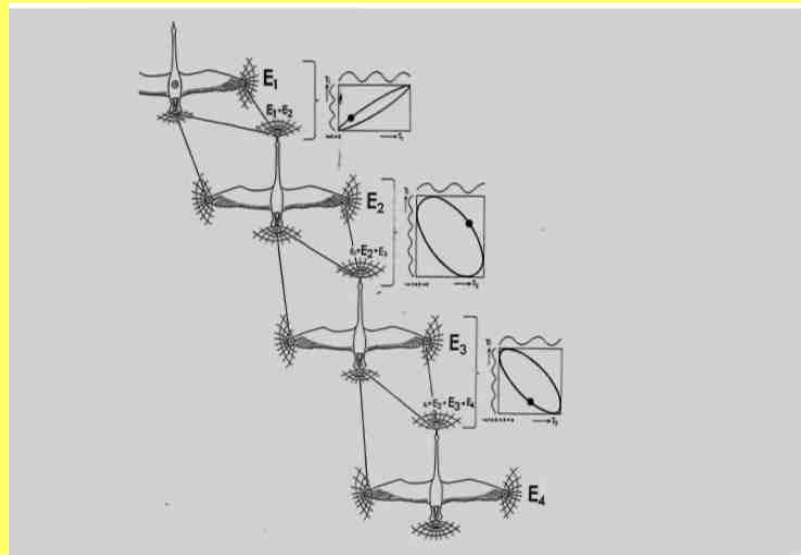


Fig. 28: V-formations can be constructed by means of an equation that I developed on the basis of physical laws. Comparisons with photographs of natural bird formations show that the assumptions are correct: The formations are given by the Coulomb forces between birds that are electrically charged in flight.
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It is significant that long necked birds in particular, tend to fly in formation. Their long neck offers the advantage that the detectors in the head region – such as the highly sensitive mechanoreceptors, which also respond to the electrical field forces, can receive signals in flight largely free from the interference of their own body. Observations of their flight behaviour show that the head region compensates for all the movements of the body itself, thereby not having any own oscillations.

Electromagnetic fields therefore have a role to play in formation flying of birds as well. They serve as orientation and navigation aid and determine the position of a single animal in the flock. Depending especially on wing width, wing span and body length, our observations and

calculations show that the biophysical relationships influence the species-typical V-formation flying of flocks. Computer calculations of the flight order allow us to predict natural formation flights. And photographic records, vice-versa, also agree well with computer simulations.

The observations demonstrate a unique information and orientation system of the animal kingdom. But they also explain why this is destroyed by the interference of technically generated electrical and magnetic fields.

Due to interference, it would not be possible to measure the magnetic field of the earth inside a flock of birds and its periodic variations caused by the individuals. The reason lies in

the moving electrical wing charges, not only generating a weak magnetic field component (induction B approx. 0.01 pT), but also inducing voltages in neighbouring matter – like an AC generator. Only the bird flying at the tip of the formation will perceive a largely undisturbed earth magnetic field component for navigation, independent of changing superpositions – provided it is sufficiently far removed from its compatriots. The remaining animals must therefore do without own navigation mechanisms and couple themselves to the birds flying in front via an electromechanical reception channel.

The birds fly straight ahead, i.e. in the desired migration direction, if the direction of the total electrical force corresponds to the direction of the connection to the head of the bird flying in front. The connecting line between heads is visible by day and can be localised by night through calls.

The recognition of direction and magnitude of the electrical total force vector is by highly sensitive mechanoreceptors at the circumferential edge of the bill. Magnetite was also found here: Through ferromagnetic resonance, magnetite is an excellent absorber of microwaves in the 0.5-10.0 GHz band. Superimposed modulations can be transformed into acoustic vibrations via the magneto-acoustic effect (KIRSCHVINK, 1996).

Correspondence of the direction of the electrical force with the head-head line assigns to each bird a prescribed position in his flock; this position can be mathematically expressed and accurately calculated. All the results of the 22 formations investigated so far confirm the theory. It may be concluded from these data that the birds' electrical characteristics have an important biological function for transfer of information (WARNKE, 1978, 1984, 1986, 1989).

3.12 Magnetite and free radicals as a magnetic compass

Artificial oscillating magnetic fields deny migrating birds the possibility to orientate themselves. The investigation covered the effect of either an electromagnetic frequency band of 0.1 – 10 MHz, or a single frequency of 7 MHz, both superimposed vertically on earth's magnetic field. These investigations again showed that not only magnetite was required for orientation and navigation but that other mechanisms such as free radicals, played an important role as well.

Because the frequencies used in the experiments correspond to the transition energy from singlet to triplet in free radicals. The animals can obviously utilise this mechanism for orientation by targeted control (RITZ et al 2004).

The following overall picture emerges: The magnetite crystals found in the bill of the animals indicate the intensity of the magnetic field. But the animals receive complementary information on the direction of orientation via the free radical levels. Using these data, they are able to know at each stage of their flight what their instantaneous location is with reference to their biological magnetic field (WILTCHKO et al. 2005) map.

If migrating birds are subjected to a stronger magnetic impulse, they will change their direction of flight. They can even be sent in the exact opposite direction with artificial fields superimposed on earth's magnetic field. Magnetic impulses convey information on the direction of migration; generated false impulses can also corrupt the migration direction (WILTCHKO et al 2006).

Summary

Bees and other insects, also birds, utilise the magnetic field of the earth and electromagnetic high frequency energy such as light. Through free radicals and simultaneously reacting magnetite conglomerates they can orientate themselves and navigate. Technically generated electromagnetic oscillations in the MHz region and low frequency magnetic impulses consistently disrupt the natural orientation and navigation mechanisms they were given through evolution.

The following can be concluded from the results of studies by other working groups and from own investigations:

1. The chitin shell of bees' and birds' feathers are semi-conducting and have piezo- and pyro- electrical properties. These body parts transform pulse-modulated high frequency into mechanical acoustic oscillation frequency. One of its important functions is the dielectric sensitivity to electromagnetic fields in the microwave region.
2. The presence of magnetite particles in the nano-range was shown in the abdomen of bees and the head region of birds. Through ferromagnetic resonance, magnetite is an excellent receiver of microwave radiated fields in the 0.5 to 10.0 GHz frequency range. In this way, pulsed microwave energy is transformed into acoustical vibrations (magneto-acoustic effect).
3. It was shown that free flying bees are capable of detecting magnetostatic fluctuations and extremely low-frequency magnetic fields with very weak inductions (from 26 nT) against the background of the 30 000 – 50 000 nT magnetic field of the earth.
4. Magnetic field impulses oriented parallel to earth's magnetic field lines, with repetition frequencies in the region of 250/sec. are responsible for clear precision errors of up to +10% in the orientation dances of bees.

5. The levels of magnetic induction in today's technically distorted environment are generally between 1 nT and 170 000 nT in the low frequency region and between some nT and a few thousand nT in the high frequency region. These values therefore generally exceed the threshold sensitivity of bees for magnetic field changes.

6. In honeybees, the NO system in the antennae has a function in the sense of smell and in learning processes. Disruptions of NO production through magnetic fields and electromagnetic oscillations have thus far been proven only in mammals. Expectations are, however, that the mechanism of disruption is the same in insects. In this case, the sense of smell and learning processes in the orientation of bees would be severely impaired.

In any event, if all the scientifically proven facts are considered, it is clear why wireless communication technologies, with their overall density of superimposed electrical, magnetic and electromagnetic fields, should disrupt the orientation and navigation of many birds and insects – above all, that of the bees.

4. Humans suffer functionality disorders

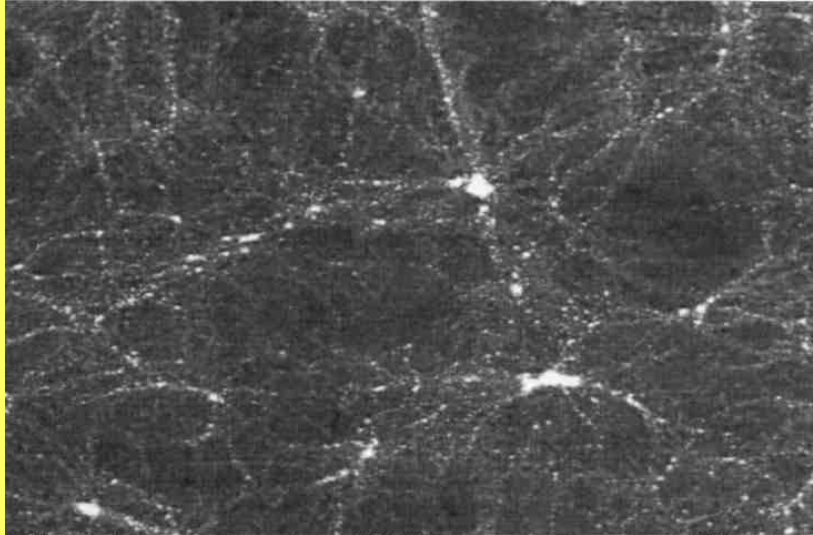


Fig. 29: All flying organisms and also other animals, including humans, are caught up in an "impenetrable" network of electromagnetic oscillations and fields. The superpositions illustrated in this model result in points of particularly high power density or field strengths.
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Humans do not have sensory organs via which electrical and magnetic energies can be detected. But these energies nevertheless envelop humans as a tightly woven net of electromagnetic oscillations and radiating fields.

We recognised the problem in the seventies already in connection with our bee experiments and called it, in our laboratory jargon, "electrosmog". The name has established itself, also via the media.

It has in the meantime been proven that humans too can transform the specific energies and forces into information, without having a specific sensory organ to do this. But the question up to now has always been: How do they do it? And to what extent can the fields damage our health?

Let us first ask what the direct effect is on humans of the high frequency energy that is spread almost uniformly across the globe for communication purposes, and then investigate whether the subjectively frequently repeated claim that this is damaging our health can possibly be true.

This requires the following steps:

1. Finding trends: *Do we have scientific literature causally correlating the epidemiologically recorded data on functional disorders and symptoms of disease in a human study group with the exposure to electromagnetic fields in the mobile radio and wireless communication range?*

2. Finding a causal mechanism: *Can a plausible mechanism be found that can explain functional disorders and disease systems as the result of exposure to these electromagnetic fields?*

3. Proof of health disorder and subsequent damage: *Can the function disorder as described be scientifically proven to be the result of subjectively described disease symptoms?*

4. Excluding a placebo effect (unfounded expectations that negatively affect health): *Do we have sound scientific procedures, such as the double-blind method, showing that the symptoms of illness are not "imaginary" and are generally rapidly reversible after the physical stress fields have been "switched off"?*

The answer to these four questions will determine whether subjectively described symptoms of illness can be ascribed to a collective placebo effect or whether those responsible are required to face consequences.

4.1 On the question of finding trends

Do we have scientific literature causally correlating the epidemiologically recorded data on functional disorders and symptoms of disease of an organism with the exposure to electromagnetic fields in the mobile radio and wireless communication range?

The answer is not treated in detail here, because it has been dealt with on several occasions elsewhere (WARNKE, 2005).

To summarise, it must be noted: There is a body of differentiated scientific literature that identified a causal correlation between epidemiologically recorded data on functional disorders and symptoms of illness of the human organism, and exposure to electromagnetic fields in the range of mobile radio and wireless communication. We therefore have an unassailable trend result.

4.2 On the effective mechanism

Can we identify a plausible effective mechanism that causally explains functional disorders and symptoms of illness as the result of exposure to electromagnetic fields?

The answer to this question does not only affect humans but analogously also birds and bees in many respects. It exposes an effective mechanism that has attracted our attention on several occasions before: The disruption of the nitrogen monoxide (NO) system. There are probably other effective mechanisms as well. But we shall only differentiate and elucidate the effective relationships of this mechanism at this point.

Nitrogen monoxide (NO) is a gas and free radical (contains unpaired electrons) that evolution has deployed as a regulator of vitality very early already – even in bacteria. This extremely important and indispensable gas is only beneficial to the organism, provided a) a certain concentration is not exceeded and b) there is no degeneration to so-called reactive nitrogens and reactive oxidative species (RNS and ROS) – i.e. no cascade-like release of newly formed free radicals and poisonous substances.

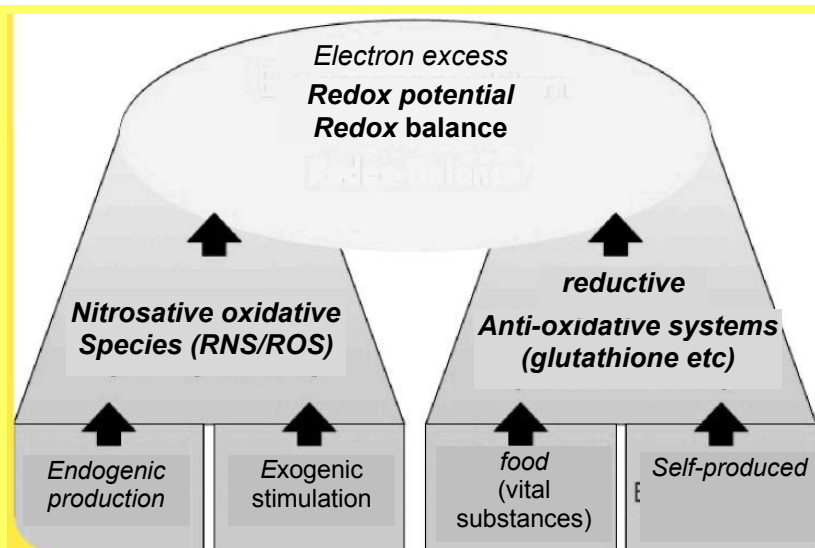


Fig. 30: Substances with an excess of electrons are indispensable for metabolism if humans and many animals want to remain healthy. Electromagnetic oscillations destroy this electron excess and form nitrosative-oxidative species (RNS/ROS). The situation is fatal to a person if anti-oxidants are also absent in the diet.
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4.2.1 Disruption of the redox balance

The NO system is closely related to the so-called redox system, which is extremely important to our molecular functions. What does this mean? Every organism needs a balanced ratio of electron excess and electron deficiency. This is also called redox balance. Oxygen compounds neutralise electron charges, causing "oxidant stress". Oxidant stress is particularly intensive if free radicals and reactive oxygen species (ROS) (e.g. superoxide anion, hydrogen-peroxide) and reactive nitrosative species (RNS) (e.g. peroxynitrite) largely prevent the antioxidative processes from re-establishing an adequate electron charge.

Shifting the redox balance towards oxidation may now result in cell damage. Oxidation may, for instance, damage unsaturated fatty acids, proteins and DNA, but particularly also the membrane – with serious consequences for heredity, energy creation and immune response.

Exposure to electrical, magnetic and electromagnetic fields disrupts the redox balance through oxidant/nitrosative stress. This can no longer be denied in the face of many in vitro and in vivo experiments – also in humans.

Latest results on the creation of oxidant/nitrosative stress through mobile radio frequencies

Human blood cells exposed to mobile radio in standby mode show increased quantities of free radicals, resulting in lipid peroxidation (MOUSTAFA et al. 2001).

In rabbits and cells of other origin, the activity of the SOD enzyme, which neutralises free radicals, increases when exposed to mobile radio (IRMAK et al. 2002, STOPCZYK et al. 2002).

The damaging oxidative processes and NO are increased in rat brains exposed to mobile radio fields; they can be alleviated again by administering antioxidants (Ginkgo biloba) (ILHAN et al. 2004).

The damaging oxidative activity is increased in the skin tissue of rats exposed to mobile radio fields; this can be alleviated by administering the melatonin hormone (AYATA et al. 2004).

Acute exposure to unmodulated 930 MHz electromagnetic fields in vitro, increases the oxidant stress level in rat lymphocytes treated with iron ions (ZMYSLONY et al. 2004).

Kidney tissue of rats shows increased levels of free radicals when exposed to mobile radio fields. The damaging effects can be alleviated through various antioxidants (OZGUNER et al. 2005). The destructive effect can be neutralised by administering melatonin hormone (OKTEM et al. 2005).

Heart tissue exposed to mobile radio fields shows an increase in the activity of free radicals. This can be reduced through antioxidants (OZGUNER et al. 2005).

When exposed to mobile radio fields, eyes show an increased activity of free radicals; this can be alleviated through administering antioxidants and melatonin hormone (OZGUNER et al. 2006).

Melatonin can limit the lipid peroxidation caused by 900 MHz mobile radio fields in the hippocampus of rats, but not in the cortex (KOYLU et al. 2006).

When exposed to mobile radio fields of base stations (SAR 11.3 mW/kg), the oxidant stress level increases; the neutralising enzyme activity is simultaneously reduced (YUREKLI et al. 2006).

Compared to controls, the mobile radio signal (GSM-DTX 2W/kg) creates increased oxidative species levels in immuno-relevant human cells (LANTOW et al. 2006).

Electromagnetic high frequencies and magnetic low frequencies create stress symptoms in lymphocytes that are similar, but not identical to heat shock (BELYAEV et al. 2005).

The effect of a 890-915 MHz mobile radio field (with 217/sec. impulse rate, 2 W max. power, SAR 0.95 W/kg) was tested on guinea pigs. The setting was on 11hr 45min stand-by and 15 min talk mode. The malonaldehyde (MDA), Glutathion (GSH), Retinol (Vitamin A), Vitamin D3, Vitamin E und catalase enzyme activity (CAT) content in the brain tissue and in the blood was chosen as the effective indicator. The MDA level rose in the brain tissue; the GSH level and CAT activity were reduced. In the blood, the MDA levels increased, as did the Vitamin A, E and D3 levels, and the CAT activity rose. The GSH level simultaneously decreased here as well. The authors conclude from this that mobile radio produces oxidant stress in the brain tissue of test animals (MERAL et al. 2007).

These results are also confirmed for the kidney in a further study (TOHUMOGLU et al. 2007).

Stimulation of the nitrogen monoxide NO free radical by electrical, magnetic and electromagnetic fields, observed for a long time, is of importance in these effects. A chronological listing below:

Electromagnetic and magnetic radiated fields promote the production of nitrogen monoxide (NO) in organisms. A chronological literature compilation

WARNKE 1979, 1980, 1984, 1993, 1994

Weak pulsating magnetic fields create an immediate effect and stimulate NO production in humans.

MIURA et al. 1993

NO increases when a weak field of high frequency radio signals is switched on; measured directly in the brain.

LAI AND SINGH 1996

DNA destroyed through electromagnetic influence; later (2004) traced back to NO stimulation.

BAWIN et al. 1996

Magnetic fields (1 or 60 Hz, 5.6, 56, μ T) had no effect when the NO synthase enzyme was pharmacologically inhibited. The effect could, on the other hand, be forced by binding NO to haemoglobin.

ADEY 1997

NO is a normal regulator of EEG rhythms and, in pathological cases, of epilepsy.

Weak magnetic fields (1 Hz, 100 μ T), modulate the NO action.

KAVALIERS et al. 1998

A 60 Hz, 141 μ T magnetic field affects the NO and NO synthase actions.

SEAMAN et al. 1999 and SEAMAN et al. 2002

Provided the body has sufficient supplies of nitrite, rapid increase of NO production when exposed to radio frequency pulses (SAR of 0.106 W/kg).

ENGSTRÖM et al. 2000

NO plays a role in the pathophysiology of oxidative stress, including Parkinson and Alzheimer disease through electromagnetic impulses.

YOSHIKAWA et al. 2000

A low frequency electromagnetic field increases the generation of NO.

PAREDI u.a. 2001

The production of NO also increases under exposure to the electromagnetic fields of mobile phones.

DINIZ et al. 2002

The increased proliferation of cells exposed to pulsating electromagnetic fields is caused by NO.

KIM et al. 2002

Pulsating electromagnetic fields amplify the neuronal NO synthase expression.

LAI AND SINGH 2004

Inhibitor of NO synthase (7-nitroindazol) blocks the effects of weak magnetic alternating fields (60 Hz, 10 μ T).

ILHAN et al. 2004

Frequencies used by mobile radio (900 MHz) cause increased activity of NO levels, increase malondialdehyde, increase xanthin oxidase, decrease superoxide dismutase and glutathione peroxidase - thereby destroying the brain of rats. Antioxidants (Ginkgo biloba) counter this.

YARIKTAS et al. 2005

The NO level in the mucosa of the nose increases when exposed to mobile radio fields (900 MHz).

AKDAG et al. 2007

The long-term effect (2 hours per day for 10 months) of a low frequency pulsed magnetic field on rats reduces the NO production below the nominal values.

It has been known for many decades already that weak low frequency magnetic fields increase the levels of free radicals. It is therefore not necessary to quote further literature at this point.

4.2.2 Primary mechanism found: Enzymes transferring electrons are magneto-sensitive

Stimulation of free radicals – including NO – through physical fields and radiated fields is therefore scientifically and reliably proven. But viewed critically, this is no proof of damage unless the underlying primary mechanism is identified. For this reason, we searched for a long time for a link to explain the damaging effect. And we have found it in one of the latest studies: The NADH oxidase enzyme exhibits a high – and quite reproducible – sensitivity for magnetic and electromagnetic fields of mobile phones (FRIEDMAN et al. 2007).

This sensitivity had been known for quite some time in connection with other oxidases such as cytochrome oxidase (BLANK et al. 1998, 2001 a/b). For a long time, it was believed that NADH oxidase was active only in certain cells such as phagocytes. But it was known for quite some time that it was sensitive to gravitation (NASA, 2006). In the meantime, homologues of NADH oxidase were discovered in various tissues and were collectively included in the NOX family (NOX1, NOX3, NOX4, NOX5, DUOX1 and DUOX2).

The NOX family is also responsible for a large range of pathological processes, especially neurodegeneration and heart diseases (BEDARD et al. 2007).

These oxidase enzymes are magnetically sensitive due to their capability of shepherding electrons through plasma membranes. When electrons move, an electrical current flows that in turn builds up its own magnetic field and also generates electromagnetic high frequency oscillations through acceleration and deceleration of electron movement. All these processes create sensitivity to external fields.

The electron transfer is finally responsible for the production of superoxide radicals and other reactive oxygen species (ROS). The consequences of this are far reaching in completely different areas, because radicals and ROS are very aggressive. In this way, the destruction of viruses and bacteria is promoted, the creation of proteins is forced through reinforced gene expression and finally cell proliferation is supported at the cost of cell differentiation.

Over-stimulation is a threat. It is analogous to a drug or medicine: Dosed correctly, the substance can be beneficial; but overdosing can be poisonous. This is exactly what happens with permanent exposure to magnetic and electromagnetic fields.

In detail, this process is as follows: It is a fact that the NADH oxidase enzyme also produces the superoxide anion ($O_2^{\cdot-}$) free radical. Superoxide anion is damaging to the NO budget, among other. NO may be deactivated and may subsequently degrade, negatively affecting various vital parameters (WARNHOLTZ et al. 1999).

What is new is the realisation that NADH oxidase also forces the generation of NO by stimulating the eNOS enzyme (SUZUKI et al. 2006, RACASAN et al. 2005). This stimulation of eNOS then becomes a further source of increased superoxide anion radical generation (SEINOSUKE et al. 2004). This is not the end of the list in this fatal loop of overstimulation, because the NADH oxidase system also stimulates the formation of toxic hydrogen peroxide (H_2O_2), which also increases NO production by up to 100% (LI et al. 2002). These two additional NO stimulants explain the abovementioned increased NO production under the influence of magnetic fields and electromagnetic radiated fields – also through mobile radio communication.

But this is the start of a vicious circle. Because overstimulation of the eNOS enzyme, that in the final analysis is also an agent for increased NO production, also increases superoxide anion radicals on its own (SEINOSUKE et al. 2004). Nature, however, also has a cleverly devised countermeasure against excessive and dangerous NO production threatening overproduction of a radical: The more stimulated hydrogen peroxide, which also increases the NO production, is an agent for de-activating eNOS co-factors, which finally prevents the NO production by affecting the membrane receptor (JAMES et al. 2001). Such a reduction of NO has also been found before under long-term exposure to stronger magnetic fields (AKDAG et al. 2007). Even if the NO now appears to be regulated, the damaging effects of ROS remain intact.

The real pathological effects arise afterwards. We have to consider, in addition, that both the NO and ROS, which includes superoxide anion, are important modulators of the vascular tonus and are architects of the adhesive interaction of leucocytes, platelets and endothelium. The two molecules of NO and of superoxide anion, however, have opposite effects: NO is normally beneficial in a healthy life cycle; ROS, however, prepares the system for special regulation when disruptions occur.

The functions are thereby flexibly adjusted. This allocation of functions disappears, however, under the influence of an external magnetic and electromagnetic field: NO and ROS now react together. In this event, their specific effective potential is destroyed and toxic substances are created, such as peroxynitrite ($ONOO^-$) (MÜNZEL et al. 1999). This peroxynitrite in turn reacts with hydrogens, creating more hydrogen peroxide.

Because this mechanism is so important, we shall summarise it in one sentence: The serious pathological disruption is caused by exposure to magnetic and radiated fields resulting in the creation of additional reactive oxygen species (ROS) such as superoxide radicals and hydrogen peroxide, that combine with the increasingly produced NO to form extremely toxic peroxynitrite, that in turn reacts with hydrogens to form more hydrogen peroxide. The consequences of the pathological process are listed further down.

Many vital substances, required for functioning of the body, are rendered useless.

If the cascade of effects is disrupted, the normal and healthy effects of NO are restored (HORNIG et al. 2001).

The NADH oxidase is important in another sense as well. It is also found in the cell nucleus where it can – depending on the redox system – control the gene expression, but can also damage genes (MASUKA, 2006).

Let us therefore state in response to the question of a conclusive effective mechanism: The existing scientific literature abundantly documents disruptions of the redox balance in organisms through reactive oxidative and nitrogenous species (ROS/RNS), causally connected to the exposure to electromagnetic fields of mobile radio and wireless communication.

An unambiguous effective damaging mechanism has therefore been found.

4.3 On the question of deterioration of health and damage

Can the functional disruptions thus explained be considered the scientifically proven reasons for the subjectively described symptoms of disease?

The proven effective mechanism is important also because it shows that the subjective ailments of many people are based on biological facts that can be explained. If you are aware of the cascades of effects described hereunder, you will better understand why "electrosmog" is damaging.

4.3.1 Functional disruptions and symptoms of disease

Electromagnetically induced excessive ROS/RNS stimulation may be differentiated into three effective phases that are passed through sequentially:

1. Stimulation of free radicals,
2. Stimulation of highly toxic peroxynitrite,
3. Stimulation of highly toxic peroxide radical.

The following processes are serious: Cell components are destroyed; the antioxidants absorbed with the food and the substances with excess electrons produced by the organism itself are spent; the harmful cholesterol increases. People feel tired, tense, battle various inflammations.

Pain is felt in places. More detail on the individual steps is given below.

First complex: Stimulation of free radicals such as superoxide $O_2^{\cdot-}$ and NO leads to

- activation of protooncogenes
- damage to the mitochondria genome
- damage to the cell nucleus genome
- damage to the membranes
- oxidation of the polyene fatty acids of the membranes; release of cardiolipins (auto antibody formation)
- oxidation of SH groups, causing enzyme blocking
- activation of proteases (cell damage)
- activation of transcription factors.

Second complex: Stimulation of highly toxic peroxynitrite from superoxide anion $O_2^{\cdot-}$ together with NO ($O_2^{\cdot-} + NO = ONOO^{\cdot-}$)

NO has three times the affinity for superoxide $O_2^{\cdot-}$ that $O_2^{\cdot-}$ has for the neutralising superoxide dismutase; the peroxynitrite

- oxidises vitamin C
- oxidises uric acid
- oxidises cholesterol
- oxidises sulfhydryl groups (destroys thioles)
- oxidises polyene fatty acids of the membranes (initiates lipid peroxidation)
- causes DNA breaks
- activates kinases (phospholipase 2)
- activates polymerase (PAPP); this destroys NAD⁺, leading to a cellular energetic catastrophe.

NO and peroxynitrite react to form nitroendioxide (NO_2); this deactivates superoxide dismutase (MnSOD), i.e. inhibiting the neutralising enzymes of the mitochondria (mt-Mn-SOD). These reactions alone result in massive metabolism disruptions already.

Third complex: Stimulation of highly toxic peroxide radical ($HO_2^{\cdot-}$) from superoxide and peroxynitrite with the involvement of hydrogen

Peroxide $HO_2^{\cdot-}$ has a redox potential of +1000 mV, making it highly oxidising.. An addition to the listing in complex 2, peroxide also oxidises:

- Polyene fatty acids
- Tocopherol (Vit E)
- Lycopene
- Co-enzyme Q 10

The functional disruptions are manifest in disease symptoms, as described in more detail below.

4.3.2 The "Acquired Energy Dyssymbiosis Syndrome" (AEDS)

The clinical picture of the "Acquired Energy Dyssymbiosis Syndrome" describes a deficiency of cell energy – with simultaneous derailment of the cell environment. This leads to "mitochondriopathy": energy creation is blocked; the power generators for cell energy are transformed into copious sources of free radicals.

The changes have serious consequences:

1. Inflammation processes spread and release further substances that are harmful when overdosed (tumour necrosis factor TNF α and time and again nitrogen monoxide). We must also not forget that inflammations are on the increase in our industrial society and that arteriosclerosis and heart attacks – the primary cause of death – are ultimately caused by inflammations. This point of view has already been accepted among the scientifically active medical fraternity today.

2. Aerobic glycolysis (glycolysis despite the presence of oxygen) is activated as "emergency power generator" – which is in turn associated with:

- stimulation of proto-oncogenes (precursors to cancer genes)
- increased release of superoxide radicals
- lactate acidosis (excessive acidification)

3. The mitochondrial genome finally mutates. But especially this pathological change can be hereditary via the female gender. It burdens the progeny for the generations to come.

Overview: Physiopathological consequences of nitrosative/oxidative stress

- I. Disruption of mitochondrial activity
- II. Disruption of sugar utilisation (pathological lactate acidosis)
- III. Disruption of the neurotransmitter function
- IV. Disruption of the cholesterol metabolism
- V. Disruption of the steroid hormone synthesis (corticoids)
- VI. Disruption of the haem system
- VII. Generation of mutations, esp. the mitochondrial DNA (hereditary)
- VIII. Disruption of apoptosis

Catalogue of symptoms and diseases (excerpt), derived from the known effective mechanisms of nitrosative/oxidative stress

- Sleep disorders
- High level of fatigue: no relaxation, recuperation times ineffective
- Psychosomatic performance lapses
- Major phases of restlessness and "panic disorder"
- Corpulence
- Chronic hypoglycemia
- Increased cholesterol and triglycerid values
- Lactate acidosis
- Fibromyalgia FMS (nitroso serotonin auto-antibody formation)
- Autoimmune diseases
- Arteriosclerosis
- M. Parkinson
- Chronic inflammation processes, especially in the nervous system, with multiple sclerosis and amyotrophic lateral sclerosis
- Haem synthesis disruptions (porphyria)
- Lactose intolerance
- Pathological energy deficit PED (WARNKE, 1989)
- Chronic immune insufficiency

(high infection susceptibility)

- Functional disruptions of the thyroid
- Myopathy
- Encephalopathy
- Polyneuropathy
- Enteropathy
- Cancer
- AIDS

To summarise, we can answer the question as to whether subjective reports of illness have an objective basis as follows: The redox balance is disturbed via the direct influence of weak magnetic and electromagnetic fields on the NADH oxidase. The result is oxidative/nitrosative stress. It leads to disruptions and prevention of vital functions. In the course of these processes, exactly those disease symptoms subjectively described by those affected and exposed to radiated fields, are in evidence.

Hereditary pathological changes passed on via the mother should, in particular, draw our attention to the effects that will only manifest themselves in generations to come.

4.4 On the exclusion of a placebo effect.

Do we have scientifically designed methods, such as the "double blind" method, proving that the symptoms of disease cannot be attributed to fears but that they are generally reversible after "switching off" the physically stressing fields (unknown to the participants), after a short period of time?

All the scientific investigations that addressed this question reply to this question with "yes":

The various problems disappear if the influence of the radiation or the ROS/RNS formation is "switched off" (e.g. ABELIN 1999, ABELIN et al. 1995, HORNIG et al. 2001, PETROV1970, TNO study 2004).

Health is not, however, restored if the disruptions have already led to serious damage such as DNA destruction or tumours.

5. Summary

For many decades, research results showing that the natural electrical and magnetic fields and their variation are a vital precondition for the orientation and navigation of a whole range of animals, have been freely available.

What has also been known to science for many decades is that we as humans depend on this natural environment for many of our vital functions.

Today, however, this natural information and functional system of humans, animals and plants has been superimposed by an unprecedented dense and energetic mesh of artificial magnetic, electrical and electromagnetic fields, generated by numerous mobile radio and wireless communication technologies.

The consequences of this development have also been predicted by the critics for many decades and can now no longer be ignored. Bees and other insects disappear, birds avoid certain areas and are disoriented in other locations. Humans suffer from functional disorders and diseases. And those that are hereditary are passed on to the next generation as existing defects.



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Glossary (GL)

Information: This concept is generally understood in everyday life and has gained a position of central importance, especially in modern biosciences. “Informed society” demands to be in a position to inform itself about everything if possible, at any time and any place in the world. Analogously, it is of cardinal importance to a living organism of any kind, not only to be in a position to communicate with its environment via **information carriers**, but the control of its internal vital functions must also be assured, which is again possible only through the exchange of “information”.

Electromagnetic (EM) fields of all kinds and magnitudes (including light, UV and infrared radiation, microwaves, etc.) were chosen by evolution as particularly suitable carriers of information because they are able to flood the living space of organisms spontaneously and fully, affording every individual immediate access to its **information content**.

This is available in the ordered structure of the EM fields themselves, described in physics as waves, which propagate at the speed of light with alternating electrical and magnetic field components. Because, in accordance with Faraday’s **law of induction** (1831), the changes in a magnetic field induce changes in an **electrical field**.

The force/field lines of magnetic and electrical fields are in the form of vectors between positive and negative poles and we can therefore describe them as electrical or magnetic flux and a **flux density** orthogonal to a unit surface area, e.g. 1 m^2 .

The actual information in an EM field resides – similar to acoustics – in the number of oscillations per second (=frequency) and also in the **amplitude** of the oscillations. If an EM field of higher frequency is interrupted at a certain rate (facilitated only through modern digital technology), **low frequency pulsed high frequency** radiation is created, whereby the cyclic rate can also be used for information purposes.

The traditional technical method of transmitting “information” is called **modulation**. In this way, a continuous low frequency **carrier wave**, subject to less interference during propagation in space, is overlaid (modulated) with the higher frequencies of music and voice, allowing the information to be transmitted over large distances.

A few common physical units:

Ampere (A): current amplitude
 Volt (V): electrical voltage
 V / meter (E): electrical field strength
 Watt (W): power (=VA)
 Joule (J): electrical energy (=W sec)
 Tesla (T): magnetic induction
 (=V sec/m²)

Number units

(k) Kilo... * 1000
 (M) Mega ... * 1000 000
 (G) Giga ... * 1000 000 000
 (T) Tera ... * 1000 000 000 000

 (m) Milli ... * 0.000
 (μ) Micro ... * 0.000 000
 (n) Nano ... * 0.000 000 000
 (p) Pico ... * 0.000 000 000 000

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On this paper

The bio-scientist Ulrich Warnke is one of the foremost experts on nature's electro-magnetic housekeeping. The paper presented here is the first in a new scientific series by independent scientists, physicians and technicians in which he demonstrates how wisely and sensitively nature has made use of electrical and magnetic fields in the development of life. But it is precisely for this reason that he is in a position to convincingly criticise our foolish and irresponsible interference in the natural scheme of things. The paper shows that we are now in the process of destroying, within a few decades, that which nature has created over millions of years.

The prognosis is even more disturbing because it is not based on hypotheses and probabilities but on the identification of verifiable and reproducible effective mechanisms. It is clear that those in politically responsible positions, constitutionally obliged to take precautions, must now consider the inevitable consequences. Whoever continues to retreat to the common and comfortable strategy of belittlement today, denying any knowledge of serious risks, only exposes an attitude in terms of which short-sighted economic interests are closer to his heart than the future of generations to come.

"This natural system of information and functionality in humans, animals and plants has today been superimposed by an unprecedented dense and energetic mesh of artificial magnetic, electrical and electromagnetic fields, generated by numerous mobile radio and wireless communication technologies. The critics have predicted the consequences of this development by for many decades and these can now no longer be ignored. Bees and other insects disappear; birds avoid certain areas and are disoriented in other locations. Humans suffer functional disorders and illnesses. And those disorders that are hereditary are passed on to the next generation as pre-programmed defects".

Prof. Dr. K. Hecht, Dr. med. M. Kern, Prof. Dr. K. Richter, Dr. med. H.-Chr. Scheiner

About the author

The main fields of activity of Dr. rer. nat. Ulrich Warnke, an internationally renowned bio-scientist of the University of the Saarland, are in bio-medicine, environmental medicine and bio-physics. His research has, for many decades, focussed especially on the effect of electromagnetic fields.

Errata:

On page 6, middle column, last paragraph, add “up to 300 GHz” after “over all frequencies”

Page 33, column.1, delete the second last paragraph: “Human immuno-relevant ..(LANTOW et al. 2006).

Page 35, first column, last word: Instead “infections” it is better to write “inflammations”.

Page 35, second column at the end: from “Peroxide HOO°also oxidises” should be in a smaller font

Page 39, first column: “Hornig Burkhard.... .. 103: 799” to be deleted (it is correctly cited earlier).

Page 39, third column: “Lantow M, Schluderer J,..... 88-94” to be deleted.

Page 40, third column, last reference: “Towne WF.....” should be as separate reference.

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Part 2: Radiofrequency Electromagnetic Fields, 2013
(Tab 13 Part 1)

NON-IONIZING RADIATION, PART 2: RADIOFREQUENCY ELECTROMAGNETIC FIELDS

VOLUME 102

IARC MONOGRAPHS
ON THE EVALUATION
OF CARCINOGENIC RISKS
TO HUMANS

NON-IONIZING RADIATION, PART 2: RADIOFREQUENCY ELECTROMAGNETIC FIELDS

VOLUME 102

This publication represents the views and expert
opinions of an IARC Working Group on the
Evaluation of Carcinogenic Risks to Humans,
which met in Lyon, 24-31 May 2011

LYON, FRANCE - 2013

**IARC MONOGRAPHS
ON THE EVALUATION
OF CARCINOGENIC RISKS
TO HUMANS**

IARC MONOGRAPHS

In 1969, the International Agency for Research on Cancer (IARC) initiated a programme on the evaluation of the carcinogenic risk of chemicals to humans involving the production of critically evaluated monographs on individual chemicals. The programme was subsequently expanded to include evaluations of carcinogenic risks associated with exposures to complex mixtures, lifestyle factors and biological and physical agents, as well as those in specific occupations. The objective of the programme is to elaborate and publish in the form of monographs critical reviews of data on carcinogenicity for agents to which humans are known to be exposed and on specific exposure situations; to evaluate these data in terms of human risk with the help of international working groups of experts in chemical carcinogenesis and related fields; and to indicate where additional research efforts are needed. The lists of IARC evaluations are regularly updated and are available on the Internet at <http://monographs.iarc.fr/>.

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NOTE TO THE READER

The term ‘carcinogenic risk’ in the *IARC Monographs* series is taken to mean that an agent is capable of causing cancer. The *Monographs* evaluate cancer hazards, despite the historical presence of the word ‘risks’ in the title.

Inclusion of an agent in the *Monographs* does not imply that it is a carcinogen, only that the published data have been examined. Equally, the fact that an agent has not yet been evaluated in a *Monograph* does not mean that it is not carcinogenic. Similarly, identification of cancer sites with *sufficient evidence* or *limited evidence* in humans should not be viewed as precluding the possibility that an agent may cause cancer at other sites.

The evaluations of carcinogenic risk are made by international working groups of independent scientists and are qualitative in nature. No recommendation is given for regulation or legislation.

Anyone who is aware of published data that may alter the evaluation of the carcinogenic risk of an agent to humans is encouraged to make this information available to the Section of IARC Monographs, International Agency for Research on Cancer, 150 cours Albert Thomas, 69372 Lyon Cedex 08, France, in order that the agent may be considered for re-evaluation by a future Working Group.

Although every effort is made to prepare the *Monographs* as accurately as possible, mistakes may occur. Readers are requested to communicate any errors to the Section of IARC Monographs, so that corrections can be reported in future volumes.

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¹ Working Group Members and Invited Specialists serve in their individual capacities as scientists and not as representatives of their government or any organization with which they are affiliated. Affiliations are provided for identification purposes only. Invited Specialists do not serve as meeting chair or subgroup chair, draft text that pertains to the description or interpretation of cancer data, or participate in the evaluations.

Each participant was asked to disclose pertinent research, employment, and financial interests. Current financial interests and research and employment interests during the past 4 years or anticipated in the future are identified here. Minor pertinent interests are not listed and include stock valued at no more than US\$1000 overall, grants that provide no more than 5% of the research budget of the expert's organization and that do not support the expert's research or position, and consulting or speaking on matters not before a court or government agency that does not exceed 2% of total professional time or compensation. All grants that support the expert's research or position and all consulting or speaking on behalf of an interested party on matters before a court or government agency are listed as significant pertinent interests.

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² René de Seze received significant research support (more than €100 000, ceased in 2009) from Fondation Santé et Radiofréquences, a research foundation created under the leadership of the French Ministry of Research and with public interest status. Half of the budget is state funded, the other half is provided by industry. The Foundation's independence and the transparency of its operations are guaranteed by its Ethics Charter. In 2009, René de Seze prepared a report for a plaintiff's lawyer on the association between radiofrequency fields and brain cancer.

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⁴Martin Rösli receives research funding for studies on adverse health effects of mobile-phone use from Forschungsstiftung Mobilfunk, a non-profit-making research foundation at ETH Zurich. Neither industry, nor nongovernmental organizations are represented on the scientific board of the foundation. He also serves as a member on the board of this foundation.

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⁶ Luc Verschaeve's research institute received a small research grant (less than €5000, ceased in March 2011) from the GSM Operators Forum on environmental effects of mobile-phone base stations.

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⁸ Niels Kuster is Director and Board member of the non-profit IT'IS Foundation that performs exposure assessments for industry and governments, and also President of the Board and shareholder of Near-Field Technology AG, a holding controlling two companies, SPEAG and ZMT, that are active in the development of near-field measurement instruments, simulation software and medical-test equipment.

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⁹ Each Observer agreed to respect the Guidelines for Observers at IARC Monographs meetings. Observers did not serve as meeting chair or subgroup chair, draft any part of a Monograph, or participate in the evaluations. They also agreed not to contact participants before the meeting, not to lobby them at any time, not to send them written materials, and not to offer them meals or other favours. IARC asked and reminded Working Group Members to report any contact or attempt to influence that they may have encountered, either before or during the meeting.

¹⁰ Joe Elder is self-employed as a radiofrequency bioeffects consultant. He was employed by Motorola (until 2009) and his wife holds stock in Motorola. His participation as an Observer in this IARC Monographs meeting is sponsored by the Mobile Manufacturers Forum representing manufacturers of mobile and wireless communication devices and the network infrastructure that supports them.

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PREAMBLE

The Preamble to the *IARC Monographs* describes the objective and scope of the programme, the scientific principles and procedures used in developing a *Monograph*, the types of evidence considered and the scientific criteria that guide the evaluations. The Preamble should be consulted when reading a *Monograph* or list of evaluations.

A. GENERAL PRINCIPLES AND PROCEDURES

1. Background

Soon after IARC was established in 1965, it received frequent requests for advice on the carcinogenic risk of chemicals, including requests for lists of known and suspected human carcinogens. It was clear that it would not be a simple task to summarize adequately the complexity of the information that was available, and IARC began to consider means of obtaining international expert opinion on this topic. In 1970, the IARC Advisory Committee on Environmental Carcinogenesis recommended ‘...that a compendium on carcinogenic chemicals be prepared by experts. The biological activity and evaluation of practical importance to public health should be referenced and documented.’ The IARC Governing Council adopted a resolution concerning the role of IARC in providing government authorities with expert, independent, scientific opinion on environmental carcinogenesis. As one means to that end, the Governing Council recommended that IARC should prepare monographs on the evaluation of carcinogenic

risk of chemicals to man, which became the initial title of the series.

In the succeeding years, the scope of the programme broadened as *Monographs* were developed for groups of related chemicals, complex mixtures, occupational exposures, physical and biological agents and lifestyle factors. In 1988, the phrase ‘of chemicals’ was dropped from the title, which assumed its present form, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*.

Through the *Monographs* programme, IARC seeks to identify the causes of human cancer. This is the first step in cancer prevention, which is needed as much today as when IARC was established. The global burden of cancer is high and continues to increase: the annual number of new cases was estimated at 10.1 million in 2000 and is expected to reach 15 million by 2020 ([Stewart & Kleihues, 2003](#)). With current trends in demographics and exposure, the cancer burden has been shifting from high-resource countries to low- and medium-resource countries. As a result of *Monographs* evaluations, national health agencies have been able, on scientific grounds, to take measures to reduce human exposure to carcinogens in the workplace and in the environment.

The criteria established in 1971 to evaluate carcinogenic risks to humans were adopted by the Working Groups whose deliberations resulted in the first 16 volumes of the *Monographs* series. Those criteria were subsequently updated by further ad hoc Advisory Groups ([IARC, 1977, 1978, 1979, 1982, 1983, 1987, 1988, 1991](#); [Vainio et al., 1992](#); [IARC, 2005, 2006](#)).

The Preamble is primarily a statement of scientific principles, rather than a specification of working procedures. The procedures through which a Working Group implements these principles are not specified in detail. They usually involve operations that have been established as being effective during previous *Monograph* meetings but remain, predominantly, the prerogative of each individual Working Group.

2. Objective and scope

The objective of the programme is to prepare, with the help of international Working Groups of experts, and to publish in the form of *Monographs*, critical reviews and evaluations of evidence on the carcinogenicity of a wide range of human exposures. The *Monographs* represent the first step in carcinogen risk assessment, which involves examination of all relevant information to assess the strength of the available evidence that an agent could alter the age-specific incidence of cancer in humans. The *Monographs* may also indicate where additional research efforts are needed, specifically when data immediately relevant to an evaluation are not available.

In this Preamble, the term ‘agent’ refers to any entity or circumstance that is subject to evaluation in a *Monograph*. As the scope of the programme has broadened, categories of agents now include specific chemicals, groups of related chemicals, complex mixtures, occupational or environmental exposures, cultural or behavioural practices, biological organisms and physical agents. This list of categories may expand as

causation of, and susceptibility to, malignant disease become more fully understood.

A cancer ‘hazard’ is an agent that is capable of causing cancer under some circumstances, while a cancer ‘risk’ is an estimate of the carcinogenic effects expected from exposure to a cancer hazard. The *Monographs* are an exercise in evaluating cancer hazards, despite the historical presence of the word ‘risks’ in the title. The distinction between hazard and risk is important, and the *Monographs* identify cancer hazards even when risks are very low at current exposure levels, because new uses or unforeseen exposures could engender risks that are significantly higher.

In the *Monographs*, an agent is termed ‘carcinogenic’ if it is capable of increasing the incidence of malignant neoplasms, reducing their latency, or increasing their severity or multiplicity. The induction of benign neoplasms may in some circumstances (see Part B, Section 3a) contribute to the judgement that the agent is carcinogenic. The terms ‘neoplasm’ and ‘tumour’ are used interchangeably.

The Preamble continues the previous usage of the phrase ‘strength of evidence’ as a matter of historical continuity, although it should be understood that *Monographs* evaluations consider studies that support a finding of a cancer hazard as well as studies that do not.

Some epidemiological and experimental studies indicate that different agents may act at different stages in the carcinogenic process, and several different mechanisms may be involved. The aim of the *Monographs* has been, from their inception, to evaluate evidence of carcinogenicity at any stage in the carcinogenesis process, independently of the underlying mechanisms. Information on mechanisms may, however, be used in making the overall evaluation ([IARC, 1991](#); [Vainio et al., 1992](#); [IARC, 2005, 2006](#); see also Part B, Sections 4 and 6). As mechanisms of carcinogenesis are elucidated, IARC convenes international scientific conferences to determine whether a broad-based consensus has emerged

on how specific mechanistic data can be used in an evaluation of human carcinogenicity. The results of such conferences are reported in IARC Scientific Publications, which, as long as they still reflect the current state of scientific knowledge, may guide subsequent Working Groups.

Although the *Monographs* have emphasized hazard identification, important issues may also involve dose–response assessment. In many cases, the same epidemiological and experimental studies used to evaluate a cancer hazard can also be used to estimate a dose–response relationship. A *Monograph* may undertake to estimate dose–response relationships within the range of the available epidemiological data, or it may compare the dose–response information from experimental and epidemiological studies. In some cases, a subsequent publication may be prepared by a separate Working Group with expertise in quantitative dose–response assessment.

The *Monographs* are used by national and international authorities to make risk assessments, formulate decisions concerning preventive measures, provide effective cancer control programmes and decide among alternative options for public health decisions. The evaluations of IARC Working Groups are scientific, qualitative judgements on the evidence for or against carcinogenicity provided by the available data. These evaluations represent only one part of the body of information on which public health decisions may be based. Public health options vary from one situation to another and from country to country and relate to many factors, including different socioeconomic and national priorities. Therefore, no recommendation is given with regard to regulation or legislation, which are the responsibility of individual governments or other international organizations.

3. Selection of agents for review

Agents are selected for review on the basis of two main criteria: (a) there is evidence of human

exposure and (b) there is some evidence or suspicion of carcinogenicity. Mixed exposures may occur in occupational and environmental settings and as a result of individual and cultural habits (such as tobacco smoking and dietary practices). Chemical analogues and compounds with biological or physical characteristics similar to those of suspected carcinogens may also be considered, even in the absence of data on a possible carcinogenic effect in humans or experimental animals.

The scientific literature is surveyed for published data relevant to an assessment of carcinogenicity. Ad hoc Advisory Groups convened by IARC in 1984, 1989, 1991, 1993, 1998 and 2003 made recommendations as to which agents should be evaluated in the *Monographs* series. Recent recommendations are available on the *Monographs* programme web site (<http://monographs.iarc.fr>). IARC may schedule other agents for review as it becomes aware of new scientific information or as national health agencies identify an urgent public health need related to cancer.

As significant new data become available on an agent for which a *Monograph* exists, a re-evaluation may be made at a subsequent meeting, and a new *Monograph* published. In some cases it may be appropriate to review only the data published since a prior evaluation. This can be useful for updating a database, reviewing new data to resolve a previously open question or identifying new tumour sites associated with a carcinogenic agent. Major changes in an evaluation (e.g. a new classification in Group 1 or a determination that a mechanism does not operate in humans, see Part B, Section 6) are more appropriately addressed by a full review.

4. Data for the *Monographs*

Each *Monograph* reviews all pertinent epidemiological studies and cancer bioassays in experimental animals. Those judged inadequate

or irrelevant to the evaluation may be cited but not summarized. If a group of similar studies is not reviewed, the reasons are indicated.

Mechanistic and other relevant data are also reviewed. A *Monograph* does not necessarily cite all the mechanistic literature concerning the agent being evaluated (see Part B, Section 4). Only those data considered by the Working Group to be relevant to making the evaluation are included.

With regard to epidemiological studies, cancer bioassays, and mechanistic and other relevant data, only reports that have been published or accepted for publication in the openly available scientific literature are reviewed. The same publication requirement applies to studies originating from IARC, including meta-analyses or pooled analyses commissioned by IARC in advance of a meeting (see Part B, Section 2c). Data from government agency reports that are publicly available are also considered. Exceptionally, doctoral theses and other material that are in their final form and publicly available may be reviewed.

Exposure data and other information on an agent under consideration are also reviewed. In the sections on chemical and physical properties, on analysis, on production and use and on occurrence, published and unpublished sources of information may be considered.

Inclusion of a study does not imply acceptance of the adequacy of the study design or of the analysis and interpretation of the results, and limitations are clearly outlined in square brackets at the end of each study description (see Part B). The reasons for not giving further consideration to an individual study also are indicated in the square brackets.

5. Meeting participants

Five categories of participant can be present at *Monograph* meetings.

(a) *The Working Group*

The Working Group is responsible for the critical reviews and evaluations that are developed during the meeting. The tasks of Working Group Members are: (i) to ascertain that all appropriate data have been collected; (ii) to select the data relevant for the evaluation on the basis of scientific merit; (iii) to prepare accurate summaries of the data to enable the reader to follow the reasoning of the Working Group; (iv) to evaluate the results of epidemiological and experimental studies on cancer; (v) to evaluate data relevant to the understanding of mechanisms of carcinogenesis; and (vi) to make an overall evaluation of the carcinogenicity of the exposure to humans. Working Group Members generally have published significant research related to the carcinogenicity of the agents being reviewed, and IARC uses literature searches to identify most experts. Working Group Members are selected on the basis of (a) knowledge and experience and (b) absence of real or apparent conflicts of interests. Consideration is also given to demographic diversity and balance of scientific findings and views.

(b) *Invited Specialists*

Invited Specialists are experts who also have critical knowledge and experience but have a real or apparent conflict of interests. These experts are invited when necessary to assist in the Working Group by contributing their unique knowledge and experience during subgroup and plenary discussions. They may also contribute text on non-influential issues in the section on exposure, such as a general description of data on production and use (see Part B, Section 1). Invited Specialists do not serve as meeting chair or subgroup chair, draft text that pertains to the description or interpretation of cancer data, or participate in the evaluations.

(c) *Representatives of national and international health agencies*

Representatives of national and international health agencies often attend meetings because their agencies sponsor the programme or are interested in the subject of a meeting. Representatives do not serve as meeting chair or subgroup chair, draft any part of a *Monograph*, or participate in the evaluations.

(d) *Observers with relevant scientific credentials*

Observers with relevant scientific credentials may be admitted to a meeting by IARC in limited numbers. Attention will be given to achieving a balance of Observers from constituencies with differing perspectives. They are invited to observe the meeting and should not attempt to influence it. Observers do not serve as meeting chair or subgroup chair, draft any part of a *Monograph*, or participate in the evaluations. At the meeting, the meeting chair and subgroup chairs may grant Observers an opportunity to speak, generally after they have observed a discussion. Observers agree to respect the Guidelines for Observers at *IARC Monographs* meetings (available at <http://monographs.iarc.fr>).

(e) *The IARC Secretariat*

The IARC Secretariat consists of scientists who are designated by IARC and who have relevant expertise. They serve as rapporteurs and participate in all discussions. When requested by the meeting chair or subgroup chair, they may also draft text or prepare tables and analyses.

Before an invitation is extended, each potential participant, including the IARC Secretariat, completes the WHO Declaration of Interests to report financial interests, employment and consulting, and individual and institutional research support related to the subject of the meeting. IARC assesses these interests to determine

whether there is a conflict that warrants some limitation on participation. The declarations are updated and reviewed again at the opening of the meeting. Interests related to the subject of the meeting are disclosed to the meeting participants and in the published volume ([Cogliano et al., 2004](#)).

The names and principal affiliations of participants are available on the *Monographs* programme web site (<http://monographs.iarc.fr>) approximately two months before each meeting. It is not acceptable for Observers or third parties to contact other participants before a meeting or to lobby them at any time. Meeting participants are asked to report all such contacts to IARC ([Cogliano et al., 2005](#)).

All participants are listed, with their principal affiliations, at the beginning of each volume. Each participant who is a Member of a Working Group serves as an individual scientist and not as a representative of any organization, government or industry.

6. Working procedures

A separate Working Group is responsible for developing each volume of *Monographs*. A volume contains one or more *Monographs*, which can cover either a single agent or several related agents. Approximately one year in advance of the meeting of a Working Group, the agents to be reviewed are announced on the *Monographs* programme web site (<http://monographs.iarc.fr>) and participants are selected by IARC staff in consultation with other experts. Subsequently, relevant biological and epidemiological data are collected by IARC from recognized sources of information on carcinogenesis, including data storage and retrieval systems such as PubMed. Meeting participants who are asked to prepare preliminary working papers for specific sections are expected to supplement the IARC literature searches with their own searches.

For most chemicals and some complex mixtures, the major collection of data and the preparation of working papers for the sections on chemical and physical properties, on analysis, on production and use, and on occurrence are carried out under a separate contract funded by the US National Cancer Institute. Industrial associations, labour unions and other knowledgeable organizations may be asked to provide input to the sections on production and use, although this involvement is not required as a general rule. Information on production and trade is obtained from governmental, trade and market research publications and, in some cases, by direct contact with industries. Separate production data on some agents may not be available for a variety of reasons (e.g. not collected or made public in all producing countries, production is small). Information on uses may be obtained from published sources but is often complemented by direct contact with manufacturers. Efforts are made to supplement this information with data from other national and international sources.

Six months before the meeting, the material obtained is sent to meeting participants to prepare preliminary working papers. The working papers are compiled by IARC staff and sent, before the meeting, to Working Group Members and Invited Specialists for review.

The Working Group meets at IARC for seven to eight days to discuss and finalize the texts and to formulate the evaluations. The objectives of the meeting are peer review and consensus. During the first few days, four subgroups (covering exposure data, cancer in humans, cancer in experimental animals, and mechanistic and other relevant data) review the working papers, develop a joint subgroup draft and write summaries. Care is taken to ensure that each study summary is written or reviewed by someone not associated with the study being considered. During the last few days, the Working Group meets in plenary session to review the subgroup drafts and develop the evaluations. As a result,

the entire volume is the joint product of the Working Group, and there are no individually authored sections.

IARC Working Groups strive to achieve a consensus evaluation. Consensus reflects broad agreement among Working Group Members, but not necessarily unanimity. The chair may elect to poll Working Group Members to determine the diversity of scientific opinion on issues where consensus is not readily apparent.

After the meeting, the master copy is verified by consulting the original literature, edited and prepared for publication. The aim is to publish the volume within six months of the Working Group meeting. A summary of the outcome is available on the *Monographs* programme web site soon after the meeting.

B. SCIENTIFIC REVIEW AND EVALUATION

The available studies are summarized by the Working Group, with particular regard to the qualitative aspects discussed below. In general, numerical findings are indicated as they appear in the original report; units are converted when necessary for easier comparison. The Working Group may conduct additional analyses of the published data and use them in their assessment of the evidence; the results of such supplementary analyses are given in square brackets. When an important aspect of a study that directly impinges on its interpretation should be brought to the attention of the reader, a Working Group comment is given in square brackets.

The scope of the *IARC Monographs* programme has expanded beyond chemicals to include complex mixtures, occupational exposures, physical and biological agents, lifestyle factors and other potentially carcinogenic exposures. Over time, the structure of a *Monograph* has evolved to include the following sections:

Exposure data
Studies of cancer in humans
Studies of cancer in experimental animals
Mechanistic and other relevant data
Summary
Evaluation and rationale

In addition, a section of General Remarks at the front of the volume discusses the reasons the agents were scheduled for evaluation and some key issues the Working Group encountered during the meeting.

This part of the Preamble discusses the types of evidence considered and summarized in each section of a *Monograph*, followed by the scientific criteria that guide the evaluations.

1. Exposure data

Each *Monograph* includes general information on the agent: this information may vary substantially between agents and must be adapted accordingly. Also included is information on production and use (when appropriate), methods of analysis and detection, occurrence, and sources and routes of human occupational and environmental exposures. Depending on the agent, regulations and guidelines for use may be presented.

(a) General information on the agent

For chemical agents, sections on chemical and physical data are included: the Chemical Abstracts Service Registry Number, the latest primary name and the IUPAC systematic name are recorded; other synonyms are given, but the list is not necessarily comprehensive. Information on chemical and physical properties that are relevant to identification, occurrence and biological activity is included. A description of technical products of chemicals includes trade names, relevant specifications and available information on composition and impurities. Some of the trade names given may be those of mixtures in

which the agent being evaluated is only one of the ingredients.

For biological agents, taxonomy, structure and biology are described, and the degree of variability is indicated. Mode of replication, life cycle, target cells, persistence, latency, host response and clinical disease other than cancer are also presented.

For physical agents that are forms of radiation, energy and range of the radiation are included. For foreign bodies, fibres and respirable particles, size range and relative dimensions are indicated.

For agents such as mixtures, drugs or lifestyle factors, a description of the agent, including its composition, is given.

Whenever appropriate, other information, such as historical perspectives or the description of an industry or habit, may be included.

(b) Analysis and detection

An overview of methods of analysis and detection of the agent is presented, including their sensitivity, specificity and reproducibility. Methods widely used for regulatory purposes are emphasized. Methods for monitoring human exposure are also given. No critical evaluation or recommendation of any method is meant or implied.

(c) Production and use

The dates of first synthesis and of first commercial production of a chemical, mixture or other agent are provided when available; for agents that do not occur naturally, this information may allow a reasonable estimate to be made of the date before which no human exposure to the agent could have occurred. The dates of first reported occurrence of an exposure are also provided when available. In addition, methods of synthesis used in past and present commercial production and different methods of production,

which may give rise to different impurities, are described.

The countries where companies report production of the agent, and the number of companies in each country, are identified. Available data on production, international trade and uses are obtained for representative regions. It should not, however, be inferred that those areas or nations are necessarily the sole or major sources or users of the agent. Some identified uses may not be current or major applications, and the coverage is not necessarily comprehensive. In the case of drugs, mention of their therapeutic uses does not necessarily represent current practice nor does it imply judgement as to their therapeutic efficacy.

(d) Occurrence and exposure

Information on the occurrence of an agent in the environment is obtained from data derived from the monitoring and surveillance of levels in occupational environments, air, water, soil, plants, foods and animal and human tissues. When available, data on the generation, persistence and bioaccumulation of the agent are also included. Such data may be available from national databases.

Data that indicate the extent of past and present human exposure, the sources of exposure, the people most likely to be exposed and the factors that contribute to the exposure are reported. Information is presented on the range of human exposure, including occupational and environmental exposures. This includes relevant findings from both developed and developing countries. Some of these data are not distributed widely and may be available from government reports and other sources. In the case of mixtures, industries, occupations or processes, information is given about all agents known to be present. For processes, industries and occupations, a historical description is also given, noting variations in chemical composition, physical properties and levels of occupational exposure with date and

place. For biological agents, the epidemiology of infection is described.

(e) Regulations and guidelines

Statements concerning regulations and guidelines (e.g. occupational exposure limits, maximal levels permitted in foods and water, pesticide registrations) are included, but they may not reflect the most recent situation, since such limits are continuously reviewed and modified. The absence of information on regulatory status for a country should not be taken to imply that that country does not have regulations with regard to the exposure. For biological agents, legislation and control, including vaccination and therapy, are described.

2. Studies of cancer in humans

This section includes all pertinent epidemiological studies (see Part A, Section 4). Studies of biomarkers are included when they are relevant to an evaluation of carcinogenicity to humans.

(a) Types of study considered

Several types of epidemiological study contribute to the assessment of carcinogenicity in humans — cohort studies, case-control studies, correlation (or ecological) studies and intervention studies. Rarely, results from randomized trials may be available. Case reports and case series of cancer in humans may also be reviewed.

Cohort and case-control studies relate individual exposures under study to the occurrence of cancer in individuals and provide an estimate of effect (such as relative risk) as the main measure of association. Intervention studies may provide strong evidence for making causal inferences, as exemplified by cessation of smoking and the subsequent decrease in risk for lung cancer.

In correlation studies, the units of investigation are usually whole populations (e.g. in

particular geographical areas or at particular times), and cancer frequency is related to a summary measure of the exposure of the population to the agent under study. In correlation studies, individual exposure is not documented, which renders this kind of study more prone to confounding. In some circumstances, however, correlation studies may be more informative than analytical study designs (see, for example, the *Monograph on arsenic in drinking-water*; [IARC, 2004](#)).

In some instances, case reports and case series have provided important information about the carcinogenicity of an agent. These types of study generally arise from a suspicion, based on clinical experience, that the concurrence of two events — that is, a particular exposure and occurrence of a cancer — has happened rather more frequently than would be expected by chance. Case reports and case series usually lack complete ascertainment of cases in any population, definition or enumeration of the population at risk and estimation of the expected number of cases in the absence of exposure.

The uncertainties that surround the interpretation of case reports, case series and correlation studies make them inadequate, except in rare instances, to form the sole basis for inferring a causal relationship. When taken together with case-control and cohort studies, however, these types of study may add materially to the judgement that a causal relationship exists.

Epidemiological studies of benign neoplasms, presumed preneoplastic lesions and other end-points thought to be relevant to cancer are also reviewed. They may, in some instances, strengthen inferences drawn from studies of cancer itself.

(b) Quality of studies considered

It is necessary to take into account the possible roles of bias, confounding and chance in the interpretation of epidemiological studies.

Bias is the effect of factors in study design or execution that lead erroneously to a stronger or weaker association than in fact exists between an agent and disease. Confounding is a form of bias that occurs when the relationship with disease is made to appear stronger or weaker than it truly is as a result of an association between the apparent causal factor and another factor that is associated with either an increase or decrease in the incidence of the disease. The role of chance is related to biological variability and the influence of sample size on the precision of estimates of effect.

In evaluating the extent to which these factors have been minimized in an individual study, consideration is given to several aspects of design and analysis as described in the report of the study. For example, when suspicion of carcinogenicity arises largely from a single small study, careful consideration is given when interpreting subsequent studies that included these data in an enlarged population. Most of these considerations apply equally to case-control, cohort and correlation studies. Lack of clarity of any of these aspects in the reporting of a study can decrease its credibility and the weight given to it in the final evaluation of the exposure.

First, the study population, disease (or diseases) and exposure should have been well defined by the authors. Cases of disease in the study population should have been identified in a way that was independent of the exposure of interest, and exposure should have been assessed in a way that was not related to disease status.

Second, the authors should have taken into account — in the study design and analysis — other variables that can influence the risk of disease and may have been related to the exposure of interest. Potential confounding by such variables should have been dealt with either in the design of the study, such as by matching, or in the analysis, by statistical adjustment. In cohort studies, comparisons with local rates of disease may or may not be more appropriate than those with national rates. Internal comparisons of

frequency of disease among individuals at different levels of exposure are also desirable in cohort studies, since they minimize the potential for confounding related to the difference in risk factors between an external reference group and the study population.

Third, the authors should have reported the basic data on which the conclusions are founded, even if sophisticated statistical analyses were employed. At the very least, they should have given the numbers of exposed and unexposed cases and controls in a case-control study and the numbers of cases observed and expected in a cohort study. Further tabulations by time since exposure began and other temporal factors are also important. In a cohort study, data on all cancer sites and all causes of death should have been given, to reveal the possibility of reporting bias. In a case-control study, the effects of investigated factors other than the exposure of interest should have been reported.

Finally, the statistical methods used to obtain estimates of relative risk, absolute rates of cancer, confidence intervals and significance tests, and to adjust for confounding should have been clearly stated by the authors. These methods have been reviewed for case-control studies ([Breslow & Day, 1980](#)) and for cohort studies ([Breslow & Day, 1987](#)).

(c) *Meta-analyses and pooled analyses*

Independent epidemiological studies of the same agent may lead to results that are difficult to interpret. Combined analyses of data from multiple studies are a means of resolving this ambiguity, and well conducted analyses can be considered. There are two types of combined analysis. The first involves combining summary statistics such as relative risks from individual studies (meta-analysis) and the second involves a pooled analysis of the raw data from the individual studies (pooled analysis) ([Greenland, 1998](#)).

The advantages of combined analyses are increased precision due to increased sample size and the opportunity to explore potential confounders, interactions and modifying effects that may explain heterogeneity among studies in more detail. A disadvantage of combined analyses is the possible lack of compatibility of data from various studies due to differences in subject recruitment, procedures of data collection, methods of measurement and effects of unmeasured co-variates that may differ among studies. Despite these limitations, well conducted combined analyses may provide a firmer basis than individual studies for drawing conclusions about the potential carcinogenicity of agents.

IARC may commission a meta-analysis or pooled analysis that is pertinent to a particular *Monograph* (see Part A, Section 4). Additionally, as a means of gaining insight from the results of multiple individual studies, ad hoc calculations that combine data from different studies may be conducted by the Working Group during the course of a *Monograph* meeting. The results of such original calculations, which would be specified in the text by presentation in square brackets, might involve updates of previously conducted analyses that incorporate the results of more recent studies or de-novo analyses. Irrespective of the source of data for the meta-analyses and pooled analyses, it is important that the same criteria for data quality be applied as those that would be applied to individual studies and to ensure also that sources of heterogeneity between studies be taken into account.

(d) *Temporal effects*

Detailed analyses of both relative and absolute risks in relation to temporal variables, such as age at first exposure, time since first exposure, duration of exposure, cumulative exposure, peak exposure (when appropriate) and time since cessation of exposure, are reviewed and summarized when available. Analyses of temporal

relationships may be useful in making causal inferences. In addition, such analyses may suggest whether a carcinogen acts early or late in the process of carcinogenesis, although, at best, they allow only indirect inferences about mechanisms of carcinogenesis.

(e) *Use of biomarkers in epidemiological studies*

Biomarkers indicate molecular, cellular or other biological changes and are increasingly used in epidemiological studies for various purposes ([IARC, 1991](#); [Vainio et al., 1992](#); [Toniolo et al., 1997](#); [Vineis et al., 1999](#); [Buffler et al., 2004](#)). These may include evidence of exposure, of early effects, of cellular, tissue or organism responses, of individual susceptibility or host responses, and inference of a mechanism (see Part B, Section 4b). This is a rapidly evolving field that encompasses developments in genomics, epigenomics and other emerging technologies.

Molecular epidemiological data that identify associations between genetic polymorphisms and interindividual differences in susceptibility to the agent(s) being evaluated may contribute to the identification of carcinogenic hazards to humans. If the polymorphism has been demonstrated experimentally to modify the functional activity of the gene product in a manner that is consistent with increased susceptibility, these data may be useful in making causal inferences. Similarly, molecular epidemiological studies that measure cell functions, enzymes or metabolites that are thought to be the basis of susceptibility may provide evidence that reinforces biological plausibility. It should be noted, however, that when data on genetic susceptibility originate from multiple comparisons that arise from subgroup analyses, this can generate false-positive results and inconsistencies across studies, and such data therefore require careful evaluation. If the known phenotype of a genetic polymorphism can explain the carcinogenic mechanism

of the agent being evaluated, data on this phenotype may be useful in making causal inferences.

(f) *Criteria for causality*

After the quality of individual epidemiological studies of cancer has been summarized and assessed, a judgement is made concerning the strength of evidence that the agent in question is carcinogenic to humans. In making its judgement, the Working Group considers several criteria for causality ([Hill, 1965](#)). A strong association (e.g. a large relative risk) is more likely to indicate causality than a weak association, although it is recognized that estimates of effect of small magnitude do not imply lack of causality and may be important if the disease or exposure is common. Associations that are replicated in several studies of the same design or that use different epidemiological approaches or under different circumstances of exposure are more likely to represent a causal relationship than isolated observations from single studies. If there are inconsistent results among investigations, possible reasons are sought (such as differences in exposure), and results of studies that are judged to be of high quality are given more weight than those of studies that are judged to be methodologically less sound.

If the risk increases with the exposure, this is considered to be a strong indication of causality, although the absence of a graded response is not necessarily evidence against a causal relationship. The demonstration of a decline in risk after cessation of or reduction in exposure in individuals or in whole populations also supports a causal interpretation of the findings.

Several scenarios may increase confidence in a causal relationship. On the one hand, an agent may be specific in causing tumours at one site or of one morphological type. On the other, carcinogenicity may be evident through the causation of multiple tumour types. Temporality, precision of estimates of effect, biological plausibility and

coherence of the overall database are considered. Data on biomarkers may be employed in an assessment of the biological plausibility of epidemiological observations.

Although rarely available, results from randomized trials that show different rates of cancer among exposed and unexposed individuals provide particularly strong evidence for causality.

When several epidemiological studies show little or no indication of an association between an exposure and cancer, a judgement may be made that, in the aggregate, they show evidence of lack of carcinogenicity. Such a judgement requires first that the studies meet, to a sufficient degree, the standards of design and analysis described above. Specifically, the possibility that bias, confounding or misclassification of exposure or outcome could explain the observed results should be considered and excluded with reasonable certainty. In addition, all studies that are judged to be methodologically sound should (a) be consistent with an estimate of effect of unity for any observed level of exposure, (b) when considered together, provide a pooled estimate of relative risk that is at or near to unity, and (c) have a narrow confidence interval, due to sufficient population size. Moreover, no individual study nor the pooled results of all the studies should show any consistent tendency that the relative risk of cancer increases with increasing level of exposure. It is important to note that evidence of lack of carcinogenicity obtained from several epidemiological studies can apply only to the type(s) of cancer studied, to the dose levels reported, and to the intervals between first exposure and disease onset observed in these studies. Experience with human cancer indicates that the period from first exposure to the development of clinical cancer is sometimes longer than 20 years; latent periods substantially shorter than 30 years cannot provide evidence for lack of carcinogenicity.

3. Studies of cancer in experimental animals

All known human carcinogens that have been studied adequately for carcinogenicity in experimental animals have produced positive results in one or more animal species ([Wilbourn *et al.*, 1986](#); [Tomatis *et al.*, 1989](#)). For several agents (e.g. aflatoxins, diethylstilbestrol, solar radiation, vinyl chloride), carcinogenicity in experimental animals was established or highly suspected before epidemiological studies confirmed their carcinogenicity in humans ([Vainio *et al.*, 1995](#)). Although this association cannot establish that all agents that cause cancer in experimental animals also cause cancer in humans, it is biologically plausible that agents for which there is *sufficient evidence of carcinogenicity* in experimental animals (see Part B, Section 6b) also present a carcinogenic hazard to humans. Accordingly, in the absence of additional scientific information, these agents are considered to pose a carcinogenic hazard to humans. Examples of additional scientific information are data that demonstrate that a given agent causes cancer in animals through a species-specific mechanism that does not operate in humans or data that demonstrate that the mechanism in experimental animals also operates in humans (see Part B, Section 6).

Consideration is given to all available long-term studies of cancer in experimental animals with the agent under review (see Part A, Section 4). In all experimental settings, the nature and extent of impurities or contaminants present in the agent being evaluated are given when available. Animal species, strain (including genetic background where applicable), sex, numbers per group, age at start of treatment, route of exposure, dose levels, duration of exposure, survival and information on tumours (incidence, latency, severity or multiplicity of neoplasms or preneoplastic lesions) are reported. Those studies in experimental animals that are judged to be irrelevant to the evaluation or judged to be inadequate

(e.g. too short a duration, too few animals, poor survival; see below) may be omitted. Guidelines for conducting long-term carcinogenicity experiments have been published (e.g. [OECD, 2002](#)).

Other studies considered may include: experiments in which the agent was administered in the presence of factors that modify carcinogenic effects (e.g. initiation–promotion studies, co-carcinogenicity studies and studies in genetically modified animals); studies in which the end-point was not cancer but a defined precancerous lesion; experiments on the carcinogenicity of known metabolites and derivatives; and studies of cancer in non-laboratory animals (e.g. livestock and companion animals) exposed to the agent.

For studies of mixtures, consideration is given to the possibility that changes in the physicochemical properties of the individual substances may occur during collection, storage, extraction, concentration and delivery. Another consideration is that chemical and toxicological interactions of components in a mixture may alter dose–response relationships. The relevance to human exposure of the test mixture administered in the animal experiment is also assessed. This may involve consideration of the following aspects of the mixture tested: (i) physical and chemical characteristics, (ii) identified constituents that may indicate the presence of a class of substances and (iii) the results of genetic toxicity and related tests.

The relevance of results obtained with an agent that is analogous (e.g. similar in structure or of a similar virus genus) to that being evaluated is also considered. Such results may provide biological and mechanistic information that is relevant to the understanding of the process of carcinogenesis in humans and may strengthen the biological plausibility that the agent being evaluated is carcinogenic to humans (see Part B, Section 2f).

(a) *Qualitative aspects*

An assessment of carcinogenicity involves several considerations of qualitative importance, including (i) the experimental conditions under which the test was performed, including route, schedule and duration of exposure, species, strain (including genetic background where applicable), sex, age and duration of follow-up; (ii) the consistency of the results, for example, across species and target organ(s); (iii) the spectrum of neoplastic response, from preneoplastic lesions and benign tumours to malignant neoplasms; and (iv) the possible role of modifying factors.

Considerations of importance in the interpretation and evaluation of a particular study include: (i) how clearly the agent was defined and, in the case of mixtures, how adequately the sample characterization was reported; (ii) whether the dose was monitored adequately, particularly in inhalation experiments; (iii) whether the doses, duration of treatment and route of exposure were appropriate; (iv) whether the survival of treated animals was similar to that of controls; (v) whether there were adequate numbers of animals per group; (vi) whether both male and female animals were used; (vii) whether animals were allocated randomly to groups; (viii) whether the duration of observation was adequate; and (ix) whether the data were reported and analysed adequately.

When benign tumours (a) occur together with and originate from the same cell type as malignant tumours in an organ or tissue in a particular study and (b) appear to represent a stage in the progression to malignancy, they are usually combined in the assessment of tumour incidence ([Huff et al., 1989](#)). The occurrence of lesions presumed to be preneoplastic may in certain instances aid in assessing the biological plausibility of any neoplastic response observed. If an agent induces only benign neoplasms that appear to be end-points that do not readily undergo

transition to malignancy, the agent should nevertheless be suspected of being carcinogenic and requires further investigation.

(b) Quantitative aspects

The probability that tumours will occur may depend on the species, sex, strain, genetic background and age of the animal, and on the dose, route, timing and duration of the exposure. Evidence of an increased incidence of neoplasms with increasing levels of exposure strengthens the inference of a causal association between the exposure and the development of neoplasms.

The form of the dose–response relationship can vary widely, depending on the particular agent under study and the target organ. Mechanisms such as induction of DNA damage or inhibition of repair, altered cell division and cell death rates and changes in intercellular communication are important determinants of dose–response relationships for some carcinogens. Since many chemicals require metabolic activation before being converted to their reactive intermediates, both metabolic and toxicokinetic aspects are important in determining the dose–response pattern. Saturation of steps such as absorption, activation, inactivation and elimination may produce nonlinearity in the dose–response relationship ([Hoel et al., 1983](#); [Gart et al., 1986](#)), as could saturation of processes such as DNA repair. The dose–response relationship can also be affected by differences in survival among the treatment groups.

(c) Statistical analyses

Factors considered include the adequacy of the information given for each treatment group: (i) number of animals studied and number examined histologically, (ii) number of animals with a given tumour type and (iii) length of survival. The statistical methods used should be clearly stated and should be the generally accepted techniques refined for this purpose ([Peto et al., 1980](#);

[Gart et al., 1986](#); [Portier & Bailer, 1989](#); [Bieler & Williams, 1993](#)). The choice of the most appropriate statistical method requires consideration of whether or not there are differences in survival among the treatment groups; for example, reduced survival because of non-tumour-related mortality can preclude the occurrence of tumours later in life. When detailed information on survival is not available, comparisons of the proportions of tumour-bearing animals among the effective number of animals (alive at the time the first tumour was discovered) can be useful when significant differences in survival occur before tumours appear. The lethality of the tumour also requires consideration: for rapidly fatal tumours, the time of death provides an indication of the time of tumour onset and can be assessed using life-table methods; non-fatal or incidental tumours that do not affect survival can be assessed using methods such as the Mantel-Haenzel test for changes in tumour prevalence. Because tumour lethality is often difficult to determine, methods such as the Poly-K test that do not require such information can also be used. When results are available on the number and size of tumours seen in experimental animals (e.g. papillomas on mouse skin, liver tumours observed through nuclear magnetic resonance tomography), other more complicated statistical procedures may be needed ([Sherman et al., 1994](#); [Dunson et al., 2003](#)).

Formal statistical methods have been developed to incorporate historical control data into the analysis of data from a given experiment. These methods assign an appropriate weight to historical and concurrent controls on the basis of the extent of between-study and within-study variability: less weight is given to historical controls when they show a high degree of variability, and greater weight when they show little variability. It is generally not appropriate to discount a tumour response that is significantly increased compared with concurrent controls by arguing that it falls within the range of historical controls,

particularly when historical controls show high between-study variability and are, thus, of little relevance to the current experiment. In analysing results for uncommon tumours, however, the analysis may be improved by considering historical control data, particularly when between-study variability is low. Historical controls should be selected to resemble the concurrent controls as closely as possible with respect to species, gender and strain, as well as other factors such as basal diet and general laboratory environment, which may affect tumour-response rates in control animals ([Haseman *et al.*, 1984](#); [Fung *et al.*, 1996](#); [Greim *et al.*, 2003](#)).

Although meta-analyses and combined analyses are conducted less frequently for animal experiments than for epidemiological studies due to differences in animal strains, they can be useful aids in interpreting animal data when the experimental protocols are sufficiently similar.

4. Mechanistic and other relevant data

Mechanistic and other relevant data may provide evidence of carcinogenicity and also help in assessing the relevance and importance of findings of cancer in animals and in humans. The nature of the mechanistic and other relevant data depends on the biological activity of the agent being considered. The Working Group considers representative studies to give a concise description of the relevant data and issues that they consider to be important; thus, not every available study is cited. Relevant topics may include toxicokinetics, mechanisms of carcinogenesis, susceptible individuals, populations and life-stages, other relevant data and other adverse effects. When data on biomarkers are informative about the mechanisms of carcinogenesis, they are included in this section.

These topics are not mutually exclusive; thus, the same studies may be discussed in more than

one subsection. For example, a mutation in a gene that codes for an enzyme that metabolizes the agent under study could be discussed in the subsections on toxicokinetics, mechanisms and individual susceptibility if it also exists as an inherited polymorphism.

(a) *Toxicokinetic data*

Toxicokinetics refers to the absorption, distribution, metabolism and elimination of agents in humans, experimental animals and, where relevant, cellular systems. Examples of kinetic factors that may affect dose-response relationships include uptake, deposition, biopersistence and half-life in tissues, protein binding, metabolic activation and detoxification. Studies that indicate the metabolic fate of the agent in humans and in experimental animals are summarized briefly, and comparisons of data from humans and animals are made when possible. Comparative information on the relationship between exposure and the dose that reaches the target site may be important for the extrapolation of hazards between species and in clarifying the role of in-vitro findings.

(b) *Data on mechanisms of carcinogenesis*

To provide focus, the Working Group attempts to identify the possible mechanisms by which the agent may increase the risk of cancer. For each possible mechanism, a representative selection of key data from humans and experimental systems is summarized. Attention is given to gaps in the data and to data that suggests that more than one mechanism may be operating. The relevance of the mechanism to humans is discussed, in particular, when mechanistic data are derived from experimental model systems. Changes in the affected organs, tissues or cells can be divided into three non-exclusive levels as described below.

(i) Changes in physiology

Physiological changes refer to exposure-related modifications to the physiology and/or response of cells, tissues and organs. Examples of potentially adverse physiological changes include mitogenesis, compensatory cell division, escape from apoptosis and/or senescence, presence of inflammation, hyperplasia, metaplasia and/or preneoplasia, angiogenesis, alterations in cellular adhesion, changes in steroidal hormones and changes in immune surveillance.

(ii) Functional changes at the cellular level

Functional changes refer to exposure-related alterations in the signalling pathways used by cells to manage critical processes that are related to increased risk for cancer. Examples of functional changes include modified activities of enzymes involved in the metabolism of xenobiotics, alterations in the expression of key genes that regulate DNA repair, alterations in cyclin-dependent kinases that govern cell cycle progression, changes in the patterns of post-translational modifications of proteins, changes in regulatory factors that alter apoptotic rates, changes in the secretion of factors related to the stimulation of DNA replication and transcription and changes in gap-junction-mediated intercellular communication.

(iii) Changes at the molecular level

Molecular changes refer to exposure-related changes in key cellular structures at the molecular level, including, in particular, genotoxicity. Examples of molecular changes include formation of DNA adducts and DNA strand breaks, mutations in genes, chromosomal aberrations, aneuploidy and changes in DNA methylation patterns. Greater emphasis is given to irreversible effects.

The use of mechanistic data in the identification of a carcinogenic hazard is specific to the mechanism being addressed and is not readily

described for every possible level and mechanism discussed above.

Genotoxicity data are discussed here to illustrate the key issues involved in the evaluation of mechanistic data.

Tests for genetic and related effects are described in view of the relevance of gene mutation and chromosomal aberration/aneuploidy to carcinogenesis ([Vainio et al., 1992](#); [McGregor et al., 1999](#)). The adequacy of the reporting of sample characterization is considered and, when necessary, commented upon; with regard to complex mixtures, such comments are similar to those described for animal carcinogenicity tests. The available data are interpreted critically according to the end-points detected, which may include DNA damage, gene mutation, sister chromatid exchange, micronucleus formation, chromosomal aberrations and aneuploidy. The concentrations employed are given, and mention is made of whether the use of an exogenous metabolic system in vitro affected the test result. These data are listed in tabular form by phylogenetic classification.

Positive results in tests using prokaryotes, lower eukaryotes, insects, plants and cultured mammalian cells suggest that genetic and related effects could occur in mammals. Results from such tests may also give information on the types of genetic effect produced and on the involvement of metabolic activation. Some end-points described are clearly genetic in nature (e.g. gene mutations), while others are associated with genetic effects (e.g. unscheduled DNA synthesis). In-vitro tests for tumour promotion, cell transformation and gap-junction intercellular communication may be sensitive to changes that are not necessarily the result of genetic alterations but that may have specific relevance to the process of carcinogenesis. Critical appraisals of these tests have been published ([Montesano et al., 1986](#); [McGregor et al., 1999](#)).

Genetic or other activity manifest in humans and experimental mammals is regarded to be of

greater relevance than that in other organisms. The demonstration that an agent can induce gene and chromosomal mutations in mammals in vivo indicates that it may have carcinogenic activity. Negative results in tests for mutagenicity in selected tissues from animals treated in vivo provide less weight, partly because they do not exclude the possibility of an effect in tissues other than those examined. Moreover, negative results in short-term tests with genetic end-points cannot be considered to provide evidence that rules out the carcinogenicity of agents that act through other mechanisms (e.g. receptor-mediated effects, cellular toxicity with regenerative cell division, peroxisome proliferation) ([Vainio et al., 1992](#)). Factors that may give misleading results in short-term tests have been discussed in detail elsewhere ([Montesano et al., 1986](#); [McGregor et al., 1999](#)).

When there is evidence that an agent acts by a specific mechanism that does not involve genotoxicity (e.g. hormonal dysregulation, immune suppression, and formation of calculi and other deposits that cause chronic irritation), that evidence is presented and reviewed critically in the context of rigorous criteria for the operation of that mechanism in carcinogenesis (e.g. [Capen et al., 1999](#)).

For biological agents such as viruses, bacteria and parasites, other data relevant to carcinogenicity may include descriptions of the pathology of infection, integration and expression of viruses, and genetic alterations seen in human tumours. Other observations that might comprise cellular and tissue responses to infection, immune response and the presence of tumour markers are also considered.

For physical agents that are forms of radiation, other data relevant to carcinogenicity may include descriptions of damaging effects at the physiological, cellular and molecular level, as for chemical agents, and descriptions of how these effects occur. 'Physical agents' may also be considered to comprise foreign bodies, such as

surgical implants of various kinds, and poorly soluble fibres, dusts and particles of various sizes, the pathogenic effects of which are a result of their physical presence in tissues or body cavities. Other relevant data for such materials may include characterization of cellular, tissue and physiological reactions to these materials and descriptions of pathological conditions other than neoplasia with which they may be associated.

(c) *Other data relevant to mechanisms*

A description is provided of any structure-activity relationships that may be relevant to an evaluation of the carcinogenicity of an agent, the toxicological implications of the physical and chemical properties, and any other data relevant to the evaluation that are not included elsewhere.

High-output data, such as those derived from gene expression microarrays, and high-throughput data, such as those that result from testing hundreds of agents for a single end-point, pose a unique problem for the use of mechanistic data in the evaluation of a carcinogenic hazard. In the case of high-output data, there is the possibility to overinterpret changes in individual end-points (e.g. changes in expression in one gene) without considering the consistency of that finding in the broader context of the other end-points (e.g. other genes with linked transcriptional control). High-output data can be used in assessing mechanisms, but all end-points measured in a single experiment need to be considered in the proper context. For high-throughput data, where the number of observations far exceeds the number of end-points measured, their utility for identifying common mechanisms across multiple agents is enhanced. These data can be used to identify mechanisms that not only seem plausible, but also have a consistent pattern of carcinogenic response across entire classes of related compounds.

(d) *Susceptibility data*

Individuals, populations and life-stages may have greater or lesser susceptibility to an agent, based on toxicokinetics, mechanisms of carcinogenesis and other factors. Examples of host and genetic factors that affect individual susceptibility include sex, genetic polymorphisms of genes involved in the metabolism of the agent under evaluation, differences in metabolic capacity due to life-stage or the presence of disease, differences in DNA repair capacity, competition for or alteration of metabolic capacity by medications or other chemical exposures, pre-existing hormonal imbalance that is exacerbated by a chemical exposure, a suppressed immune system, periods of higher-than-usual tissue growth or regeneration and genetic polymorphisms that lead to differences in behaviour (e.g. addiction). Such data can substantially increase the strength of the evidence from epidemiological data and enhance the linkage of in-vivo and in-vitro laboratory studies to humans.

(e) *Data on other adverse effects*

Data on acute, subchronic and chronic adverse effects relevant to the cancer evaluation are summarized. Adverse effects that confirm distribution and biological effects at the sites of tumour development, or alterations in physiology that could lead to tumour development, are emphasized. Effects on reproduction, embryonic and fetal survival and development are summarized briefly. The adequacy of epidemiological studies of reproductive outcome and genetic and related effects in humans is judged by the same criteria as those applied to epidemiological studies of cancer, but fewer details are given.

5. Summary

This section is a summary of data presented in the preceding sections. Summaries can be

found on the *Monographs* programme web site (<http://monographs.iarc.fr>).

(a) *Exposure data*

Data are summarized, as appropriate, on the basis of elements such as production, use, occurrence and exposure levels in the workplace and environment and measurements in human tissues and body fluids. Quantitative data and time trends are given to compare exposures in different occupations and environmental settings. Exposure to biological agents is described in terms of transmission, prevalence and persistence of infection.

(b) *Cancer in humans*

Results of epidemiological studies pertinent to an assessment of human carcinogenicity are summarized. When relevant, case reports and correlation studies are also summarized. The target organ(s) or tissue(s) in which an increase in cancer was observed is identified. Dose–response and other quantitative data may be summarized when available.

(c) *Cancer in experimental animals*

Data relevant to an evaluation of carcinogenicity in animals are summarized. For each animal species, study design and route of administration, it is stated whether an increased incidence, reduced latency, or increased severity or multiplicity of neoplasms or preneoplastic lesions were observed, and the tumour sites are indicated. If the agent produced tumours after prenatal exposure or in single-dose experiments, this is also mentioned. Negative findings, inverse relationships, dose–response and other quantitative data are also summarized.

(d) *Mechanistic and other relevant data*

Data relevant to the toxicokinetics (absorption, distribution, metabolism, elimination) and

the possible mechanism(s) of carcinogenesis (e.g. genetic toxicity, epigenetic effects) are summarized. In addition, information on susceptible individuals, populations and life-stages is summarized. This section also reports on other toxic effects, including reproductive and developmental effects, as well as additional relevant data that are considered to be important.

6. Evaluation and rationale

Evaluations of the strength of the evidence for carcinogenicity arising from human and experimental animal data are made, using standard terms. The strength of the mechanistic evidence is also characterized.

It is recognized that the criteria for these evaluations, described below, cannot encompass all of the factors that may be relevant to an evaluation of carcinogenicity. In considering all of the relevant scientific data, the Working Group may assign the agent to a higher or lower category than a strict interpretation of these criteria would indicate.

These categories refer only to the strength of the evidence that an exposure is carcinogenic and not to the extent of its carcinogenic activity (potency). A classification may change as new information becomes available.

An evaluation of the degree of evidence is limited to the materials tested, as defined physically, chemically or biologically. When the agents evaluated are considered by the Working Group to be sufficiently closely related, they may be grouped together for the purpose of a single evaluation of the degree of evidence.

(a) Carcinogenicity in humans

The evidence relevant to carcinogenicity from studies in humans is classified into one of the following categories:

Sufficient evidence of carcinogenicity: The Working Group considers that a causal

relationship has been established between exposure to the agent and human cancer. That is, a positive relationship has been observed between the exposure and cancer in studies in which chance, bias and confounding could be ruled out with reasonable confidence. A statement that there is *sufficient evidence* is followed by a separate sentence that identifies the target organ(s) or tissue(s) where an increased risk of cancer was observed in humans. Identification of a specific target organ or tissue does not preclude the possibility that the agent may cause cancer at other sites.

Limited evidence of carcinogenicity: A positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence.

Inadequate evidence of carcinogenicity: The available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association between exposure and cancer, or no data on cancer in humans are available.

Evidence suggesting lack of carcinogenicity: There are several adequate studies covering the full range of levels of exposure that humans are known to encounter, which are mutually consistent in not showing a positive association between exposure to the agent and any studied cancer at any observed level of exposure. The results from these studies alone or combined should have narrow confidence intervals with an upper limit close to the null value (e.g. a relative risk of 1.0). Bias and confounding should be ruled out with reasonable confidence, and the studies should have an adequate length of follow-up. A conclusion of *evidence suggesting lack of carcinogenicity* is inevitably limited to the cancer sites, conditions and levels of exposure, and length of observation covered by the available studies. In

addition, the possibility of a very small risk at the levels of exposure studied can never be excluded.

In some instances, the above categories may be used to classify the degree of evidence related to carcinogenicity in specific organs or tissues.

When the available epidemiological studies pertain to a mixture, process, occupation or industry, the Working Group seeks to identify the specific agent considered most likely to be responsible for any excess risk. The evaluation is focused as narrowly as the available data on exposure and other aspects permit.

(b) *Carcinogenicity in experimental animals*

Carcinogenicity in experimental animals can be evaluated using conventional bioassays, bioassays that employ genetically modified animals, and other in-vivo bioassays that focus on one or more of the critical stages of carcinogenesis. In the absence of data from conventional long-term bioassays or from assays with neoplasia as the end-point, consistently positive results in several models that address several stages in the multi-stage process of carcinogenesis should be considered in evaluating the degree of evidence of carcinogenicity in experimental animals.

The evidence relevant to carcinogenicity in experimental animals is classified into one of the following categories:

Sufficient evidence of carcinogenicity: The Working Group considers that a causal relationship has been established between the agent and an increased incidence of malignant neoplasms or of an appropriate combination of benign and malignant neoplasms in (a) two or more species of animals or (b) two or more independent studies in one species carried out at different times or in different laboratories or under different protocols. An increased incidence of tumours in both sexes of a single species in a well conducted study, ideally conducted under Good Laboratory Practices, can also provide *sufficient evidence*.

A single study in one species and sex might be considered to provide *sufficient evidence of carcinogenicity* when malignant neoplasms occur to an unusual degree with regard to incidence, site, type of tumour or age at onset, or when there are strong findings of tumours at multiple sites.

Limited evidence of carcinogenicity: The data suggest a carcinogenic effect but are limited for making a definitive evaluation because, e.g. (a) the evidence of carcinogenicity is restricted to a single experiment; (b) there are unresolved questions regarding the adequacy of the design, conduct or interpretation of the studies; (c) the agent increases the incidence only of benign neoplasms or lesions of uncertain neoplastic potential; or (d) the evidence of carcinogenicity is restricted to studies that demonstrate only promoting activity in a narrow range of tissues or organs.

Inadequate evidence of carcinogenicity: The studies cannot be interpreted as showing either the presence or absence of a carcinogenic effect because of major qualitative or quantitative limitations, or no data on cancer in experimental animals are available.

Evidence suggesting lack of carcinogenicity: Adequate studies involving at least two species are available which show that, within the limits of the tests used, the agent is not carcinogenic. A conclusion of *evidence suggesting lack of carcinogenicity* is inevitably limited to the species, tumour sites, age at exposure, and conditions and levels of exposure studied.

(c) *Mechanistic and other relevant data*

Mechanistic and other evidence judged to be relevant to an evaluation of carcinogenicity and of sufficient importance to affect the overall evaluation is highlighted. This may include data on preneoplastic lesions, tumour pathology, genetic and related effects, structure-activity relationships, metabolism and toxicokinetics,

physicochemical parameters and analogous biological agents.

The strength of the evidence that any carcinogenic effect observed is due to a particular mechanism is evaluated, using terms such as ‘weak’, ‘moderate’ or ‘strong’. The Working Group then assesses whether that particular mechanism is likely to be operative in humans. The strongest indications that a particular mechanism operates in humans derive from data on humans or biological specimens obtained from exposed humans. The data may be considered to be especially relevant if they show that the agent in question has caused changes in exposed humans that are on the causal pathway to carcinogenesis. Such data may, however, never become available, because it is at least conceivable that certain compounds may be kept from human use solely on the basis of evidence of their toxicity and/or carcinogenicity in experimental systems.

The conclusion that a mechanism operates in experimental animals is strengthened by findings of consistent results in different experimental systems, by the demonstration of biological plausibility and by coherence of the overall database. Strong support can be obtained from studies that challenge the hypothesized mechanism experimentally, by demonstrating that the suppression of key mechanistic processes leads to the suppression of tumour development. The Working Group considers whether multiple mechanisms might contribute to tumour development, whether different mechanisms might operate in different dose ranges, whether separate mechanisms might operate in humans and experimental animals and whether a unique mechanism might operate in a susceptible group. The possible contribution of alternative mechanisms must be considered before concluding that tumours observed in experimental animals are not relevant to humans. An uneven level of experimental support for different mechanisms may reflect that disproportionate resources

have been focused on investigating a favoured mechanism.

For complex exposures, including occupational and industrial exposures, the chemical composition and the potential contribution of carcinogens known to be present are considered by the Working Group in its overall evaluation of human carcinogenicity. The Working Group also determines the extent to which the materials tested in experimental systems are related to those to which humans are exposed.

(d) Overall evaluation

Finally, the body of evidence is considered as a whole, to reach an overall evaluation of the carcinogenicity of the agent to humans.

An evaluation may be made for a group of agents that have been evaluated by the Working Group. In addition, when supporting data indicate that other related agents, for which there is no direct evidence of their capacity to induce cancer in humans or in animals, may also be carcinogenic, a statement describing the rationale for this conclusion is added to the evaluation narrative; an additional evaluation may be made for this broader group of agents if the strength of the evidence warrants it.

The agent is described according to the wording of one of the following categories, and the designated group is given. The categorization of an agent is a matter of scientific judgement that reflects the strength of the evidence derived from studies in humans and in experimental animals and from mechanistic and other relevant data.

Group 1: The agent is carcinogenic to humans.

This category is used when there is *sufficient evidence of carcinogenicity* in humans. Exceptionally, an agent may be placed in this category when evidence of carcinogenicity in humans is less than *sufficient* but there is *sufficient evidence of carcinogenicity* in experimental

animals and strong evidence in exposed humans that the agent acts through a relevant mechanism of carcinogenicity.

Group 2.

This category includes agents for which, at one extreme, the degree of evidence of carcinogenicity in humans is almost *sufficient*, as well as those for which, at the other extreme, there are no human data but for which there is evidence of carcinogenicity in experimental animals. Agents are assigned to either Group 2A (*probably carcinogenic to humans*) or Group 2B (*possibly carcinogenic to humans*) on the basis of epidemiological and experimental evidence of carcinogenicity and mechanistic and other relevant data. The terms *probably carcinogenic* and *possibly carcinogenic* have no quantitative significance and are used simply as descriptors of different levels of evidence of human carcinogenicity, with *probably carcinogenic* signifying a higher level of evidence than *possibly carcinogenic*.

Group 2A: The agent is probably carcinogenic to humans.

This category is used when there is *limited evidence of carcinogenicity* in humans and *sufficient evidence of carcinogenicity* in experimental animals. In some cases, an agent may be classified in this category when there is *inadequate evidence of carcinogenicity* in humans and *sufficient evidence of carcinogenicity* in experimental animals and strong evidence that the carcinogenesis is mediated by a mechanism that also operates in humans. Exceptionally, an agent may be classified in this category solely on the basis of *limited evidence of carcinogenicity* in humans. An agent may be assigned to this category if it clearly belongs, based on mechanistic considerations, to a class of agents for which one or more members have been classified in Group 1 or Group 2A.

Group 2B: The agent is possibly carcinogenic to humans.

This category is used for agents for which there is *limited evidence of carcinogenicity* in humans and less than *sufficient evidence of carcinogenicity* in experimental animals. It may also be used when there is *inadequate evidence of carcinogenicity* in humans but there is *sufficient evidence of carcinogenicity* in experimental animals. In some instances, an agent for which there is *inadequate evidence of carcinogenicity* in humans and less than *sufficient evidence of carcinogenicity* in experimental animals together with supporting evidence from mechanistic and other relevant data may be placed in this group. An agent may be classified in this category solely on the basis of strong evidence from mechanistic and other relevant data.

Group 3: The agent is not classifiable as to its carcinogenicity to humans.

This category is used most commonly for agents for which the evidence of carcinogenicity is *inadequate* in humans and *inadequate* or *limited* in experimental animals.

Exceptionally, agents for which the evidence of carcinogenicity is *inadequate* in humans but *sufficient* in experimental animals may be placed in this category when there is strong evidence that the mechanism of carcinogenicity in experimental animals does not operate in humans.

Agents that do not fall into any other group are also placed in this category.

An evaluation in Group 3 is not a determination of non-carcinogenicity or overall safety. It often means that further research is needed, especially when exposures are widespread or the cancer data are consistent with differing interpretations.

Group 4: The agent is probably not carcinogenic to humans.

This category is used for agents for which there is *evidence suggesting lack of carcinogenicity*

in humans and in experimental animals. In some instances, agents for which there is *inadequate evidence of carcinogenicity* in humans but *evidence suggesting lack of carcinogenicity* in experimental animals, consistently and strongly supported by a broad range of mechanistic and other relevant data, may be classified in this group.

(e) Rationale

The reasoning that the Working Group used to reach its evaluation is presented and discussed. This section integrates the major findings from studies of cancer in humans, studies of cancer in experimental animals, and mechanistic and other relevant data. It includes concise statements of the principal line(s) of argument that emerged, the conclusions of the Working Group on the strength of the evidence for each group of studies, citations to indicate which studies were pivotal to these conclusions, and an explanation of the reasoning of the Working Group in weighing data and making evaluations. When there are significant differences of scientific interpretation among Working Group Members, a brief summary of the alternative interpretations is provided, together with their scientific rationale and an indication of the relative degree of support for each alternative.

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GENERAL REMARKS

This one-hundred-and-second volume of the *IARC Monographs* contains evaluations of the carcinogenic hazard to humans of radiofrequency electromagnetic fields. This is the second volume on non-ionizing radiation, after Volume 80 (Static and Extremely Low-Frequency (ELF) Electric and Magnetic Fields; IARC, 2002), and the fourth and last in a series on physical agents, after Volume 75 (Ionizing Radiation, Part 1: X- and Gamma-radiation, and Neutrons; IARC, 2000) and Volume 78 (Ionizing Radiation, Part 2: Some Internally Deposited Radionuclides; IARC, 2001). Solar radiation and ultraviolet radiation were evaluated in Volume 55 (IARC, 1992). The types of radiation evaluated as human carcinogens (Group 1) were revisited in Volume 100D (IARC, 2012). A summary of the findings in the present volume has appeared in *The Lancet Oncology* (Baan *et al.*, 2011).

The topic of this *Monograph* is the evaluation of the carcinogenicity of radiation in the radiofrequency (RF) range (30 kHz to 300 GHz) of the electromagnetic spectrum. This type of radiation is emitted by devices used in wireless telecommunication, including mobile phones, and by many other sources in occupational and general environmental settings. Exposures are ubiquitous in more developed countries and rapidly increasing in developing countries, in particular with respect to the use of mobile phones. There is rising concern as to whether exposure to RF radiation emitted by a mobile phone affects human health and, specifically, whether mobile-phone use increases the risk of cancer of the brain. The general public, manufacturers, regulatory authorities and public health agencies are seeking evidence on the safety of mobile-phone use. Consequently, there has been intense interest in the development and outcome of this *IARC Monograph*. This interest reflects the high prevalence of exposure (which increasingly extends to children), the vast scope of the telecommunications industry, the findings of some epidemiological studies that suggest an increased risk of cancer, and a high level of media coverage of the topic of mobile phones and cancer.

Although the preparation of this *Monograph* had been scheduled so as to include the results of the large international case-control study INTERPHONE on mobile-phone use (conducted in 2000–2004; published in 2010), it should be emphasized that the evaluations in this volume address the general question of whether RF radiation causes cancer in humans or in experimental animals: it does not specifically or exclusively consider mobile phones, but rather the type of radiation emitted by mobile phones and various other sources. Furthermore, this *Monograph* is focused on the potential for an increased risk of cancer among those exposed to RF radiation, but does not provide a quantitative assessment of any cancer risk, nor does it discuss or evaluate any other potential health effects of RF radiation.

The Working Group recognized that mobile-phone technology has transformed the world, making wireless communication rapidly available, especially in less developed countries, with important

benefits to society. With this, an increasingly large population will be exposed, and for longer and longer periods of time. Undoubtedly, questions will continue to arise about the health risks of mobile-phone use and possibly other emerging sources of exposure to RF radiation. This *Monograph* is a comprehensive review of the currently published evidence that also identifies gaps in the available information. These gaps should be resolved with further research if ongoing concerns about the health risks of mobile-phone use are to be addressed with greater certainty.

The Working Group agreed to consider three categories of human exposure to RF radiation: (a) environmental sources such as mobile-phone base stations, broadcast antennae, smart meters, and medical applications; (b) occupational sources such as high-frequency dielectric and induction heaters, and high-power pulsed radars; and (c) the use of personal devices such as mobile phones, cordless phones, Bluetooth devices, and amateur radios.

The general population receives the highest exposure from transmitters close to the body, including hand-held devices such as mobile phones, which deposit most of the RF energy in the brain. Holding a mobile phone to the ear to make a voice call can result in high specific rates of absorption (SAR) of RF energy in the brain, depending on the design and position of the phone and its antenna in relation to the head, the anatomy of the head, and the quality of the connection with the base-station antenna: the better the connection, which is ensured by a dense network of base stations, the lower the energy output from the phone. In children using mobile phones, the average deposition of RF energy may be two times higher in the brain and up to ten times higher in the bone marrow of the skull than in adult users. The use of hands-free kits lowers exposure of the brain to less than 10% of the exposure from use at the ear, but it may increase exposure to other parts of the body.

Typical environmental exposures to the brain from mobile-phone base stations on rooftops and from television and radio stations are several orders of magnitude lower than those from GSM (Global System for Mobile communications) handsets. The average exposure from DECT (Digital Enhanced Cordless Telecommunications) phones is around five times lower than that measured for GSM phones, and third-generation (3G) phones emit, on average, about 100 times less RF energy than second-generation GSM phones, when signals are strong. Similarly, the average output power of Bluetooth wireless hands-free kits is estimated to be around 100 times lower than that of mobile phones. In occupational settings, exposure to high-power sources may involve higher cumulative deposition of RF energy in the body than with exposure to mobile phones, but the energy deposited locally in the brain is generally less.

Epidemiological evidence of an association between RF radiation and cancer comes from time-trend, cohort, and case-control studies. The populations in these studies were exposed to RF radiation in occupational settings, from sources in the general environment, and from use of wireless (mobile and cordless) phones. Two sets of data from case-control studies were considered by the Working Group as the principal and most informative basis for their evaluation of the human evidence, i.e. the INTERPHONE study and the Swedish case-control studies; both sets of data focused on brain tumours among mobile-phone users.

The Working Group recognized not only the rapid increase worldwide in the use of wireless communication systems – both in number of users and in duration of use – but also the considerable technological developments in this area, with the introduction of third- and fourth-generation (3G and 4G) devices during the past decade. It is of interest to note that the key epidemiological studies mentioned above were conducted in the late 1990s and the early 2000s. In the INTERPHONE study, all participating countries in Europe had GSM networks. It is worth mentioning that the 3G and 4G

mobile phones commercially available today – equipped with adaptive power control – emit considerably less RF energy than the GSM phones used more than a decade ago.

Experimental evidence from cancer bioassays was evaluated by the Working Group after reviewing more than 40 studies that assessed the incidence of tumours in rodents exposed to RF radiation at various frequencies, some of which simulated emissions from mobile phones. In the evaluation of studies of cancer in experimental animals, exposure assessment deserves critical consideration. In this regard, the conduct of cancer bioassays with RF radiation presents challenges that are not ordinarily encountered in studies with chemical or other physical agents. For example, the radiation frequency is an important determinant of the specific absorption rate (SAR). The whole-body SAR provides little information about spatial or organ-specific energy deposition, as it strongly depends on field polarization and animal posture. Furthermore, long-term exposure to RF radiation at a fixed frequency and power density will result in substantial changes in SAR over time as an animal gains body weight. Even if the power is adjusted for body weight changes, the spatial distribution can vary. Full dosimetric analyses of all these variables are only available in a few studies. Furthermore, SARs to which animals can be exposed without the induction of systemic toxicity are generally limited by the induction of thermal effects; increases in body temperature may induce biological responses that are not seen at the (generally much lower) levels of RF radiation to which humans may be exposed. In a substantial number of studies, exposure was at SAR values below the maximum tolerated dose (MTD); nonetheless, these studies were considered to provide useful data, and were included in the evaluation.

Several cancer bioassays with RF radiation were conducted with exposure systems in which animals were restrained (usually in tubes) or non-restrained (in cages) during exposure. In this *Monograph*, study designs involving animal restraint were identified as such. Exposures involving animal restraint are generally limited to periods of no more than 4 hours per day. They have the advantage of optimal exposure uniformity and maximal local delivery of RF-radiation energy to the head or other selected body parts. Exposure of animals in cages – whole-body exposure – can be for up to 24 hours per day. The design of some bioassays with restrained animals included both sham-exposed and cage-control animals; because of the possibly confounding effects of restraint stress, the Working Group compared tumour responses in the exposed groups only to the responses in sham-exposed controls. Lack of a sham-exposed control group was considered a serious flaw in the study design.

The Working Group reviewed a large number of studies with end-points relevant to mechanisms of carcinogenesis, including genotoxicity, effects on immune function, gene and protein expression, cell signalling, oxidative stress, and apoptosis. Studies on the possible effects of RF radiation on the blood–brain barrier, and on a variety of effects in the brain itself were also considered. The Working Group found several studies inadequately controlled for the thermal effects of RF radiation, but also noted well conducted studies showing aneuploidy, spindle disturbances, altered microtubule structures or induction of DNA damage. While RF radiation has insufficient energy to directly produce genetic damage, other changes such as induction of oxidative stress and production of reactive oxygen species may explain these results. Indeed, several studies *in vitro* evaluated the possible role of RF radiation in altering levels of intracellular oxidants or activities of antioxidant enzymes. While the overall evidence was inconclusive, the Working Group expressed concern about the results from several of these studies.

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1. EXPOSURE DATA

1.1 Introduction

This chapter explains the physical principles and terminology relating to sources, exposures and dosimetry for human exposures to radiofrequency electromagnetic fields (RF-EMF). It also identifies critical aspects for consideration in the interpretation of biological and epidemiological studies.

1.1.1 *Electromagnetic radiation*

Radiation is the process through which energy travels (or “propagates”) in the form of waves or particles through space or some other medium. The term “electromagnetic radiation” specifically refers to the wave-like mode of transport in which energy is carried by electric (E) and magnetic (H) fields that vary in planes perpendicular to each other and to the direction of energy propagation.

The variations in electric and magnetic field strength depend only on the source of the waves, and most man-made sources of electromagnetic radiation produce waves with field strengths that vary sinusoidally with time, as shown in Fig. 1.1. The number of cycles per second is known as the frequency (f) and is quantified in the unit hertz (Hz). The waves travel at the speed of light (c) in free space and in air, but more slowly in dielectric media, including body tissues. The wavelength (λ) is the distance between successive peaks in

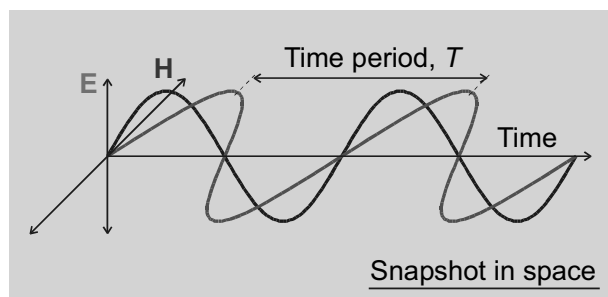
a wave (Fig. 1.1) and is related to the frequency according to $\lambda = c/f$ ([ICNIRP, 2009a](#)).

The fundamental equations of electromagnetism, Maxwell’s equations, imply that a time-varying electric field generates a time-varying magnetic field and vice versa. These varying fields are thus described as “interdependent” and together they form a propagating electromagnetic wave. The ratio of the strength of the electric-field component to that of the magnetic-field component is constant in an electromagnetic wave and is known as the characteristic impedance of the medium (η) through which the wave propagates. The characteristic impedance of free space and air is equal to 377 ohm ([ICNIRP, 2009a](#)).

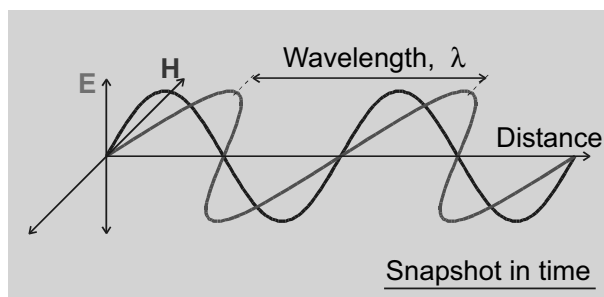
It should be noted that the perfect sinusoidal case shown in Fig. 1.1, in which a wave has a sharply defined frequency, is somewhat ideal; man-made waves are usually characterized by noise-like changes in frequency over time that result in the energy they carry being spread over a range of frequencies. Waves from some sources may show purely random variation over time and no evident sinusoidal character. Some field waveforms, particularly with industrial sources, can have a distorted shape while remaining periodic, and this corresponds to the presence of harmonic components at multiples of the fundamental frequency ([ICNIRP, 2009a](#)).

The quantities and units used to characterize electromagnetic radiation are listed in [Table 1.1](#).

Fig. 1.1 A sinusoidally varying electromagnetic wave viewed in time at a point in space (a) and in space at a point in time (b)



(a)



(b)

E, electric field; H, magnetic field.
Prepared by the Working Group

1.1.2 The electromagnetic spectrum

The frequency of electromagnetic radiation determines the way in which it interacts with matter; a variety of different terms are used to refer to radiation with different physical properties. The electromagnetic spectrum, describing the range of all possible frequencies of electromagnetic radiation, is shown in Fig. 1.2.

For the purposes of this *Monograph*, radio-frequency (RF) electromagnetic radiation will be taken as extending from 30 kHz to 300 GHz, which corresponds to free-space wavelengths in the range of 10 km to 1 mm. Electromagnetic fields (EMF) in the RF range can be used readily for communication purposes as radio waves. As shown in Fig. 1.2, the International Telecommunications Union (ITU) has developed a categorization for radio waves according to their frequency decade: very low frequency (VLF); voice frequency (VF); low frequency (LF); medium frequency (MF); high frequency (HF); very high frequency (VHF); ultra-high frequency (UHF); super-high frequency (SHF); and extremely high frequency (EHF) ([ITU, 2008](#)).

Radio waves with frequencies in the range 300 MHz to 300 GHz can be referred to as microwaves, although this does not imply any sudden change in physical properties at 300 MHz. The

photon energy would be about 1 μeV (micro-electronvolt) at 300 MHz.

Above the frequencies used by radio waves are the infrared, visible ultraviolet (UV), X-ray and gamma-ray portions of the spectrum. At RF and up to around the UV region, it is conventional to refer to the radiation wavelength, rather than frequency. Photon energy is generally referred to in the X-ray and gamma-ray regions, and also to some extent in the UV range, because the particle-like properties of the EMFs become more obvious in these spectral regions.

Below the RF portion of the spectrum lie EMFs that are used for applications other than radiocommunication. The interdependence of the electric- and magnetic-field components also becomes less strong and they tend to be considered entirely separately at the frequency (50 Hz) associated with distribution of electricity ([IARC, 2002](#)).

1.1.3 Exposures to EMF

RF fields within the 30 kHz to 300 GHz region of the spectrum considered in this *Monograph* arise from a variety of sources, which are considered in Section 1.2. The strongest fields to which people are exposed arise from the intentional use of the physical properties of fields, such as

Table 1.1 Quantities and units used in the radiofrequency band

Quantity	Symbol	Unit	Symbol
Conductivity	σ	siemens per metre	S/m
Current	I	ampere	A
Current density	J	ampere per square metre	A/m ²
Electric-field strength	E	volt per metre	V/m
Frequency	f	hertz	Hz
Impedance	Z or η	ohm	Ω
Magnetic-field strength	H	ampere per metre	A/m
Permittivity	ϵ	farad per metre	F/m
Power density	S or Pd	watt per square metre	W/m ²
Propagation constant	K	per metre	m ⁻¹
Specific absorption	SA	joule per kilogram	J/kg
Specific absorption rate	SAR	watt per kilogram	W/kg
Wavelength	λ	metre	m

Adapted from [ICNIRP \(2009a\)](#)

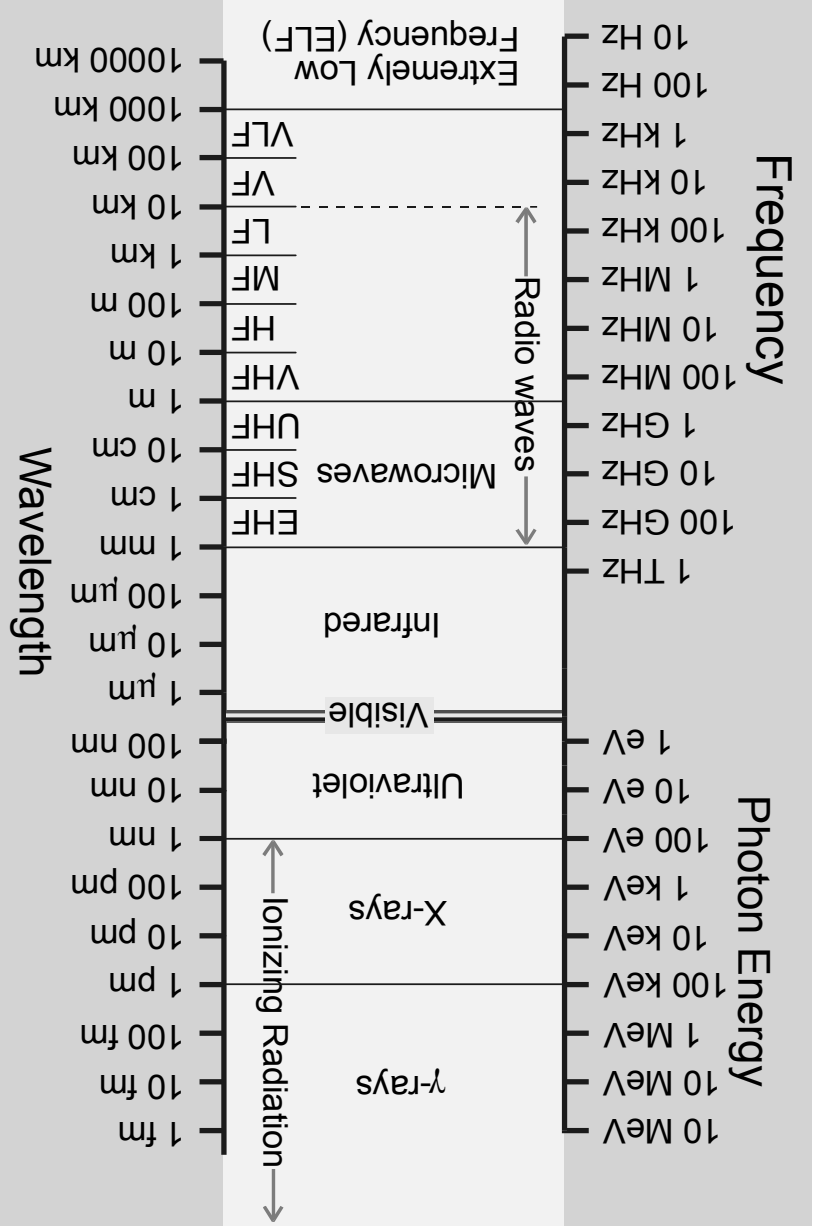
induction heating (including the industrial heating of materials and cooking hobs), remote detection of objects and devices (anti-theft devices, radar, radiofrequency identification [RFID]), telecommunications (radio, television, mobile phones, wireless networks), medical diagnostics and therapy (magnetic resonance imaging [MRI], hyperthermia), and many more. There are also unintentionally generated fields, such as those associated with the electrical ballasts used for fluorescent lighting, electronic circuits, processors and motors.

When considering human exposures it is important to recognize that, in addition to the EMFs associated with energy being radiated away from a source, there are electric and magnetic fields associated with energy stored in the vicinity of the source, and this energy is not propagating. The reactive fields associated with this stored energy are stronger than the radiated fields within the region known as the reactive near field, which extends to a distance of about a wavelength from the source. The wave impedance in the reactive near field may be higher than the impedance of free space if a source is capacitive in nature and lower if a source is inductive in nature ([AGNIR, 2003](#)).

Beyond the near field region lies the far field, where the RF fields have the characteristics of radiation, i.e. with planar wave fronts and E and H components that are perpendicular to each other and to the direction of propagation. The power density of the radiation, P_d , describes the energy flux per unit area in the plane of the fields expressed as watts per square metre (W/m²) and decreases with distance squared (the inverse square law). Power density can be determined from the field strengths (see Glossary) ([AGNIR, 2003](#)).

Sources that are large relative to the wavelength of the RF fields they produce, e.g. dish antennae, also have a region known as the radiating near field that exists in between the reactive near field and the far field. In this region the wave impedance is equal to 377 ohm, but the wave fronts do not have planar characteristics: there is an oscillatory variation in power density with distance and the angular distribution of the radiation also changes with distance. Since the radiating near field is taken to extend to a distance of $2D^2/\lambda$ (where D is the largest dimension of the antenna) from the source, it is therefore necessary to be located beyond both this distance and

Fig. 1.2 The electromagnetic spectrum



The figure shows frequency increasing from left to right expressed in hertz (Hz) and in kHz (kilo-), MHz (mega-), GHz (giga-) and THz (tera-) (denoting multipliers of 10³, 10⁶, 10⁹ and 10¹²). Electromagnetic fields in the radiofrequency (RF) range can be used for communication purposes as radio waves. Mobile phones operate in the low-microwave range, around 1 GHz. The terms VLF, VF, LF, MF, HF, VHF, UHF, SHF, EHF denote very low frequency, voice frequency, low frequency, medium frequency, high frequency, very high frequency, ultra-high frequency, super-high frequency, and extremely high frequency, respectively. Beyond the frequencies used by radio waves follow the infrared, visible, ultraviolet, X-ray and gamma-ray portions of the spectrum. Above radiofrequencies and up to around the ultraviolet region, it is conventional to refer to the wavelength (expressed in metres and its multipliers) of the radiation, rather than frequency. Below the radiofrequency portion of the spectrum lie electromagnetic fields that are used for applications other than radiocommunications. Photon energy is expressed in electronvolts (eV and its multipliers). Prepared by the Working Group

about a wavelength from a source to be in the far-field region ([AGNIR, 2003](#)).

The incident EMFs (external fields when the body is not present) interact or couple with the humanbodyandinduceEMFsandcurrentswithin the body tissues. A different interaction mechanism exists for the electric- and magnetic-field components, as discussed in detail in Section 1.3.

In general, both quantities must be determined to fully characterize human exposure, unless the exposure is to pure radiating fields. The coupling depends on the size of the wavelength relative to

the dimensions of the human body and, therefore, dosimetric interactions are often considered in three different frequency ranges: 30 kHz to 10 MHz (body larger than the wavelength), 10 MHz to 10 GHz (body dimensions comparable to the wavelength), and 10 GHz to 300 GHz (body dimensions much larger than the wavelength).

1.2 Sources of exposure

This section describes natural and man-made sources of RF fields to which people are exposed during their everyday lives at home, work and elsewhere in the environment. Fields from natural and man-made sources differ in their spectral and time-domain characteristics and this complicates comparisons of their relative strengths. The fields produced by natural sources have a much broader frequency spectrum than those produced by man-made sources and it is necessary to define a bandwidth of interest for comparison. In a bandwidth of 1 MHz, man-made fields will typically appear to be orders of magnitude stronger than natural ones, whereas if the entire bandwidth of 300 GHz of interest to this *Monograph* is chosen, natural fields may appear to be stronger than man-made ones at typical environmental levels ([ICNIRP, 2009a](#)).

When considering sources, it is helpful to clearly delineate the concepts of emissions, exposures and dose:

Emissions from a source are characterized by the radiated power, including its spectral and time-domain distributions: the polarization and the angular distribution (pattern) of the radiation. For sources that are large relative to their distance from a location where a person is exposed, it also becomes necessary to consider the spatial distribution of the emitted radiation over the entire structure of the source to fully describe it as an emitter.

Exposure describes the EMFs from the source at a location where a person may be present in terms of the strength and direction of the electric and magnetic fields. If these vary over the volume occupied by a person (non-uniform exposure), possibly because the source is close to them, or has strongly directional characteristics, it becomes necessary to quantify the RF fields over the space occupied by the person. The exposure depends not only on the source emissions and the geometrical relationship to the

source (distance, angular direction), but also on the effect of the environment on the radiated fields. This can involve processes such as reflection, shielding, and diffraction, all of which can modify the fields substantially.

Dose is concerned with quantities of effects inside the body tissues that are induced by the exposure fields. These include the electric- or magnetic-field strength in the body tissues and the specific energy absorption rate (SAR) (see Section 1.3.2, and Glossary). The strength of the electric fields within the body tissues is generally much smaller than that of the exposure fields outside the body, and depends on the electrical parameters of the tissues ([Beiser, 1995](#)).

In most situations, the concept of emissions leading to exposure and then dose is helpful, but there are situations in which the presence of an exposed individual and the dose received affect the emissions from a source. This means that the intermediate concept of exposure cannot be isolated meaningfully, and dose has to be assessed directly from the source emissions either through computational modelling or via measurement of fields inside the body tissues. When the way in which a source radiates is strongly affected by the presence of an exposed person, the source and the exposed person are described as “mutually coupled”; a classic example of this is when a mobile phone is used next to the body.

1.2.1 Natural fields

The natural electromagnetic environment originates from the Earth (terrestrial sources) and from space (extraterrestrial sources) (Fig. 1.3). Compared with man-made fields, natural fields are extremely small at RFs ([ICNIRP, 2009a](#)).

The energy of natural fields tends to be spread over a very wide range of frequencies. Many natural sources emit RF radiation and optical radiation according to Planck’s law of “black-body radiation” (see Fig. 1.4; [Beiser, 1995](#)).

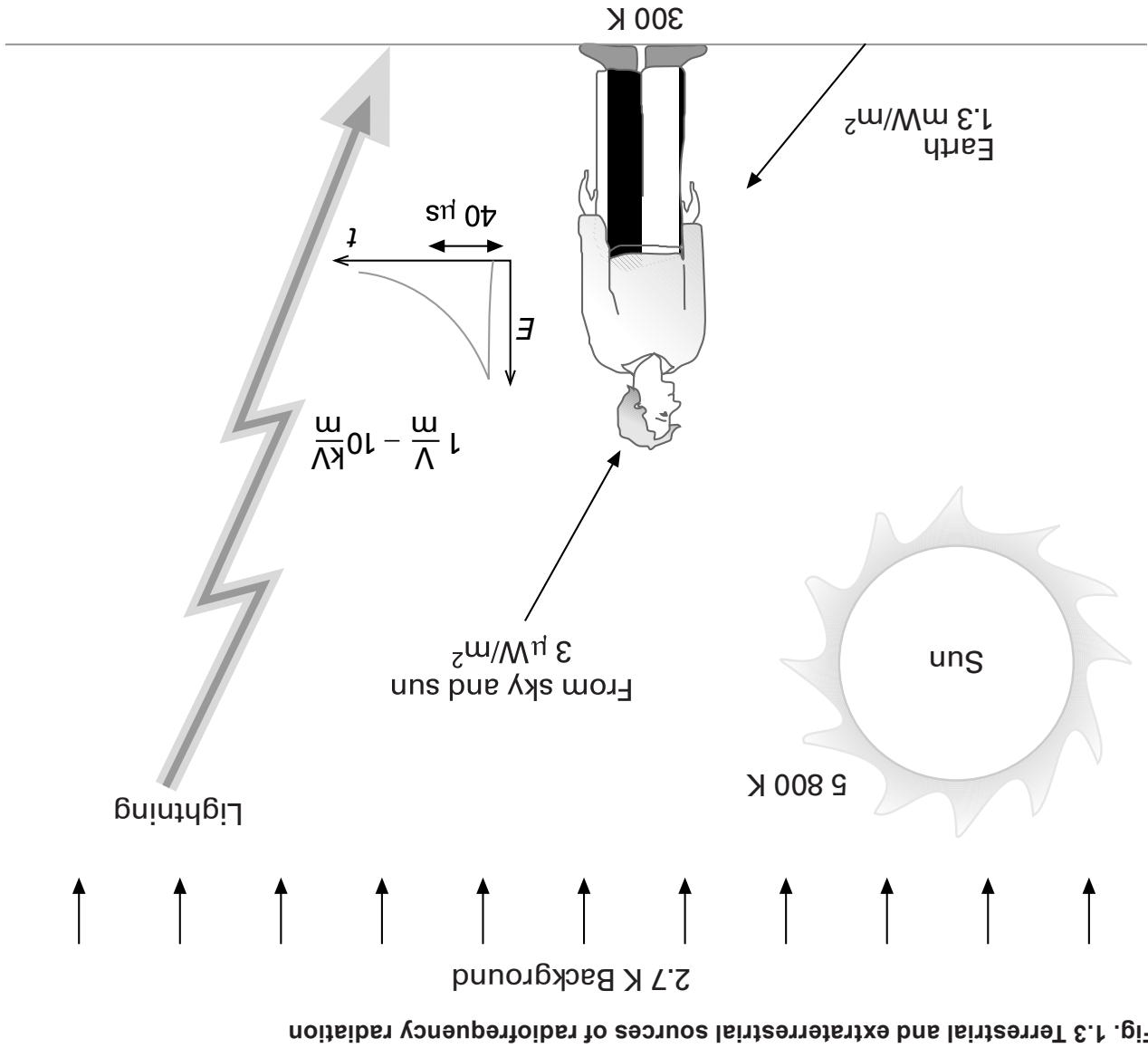


Fig. 1.3 Terrestrial and extraterrestrial sources of radiofrequency radiation

E, electric field strength; K, Kelvin; kV, kilovolt; m, metre; μs , microsecond; t, time; V, volt; W/m^2 , watt per square metre. The solar radiation spectrum is similar to that of a black body with a temperature of about 5800 °K. The sun emits radiation across most of the electromagnetic spectrum, i.e. X-rays, ultraviolet radiation, visible light, infrared radiation, and radio waves. The total amount of energy received by the Earth at ground level from the sun at the zenith is approximately 1000 W/m^2 , which is composed of approximately 53% infrared, 44% visible light, 3% ultraviolet, and a tiny fraction of radio waves (3 $\mu\text{W/m}^2$). From ICNIRP (2009a) <http://www.icnirp.de>

Fig. 1.4 Equations used in calculating energy and emitted power of black-body radiators

$$S(f,T) = \frac{2hf^2}{c^2} \frac{1}{e^{\frac{hf}{kT}} - 1}$$

$$J^* = \sigma T^4$$

(a) Planck's Law of "black body radiation"

$S(f,T)$ is the power radiated per unit area of emitting surface in the normal direction per unit solid angle per unit frequency by a black body at temperature T .

h is the Planck constant, equal to 6.626×10^{-34} Js.

c is the speed of light in a vacuum, equal to 2.998×10^8 m/s.

k is the Boltzmann constant, equal to 1.381×10^{-23} J/K.

f is the frequency of the electromagnetic radiation in hertz (Hz).

T is the temperature of the body in Kelvin (K).

(b) Stefan-Boltzmann Law

J^* , the black-body irradiance or emissive power, is directly proportional to the fourth power of the black-body thermodynamic temperature T (also called absolute temperature).

σ , the constant of proportionality, called Stefan-Boltzmann constant.

The total power emitted per unit surface area of a black-body radiator can be evaluated by integrating Planck's law over all angles in a half-space (2π steradians) and over all frequencies. This yields the Stefan-Boltzmann law (see Fig. 1.4), which describes how the power emitted by a black-body radiator increases with the fourth power of the absolute temperature ([Beiser, 1995](#)).

(a) Extraterrestrial sources

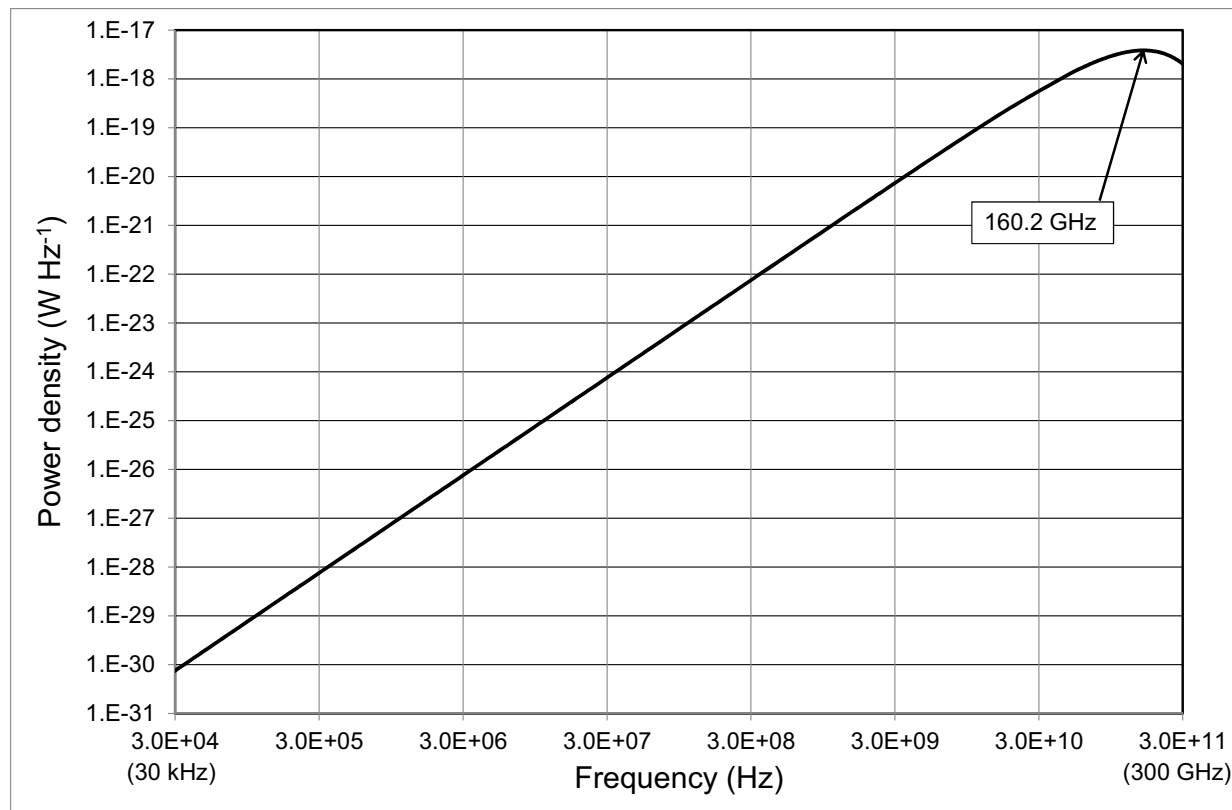
Extraterrestrial sources include electrical discharges in the Earth's atmosphere, and solar and cosmic radiation. Heat remaining from the "big bang" at the formation of the universe is evident as the cosmic microwave background (CMB), which presents as black-body radiation from all directions towards the Earth. The observed peak in the CMB spectrum is at a frequency of 160.2 GHz, which according to Planck's law (see Fig. 1.4) implies a temperature of 2.725 K ([Fixsen, 2009](#)). Fig 1.5 shows the results of evaluating Planck's law over the frequency range 30 kHz to 300 GHz. The total power density in this frequency range represents 80% of the total power density across all frequencies. Applying this factor to the results from Stefan-Boltzmann's

law at 2.725 K gives the power density at the surface of the Earth as $2.5 \mu\text{W}/\text{m}^2$.

The sun is also a black-body radiator and its spectrum shows a peak at 3.4×10^{14} Hz, a wavelength of 880 nm, commensurate with a surface temperature of 5778 K ([NASA, 2011](#)). Based on Planck's law, most of the sun's radiation is in the infrared region of the spectrum. Only a small proportion is in the frequency range 30 kHz to 300 GHz; this fraction represents about $5 \mu\text{W}/\text{m}^2$ of the total power density of $1366 \text{ W}/\text{m}^2$ incident on the Earth. This value is similar to that from the CMB, which contributes power from all directions, but the RF power from the sun is predominantly incident from the direction of the sun, and hence much reduced at night ([ICNIRP, 2009a](#)).

The atmosphere of the Earth has a marked effect on RF fields arriving from space. The ionosphere, which extends from about 60 km to 600 km above the Earth's surface, contains layers of charged particles and reflects RF fields at frequencies of up to about 30 MHz. Above a few tens of gigahertz, atmospheric water vapour and oxygen have an attenuating effect on RF fields, due to absorption. These effects mean that the RF power density incident at the Earth's surface

Fig. 1.5 Power density spectrum of the cosmic microwave background in the radiofrequency range (30 kHz to 300 GHz)



Prepared by the Working Group

from the sun and the CMB will be somewhat less than the $5 \mu\text{W}/\text{m}^2$ values given for each above. The International Commission on Non-Ionizing Radiation Protection (ICNIRP) gives the total power density arising from the sky and the sun as $3 \mu\text{W}/\text{m}^2$ at the surface of the Earth (see Fig. 1.3; [ICNIRP, 2009a](#)).

(b) Terrestrial sources

The Earth itself is a black-body radiator with a typical surface temperature of about 300 K (see Fig. 1.3). Most emissions from Earth are in the infrared part of the spectrum and only 0.0006% of the emitted power is in the RF region, which amounts to a few milliwatts per square metre from the Earth's surface. This is about a thousand times larger than the RF power density arising from the sky and the sun ([ICNIRP, 2009a](#)).

People also produce black-body radiation from their body surfaces (skin). Assuming a surface temperature of 37°C , i.e. 310 K, the power density for a person would be $2.5 \text{ mW}/\text{m}^2$ in the RF range. With a typical skin area of 1.8 m^2 , the total radiated power from a person is about 4.5 mW.

As mentioned above, the ionosphere effectively shields the Earth from extraterrestrially arising RF fields at frequencies below 30 MHz. However, lightning is an effective terrestrial source of RF fields below 30 MHz. The fields are generated impulsively as a result of the time-varying voltages and currents associated with lightning, and the waveguide formed between the surface of the Earth and the ionosphere enables the RF fields generated to propagate over large distances around the Earth.

On average, lightning strikes the Earth 40 times per second, or 10 times per square kilometre per year. Maps of annual flash rates based on observations by National Aeronautics and Space Administration (NASA) satellites can be consulted on the National Oceanic and Atmospheric Administration (NOAA) web site ([NOAA, 2011](#)). The EMFs from lightning are impulsive and vary depending on the nature of each stroke and also according to the distance at which they are measured. A typical pulse-amplitude of 4 V/m at 200 km corresponds to a peak power density of 42 mW/m², and a total pulse energy density of 2.5 mJ/m² ([ICNIRP, 2009a](#)). [Cooray \(2003\)](#) has described various mathematical models for return strokes, which are the strongest sources of RF-EMF associated with lightning. Peak electric-field strengths of up to 10 kV/m are possible within 1 km from where the lightning strikes. At distances greater than 100 km, the field strength decreases rapidly to a few volts per metre, with peak dE/dt of about 20 V/m per μ s, and then further decreases over a few tens of microseconds. [Willett et al. \(1990\)](#) measured the electric-field strength during return strokes as a function of time and conducted Fourier analysis to determine the average spectrum between 200 kHz and 30 MHz. The energy spectral density reduced according to $1/f^2$ at frequencies of up to about 10 MHz and more rapidly thereafter.

1.2.2 Man-made fields

There are numerous different sources of man-made RF fields. The more common and notable man-made sources of radiation in the RF range of 30 kHz to 300 GHz are presented in Fig. 1.6.

Sometimes such fields are an unavoidable consequence of the way systems operate, e.g. in the case of broadcasting and telecommunications, where the receiving equipment is used at locations where people are present. In other situations, the fields are associated with energy

waste from a process, e.g. in the case of systems designed to heat materials ([ICNIRP, 2009a](#)).

The typical emission characteristics of sources will be summarized here, along with exposure and dose information where available. However, it is important to recognize that fields typically vary greatly in the vicinity of sources and spot measurements reported in the literature may not be typical values. This is because assessments are often designed to identify the maximum exposures that can be reasonably foreseen, e.g. for workers near sources, and to ensure that these do not exceed exposure limits.

(a) Radio and television broadcasting

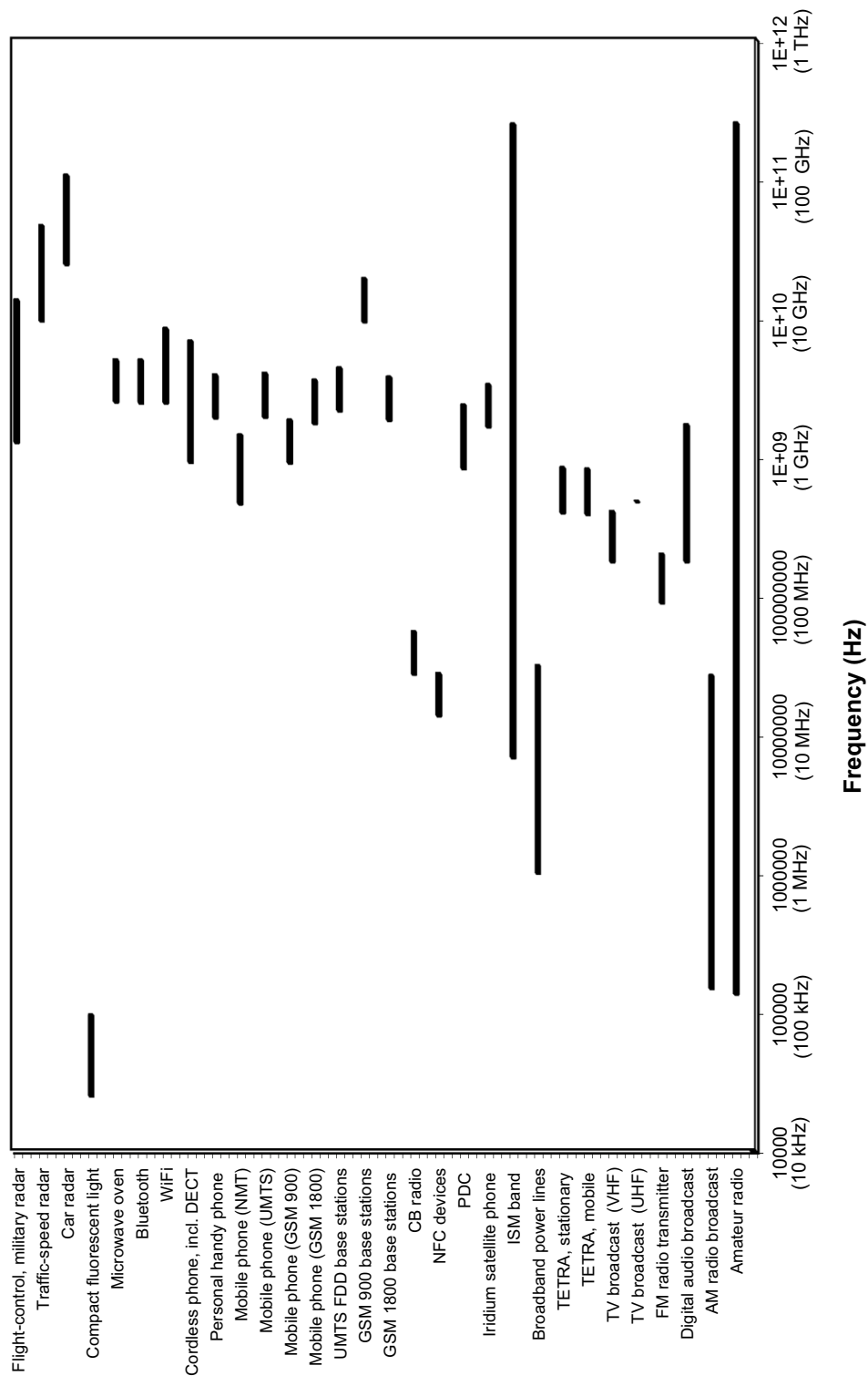
The frequency bands used for broadcasting of radio and television signals are broadly similar across countries and are shown in [Table 1.2](#).

Analogue broadcast radio has been available for many years and uses amplitude modulation (AM) in the long, medium and short-wave bands, but the sound quality is not as good as with frequency modulation (FM) in band II, which became available later and is now more popular for listening. The short-wave band continues to be important for international radio broadcasting, because signals in this frequency band can be reflected from the ionosphere to travel around the world and reach countries thousands of kilometres away ([AGNIR, 2003](#)).

Band III was the original band used for television broadcasting and continues to be used for this purpose in some countries, while others have transferred their television services to bands IV and V. Band III is also used for digital audio broadcasting (DAB), exclusively so in countries that have transferred all their television services to bands IV and V. Analogue and digital television transmissions presently share bands III, IV and V, but many countries are in the process of transferring entirely to digital broadcasting ([ICNIRP, 2009a](#)).

[AGNIR \(2003\)](#) have described broadcasting equipment in the United Kingdom in terms of

Fig. 1.6 Man-made sources of radiation in the radiofrequency range (30 kHz to 300 GHz)



AM, amplitude-modulated; CB, citizen band; DECT, digital enhanced cordless telecommunications; FDD, frequency-division duplex; FM, frequency-modulated; GSM, Global System for Mobile communications; ISM, industrial, scientific and medical; NFC, near-field communication; NMT, Nordic Mobile Telephony; PDC, personal digital cellular; TETRA, Terrestrial Trunked Radio; TV, television; UHF, ultra-high frequency; UMTS, Universal Mobile Telecommunications System; VHF, very high frequency; WiFi, standard wireless local area network (WLAN) technology.

Prepared by the Working Group

Table 1.2 Frequency bands used for broadcasting of television and radio signals

Designation	Frequency range	Usage
Long wave	145.5 – 283.5 kHz	AM radio
Medium wave	526.5 – 1606.5 kHz	AM radio
Short wave	3.9 – 26.1 MHz	International radio
UHF (Bands IV and V)	470 – 854 MHz	Analogue and digital TV
VHF (Band II)	87.5 – 108 MHz	FM radio
VHF (Band III)	174 – 223 MHz	DAB and analogue/digital TV

AM, amplitude modulation; DAB, digital audio broadcasting; FM, frequency modulation; TV, television; UHF, ultra high frequency; VHF, very high frequency

Adapted from [AGNIR \(2003\)](#)

the numbers of transmitters operating at a given power level in each frequency band ([Table 1.3](#)). The overall trends are probably similar in other countries and the main change since that time is likely to have been a growth in the number of digital transmitters for radio and television ([ICNIRP, 2009a](#)).

(i) *Long-, medium- and short-wave bands*

Antennae broadcasting in the long- and medium-wave bands tend to be constructed as tall metal towers, with cables linking the towers to each other and to the ground. Often, a single low-frequency (LF) or medium-frequency (MF) radiating structure may involve several closely located towers that are fed in such a way that a directional beam pattern is formed. Some towers are energized and insulated from the ground, while others are grounded and act as reflectors. Transmitters designed to provide local radio services, e.g. around cities, use powers in the range of 100 W to 10 kW, while a small number of transmitters that provide national services over large distances radiate up to a few hundred kilowatts ([ICNIRP, 2009a](#)).

The high-frequency (HF) band is used for international broadcasting and comprises wavelengths that are somewhat shorter than those in the long- and medium-wave bands. Curtain arrays, composed of multiple horizontal dipole antennae suspended between towers, are used to form narrow beams directed upwards towards

the required azimuth and elevation angles. The beams reflect off the ionosphere and provide services to distant countries without the need for any intermediate infrastructure. Typical curtain arrays can be up to 60 m in height and width, and might, for example, involve 16 dipoles arranged as four vertically stacked rows of four with a reflecting wire mesh screen suspended behind them. Given the transmission distances required, the powers are high, typically around 100–500 kW. The HF band has the fewest transmitters of any of the broadcast bands ([ICNIRP, 2009a](#)). [Allen et al. \(1994\)](#) reported 25 HF transmitters with powers in the range 100–500 kW and three with powers greater than 500 kW in the United Kingdom.

Broadcast sites can be quite extensive, with multiple antennae contained within an enclosed area of several square kilometres. A building containing the transmitters is generally located on the site and RF feeder cables are laid from this building to the antennae. On HF sites, switching matrices allow different transmitters to be connected to different antennae according to the broadcast schedule. The feeders may be either enclosed in coaxial arrangements or open, e.g. as twin lines having pairs of conductors around 15 cm apart suspended about 4 m above ground level.

In considering reported measurements of RF fields at MF/HF broadcast sites, it is important to note that workers may spend much of their time

Table 1.3 Approximate number of broadcast transmitters in the United Kingdom^a

Service class	Effective radiated power (kW)					
	0–0.1	> 0.1–1.0	> 1.0–10	> 10–100	> 100–500	> 500
Analogue TV	3496	589	282	122	86	19
DAB	4	126	121	–	–	–
Digital TV	134	177	192	2	–	–
MW/LW radio	14	125	38	19	12	–
VHF FM radio	632	294	232	98	72	–

^a For TV sites, each analogue channel (e.g. BBC1) or each digital multiplex counts as one transmitter.

DAB, digital audio broadcasting; FM, frequency modulation; LW, long wave; MW, medium wave; TV, television; VHF, very high frequency

Adapted from [AGNIR \(2003\)](#)

in offices, workshops or the transmitter halls. Such locations can be far from the antennae, resulting in exposure levels that are much lower than when personnel approach the antennae to carry out maintenance and installation work.

[Jokela et al. \(1994\)](#) investigated the relationship between induced RF currents flowing through the feet to ground and the RF-field strengths from MF and HF broadcast antennae. The MF antenna was a base-fed monopole, 185 m high, transmitting 600 kW at 963 MHz. At distances of 10, 20, 50, and 100 m from the antenna, the electric-field strength at 1 m height was around 420, 200, 60 and 30 V/m, respectively. At the same distances, currents in the feet were around 130, 65, 30 and 10 mA. The HF antenna was a 4 × 4 curtain array suspended between 60 m towers and radiating 500 kW at 21.55 MHz. The total field in front of the antenna at 1 m height ranged from about 32 V/m at 10 m through a maximum of 90 V/m at 30 m, a minimum of 7 V/m at 70 m and thereafter rose to around 20 V/m at distances in the range 100–160 m.

[Mantiply et al. \(1997\)](#) have summarized measurements of RF fields from MF broadcast transmitters contained in several technical reports from the mid-1980s to early 1990s from government agencies in the USA. A study based on spot measurements made at selected outdoor locations in 15 cities and linked to population statistics showed that 3% of the urban population

were exposed to electric-field strengths greater than 1 V/m, while 98% were exposed to field strengths above 70 mV/m and the median exposure was 280 mV/m. RF-field strengths were also measured near eight MF broadcast antennae, one operating at 50 kW, three at 5 kW and four at 1 kW. The measurements were made as a function of distance along three radials at most of the sites. At distances of 1–2 m, the electric-field strengths were in the range 95–720 V/m and the magnetic-field strengths were in the range 0.1–1.5 A/m, while at 100 m, electric-field strengths were 2.5–20 V/m and magnetic-field strengths were in the range 7.7–76 mA/m.

[Mantiply et al. \(1997\)](#) also reported field measurements near short-wave (HF) broadcast antennae. As mentioned earlier, these are designed to direct the beams upwards at low elevation angles. Hence, the field strengths at locations on the ground are determined by sidelobes (see Glossary) from the antennae and they vary unpredictably with distance and from one antenna to another. Measurements were made at four frequencies in the HF band and at six locations in a community around 10 km from an HF site, which was likely to have transmitted 250 kW power. Electric- and magnetic-field strengths at individual frequencies varied in the ranges 1.5–64 mV/m and 0.0055–0.16 mA/m, while the maximum field strengths just outside the site boundary were 8.6 V/m and 29 mA/m.

Field strengths measured at a distance of 100 m along a “traverse” tangential to the beam from a curtain array transmitting at 100 kW were in the ranges 4.2–9.2 V/m and 18–72 mA/m. A final set of measurements was made at a distance of 300 m from another curtain array transmitting at 100 kW, while the beam was steered through $\pm 25^\circ$ in azimuth. The field strengths were in the ranges 1.7–6.9 V/m and 14–29 mA/m.

(ii) *VHF and UHF bands*

The powers used for broadcasting in the VHF and UHF bands vary widely according to the area and terrain over which coverage is to be provided ([Table 1.2](#)). UHF transmissions are easily affected by terrain conditions, and shadowed areas with poor signal strength can occur, e.g. behind hills and in valleys. For this reason, in addition to a main set of high-power transmitters, large numbers of local booster transmitters are needed that receive signals from the main transmitters and rebroadcast them into shadowed areas. The main transmitters are mounted at the top of masts that are up to several hundreds of metres high and have effective radiated powers (ERPs) (see Glossary) of up to about 1 MW, while the booster transmitters have antennae that are mounted much nearer to the ground and mostly have powers of less than 100 W. VHF signals are less affected by terrain conditions and fewer booster transmitters are needed.

Typical high-power broadcast transmitter masts are shown in Fig. 1.7.

Access to the antennae on high-power VHF/UHF masts is gained by climbing a ladder inside the tower; reaching the antennae at the top involves passing in close proximity to radiating antennae at lower heights. The VHF transmissions have wavelengths of similar dimensions to the structures that form the tower itself, e.g. the lengths of the steel bars or the spaces between them, and hence tend to excite RF current flows in these items. Standing waves (see Glossary) can be present within the tower, and the measured

field strengths can be strongly affected by the presence of a person taking measurements. Thus, measurements of field strength can seem unstable and difficult to interpret. Currents flowing within the body can be measured at the wrist or ankle and these are more directly related to the specific absorption rate (SAR; dose) in the body than the fields associated with the standing waves. Hence, it can be preferable to measure body current (see Section 1.3) rather than field strength on towers with powerful VHF antennae.

Several papers discussed by [ICNIRP \(2009a\)](#) have reported measurement results in the range of tens to hundreds of volts per metre within broadcast towers, but it is not clear how representative these spot measurements are of typical worker exposures. [Cooper et al. \(2004\)](#) have used an instrument worn on the body as personal dosimeter to measure electric- and magnetic-field strengths during work activities at a transmitter site. They reported that a wide temporal variation in field strengths was typically found within any single record of exposure to electric or magnetic fields during work on a mast or tower used for high-power VHF/UHF broadcasts. Fig 1.8 shows a typical trace that was recorded for a worker during activities near the VHF antennae while climbing on a high-power VHF/UHF lattice mast. The field strength commonly ranged from below the detection threshold of about 14 V/m to a level approaching or exceeding the upper detection limit of about 77 V/m. The highest instantaneous exposures usually occurred when the subject was in the vicinity of high-power VHF antennae or when a portable VHF walkie-talkie radio was used to communicate with other workers.

Field strengths around the foot of towers/masts have also been reported and seem quite variable. [Mantiply et al. \(1997\)](#) described values in the range of 1–30 V/m for VHF television, 1–20 V/m for UHF television and 2–200 V/m for VHF FM radio sites. Certain designs of antennae have relatively strong downward-directed sidelobes,

Fig. 1.7 Typical antenna masts for power broadcasting of radio and television signals



(a)



(b)

(a) A concrete tower, 368 m high, with a spherical structure at just above 200 m. This is accessed by lifts from ground level and contains various equipment as well as a public restaurant. The radiating antennae are above the sphere and the antennae operating at the highest frequencies are nearest to the top. Multiple dipole antennae protrude through the wall of the red/white cylinder to provide FM radio services in band II, and television and DAB services in band III. Contained within the top-most section of the tower are the band IV and V antennae for more television services.

(b) A steel-lattice tower with the television antennae in the white cylinder at the top. Antennae for VHF and DAB broadcast radio services are mounted on the outside of the tower just below the television antenna and there are multiple antennae for other communications purposes at lower heights. The transmitters are in a building near the base of the tower and the coaxial cables carrying the RF to the transmitting antennae pass up inside the tower.

Courtesy of the Health Protection Agency, United Kingdom

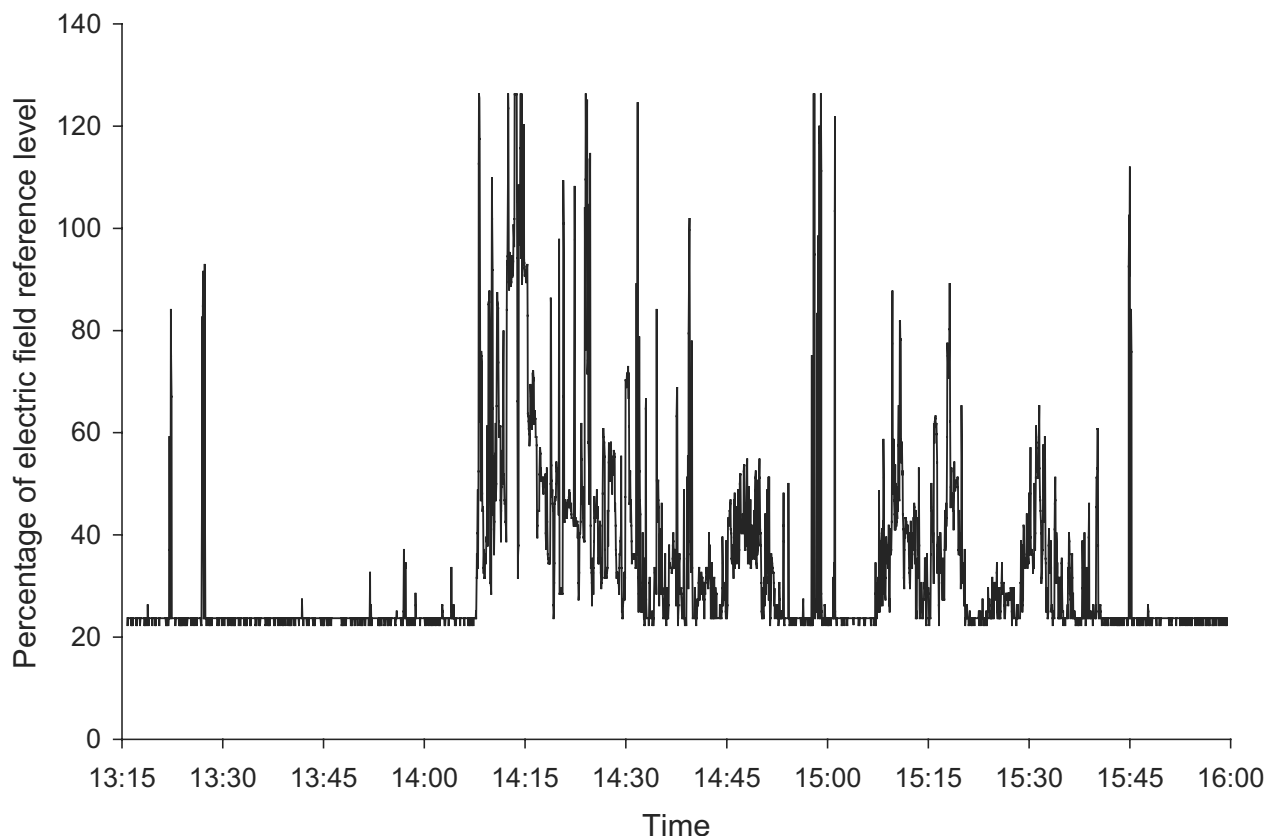
known as grating lobes, which is a possible explanation for such variability.

VHF/UHF broadcast antennae are designed to direct their beams towards the horizon, usually in all directions around the tower. Hence, field strengths at ground level and in communities near the tower are much lower than at comparable distances within the beam. When the beams do eventually reach ground level, they have spread out considerably, again implying that exposures for the general public are substantially lower than

those for workers at locations to which they have access, as summarized above ([ICNIRP, 2009a](#)).

[Mantiply *et al.* \(1997\)](#) report studies of population exposure in the USA conducted during the 1980s and based on spot measurements at selected outdoor locations. An estimated 50%, 32% and 20% of the population were exposed at greater than 0.1 V/m from VHF radio, VHF television and UHF television signals, respectively. VHF radio and television caused exposures to 0.5% and 0.005% of the population at greater than

Fig. 1.8 Relative electric-field strength recorded for an engineer operating on a mast supporting antennae for high-power VHF/UHF broadcast transmissions



The reference level is 61 V/m, as taken from the [ICNIRP \(1998\)](#) exposure guidelines for workers over the relevant frequency range (10–400 MHz). UHF, ultra high frequency; VHF, very high frequency
From [Cooper et al. \(2004\)](#). By permission of Oxford University Press.

2 V/m, while UHF television caused exposure to 0.01% of the population at greater than 1 V/m.

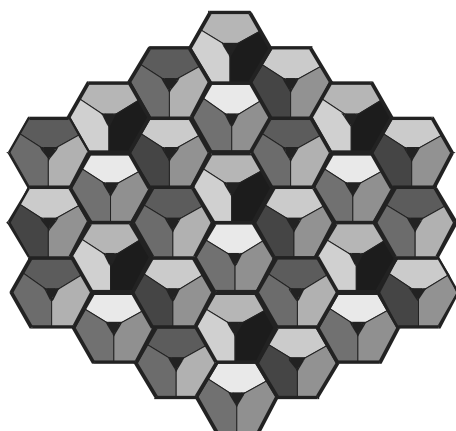
Field strengths associated with VHF/UHF radio and television broadcast signals were measured at 200 statistically distributed locations in residential areas around Munich and Nuremberg in Germany ([Schubert et al., 2007](#)). The aim of the study was to investigate whether the levels had changed as a result of the switch-over from analogue to digital broadcasting, and measurements were made before and after this change occurred at each location. The median power density was $0.3 \mu\text{W}/\text{m}^2$ (11 mV/m) for the analogue signals and $1.9 \mu\text{W}/\text{m}^2$ (27 mV/m) for the digital signals. FM radio signals had median

power densities of $0.3 \mu\text{W}/\text{m}^2$ (11 mV/m), similar to the analogue television signals, and the values ranged over approximately two orders of magnitude on either side of the medians for all types of broadcast signal. It is interesting to note that these values seem to be lower than those reported in the USA during the 1980s.

(b) Cellular (mobile-phone) networks

Unlike broadcasting, for which high-power transmitters are used to cover large areas extending 100 km or more from the transmitter, cellular networks employ large numbers of low-power transmitters, known as base stations, which are scattered throughout an area where coverage

Fig. 1.9 Example of a coverage plan for a cellular network



Each cell is hexagonal, with a base station at its centre and configured to provide signals over three sectors of 120 degrees. The shading show how coverage is provided everywhere by use of 12 frequency channels, none of which are used in the adjacent cells.

Courtesy of the Health Protection Agency, United Kingdom

is to be provided. This is because communications are two-way (duplex) in cellular networks, with each user requiring their own dedicated communication channels, both for the uplink (phone to base station) and for the downlink (base station to the phone). Each base station has limited capacity in terms of the number of calls it can serve simultaneously, so the transmitters are closer together in locations where there is a high density of users. For example, the transmitters may be about 10 km apart in sparsely populated areas, but 100 m or less apart in city centres.

An important consideration in the design of cellular networks is that operators have a limited spectrum window available and have to reuse their frequency channels to provide coverage everywhere. A typical frequency map illustrating how coverage can be provided with 12 frequency channels is shown in Fig. 1.9. Signals that use the same frequency in different cells can potentially interfere with each other, but the signal strength diminishes with increasing distance from base stations and frequencies are not reused in adjacent cells/sectors. Hence, services can be provided without interference, provided that

the radiated powers of phones and base stations are minimized during calls. This principle has important consequences for the RF exposures of people using phones and living near base stations ([ICNIRP, 2009a](#)).

Developments in mobile-phone technology are broadly categorized according to four different generations ([Table 1.4](#)). The first-generation networks (1G) were rolled-out in the mid-1980s and included Advanced Mobile Phone System (AMPS) in North America, Total Access Communication Systems (TACS) in much of Europe, Nippon Telegraph and Telephone (NTT) in Japan, and Nordic Mobile Telephony (NMT) in Scandinavia. The systems were based on analogue technology and used frequency modulation to deliver voice-communication services. These networks mostly closed down from around the year 2000, as users moved to later generations of the technology ([ICNIRP, 2009a](#)).

Second-generation networks (2G) were established in the early 1990s and continue to operate. They are based on digital technology and use voice coding to improve spectral efficiency. Many systems use time-division multiple access (TDMA) within their frequency channels and such systems include Global System for Mobile (GSM) in Europe, Personal Digital Cellular (PDC) in Japan, and both Personal Communication Systems (PCS) and D-AMPS (digital AMPS, also known as “TDMA”) in North America. Other north-American systems are known as CDMA, because they use code-division multiple access. 2G systems were extended to include some basic data services, but subsequent systems with enhanced data services were usually termed 2.5G ([ICNIRP, 2009a](#)).

The third generation of mobile phones (3G), with comprehensive data services, became available in the early 2000s. These phones have developed to become today’s “smartphones,” although it is important to recognize that they are fully backward-compatible with 2G networks and whether 2G or 3G is used at any given time

Table 1.4 Frequency bands originally used by different mobile-phone systems

Generation	Start date of commercial availability ^a	Main geographical region	System ^b	Handset band (MHz)	Base-station band (MHz)	Channel spacing (kHz)
1	1981	Nordic countries	NMT450	453.5 – 457.5	463.5 – 467.5	25
	1986		NMT900	890 – 915	935 – 960	12.5
	1985	Europe	TACS/ETACS	872 – 915	917 – 960	25
	1989	Japan	JTACS/NTACS	898 – 925	860 – 870	25/12.5
	1985	Germany	NET-C	451.3 – 455.74	461.3 – 465.74	20
	1985	USA & Canada	AMPS	824 – 849	869 – 894	30
	1985		N-AMPS	824 – 849	869 – 894	10
	1987	Japan	NTT	925 – 940	870 – 885	25
2	1992	USA & Canada	TDMA800	824 – 849	869 – 894	30
	1998		TDMA1900	1850 – 1910	1930 – 1990	30
	1992	Europe	GSM900	890 – 915	935 – 960	200
	1993		GSM1800	1710 – 1785	1805 – 1880	200
	2001	USA & Canada	GSM1900 (PCS)	1850 – 1910	1930 – 1990	200
	1993	Japan	PDC800	940 – 956	810 – 826	25
	1994		PDC1500	1429 – 1465	1477 – 1513	25
	1998	USA & Canada	CDMA800	824 – 849	869 – 894	1250
3	2001	World	IMT-2000 (W-CDMA)	1920 – 1980 ^c	2110 – 2170 ^c	5000
4		World	LTE	Many possible	Many possible	Various

^a The start dates of use will be different depending on country.

^b For abbreviations, see [Cardis et al. \(2011b\)](#) and [Singal \(2010\)](#).

^c Technical standards for a 2001 version for the 3G systems (IMT-2000). Note that standards for the 3G systems evolve quickly.

Compiled by the Working Group and adapted mainly from the references mentioned in footnote b

depends on network coverage and how operators have chosen to manage call/data traffic within their network. The systems use CDMA radio-access methods ([ICNIRP, 2009a](#)).

A fourth generation (4G) of the technology is just starting to be rolled out to meet the increasing demand for data services. Some systems are known as Long-term Evolution (LTE) and use orthogonal frequency-division multiplexing (OFDM), while others are based on Worldwide Interoperability for Microwave Access (WiMax). As with 3G services, this technology will be overlaid on other services, and phones will be able to support multiple access modes (4G, 3G and 2G) ([Buddhikot et al., 2009](#)).

The frequency bands originally used by cellular networks in various parts of the world

are shown in [Table 1.4](#). It is important to note that spectrum liberalization is ongoing at present, such that operators who hold a license for a particular part of the spectrum may choose to use it to provide services with any technology they wish. For example, bands originally reserved for 2G services such as GSM are being made available for 3G/4G services in many countries as demand shifts from 2G to systems with more capacity for data services. Also, with the move to digital-television broadcasting, the spectrum in the frequency range of 698 to 854 MHz is becoming available and being reallocated to 3G/4G cellular services ([Buddhikot et al., 2009](#)).

(i) Mobile-phone handsets

The output powers and – where TDMA is used – the burst characteristics of various types of mobile phones are summarized in [Table 1.5](#). Analogue mobile phones were specified to have maximum equivalent isotropically radiated powers (EIRP) of 1 W, but the antennae were not isotropic and would have had gains of around 2 dB. This implies the radiated powers would have been around 600 mW. 2G mobile phones that use TDMA have time-averaged powers that are less than their peak powers according to their duty factors, i.e. the time they spend transmitting, as a proportion of the total. For example, GSM phones that transmit at a power level of 2 W in the 900 MHz band (GSM900) have time-averaged powers that are 12% of this, i.e. 240 mW. Maximum time-averaged output powers are generally in the range of 125–250 mW for 2G onwards.

Mobile phones are generally held with their transmitting antennae around 1–2 cm from the body, so the RF fields they produce are highly non-uniform over the body and diminish rapidly in strength with increasing distance. The fields penetrate body tissues, leading to energy absorption, which is described by the SAR. SAR values are derived by phone manufacturers under a series of prescribed tests and the maximum value recorded under any of the tests is reported in the product literature. Values in normal usage positions should be lower than the values declared by manufacturers because the positions used in the testing standards are designed to mimic near-worst-case conditions.

While [Table 1.5](#) gives maximum output powers for phones, the actual power used at any point during a call is variable up to this maximum. As mentioned above, to minimize interference in the networks, the power is dynamically reduced to the minimum necessary to carry out calls. [Vrijheid et al. \(2009a\)](#) found that the reduction was on average to around 50% of the maximum

with GSM phones, whereas [Gati et al. \(2009\)](#) reported that 3G phones only operated at a few percent of the maximum power.

Another consideration is that GSM phones employ a mode called discontinuous transmission (DTX), under which their transmission-burst pattern changes to one with a lower duty factor during the periods of a conversation when the mobile-phone user is not talking. [Wiart et al. \(2000\)](#) found that DTX reduced average power by about 30% for GSM phones.

(ii) Time trends in SAR for mobile phones

As shown in [Table 1.5](#), analogue mobile phones had higher specified maximum radiated powers than digital ones (typically 0.6 W versus 0.1–0.25 W). While these systems are no longer in use and few data on exposure are available, it is of interest to consider whether exposures from these phones would have been higher than with present-day phones. Key differences, aside from relative power levels, are that analogue phones were larger than their modern digital counterparts and that they generally had larger antennae, e.g. extractable whip antennae rather than the compact helices and patch antennae used nowadays. The increased distance between the antenna and the head would have reduced the SAR level overall, and the larger size of the antenna would have led to a more diffuse distribution of SAR in the head.

The evolution of localized SAR values over time is also interesting to consider. [Cardis et al. \(2011b\)](#) assembled a database of reported peak 1-g and 10-g SARs for phones from a range of publications and web sites. Most data covered the years 1997–2003, and no significant upward or downward trends over this time period were found for the 900 MHz or 1800 MHz bands.

In summary, the peak spatial SARs (psSAR) do not seem to have changed significantly over time as analogue phones have been replaced by digital ones. However, the more diffuse nature of the distributions produced by analogue phones

Table 1.5 Output powers and TDMA characteristics of various types of mobile phone

System	Peak power (W)		Burst duration (ms)	TDMA duty factor	Average power (W)
	EIRP	Output			
GSM900	-	2.0	0.5769	0.12	0.24
GSM1800	-	1.0	0.5769	0.12	0.12
PCS1900	-	1.0	0.5769	0.12	0.12
NMT450	1.5	0.9	-	NA	0.9
PDC	-	0.8	3.333 or 6.666	1/6 or 1/3	0.133 or 0.266
NMT900	1.0	0.6	-	NA	0.6
TACS/ETACS	1.0	0.6	-	NA	0.6
AMPS/NAMPS	1.0	0.6	-	NA	0.6
TDMA800	-	0.6	6.666	1/3	0.2
TDMA1900	-	0.6	6.666	1/3	0.2
CDMA800	-	0.25	-	NA	0.25
CDMA1900	-	0.25	-	NA	0.25
IMT-2000	-	0.25	-	NA	0.25

EIRP, equivalent isotropically radiated power; NA, not applicable; TDMA, time-division multiple access
Compiled by the Working Group

would likely have led to a greater overall SAR in the head, including the brain.

(iii) Phones not making calls

The emitted powers from phones when they are on standby and not making calls are also of interest. Systematic studies have not been published on this topic, but transmissions under these conditions are brief and infrequent, and exposure is expected to be very small when averaged over time.

Phones equipped for data services such as e-mail will transmit for longer time periods than ordinary phones because they will be checking e-mail servers and synchronizing databases held on the phone with those on remote servers. Also, uploading large files such as videos and photographs may take many minutes. The phone is unlikely to be held against the user's head while this is taking place, although it may be in the user's pocket or elsewhere on the body, which may lead to local emissions at a higher power level than during calls, e.g. if general packet radio service (GPRS) is used, involving multislots transmission with GSM.

The sending of a text message from a mobile phone involves a short period of transmission. [Gati et al. \(2009\)](#) showed that a long text message would take at most 1.5 seconds to send with GSM systems.

(iv) Hands-free kits and Bluetooth earpieces

A phone may sometimes be used with a wired hands-free kit, in which case parts of the body other than the head may be exposed to maximal localized SARs, e.g. if the phone is placed in the user's pocket during the call. While one might expect that the audio cable to the ear-piece would not efficiently guide RF fields to the ear-piece, and that the use of wired hands-free kits would lead to greatly reduced SARs in the head due to the increased distance of the phone from the head, there have been suggestions that this is not always the case.

[Porter et al. \(2005\)](#) showed that the layout of the cables of the hands-free kit was a critical factor in determining head exposures and that certain geometries could result in appreciably more power being coupled into the audio cable than others. However, in all of the combinations

tested, the maximum value for SAR 10 g was lower when a hands-free kit was used than when it was not. [Kühn *et al.* \(2009a\)](#) further developed procedures for the testing of hands-free kits under worst-case and realistic conditions of use and applied them to a set of phones and kits. The authors concluded that exposure of the entire head was lower when a hands-free kit was used than when the phone was held directly against the head, but that there might be very localized increases in exposure in the ear.

Wireless hands-free kits are available that use the Bluetooth RF communications protocol to link to a mobile-phone handset located within a few metres of the body. This protocol provides for RF transmissions in the frequency range 2.4–2.5 GHz at power levels of 1, 2.5 or 100 mW. Only the lowest of these power levels would be used with a wireless hands-free kit and these are around a hundred times lower than the maximum output powers of mobile phones. In the study on wired hands-free kits mentioned above, [Kühn *et al.* \(2009a\)](#) also tested Bluetooth wireless hands-free kits and concluded that they are responsible for a low but constant exposure.

(v) *Mobile-phone base stations*

The base stations that provide mobile-phone services to come in many different sizes and shapes, according to their individual coverage requirements.

The radiated powers and heights of mobile-phone base-station antennae are highly variable. [Cooper *et al.* \(2006\)](#) collected data on base-station antenna height and power from all cellular operators in the United Kingdom, a total of 32 837 base stations, for the year 2002. The data are presented in Fig. 1.10 and show that base-station powers typically vary from about 0.1 W to 200 W and that heights range from about 3 m to 60 m above ground level. There is a large group of base stations with heights in the range 15–25 m and powers in the range 20–100 W, and a second group with heights in the range 2–6 m

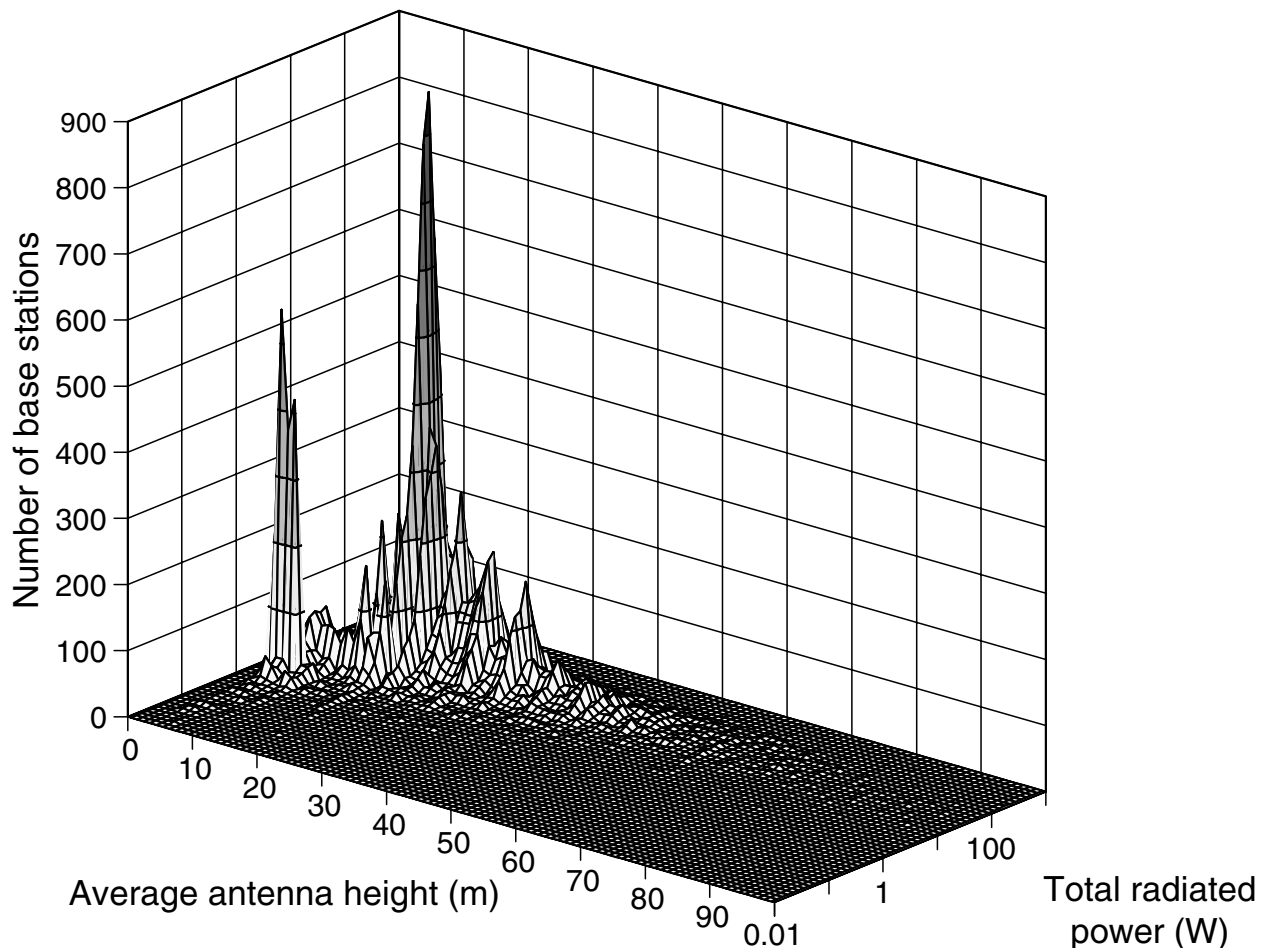
and powers of about 2 W. Cooper *et al.* concluded that the base stations in the first group are likely to serve macrocells and provide the main coverage for cellular networks, while those in the second group are likely to be microcells and provide a second layer of coverage, e.g. in densely populated areas.

Numerous spot measurements have been carried out to determine levels of exposure in the vicinity of mobile-phone base stations, often within national campaigns to address public concerns. Generally, these spot measurements take into account exposure contributions from all signals in the bands used by the base station at the time of measurement, but ignore other parts of the spectrum, such as those used by broadcast transmitters. [Mann \(2010\)](#) summarized the United Kingdom audit programme, which encompassed 3321 measurements at 541 sites comprising 339 schools, 37 hospitals and 165 other locations. Exposure quotients, describing the fraction of the ICNIRP general public reference level ([ICNIRP, 1998](#)) that is contributed collectively by the signals measured, are shown in Fig. 1.11 as a cumulative distribution.

Fig. 1.11 includes a log-normal curve fitted optimally (least squares) to the data. The curve suggests that the data are approximately log-normally distributed, although with a longer tail towards the lower values. The quotient values are 8.1×10^{-6} ($3.0 \times 10^{-8} - 2.5 \times 10^{-4}$), where the first figure is the median value and the values in parentheses indicate the range from the 5th to the 95th percentile. About 55% of the measurements were made outdoors and these were associated with higher exposure quotients than the indoor measurements. The median quotients for the outdoor and indoor measurements were 1.7×10^{-5} and 2.8×10^{-6} respectively, i.e. the outdoor median was around six times higher than the indoor median ([Mann, 2010](#)).

The exposure quotients may be converted to electric-field strengths or power densities by assuming a value for the reference level, but the

Fig. 1.10 Distribution of 32 837 base stations in the United Kingdom according to average antenna height and total radiated power



Antenna height is given as an average value since some base stations with multiple antennae have the antennae mounted at different heights.
From [Cooper et al. \(2006\)](#)

latter varies from 2 to 10 W/m² over the frequency range considered in the measurements (TETRA at 390 MHz to UMTS at 2170 MHz). The variation of the reference level is, however, very much less than the variation in the exposure quotients, so taking 4.5 W/m² as the reference level (the value at 900 MHz) still yields useful data. The power densities and electric-field strengths based on this assumed value are shown in [Table 1.6](#).

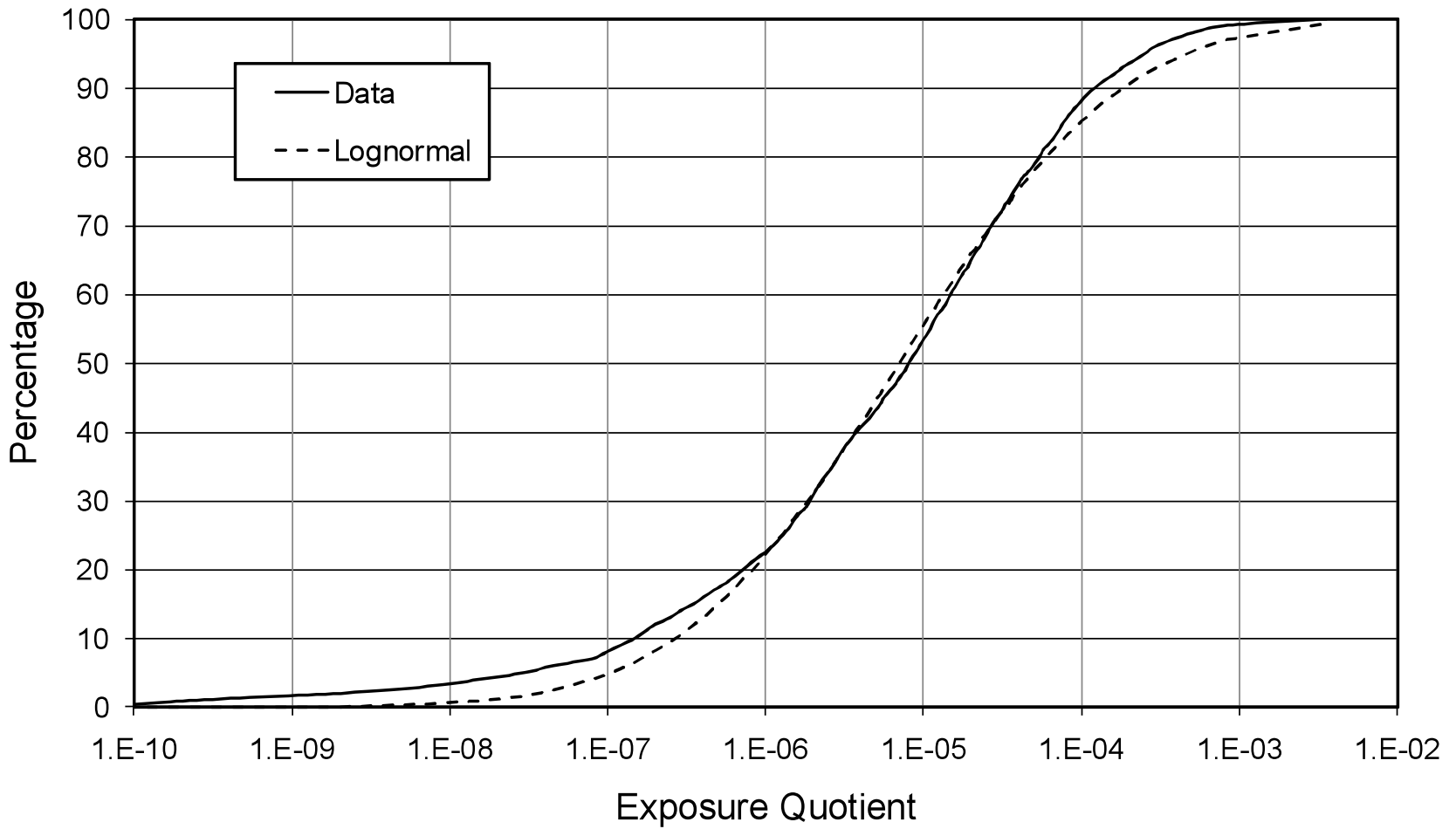
[Table 1.6](#) shows electric-field strengths that range from about ten to a few hundred millivolts per metre indoors, where people spend most of their time. However, in considering these data it

is important to recognize that the indoor sites in this study were selected according to public concern regarding a nearby base station; these field strengths may thus be higher than would be found at locations representative of exposure of the general population.

[Petersen & Testagrossa \(1992\)](#) published measurements of power densities around analogue base-station sites in the USA, transmitting in the frequency range 869–894 MHz. A basic start-up site would serve a cell with a range of up to 12–16 km and provide up to 16 signals (each serving one phone call) from a

58

Fig. 1.11 Cumulative distribution of exposure quotients corresponding to 3321 spot measurements made by Office of Communications at 499 sites where public concern had been expressed about nearby base stations



The exposure quotients were calculated by dividing the power density of each individually measured signal by the general public reference level at its frequency according to [ICNIRP \(1998\)](#) and then summing these individual signal quotients to obtain a total quotient of the reference level. The figure shows a log-normal curve fitted to the data. From [Mann \(2010\)](#). Copyright © 2010. Published by Elsevier Masson SAS on behalf of Académie des sciences. All rights reserved.

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Table 1.6 Summary of exposure quotients measured in the United Kingdom

Category	No. of measurements	Exposure quotient ($\times 10^{-6}$)		Power density ($\mu\text{W}/\text{m}^2$)		Electric-field strength (mV/m)	
		Median	Range ^a	Median	Range ^a	Median	Range ^a
All data	3321	8.1	0.03 – 250	37	0.13 – 1100	120	7.1 – 650
Outdoor	1809	17	0.052 – 314	77	0.23 – 1400	170	9.3 – 730
Indoor	1516	2.8	0.024 – 124	13	0.11 – 560	69	6.4 – 460

^a Range from 5th to 95th percentiles

These data are from an audit of base stations up to the end of 2007. Equivalent power densities and electric-field strengths are given assuming a reference level of $4.5 \text{ W}/\text{m}^2$.

Adapted from [Mann \(2010\)](#)

single omni-directional antenna. As demand grew, sites could be expanded to split cells into three sectors with up to six antennae mounted on a triangular mast head. Again, each antenna would provide up to 16 signals, so there would be a maximum of 96 signals available, 32 of which would have been directed into each sector. Values for nominal ERP (see Glossary) were about 100 W and so the radiated power would have been of the order of 10 W per signal from omni-directional and sectorised sites, with typical antenna gains in the range of 9–10 dB and 8–12 dB, respectively.

For four masts ranging from 46 to 82 m in height, measurements were made at intervals along radials from the bases of the masts out to distances of a few hundred metres. Individual signals from a given antenna were found to vary in strength at any given measurement position and the sidelobe structure of the antenna was evident in that the signal strength had an oscillatory dependence on distance. The maximum power density per signal was $< 100 \mu\text{W}/\text{m}^2$, except in proximity to metal structures near the foot of the tower. Thus, even for 96 signals transmitted simultaneously, the maximum aggregate power density possible would have been $< 10 \text{ mW}/\text{m}^2$.

[Henderson & Bangay \(2006\)](#) reported on a survey of exposures around 60 base station sites in Australia transmitting CDMA800 (29 sites), GSM900 (51 sites), GSM1800 (12 sites) and 3G UMTS (35 sites) signals. Initially, computer modelling was carried out to identify

the direction from the mast where maximum exposures were expected. Measurements were then made at distances of 50, 200 and 500 m, and further measurements were then made at the distance where maximum exposures were predicted, which varied from 14 to 480 m from the mast as a consequence of antenna height, pattern and tilt. The maximum recorded power density of $7.8 \text{ mW}/\text{m}^2$ corresponded to an exposure quotient of 0.002 (0.2%) relative to the ICNIRP public reference level (identical to the Australian standard at the frequencies concerned). The cumulative distributions also reported in this paper showed roughly similar median exposure quotients of about 0.0015 at 50 and 200 m, 0.0001 at 500 m and 0.004 at the maximum.

The study by [Cooper et al. \(2006\)](#) mentioned above focused on measurements around 20 GSM base stations with powers $< 5 \text{ W}$ and heights $< 10 \text{ m}$, selected randomly from all base stations in the United Kingdom. From the total of 32 837 base stations, 3008 eligible stations were identified. The antennae of the selected base stations were often fixed to the walls of buildings at a minimum height of 2.8 m. Theoretical calculations based on the radiated powers showed that the minimum height at which the reference level could be reached was 2.4 m above ground. Exposure measurements were made as a function of distance at 10 of the 20 sites and at 610 locations in total, ranging from 1 to 100 m from the antenna. The highest spot measurement at

an accessible location represented 8.6% of the reference level and the exposures more generally ranged from 0.002% to 2% of the ICNIRP public reference level. Empirical fits showed that the exposure quotients decreased in a way that was inversely proportional to the distance, for distances up to about 20 m from the antennae and thereafter diminished with the fourth power of distance. Exposures close to microcell base stations were found to be higher than close to macrocell base stations, because the antennae were at lower heights and could be approached more closely by the public.

[Kim & Park \(2010\)](#) made measurements at 50 locations between 32 and 422 m from CDMA800 and CDMA1800 base stations in the Republic of Korea. The base stations were selected to represent locations where concern had been expressed by the local population. The highest reported electric field level was 1.5 V/m, equivalent to an exposure quotient of 0.0015 (0.15%) compared with the reference level, and the median exposure quotient was below 0.0001 (0.01%).

The most recent studies have used personal-exposure meters worn for periods of up to several days by groups of volunteers. These studies are covered in Section 1.6.1, and provide information not only on exposure from base stations, but also from other environmental transmitters during typical activities.

(vi) *Terrestrial Trunked Radio (TETRA)*

TETRA is a cellular radio system designed to meet the needs of professional users and emergency services. The handsets can be used like mobile phones, but are normally used as walkie-talkies, held in front of the face and in push-to-talk (PTT) mode. Remote speaker microphones and a variety of covert add-ons are also available. When the handsets are used with accessories, the transmitting handset may be mounted on the belt, on the chest, or elsewhere on the body. Systems for use in vehicles with the transmitting antennae mounted externally are also available.

The operating principles and the detailed characteristics of the signals involved are described in a review by [AGNIR \(2001\)](#).

Several frequency bands are available between 380 and 470 MHz, as well as one set of bands near 900 MHz. Handsets can have peak emitted powers of 1 W or 3 W, while vehicle-mounted transmitters can have powers of 3 W or 10 W. Base stations have similar powers to those used for mobile-phone networks, i.e. a few tens of watts. The system uses TDMA, although the frame rate is slower than that of the TDMA systems involved with mobile phones. There are four slots per frame and 17.6 frames per second. Hence, the bursts from handsets occupy slots with a duration of 14.2 ms and the time-averaged power is a quarter of the peak powers mentioned earlier in this paragraph. The base stations transmit continuous signals [AGNIR \(2001\)](#).

The AGNIR review refers to SARs measured from 1 W and 3 W handsets held to either side of the head and in front of the face in a model of the head. With spatial averaging over 10 g, as per ICNIRP and IEEE exposure guidelines, the 1 W radio produced SARs of 0.88, 0.89 and 0.24 W/kg on the left, right and front of the face, respectively, while the 3 W radio produced SARs of 2.88, 2.33 and 0.53 W/kg, respectively, under the same conditions.

[Dimbylow *et al.* \(2003\)](#) developed a numerical model of a commercially available TETRA handset and calculated SARs in an anatomically realistic numerical model (resolution, 2 mm) of the head developed from MRI images. The handset was modelled as a metal box of dimensions 34 × 50 × 134 mm, and with either a helical (pitch, 4 mm; diameter, 8 mm) or a monopole antenna mounted on its top face, and resonant at 380 MHz. For the handset held vertically in front of the face in the position that was considered to be most representative of practical use, the averaged SARs at 10 g were 1.67 W/kg and 2.37 W/kg per watt of radiated power with the monopole and helical antennae, respectively. Various positions

were considered with the handset held to the sides of the head and the maximum SARs with the two antennae were 2.33 and 3.90 W/kg per watt. These values suggest SARs with 3 W handsets (3/4 W time-averaged) having a helical antenna could exceed the 2 W/kg restriction on exposure for the general public, if the handsets were to transmit at full power for 6 minutes while held to the side of the head.

(vii) Cordless phones

Cordless phones are used to make voice calls and are held against the head just like mobile phones. Hence, the antenna inside the phone is in close proximity to the head and its radiated fields deposit energy inside the head tissues near to the phone, in a similar way to the fields from mobile phones. With cordless phones, communications are made over shorter distances than with mobile phones and so the radiated powers used are lower, but cordless phones do not use adaptive power control, which means that, unlike mobile phones, they do not continually adapt their radiated power to the minimum necessary for satisfactory communication ([ETSI, 2010](#)).

With simple cordless installations, the phones are typically placed back on a desk or charging point after a call has finished. However, there are also more complicated installations in which multiple base stations are installed throughout a building and the phones are carried by the user as a personal phone. The radio communications are over distances of a few tens of metres and to the nearest base station, which provides the link into the main wired telephone system.

The first cordless phones used analogue technology and operated to a range of different technical standards, with continuous emitted power levels of about 10 mW during calls. Frequencies were generally in the range 30–50 MHz and therefore about 20 times lower than the frequencies used by mobile phones. Some phones used telescopic antennae of about 15–30 cm in length, while others used helical

antennae of about 5 cm in length. The lower frequencies and the greater size of the antennae used with analogue cordless phones would have resulted in a smaller proportion of the radiated power being absorbed, and also in a more diffuse pattern of absorption in the head than occurs with mobile phones ([ETSI, 2010](#)).

Modern cordless phones use digital technology, including the digital enhanced cordless telecommunications (DECT) technical standard, which operates in the frequency band 1880–1900 MHz and is the main system used in Europe. In other parts of the world, systems operating around 900, 2400 and 5800 MHz are used as well as DECT ([ETSI, 2010](#)).

DECT systems produce discontinuous emissions due to their use of TDMA. The signals from the phone and base station during calls are in the form of 100 bursts every second, each of about 0.4 ms in duration. These bursts are emitted at a peak power level of 250 mW, but the time-averaged power is 10 mW because each device only transmits for 1/24 of the time (duty factor of 4%). Handsets do not transmit unless calls are being made, but when on “standby” most base stations produce 100 beacon pulses per second, each pulse being 0.08 ms in duration. This implies a duty factor of 0.8% ([ETSI, 2010](#)).

(viii) Professional mobile radio systems

A variety of professional mobile radio systems, also called private mobile radio (PMR), have been developed over the years and these are generally licensed to professional users by spectrum-management agencies in the countries where they are used. In many countries, the emergency services (police, fire, ambulance, etc.) are converting to the use of digital cellular systems, such as TETRA, although analogue systems – which were the norm before roll-out of TETRA systems – are also used.

The PMR systems use frequencies in the VHF and UHF parts of the spectrum; VHF generally propagates further for a given radiated power

and is, therefore, preferred for longer-distance communications. On the other hand, UHF systems have smaller antennae and present as more compact terminals.

Systems exist in the form of walkie-talkies that are held in front of the face and used in push-to-talk (PTT) mode; they may be built into vehicles with external, e.g. roof-mounted, antennae or be worn on the body. The transmitting antennae can be on the handset itself, on the vehicle, or carried on the chest or waist. The radiated powers are typically in the range 1–5 W, but it is important to take into account the duty factor associated with how they are used: the PTT mode will involve only a few seconds of transmission during the time that the button is pressed down and the user is speaking.

(c) *Wireless networks*

Wireless networking has developed rapidly since about 2000 and is becoming the method of choice for connecting mobile devices such as laptop computers and mobile phones to other electronic systems and to the Internet. The networks are found in homes, schools, public places such as cafés and transport hubs, and in the workplace. The systems operate to the IEEE802.11 family of technical standards and are often known as “Wi-Fi,” after the Wi-Fi Alliance, an organization that certifies inter-operability of devices on the market.

The original version of IEEE802.11 was published in 1997 and provided for data-transfer rates of up to 2 Mbit/s through frequency channels between 2.4 and 2.5 GHz. Subsequent developments using this band were IEEE802.11b and IEEE802.11g, allowing for rates up to 11 and 54 Mbit/s, respectively. Several frequency bands between 5 and 6 GHz are exploited by IEEE802.11a and provide for 54 Mbit/s communications. The latest devices operate according to IEEE802.11n and provide up to 72 Mbit/s in a single frequency channel, but the standard allows for devices that can use multiple frequency

channels simultaneously to deliver much higher data rates ([ICNIRP, 2009a](#)).

The IEEE802.11 standard specifies maximum radiated powers, but these are above the values permitted by regulatory agencies in many parts of the world. For example, in Europe the technical standards EN300328 and EN301893 limit the EIRP to 100 mW in the 2.4-GHz band and 200 mW in the 5 GHz band, respectively. [Peyman et al. \(2011\)](#) measured the actual power radiated by a selection of Wi-Fi devices marketed among schools in the United Kingdom. The spherically integrated radiated power (IRP) ranged from 5 to 17 mW for fifteen laptops in the 2.45 GHz band and from 1 to 16 mW for eight laptops in the 5 GHz band. For practical reasons and because access points are generally wall-mounted with beams directed into the room, their powers were integrated over a hemisphere. These ranged from 3 to 28 mW for twelve access points at 2.4 GHz and from 3 to 29 mW for six access points at 5 GHz. Thus the radiated powers of laptops seem to range from a few mW up to about 30 mW. In principle, these measurements imply that the powers of access points could range from a few mW up to around 60 mW, if their patterns extend symmetrically into the unmeasured hemisphere, which seems unlikely.

The RF emissions from Wi-Fi devices are in the form of short bursts containing portions of the data being transmitted and other information, such as acknowledgements that data have been successfully received. Unlike the emissions from mobile phones using TDMA, the bursts are irregular in terms of timing and duration. Typical bursts range from about 10 μ s to about 1 ms in duration. If data are lost or corrupted during transmission, bursts are retransmitted until they are successfully received. Also, under conditions where communications are poor, e.g. due to weak signal strength, the systems can lower their data-transfer rates to have better signal-to-noise ratios and improved reliability. This increases the cumulative time that it takes to transmit a given

amount of data. Thus, high signal strengths from Wi-Fi devices (during transmission of bursts) do not necessarily translate to higher exposures, because this results in lower duty factors ([Mann, 2010](#)).

Comprehensive data are yet to be published regarding the duty factors of Wi-Fi equipment during normal use; however, [Khalid et al. \(2011\)](#) has reported initial results from the use of data-traffic capturing and packet-counting equipment in school networks. Transmitted bursts were captured to determine the proportion of time during which Wi-Fi devices transmitted while children were using laptops during their lessons. The laptops were mostly used for receiving traffic from the access points and therefore laptop-transmit times were low. Duty factors for the monitored laptops were consistently less than 1% and those of access points were less than 10%. Baseline duty factors of access points (with no data being transferred) are about 1%, due to beacon pulses of duration 1 ms that are produced at a rate of ten pulses per second ([Mann, 2010](#)).

The SAR values produced when using laptop computers equipped with Wi-Fi transmitters have been evaluated by several authors. Most devices now have built-in antennae located around and along the top edge of the screen, which are therefore at greater distances from the body than a mobile phone held against the head. The rapid reduction in field strength that occurs with increasing distance means that SARs can be expected to be much lower than from mobile phones under such scenarios. Based on a continuous radiated power of 100 mW under a range of such scenarios, [Findlay & Dimbylow \(2010\)](#) calculated a maximum 10 g averaged SAR of 5.7 mW/kg in the head.

When Wi-Fi devices are able to transmit continuously with their antennae in close proximity to the body, the SARs may be higher than in the scenario described above. For example, [Kühn et al. \(2007a\)](#) measured a SAR of 0.81 W/kg in a flat phantom with the antennae of a Wi-Fi

access point in close proximity and [Schmid et al. \(2007b\)](#) measured a SAR of 0.05 W/kg under similar conditions from a Wi-Fi equipped PCI card inserted into a laptop. The value reported by Kühn et al. is within the range of maximum localized SARs from mobile phones ([ICNIRP, 1998](#)).

Studies have also examined the general field strengths in environments where Wi-Fi networks are installed. [Foster \(2007\)](#) measured RF fields at 55 public and private sites in the USA and Europe (4 countries), which included private residences, commercial spaces, and health-care and educational institutions. In nearly all cases, the measured Wi-Fi signal levels were far lower than other RF signals in the same environment. The maximum time-averaged power density in the 2.4-GHz band measured at 1 m distance from a laptop uploading and downloading a file was 7 mW/m², which is far less than the [ICNIRP \(1998\)](#) reference level value of 10 W/m² for the general public.

[Schmid et al. \(2007a\)](#) investigated the typical exposure caused by wireless local area network (WLAN) applications in small and large indoor public areas (e.g. Internet cafés, airports). Outdoor scenarios were also considered where the exposure was measured in the vicinity of access points serving residential areas and public places. Exposure was assessed by computational methods and by on-site measurements. The highest values for indoor exposure were found close to the transmitting devices (access points or clients) where, at a distance of about 20 cm, spatial and temporal peak values of power density were found to reach about 100–200 mW/m². In general, the exposure values were several orders of magnitude below the [ICNIRP \(1998\)](#) reference levels.

(d) Industrial applications

There are several industrial applications for RF-EMF, many of which are described in review reports and papers. On the whole, the

literature is rather old and difficult to interpret since reported field values have generally been taken in the context of compliance assessments rather than epidemiological studies, so it is hard to judge what the typical exposures of workers may have been. Only a brief description of some of the sources producing the highest exposures is included here.

(i) *Industrial induction heating*

Industrial induction heating involves the use of induction furnaces equipped with large coils that produce strong magnetic fields. Conducting materials for treatment are placed inside the coils and the magnetic fields cause eddy currents, resulting in heating of the conducting materials. Typical applications include surface hardening, softening and melting metals, mixing alloys and heating gaseous conductors such as plasmas. The frequencies used span a wide range, from 50 Hz through to a few megahertz, so not all applications fall within the scope of this *Monograph*. The fields can be considerable and worker exposures are greatest for tasks that involve approaching the coils, e.g. when taking samples from within the coils of open furnaces. The coil impedances increase with frequency and electric fields can become the dominant contributor to exposure (rather than magnetic fields) at frequencies above about 100 kHz ([ICNIRP, 2009a](#)). [Allen et al. \(1994\)](#) have provided a review of measured exposures, drawing on peer-reviewed papers from several countries and measurements made in the United Kingdom.

(ii) *Dielectric heating*

RF heating and drying equipment has been used for many years and applications include pre-heating, wood-glueing and polyvinyl chloride (PVC) welding. These materials are lossy dielectrics and their conductivity at radiofrequencies means that they can become heated-up when placed in a strong electric field. Typical heaters are designed to use the industrial, scientific and

medical (ISM) bands at 13.56, 27.12 and 40.68 MHz, but reported measurements show that frequencies are variable within the range 10–80 MHz. Powers range from less than a kilowatt to tens of kilowatts for typical heat sealers, while for glue-dryers the maximum power may exceed 100 kW ([ICNIRP, 2009a](#)).

The greatest source of operator exposure comes from the use of manually actuated PVC dielectric machines, where the operator manipulates material to be welded by hand and then clamps it between a pair of electrodes between which the power is applied. Measurements and other details from studies carried out in the United Kingdom and elsewhere are described by [Allen et al. \(1994\)](#). The field strengths from dielectric heaters at the operator locations can be in excess of the [ICNIRP \(1998\)](#) reference levels, but they are non-uniform and it is necessary to evaluate the SAR in the body to determine compliance with the guidelines. [Kännälä et al. \(2008\)](#) have developed an assessment method based on measuring induced limb currents and relating these to localized and whole-body SARs (wbSARs).

(e) *Medical applications*

RF fields have several medical applications. In general, exposure for the clinician will be lower than for the patient, since the RF source will generally be located closer to the patient, but this is not always the case. RF fields can also be applied for therapeutic purposes, for moderate heating of tissue, or for much greater heating for the cutting and destruction of tissue during surgery.

(i) *Magnetic resonance imaging (MRI)*

Performing an MRI scan for diagnostic purposes involves strong RF fields. MRI uses a combination of EMFs to produce exceptionally clear images of tissue structures inside the human body, to assist with medical diagnoses. Hydrogen atoms associated with water in the

body tissues are made to resonate in a strong magnetic field such that they emit RF radiation at the resonant frequency. Therefore, variations in the water content of tissues are the basis of the contrast in the images obtained ([HPA, 2008](#)).

A permanent uniform static magnetic field, typically in the range 1–3 T, but sometimes up to 8 T or more with specialized systems, is applied over the body and causes splitting of the energy states associated with protons (hydrogen atoms). The difference between the energy states is such that protons will transfer from the lower to the upper energy state in response to an applied RF signal at the resonant frequency. Protons will also fall back to the lower energy state spontaneously, and in doing so emit RF radiation at the Larmor frequency. The Larmor frequency is given by 42.57 times the static magnetic-field strength. Thus a 1.5 T, an MRI scan involves the application and measurement of RF fields at 64 MHz ([HPA, 2008](#)).

During an MRI scan, multiple RF pulses (hundreds to thousands per second) are applied over either the whole body or the part of the body being visualized. The RF dose (SAR) received by patients inside the MRI scanners is reported by the system and can vary from < 0.1 W/kg to about 4 W/kg for more complex settings ([HPA, 2008](#)). The desire to limit temperature increases and prevent harm to the patient can be a limiting factor in how quickly scans can be performed in practice. Clinicians and any other personnel who are near to the magnet during the scans will be exposed to the RF fields, but the strength of the RF fields will diminish rapidly with increasing distance from the RF coils and the space between them inside the scanner.

(ii) Diathermy

Short-wave and microwave diathermy are used to gently warm muscles, tendons and joints to alleviate a variety of medical conditions. Short-wave equipment operates at frequencies of 13.56 MHz or 27.12 MHz and powers of about

400 W. Applicators for microwave diathermy operate at 2.45 GHz with powers of about 200 W and tend to take the form of a radiating antenna surrounded by reflectors that direct the emitted energy in a forward direction. While exposure of the patient is intentional, the scanner operators close to the equipment may be exposed involuntarily in areas where field strengths are high, unless they move away while the equipment is in operation ([ICNIRP, 2009a](#)).

(iii) Surgical diathermy and ablation by radiofrequency

RF fields and currents are widely used during surgical procedures. In surgical diathermy or electrosurgery, a small hand-held electrode acts as a cutting or coagulation instrument. The basic operating frequency is typically about 500 kHz and there are harmonics produced at frequencies up to around 20 MHz. Current densities in tissues can be as high as 10 A/cm² with source powers of up to 200 W ([IPEM, 2010](#)). Some more recent systems use a frequency of 9.2 GHz and powers of about 20 W delivered through needle-like electrodes containing coaxial lines. These systems are employed for minimally invasive surgery, e.g. focal tumour ablation and the treatment of menorrhagia by endometrial ablation ([IPEM, 2010](#)).

(f) Domestic sources

There are few powerful sources of RF in the home; however, among these, induction cooking hobs and microwave ovens are of note. Less powerful sources include remote-controlled toys, baby monitors, and the mobile/cordless phones and the Wi-Fi systems described earlier.

Induction cooking hobs feature coils that produce a magnetic field beneath the metal cooking pans that are placed on them. The magnetic fields produce eddy currents in the pans, which are thereby heated. The powers transferred to the pans can be several kilowatts and the frequencies involved are in the range

20–50 kHz. Magnetic fields can be in the order of the ICNIRP reference levels, but vary greatly with user position and also depend on the placement of the pan. [ICNIRP \(2009a\)](#) reviews studies that have investigated these exposures.

Microwave ovens are standard fixtures in many homes and contain microwave sources operating at a frequency of 2.45 GHz and producing powers between 500 W and 2 kW. The design of such ovens is such that leakage is kept to a minimum and a product-performance technical standard requires that microwave-power density levels fall below 50 W/m² at a distance of 5 cm. Several large surveys of leakage levels have been performed, as described in [ICNIRP \(2009a\)](#), and these indicate that approximately 99% of ovens comply with the emission limit. According to the measurements of [Bangay & Zombolas \(2003\)](#), the maximum local SAR values at the emission limit are 0.256 W/kg and the maximum 10 g averaged SAR is 0.0056 W/kg.

A new source of RF that is currently being introduced and that seems set to enter many homes is the transmitter associated with “smart” metering of electricity consumption and potentially metering for other services such as water and gas. There is no global approach to gathering information from smart meters and relaying it back to the utility companies, but it is clear that radio communications will be involved. Some systems may use mobile-phone networks for this purpose, while others may use dedicated radio infrastructures. Some systems may also involve a home area network (HAN) within which individual electrical devices in the home can relay information about usage to a central collection point, allowing residents to examine the information and make decisions about their energy consumption. Two recent investigations commissioned by the Electric Power Research Institute (available on the EPRI webpage) suggest that the power level of radio transmissions will be similar to that of mobile phones, but that the duty factors will be low (on average, such devices

will transmit for a small proportion of time only). Low duty factors, combined with the greater distances of these devices from people compared with mobile phones, imply that exposures will be low when compared with exposure guidelines.

(g) Security and safety applications, including radar and navigation

A variety of systems used for security purposes involve the application of RF, including systems for asset tracking and identification. These sources and exposures have been reviewed in [ICNIRP \(2009a\)](#).

Radar systems operate across a broad range of frequencies, mostly in the range 1–10 GHz, with some short-range applications in the range of tens of gigahertz. Emissions from these systems represent an extreme form of pulse modulation, the TDMA scheme used by some mobile phones being a less extreme example. The duty factor in a GSM TDMA signal is 1/8, whereas it is typically around 1/1000 with a radar signal. The typical duration of a pulse might be about a microsecond, while a typical pulse period might be about a millisecond, although these parameters do vary and depend on the type of radar involved. Very high power densities can be produced in the antenna beams during the pulses, and powers can still be high after duty factors are taken into account to determine the average power. To assess human exposure from radar systems it is necessary to take into account:

- The exposure metric of interest (to account for the pulsing, or simply based on the average power);
- People’s juxtaposition to the beams (are the beams going over people’s heads?);
- The duty factor associated with the pulsing;
- The duty factor associated with rotation (equal to the beam width in azimuth divided by 60 degrees; probably around 200 : 1 in the direction that a rotating beam sweeps through).

Information about radar systems can be found in the following review reports: [Allen et al. \(1994\)](#), [Cooper \(2002\)](#) and [ICNIRP \(2009a\)](#).

(i) Air traffic control

The most familiar application of radar is for navigation and the tracking of aircraft movements from rotating ground-based antennae, e.g. at airports. Long-range systems operate over 1–2 GHz, while moderate-range systems operate over 2–4 GHz. The antennae tend to be mounted sufficiently high that buildings cannot obstruct their view of the sky and they form narrow beams of about a degree in the horizontal plane that sweep around 360 degrees once every few seconds. Beams are broader in the vertical plane and tail off in strength towards low elevation angles to avoid reflections from objects on the ground. Aviation radar systems have quite high emitted power levels during the pulses, typically from tens of kilowatts to a few megawatts. Taking the duty factors into account leads to time-averaged emitted powers of about 100 W to a few kilowatts ([AGNIR, 2003](#)).

(ii) Marine radar

Marine radar systems are used to inform the crew of a ship of the presence of other vessels and thus avoid collisions. The range of these systems is shorter than that of aviation systems. It is known that targets will be at ground (sea) level, so the beam profile extends to ground level in the plane of elevation. The rotating antennae are mounted at height to allow a view of the sea that is unobstructed by the structure of the ship/vessel on which they are carried. Operating frequencies are in the ranges of 2–4 or 8–12 GHz. Mean powers are in the range 1–25 W and peak powers can be up to about 30 kW ([ICNIRP, 2009a](#)).

(iii) Tracking radar

Tracking radar is used in military systems to lock-on to and follow targets such as aircraft and missiles. The antennae can rotate, execute a nodding motion, point in a fixed direction, or

follow a target. Targets are not expected to assist with being tracked and may even be designed with stealth in mind and to suppress the extent to which they reflect radar pulses. Hence, tracking radar systems generally involve higher powers than navigation systems and use peak powers of up to several megawatts. Systems mostly operate between 2 and 8 GHz. Certain tracking radar systems can produce mean power densities $> 100 \text{ W/m}^2$ at distances in excess of a kilometre, even after duty-cycle correction ([ICNIRP, 2009a](#)).

(iv) Whole-body security scanners

Whole-body security scanners are used in places such as airports to generate images of objects carried under people's clothing without the need for physical contact. Active systems transmit either ionizing (X-rays) or non-ionizing (RF) radiation towards the body and then analyse the scattered radiation. Passive systems simply monitor the "black body" (thermal) radiation given off by the body in the RF spectrum and do not emit any radiation. Current active RF systems typically operate at about 30 GHz, although in the future systems may use frequencies of up to several hundred gigahertz. ([European Commission, 2010](#)). A note published by [AFSSET \(2010\)](#) described an assessment of an active scanner operating in the frequency range 24–30 GHz. Power densities incident on the body were reported as between 60 and 640 $\mu\text{W/m}^2$.

(v) Other systems

Various other radar systems include those used for monitoring weather, traffic speed, collision avoidance with vehicles and ground penetration.

1.3 Dosimetry

1.3.1 Introduction

Incident EMFs are defined as external fields in the absence of – i.e. without interaction with – the human body, animals, or tissue samples. Incident fields couple with the human body and induce EMFs and currents inside the body tissues.

Macrodosimetry is the science of quantifying the three-dimensional distribution of EMFs inside tissues and organs of biological bodies, with averaged induced fields across submillimetre tissue structures (e.g. cells). The term is also applied to measurements in media that have dielectric characteristics similar to those of biological bodies, e.g. cell cultures, tissue-simulating media, etc. The induced fields are the only exposure parameters that can interact with biological processes and, therefore, provide the primary exposure metric ([Kühn, 2009](#)).

Microdosimetry refers to the assessment of fields at subcellular resolution (e.g. across membranes, proteins, etc.). This is a relatively new research area that faces various basic problems, such as material models and transitions between classical and quantum electrodynamics. In all cases, however, macrodosimetry is the first step, since microdosimetry can only be developed from the locally averaged induced fields. This *Monograph* does not cover microdosimetry, and “dosimetry” used hereafter thus refers to macrodosimetry. Dosimetry studies of differences in dielectric properties of tissues in human and animals models published since 1984 are described in [Table 1.7](#).

The coupling mechanisms of the electric and magnetic incident-field components are different. Hence, both must be determined separately to fully characterize human exposure. Since coupling with the human body also depends on the ratio of wavelength versus body size, the RF-EMF spectrum is often divided

into at least three ranges, e.g. 30 kHz–10 MHz (below body resonance); 10 MHz to 2 GHz (body and partial body resonances); and 2 GHz to 300 GHz (surface-dominated absorption) ([ICNIRP, 2009a](#)). Furthermore, the distribution of the induced field strongly depends on various parameters, such as source (strength, frequency, polarization, direction of incidence, size, shape, etc.), distance and location of the source with respect to the body, outer anatomy, inner anatomy, body posture, and environment of the body (e.g. reflective objects).

The field variations within the body are generally large and may well exceed a factor of thousand for the locally absorbed energy. In general, field distributions change considerably between different postures and orientations of the body with respect to the field. For example, the exposure of the brain may change even though the whole-body average and the peak spatial absorption remain the same.

1.3.2 Dosimetric exposure

It has only recently become technically possible to achieve a detailed characterization of exposure to EMFs. Hence, research on dosimetry during the past 30 years has been focused on reliable determination of the exposure metric as defined in the safety guidelines, namely, the maximum average whole-body values and the maximum locally-induced field values. The most commonly used metrics are defined below.

At frequencies greater than 100 kHz, SAR is the main measure of exposure used. SAR is the absorbed electromagnetic energy per tissue mass and can be calculated directly from the electric energy loss, which is proportional to the square of the locally induced root-mean-square value (rms) of the electric field strength, the induced current density and the temperature increase (see Glossary for detailed equations). The assessment on the basis of the initial rise in temperature is only valid if the exposed body is in thermal

Table 1.7 Dosimetry studies of differences in dielectric properties of tissues

Reference	Description of the model	Main results and comments
Thurai et al. (1984)	Variation of dielectric properties of brain tissue of the mouse	Measurements were made on the cerebral cortex at frequencies of 10 MHz to 5 GHz in six groups of mice aged 3, 5, 19, 26, 33 or 58 days. Values of relative permittivity and conductivity are shown.
Thurai et al. (1985)	Dielectric properties of the developing rabbit brain	The dielectric properties of developing rabbit brain were measured at 37 °C, at frequencies between 10 MHz and 18 GHz, with time-domain and frequency-domain systems. Water dispersion in the brain becomes more complex with age.
Kuster & Balzano (1992)	Mechanism of energy absorption by biological bodies in the near field	Heterogeneous tissues and larger biological bodies of arbitrary shape are generalized for frequencies above 300 MHz. The SAR is found to be mainly proportional to the square of the incident H-field, which implies that in the close near field, the psSAR is related to the antenna current and not to the input power.
Lu et al. (1994)	Dielectric properties of human erythrocytes at radiofrequency	Dielectric properties of human erythrocytes in suspension (haematocrit, 50%) from 243 healthy persons (120 men, 123 women) were measured at 25 °C, at frequencies of 1–500 MHz, with a coaxial transmission line-reflection method (one-side measurement). A statistically significant age-dependence was found, with a critical age of about 50 yr, above which permittivity and conductivity of human erythrocytes in suspension decreased significantly.
Peyman et al. (2001)	Variation of dielectric properties of rat tissues by age, at microwave frequencies	The dielectric properties of tissues from rats of six different age-groups were measured at 37 °C in the frequency range 130 MHz to 10 GHz, with an open-ended coaxial probe. The percentage decrease in the dielectric properties of certain tissues in rats aged 30–70 days at mobile-phone frequencies was tabulated. These data contribute to rigorous dosimetry in lifetime-exposure animal experiments, and provide insight into possible differences in assessment of exposure for children and adults.
Peyman & Gabriel (2002)	Variation of dielectric properties of biological tissue, by age	Dielectric properties of the bone marrow generally decrease with age, due to changes in water content.
Jaspard et al. (2003)	Dielectric properties of blood, by haematocrit value	Two dielectric parameters appeared to be strongly dependent on the haematocrit value. The permittivity vs frequency decreases then increases when the haematocrit decreases. The conductivity increases in the whole frequency range when the haematocrit decreases.
Schmid et al. (2003)	Pre- and post-mortem dielectric properties of porcine brain tissue	Conductivity declined 15% (at 900 MHz) and 11% (at 1800 MHz) within 1 h after death. The decline in permittivity was 3–4%, and almost frequency-independent. In-vitro measurements of dielectric properties of brain tissue underestimate conductivity and permittivity of living tissue. These findings may affect generally accepted data on dielectric properties of brain tissue widely used in RF dosimetry.
Gabriel (2005)	Variation of dielectric properties of rat tissues, by age	Age-related dielectric data for 9 of 34 rat tissues were incorporated in a numerical dosimetry study on anatomically heterogeneous animals with body sizes corresponding to the ages of 10, 30, and 70 days, exposed to plane waves at spot frequencies from 27 to 2000 MHz. The variation in the dielectric properties affect the wBSAR by < 5%; the most conservative value (highest SAR) is obtained when 70-days properties are used. The dielectric properties of whole brain, skin, and skull were determined experimentally in the frequency range 300 KHz to 300 MHz.

Table 1.7 (continued)

Reference	Description of the model	Main results and comments
Lee et al. (2006)	Development of a body model for the Korean adult male	The dimensions of the human body vary by age, sex, and race. The internal structure and outer dimensions of a body exposed to an electromagnetic field are important for accurate dosimetry. Two volunteers with body dimensions representative of the average Korean adult male were recruited and scanned for phantom development by use of magnetic resonance and computed tomography. About 30 different tissues were manually classified by an anatomist on the raw images. The whole-body phantom can be used for radiation protection dosimetry.
Christ et al. (2006)	Electromagnetic near-field absorption in layered biological tissue in the frequency range 30–6000 MHz	The increase in SAR depends mainly on the thickness of the fat tissue and the frequency. For frequencies between 236 MHz and 5.8 GHz, the peak spatial average SAR can increase by a factor between 1.6 and 3.5 compared with homogeneous tissue-simulating liquid. In the near-field zone, reactive E-field components give rise to increased peak spatial averaged SAR, due to high absorption in the skin
Peyman & Gabriel (2007)	Development and characterization of tissue-equivalent liquids	Dielectric properties of two tissue-equivalent liquids were measured in the frequency range 30–3000 MHz. A sucrose-based solution had a permittivity of 61.3 ± 1.0 and a conductivity of 0.63 ± 0.02 S/m at 30 MHz. An aqueous diacetone solution had a permittivity of 54.2 ± 1.2 and a conductivity of 0.75 ± 0.01 S/m at 30 MHz. At 150 and 300 MHz, the two liquids met the specified target to within 5% and 10%, respectively.
Peyman et al. (2007)	Dielectric properties of porcine cerebrospinal tissues <i>in vivo</i> , <i>in vitro</i> , and by systematic variation of age	Dielectric properties of pig cerebrospinal tissue were measured <i>in vivo</i> and <i>in vitro</i> , in the frequency range of 50 MHz to 20 GHz. The study <i>in vivo</i> included tissues from pigs of different ages, weighing about 10, 50 and 250 kg. Dielectric properties of white matter and spinal chord, but not grey matter, showed significant variation with age.
Lee et al. (2009)	Development of a body model for a Korean child aged 7 yr	A whole-body voxel model of a 7-yr-old male volunteer was developed from 384 axial MRIs. The model was adjusted to the physical average of Korean boys aged 7 yr. The body weight of the adjusted model, calculated with the mass-tissue densities, is within 6% of the 50th percentile weight.
Peyman et al. (2009)	Dielectric properties of tissues and SAR in children exposed to walkie-talkie devices	Dielectric properties of porcine tissues <i>in vitro</i> – measured from 50 MHz to 20 GHz – show significant reduction with age. Both permittivity and conductivity decreased in 10 out of 15 tissues measured, mainly due to reduction in the water content of tissues in the ageing animal. The results were then used to calculate the SAR values in children aged 3–7 yr exposed to RF induced by walkie-talkie devices. No significant differences between the SAR values for the children of either age or for adults were observed.

MRI, magnetic resonance imaging; RF, radiofrequency; SAR, specific absorption rate; wk, week or weeks; yr, year or years

equilibrium or in a steady thermal state at the beginning of the exposure.

The SARs usually reported are values averaged over time, either over the periodicity of the signal or over any period of 6 minutes. Two metrics are most often determined:

- The whole-body-averaged SAR (wbSAR) is the total electromagnetic power absorbed by a body divided by its mass.
- The maximum peak spatial SAR (psSAR) averaged over any cube inside the body with a tissue mass of 1 g (psSAR-1 g) or 10 g (psSAR-10 g). Specific evaluation rules have been defined in which the cube is grown around the observation point, whereas special rules apply in case of air interfaces (see [ANSI/IEEE, 2002a](#)). This value is usually reported independently of the exposed tissue.

In recent years, the focus has shifted towards more tissue-specific measures of exposure that can be correlated with biological effects ([Kuster et al., 2006](#); [Boutry et al., 2008](#)). Examples are:

- Instant, time-averaged or cumulative organ- and tissue-specific SAR;
- Distributions and histograms of the spatially averaged SAR (sSAR) values over a mass of 1 g or 10 g of tissue in the shape of a cube (sSAR-1 g or sSAR-10 g) or 10 g of contiguous tissue (sSAR-10 g c) (see also [Ebert, 2009](#)).

At frequencies below 10 MHz, the following quantities are used:

- Current density averaged over any 1 cm² of tissue from the central nervous system (CNS) perpendicular to the current direction ([ICNIRP, 1998](#));
- Electric field integrated over any line segment of 5 mm in length oriented in any direction within the tissue ([IEEE, 2005](#));
- Electric field averaged in any 2 × 2 × 2 mm³ volume ([ICNIRP, 2010](#)).

1.3.3 Coupling of incident fields with the body

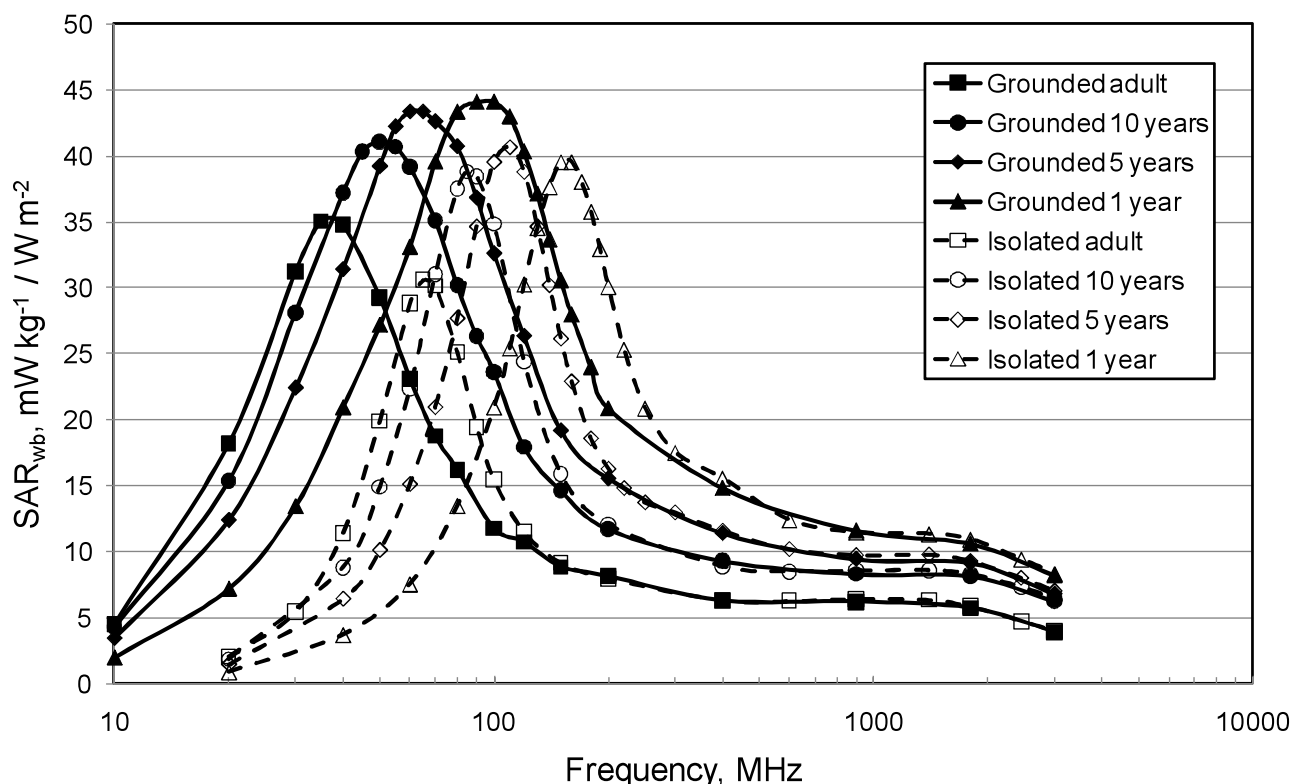
(a) Body-mounted devices

For transmitters operating at frequencies greater than 300 MHz, the absorption in proximate human tissue is approximately proportional to the square of the incident magnetic field (H_{inc}) at the skin surface of the person exposed ([Kuster & Balzano, 1992](#)). H_{inc} is approximately given by the square of the equivalent RF current in the device (I_{RF}) divided by its distance from the human body (d).

The equations presented by these authors explain many aspects of human exposure to radiation from mobile phones discussed in this *Monograph*, namely:

- Mobile phones close to the body ($d < 0.01$ m) are the dominant source of exposure, particularly of the brain, when the phone is held at the ear, compared with exposure from the more powerful base stations at larger distances ($d > 10$ m).
- Exposure from a mobile phone operated by a bystander ($d < 1$ m) may still exceed the exposure from a base station at moderate distance.
- The absorption of energy by different tissues is strongly dependent on the design of the phone, and may vary more than 20-fold according to, e.g. the location of the antenna, and the current distribution with respect to the tissue ([Kuster et al., 2004](#)).
- The level of local exposure is also relatively strongly dependent on the position of the phone at the head, and may vary by a factor of more than 10 ([Wiert et al., 2007](#); [Gosselin et al., 2011](#)).
- The exposure of children is higher than that of adults by a factor of approximately two due to the different shape of children's heads, which brings the phone geometrically closer to the brain in children

Fig. 1.12 Variation in the whole-body specific absorption rate (SAR) produced per unit power density as a function of frequency in the adult male phantom NORMAN, and child phantoms of three different ages, standing on a conductive floor (grounded) and insulated



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than in adults (see Section 1.6.1 (ii); [Wiart et al., 2008](#)).

- Hand-free kits reduce the psSAR in head tissue by a factor of 100 and more ([Porter et al., 2005](#); [Kühn et al., 2009b](#); also see Section 1.2.2).
- Bluetooth headsets operate at 1 mW and the maximum psSAR is a factor of about 100 lower than that for a mobile phone operating at the ear ([Kühn et al., 2007a](#)).
- WLAN transmitters in a laptop computer also result in lower exposures to the brain than a mobile phone operated at the ear.
- Exposures from DECT base-station antennae located in the same room as the person are similar to those from mobile-phone base stations in the neighbourhood ([Kühn et al., 2007a](#)).

(b) Whole-body and partial-body resonances

The human body can be described as an elongated poor conductor. Therefore, it couples energy best if the electric field is polarized along the long body axis and when the electrical length of the body is resonant, i.e. approximately half a wavelength ($\lambda/2$) for an ungrounded body and one quarter wavelength ($\lambda/4$) for a person standing on a grounded floor. This was first investigated with ellipsoids and recently refined with newly available human models (e.g. [Dimbylow, 2007a](#); [Conil et al., 2008](#); [Kühn et al., 2009b](#)). The typical variation in wbSAR as a function of frequency is shown in Fig. 1.12. The same effects have been investigated for partial-body resonances ([Kühn et al., 2009b](#)). The results of these modelling studies explain the main characteristics

Table 1.8 Depth of penetration of muscle and fat by radiofrequency fields at typical telecommunication frequencies

Frequency (MHz)	Muscle			Fat		
	Relative permittivity	Conductivity (S/m)	Penetration depth (mm)	Relative permittivity	Conductivity (S/m)	Penetration depth ^a (mm)
400	57.13	0.80	52	5.58	0.041	310
900	55.03	0.94	42	5.46	0.051	244
1800	53.55	1.34	29	5.35	0.078	158
2450	52.73	1.74	22	5.28	0.105	116
5200	49.28	4.27	8.8	5.01	0.255	47

^a Penetration depths have been calculated based on the equation given in the Glossary.

MHz, megahertz; mm, millimetre; S/m, siemens per metre

Compiled by the Working Group from *Tissue Properties Database: Dielectric Properties* by IT'IS Foundation: <http://www.itis.ethz.ch/itis-for-health/tissue-properties/database/dielectric-properties/>

of far-field exposures of between 10 MHz and 2 GHz, i.e. a strong dependence on body size and posture, and on polarization.

(c) Below whole-body and partial-body resonances

At exposures below the body-resonance frequency, i.e. < 10 MHz, the body can be described as a short poor conductor. The dominant exposures of concern are from near-field sources that generally have strong field gradients. Under these conditions, the energy is capacitively coupled in the case of a dominant electric-field source (dielectric heaters, diathermy applicators, etc.) or inductively coupled in the case of a dominant magnetic-field source (e.g. inductive cooking hobs, anti-theft systems, wireless power transfer systems, MRI, etc.). Strong induced currents are also caused by touching metallic objects such as fences or towers exposed to fields from transmitting antennae (contact currents).

(d) Above whole-body and partial-body resonances

At exposures above the body-resonance frequency, i.e. > 2 GHz, the body can be described as a dielectric object that is large with respect to the wavelength and the penetration depth (see [Table 1.8](#)). Therefore, the absorption

is approximately proportional to the exposed surface area of the body ([Gosselin et al., 2011](#)). In this case, the wbSAR is proportional to the largest ratio of body surface and weight ([Kühn, 2009](#)), whereas the RF energy is predominantly absorbed at the body surface.

1.3.4 Dependence on local anatomy

(a) General

Local exposure is altered by local anatomy due to inhomogeneity of the body tissues. In particular, local enhancements or hot spots can be expected as a result of impedance matching on layered structures, e.g. skin–fat–muscle layers ([Christ et al., 2006](#)), and due to narrowing cross-sections of highly conductive tissues. An example of the latter is high exposure in the ankles when the body is grounded and the electric-field frequency is in the range of or below body resonance; the ankle consists mostly of low-conductive cartilage and the integrated current is largest close to the feet of the grounded person ([Dimbylow, 2005](#)).

(b) Mobile phones

During the last decade, the dosimetric analysis of exposure to radiation from mobile phones has focused on reliable compliance testing of the phones with respect to the limits defined

for psSAR-1 g and psSAR-10 g. The absorption values for different mobile phones are determined in homogeneous head phantoms, i.e. the specific anthropometric mannequin (SAM) in touch and tilted positions. The SAR values for different phone positions have been compared in various anatomical models of the head of adults and children. Reviews of these studies concluded that the psSAR assessed with the SAM is a conservative measure of exposure of both adults and children ([Christ & Kuster, 2005](#); [Martens, 2005](#); [Wiart *et al.*, 2005](#)) and that variations in psSAR among different models can be attributed to individual anatomical differences, but not to age-dependent changes in head size ([Kainz *et al.*, 2005](#)).

The effects of age-dependent changes in tissue conductivity have been studied by several authors in various rodent species ([Thurai *et al.*, 1984, 1985](#); [Peyman *et al.*, 2001](#); [Gabriel, 2005](#); [Schmid & Überbacher, 2005](#)).

[Christ *et al.* \(2010a\)](#) investigated the effect of the anatomical differences on specific tissue exposures in humans. These studies concluded that:

- Exposure of regions inside the brain of young children (e.g. hippocampus, hypothalamus, etc.) can be higher by 1.6–3-fold than that in adults.
- Exposure of the bone marrow in the skull of children can exceed that in adults by a factor of about 10, which is due to the high electric conductivity of this tissue at a young age.
- Exposure of the eyes of children is higher than that of adults. Regarding thermal effects, however, this does not present a problem as exposure to the eyes from mobile phones is very low, i.e. < 10% of the psSAR.
- Because of their different locations relative to the ear, brain regions close to the surface of the skull can exhibit large differences in exposure between adults and children. The cerebellum of children can

show a psSAR that is > 2.5-fold that of the local exposure of the cortex of adults. It should be noted that these differences are strongly dependent on the current distribution in the phone, i.e. on the phone design.

- Tissues or anatomical regions that are located at a comparable distance from the phone in adults and children, e.g. the pineal glands, do not show age-dependent variations in exposure.

1.3.5 Estimation of local tissue temperature based on psSAR

In general, the relationship between tissue temperature and psSAR depends strongly upon blood perfusion of the tissue, which varies across the body. In addition, local hot spots (points of elevated temperature) are influenced by thermal conductivity.

The correlation between psSAR and the increase in temperature for exposures to dipoles and mobile phones operated close to the head has been studied ([Hirata *et al.*, 2003](#); [Fujimoto *et al.*, 2006](#); [Hirata *et al.*, 2006a, b, 2008](#)). The results of these studies show that the correlation for a given frequency and exposure type is often good, but that the scaling factor strongly depends on the frequency, the spatial averaging scheme and mass, the tissue perfusion, and geometrical aspects such as anatomical surface curvature. The correlation between local averaged SAR and temperature elevation is weak when multiple tissues are involved. In the brain, the relationship between psSAR and peak temperature is found to be poor, and the tissue distribution and the exact exposure situation have a strong impact on brain heating, with thermo-physiological tissue properties particularly affecting the temperature increase in the head for a given psSAR ([Samaras *et al.*, 2007](#); [McIntosh & Anderson, 2010](#)).

The temperature increase for multiple anatomical models was estimated over a wide

range of frequencies (0.01–5.6 GHz) for plane waves with different polarization and incident angles. The peak temperature increase for a given psSAR was strongly dependent on anatomy and frequency, with variations of one order of magnitude for the cases investigated ([Bakker et al., 2010](#)).

A comparative analysis of seven publications on the increase in brain temperature during mobile-phone use found a high variation (66% at 1800 MHz) in the peak increase in brain temperature relative to the peak averaged SAR in the head ([Samaras et al., 2007](#)). These results confirm the finding that the peak temperature increase in the brain should therefore be correlated with peak averaged SAR in the brain and not with the peak averaged SAR in the whole head. Generally, this peak temperature increase in the brain is strongly influenced by absorption in the neighbouring tissues, thus tissue distribution in that anatomical region is important (e.g. the impact of the cerebrospinal fluid) ([Hirata et al., 2003](#)).

1.3.6 Dosimetry methods

To demonstrate compliance with safety guidelines, wbSAR and psSAR values are estimated conservatively. In most cases, psSAR values are not correlated with a specific tissue or with typical exposures and, therefore, they can only be used for epidemiological studies when additional assessments and considerations are taken into account.

It is practically impossible to measure EMFs non-invasively or *in vivo*; thus, measurements can only be obtained post mortem. The limitations associated with post-mortem evaluations include: (1) accessibility to certain tissues only; (2) field distortions caused by the invasively introduced probe and dielectric changes due to decreased tissue temperature and blood content; and (3) large uncertainties associated with obtaining accurate measurements near and

across tissue boundaries. Only the integrated, total absorbed power can be determined relatively easily by means of the calorimetric method (see Section 1.4.4).

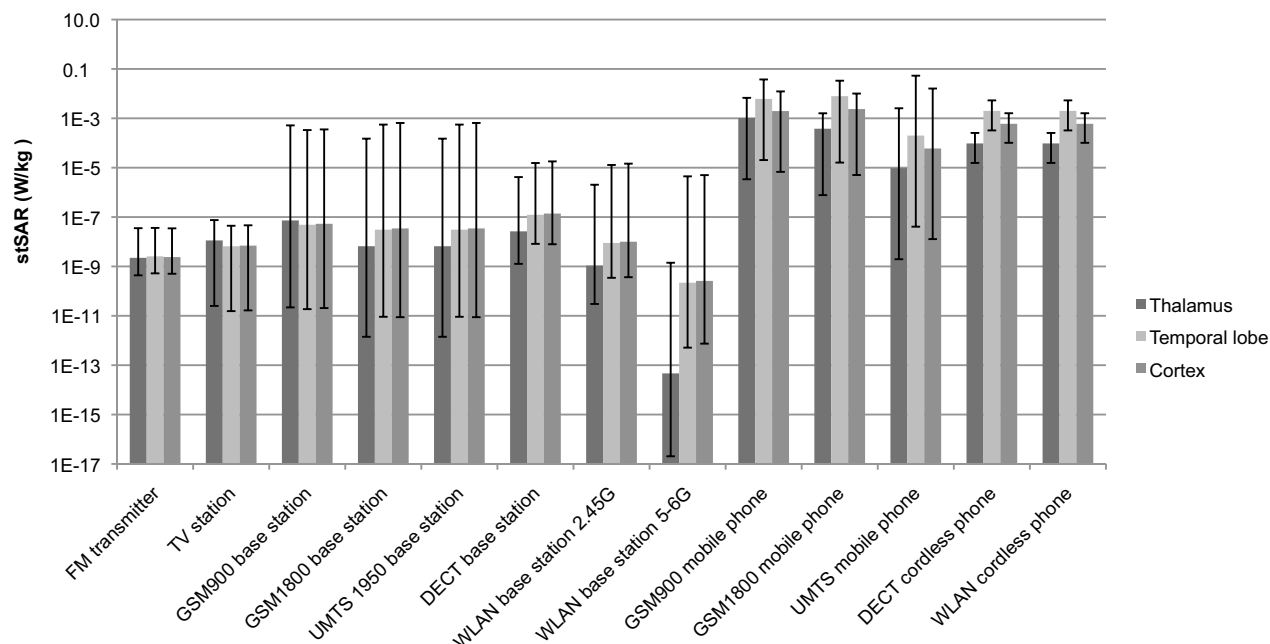
Progress in computational electromagnetics and the exponential growth of computational power and computer memory have facilitated the determination of field distributions in full anatomical models of human bodies with resolutions much smaller than 1 mm³. The dissipative properties and the low quality-factor of complex anatomical structures pose no special problem for numerical analyses such as the finite-difference time-domain (FDTD) method. A grid resolution of less than 0.2 mm in a specific region of the body and of 0.5–1 mm for uniform resolution is the standard for today's FDTD computations. Finite-element methods (FEM) are also increasingly used, especially for evaluation of exposures below 10 MHz. Approaches such as the combination of the method of moment (MoM) with FDTD, are also regularly applied ([Meyer et al., 2003](#)).

Numerical techniques have also become more powerful with the availability of human models that will soon represent the full range of anatomical variation within the human population. Reviews of these models are available ([Dimbylow et al., 2009](#); [Christ et al., 2010b](#); [Wu et al., 2011](#)). In some of these models, body posture can be varied. These models are applied to assess typical exposures, to determine interaction mechanisms, and to derive simplified phantoms for compliance testing.

(a) *Methods to demonstrate compliance with guidelines*

For compliance testing of commercial mobile telecommunication devices that operate very close to the human body, experimental dosimetry is often superior to numerical approaches. The measurement instruments and methods are described in Section 1.4. The sources usually consist of highly resonant components assembled

Fig. 1.13 Estimated tissue-averaged specific absorption rate (stSAR) of the thalamus, temporal lobe and cortex of the brain, induced by various transmission sources



Produced by the Working Group from [Kühn et al. \(2010\)](#)

with other electronic and metallic structures. It is difficult to use simulations to predict with certainty if and how secondary resonant structures may be excited, especially in view of the effect of the reflected field of biological bodies on the performance of the device. Small spatial differences can easily result in deviations of more than a factor of two from the actual value. Only when the structure is electromagnetically well defined can a good agreement between simulation and measurement be achieved, i.e. with deviations of less than 20% ([Chavannes et al., 2003](#)). It should be noted that detailed information about field distributions inside anatomical bodies is often irrelevant because it cannot be generalized and because differences in anatomy and posture can result in significantly different SAR distributions. However, for safety reasons, the upper boundary (typically the 95th percentile) of the exposure for the entire population is relevant, rather than individual exposure levels. Hence, worst-case phantoms, derived by means

of the numerical methods mentioned above, are often applied to assess the upper exposure limits for specific exposure conditions, e.g. during the use of mobile-phone handsets.

(b) Methods to estimate typical exposures

Estimation of typical exposures for specific tissues requires the numerical evaluation of the user's anatomy and usage pattern for the average output power, including its variations. Procedures to make such estimations for different brain regions exposed to mobile-phone radiation have recently been developed ([Gosselin et al., 2011](#)). Similar procedures can be applied for other sources. Quantitative estimates are given in Fig. 1.13, which illustrates the estimated tissue-averaged SARs for the thalamus, temporal lobe and cortex when induced by various transmission sources. The typical minimal and maximal values are also given. The basis for these values is shown in [Table 1.9](#). The largest exposure is

Table 1.9 Estimated minimum, maximum and average exposures in the brain from various sources of radiofrequency radiation

Source	Frequency (MHz)	Exposure			
		Average	Minimum	Maximum	Unit
FM transmitter	100	0.02	0.01	0.07	V/m
TV station	700	0.02	0.001	0.05	V/m
GSM900 base station	950	0.05	0.001	4	V/m
GSM1800 base station	1850	0.05	0.001	6	V/m
DECT base station	1890	0.1	0.03	1	V/m
UMTS 1950 base station	2140	0.05	0.001	6	V/m
WLAN base station	2450	0.03	0.007	1	V/m
WLAN base station	5200/5800	0.01	0.001	1	V/m
GSM900 mobile phone	900	50	0.2	250	mW
GSM1800 mobile phone	1750	40	0.1	125	mW
DECT cordless phone	1890	10	3	20	mW
UMTS mobile phone	1950	1	0.0003	200	mW
WLAN cordless phone	2450	10	3	20	mW

Note: Far-field exposures are estimated in terms of incident-field values and exposures from handsets are calculated from time-averaged output power.

Compiled and calculated by the Working Group from [Kühn et al. \(2010\)](#)

caused by the GSM mobile telephone, followed by exposures from DECT and WLAN cordless handsets. The new wideband code-division multiple access (WCDMA) systems result in much lower exposure values. It should be noted that the maximum exposure level is very similar for all mobile handsets. The averaged induced fields in the brain resulting from exposure to electromagnetic radiation from base stations of any technology are more than four orders of magnitude lower than those from a handset.

1.3.7 Exposure set-ups for laboratory studies

Properly designed laboratory exposure set-ups with sensitive monitoring systems are critical for producing reliable and reproducible results on the potential health effects of RF radiation. The selection of an exposure set-up is intimately linked to the design and objectives of the study, and includes factors such as the efficiency of the coupling of the incident field with the biological system, the number of animals or cell-culture samples needed per exposure level

for statistical analyses, the daily exposure times, and the overall duration of the study. Examples of exposure systems used for this type of study include:

- *Far-field/anechoic chamber*: a room designed to minimize reflections of either sound or electromagnetic waves. To prevent the latter, the inner walls of the chamber are covered with pyramid-shaped RF radiation-absorbent material ([Chou & Guy, 1982](#)). Animals or tissue-culture dishes are exposed to RF radiation via an antenna (e.g. horn antenna).
- *Near-field systems*: antennae are used to obtain partial body exposures. In the Carousel system, the animals in restraining tubes are oriented radially around a central antenna at a fixed distance between the nose of the animal and the antenna ([Adey et al., 1999](#)). Loop antennae have been used to predominantly expose a particular part of the brain ([Lévêque et al., 2004](#)).

- *Transverse electromagnetic (TEM) cell*: an RF-shielded box in which tissue cultures are positioned with a defined orientation relative to the direction of wave propagation and the electric field. A rectangular coaxial transmission line tapered at both ends provides a uniform incident plane wave when RF energy is coupled to the line ([Crawford, 1974](#)). Studies with cell cultures and animal models have been conducted in various modified TEM cells ([Nikoloski et al., 2005](#)).
- *Waveguide*: a structure that guides and confines electromagnetic waves to propagation in one dimension within a round or rectangular metallic tube. Waveguides have the advantage that only the fundamental mode can propagate within a certain frequency band, correlated with its dimensions. Therefore, resonant systems can be easily used. The power losses of the propagating wave must be carefully evaluated if larger objects are exposed. Standing waves must be appropriately used in case of resonant waveguides or waveguides terminated by a short circuit. Waveguides are widely used for *in-vitro* systems (e.g. [Schuderer et al., 2004b](#)). Non-resonant waveguides have also been used to expose rodents (e.g. [Guy et al., 1979](#)) and a cascade of 17 sectorial resonant waveguides excited by one quad-loop antenna have been employed to expose one rat per waveguide ([Kainz et al., 2006](#)).
- *Radial transmission line (RTL)*: a structure that confines the wave to propagate in two dimensions with two parallel metal plates excited between their centres by an antenna. In the case of a non-resonant application, the wave is terminated at the perimeter of the lateral plates with absorbers ([Hansen et al., 1999](#); [Moros et al., 1999](#)). The system has been used for studies *in vivo* or *in vitro* by placing the

cell cultures or animals at a fixed distance from the antenna. RTL has also been used as a resonant structure in which the wave is terminated with metallic rods instead of absorbers. This configuration has also been called a “Ferris wheel,” whereby the animals in restraining tubes are positioned at a fixed distance to the reflecting rods ([Balzano et al., 2000](#)). Several improvements have been suggested and implemented ([Ebert, 2009](#)).

- *Reverberation chamber*: a shielded room with minimal absorption of electromagnetic energy. To create statistically homogeneous fields inside the chamber when exposure is averaged over time, rotating metallic reflectors (stirrers) constantly create changing boundary conditions. Animals are unrestrained during exposure ([Jung et al., 2008](#)).

Regardless of the type of exposure system, for a correct interpretation of the findings and replication of the experiments in other laboratories, it is important that all pertinent electromagnetic-field exposure characteristics (particularly dosimetry) and biological parameters be fully addressed in the experimental design, and properly described in the study reports ([Valberg, 1995](#); [Kuster & Schönborn, 2000](#); [Kuster et al., 2006](#); [Belyaev, 2010](#)). These factors are briefly discussed in the next two sections.

1.3.8 Exposure characterization in laboratory studies

The experimental conditions during studies on the effects of exposure to EMF should be described in detail as listed below:

- Signal characteristics should include: carrier frequency, modulation scheme, power level and stability;
- Zone of exposure (near field or far field);

- Polarization (e.g. linear or circular polarization) of the induced EMF with respect to the biological system;
- Performance of the setup: determination of induced electric- and magnetic-field strengths and SAR levels and distribution (numerical dosimetry) in the cell culture, or per organ site in animal experiments; this part should also include an uncertainty analysis;
- Field distribution: should be homogeneous ($SD < 30\%$ in cell cultures) and variations in the exposure levels of individual tissues of the exposed animals should be characterized, including details on animal age, movement, posture, weight, etc.;
- The increase in temperature caused by the RF field must be well characterized and reported;
- Control of acoustic noise/vibration level and exposure to ambient RF fields and static fields;
- Monitoring: should include verification of incident field strengths and homogeneity, induced fields, and any changes in the performance of elements of the exposure system over the duration of the experiment, including the long-term reliability of monitoring equipment;
- Experimental design requirements: duration of exposure (hours per day and total number of days), continuous or intermittent (on/off cycles), time of day;
- Inclusion of a sham-exposure group.

1.3.9 Biological factors in studies in experimental animals

The biological factors that may affect the study results are briefly described below:

(a) *Studies in vivo*

- Identification and justification of the selected animal model (species, strain, sex, age at start and end of study, genotype and phenotype, exposure to other agents);
- Animal husbandry: diet (ingredients, nutrient composition and contaminant levels), drinking-water source and treatment, availability or restriction of feed and water during exposures, absence of specific pathogens, caging (cage material, number of animals per cage, bedding material), prevention of exposure to electric currents from water supply, absence/presence of animal restraining devices;
- Environmental controls: temperature, relative humidity, lighting (on/off cycle, intensity), airflow, noise, and background fields;
- Characterization of animal weight, positioning/orientation, movement in the exposure system, and proximity of other animals and cage boundaries during exposure periods.

(b) *Studies in vitro*

- Composition of the incubation media, including antioxidant levels, free-radical scavengers, presence of magnetic particles;
- Source and/or derivation of the cell system and its characteristics: cell type, species, strain, sex, age, genotype and phenotype;
- Quality of the cell-culture system and its functional condition: cell viability, growth phase and cell-cycling rate, metabolic status, and cell density (which may affect cell-cell interactions);
- Size, shape, and position of the cell-culture vessel;
- Environmental controls, including temperature, oxygen/carbon dioxide levels, air flow.

Table 1.10 Summary of studies on models of partial body exposure

Reference	Description of the model	Main results and comments
Gandhi <i>et al.</i> (1999)	EM absorption in the head and neck region	A FDTD method and a new phantom model of the human body at millimetre resolution were used to study EM energy coupled to the head from mobile phones at 835 and 1900 MHz. Homogeneous models are shown to grossly overestimate both the peak 1-voxel and 1-g SARs. It is possible to use truncated one-half or one-third models of the human head with negligible errors in the calculated SAR distributions.
Hombach <i>et al.</i> (1996)	EM energy absorption upon modelling of the human head at 900 MHz	Dependence on anatomy and modelling in the human head were investigated for EM energy absorption at 900 MHz from RF sources operating very close to the head. Different head phantoms based on MRI scans of three adults were used with voxel sizes down to 1 mm ³ . The phantoms differ greatly in terms of shape, size, and internal anatomy. The results demonstrate that size and shape are of minor importance. The volume-averaged psSAR obtained with the homogeneous phantoms only slightly overestimates that of the worst-case exposure in the inhomogeneous phantoms.
Schönborn <i>et al.</i> (1998)	Energy absorption in the head of adults and children	The levels of EM energy absorbed at 835 MHz and 1900 MHz in the heads of mobile-phone users were compared for adults and children. No significant differences between adults and children were found in the absorption of EM radiation in the near field of sources. The same conclusion holds when children are approximated as scaled adults.
Bit-Babik <i>et al.</i> (2005)	Estimation of SAR in the head of adults and children	The peak local average SAR over 1 g and 10 g of tissue and the EM energy penetration depths are about the same in all of the head models under the same exposure conditions.
Wang & Fujiwara (2003)	Evaluation of EM absorption in head models of adults and children	Based on statistical data on external shape of the head in Japanese children, two models were developed to assess SAR values in the head of a child exposed to RF radiation. Compared with the local peak SAR in the model of the adult head, there was a considerable increase in the child's head when the output power of the monopole-type antenna was fixed, but no significant difference when the effective current of the dipole-type antenna was fixed.
Anderson (2003)	Peak SAR levels in head models of children and adults	Multipole analysis of a three-layered (scalp/cranium/brain) spherical head model exposed to a nearby 0.4-lambda dipole at 900 MHz was used to assess differences in SAR in the brain of adults and children. Compared with an average adult, the peak SAR-10 g in the brain of children with a mean age of 4, 8, 12 or 16 yr is increased by a factor of 1.31, 1.23, 1.15 and 1.07, respectively. The maximum rise in brain temperature is about 0.14 °C for an average child aged 4 yr, i.e. well within safe levels and normal physiological parameters.
Martínez-Búrdalo <i>et al.</i> (2004)	Peak SAR levels in head models of adults and children	The FDTD method was used to assess differences in SAR in the brain of adults and children, at 900 and 1800 MHz. Peak SAR-1 g and peak SAR-10 g all decrease with decreasing head size, but the percentage of energy absorbed in the brain increases.
Fernández <i>et al.</i> (2005)	EM absorption in head models of adults and children	The peak SAR in the head model for a child aged 10 yr was 27% higher than that in the phantom of an adult head, when dielectric properties of a child's tissue were applied, based on fitted parameters. For the peak SAR-10 g, an increase of 32.5% was observed.
Keshvari & Lang (2005)	RF energy absorption in the ear and eye of children and adults	The FDTD computational method was used to calculate a set of SARs in the ear and eye region for anatomically correct head models for adults and children. A half-wave dipole was used as an exposure source at 900, 1800 and 2450 MHz. The head models were greatly different in terms of size, external shape and internal anatomy. The SAR difference between adults and children is more likely to be caused by general differences in the head anatomy and geometry of the individuals rather than by age.
Christ & Kuster (2005)	RF energy absorption in the head of adults and children	The conclusions of this review do not support the assumption that energy exposure to children is increased due to smaller size of the head compared with adults, but points at dielectric tissue parameters and the thickness of the pinna (the external part of the ear) as factors that determine RF energy absorption.

Table 1.10 (continued)

Reference	Description of the model	Main results and comments
Hadjem <i>et al.</i> (2005a)	SAR induced in models of the head of children and adults	The FDTD computational method was used to calculate SARs for two child-size head models and two adult-size head models, with a dual-band mobile phone. No important difference was observed in the peak SAR-10 g between the two adult models, or between the two child models.
Hadjem <i>et al.</i> (2005b)	Ear morphology and SAR induced in a child's head	Using the FDTD method, SARs induced in the heads of children aged 12 yr were calculated for different ear dimensions, at 900 and 1800 MHz. Exposure to the brain was dependent on the morphology of the ear.
Wiart <i>et al.</i> (2005)	Modelling of RF exposure in a child's head	Parameters that influence the SAR in children's heads, such as the evolution of head shape and the growth of, e.g. skull thickness, were analysed. The SAR-1 g in specific tissue was assessed in different models of a child's head based on MRI and on non-uniformly down-scaled adult heads. A handset with a patch antenna operating at 900 MHz was used as the exposure source.
Kainz <i>et al.</i> (2005)	Dosimetric comparison of SAM to anatomical models of the head	The SAM was used to estimate exposure in anatomically correct head models for head-only tissue. Frequency, phone position and head size influence the calculated SAR-10 g, which in the pinna can be up to 2.1 times greater than the psSAR.
de Salles <i>et al.</i> (2006)	Absorption of RF radiation in the head of adults and children	The SAR produced by mobile phones in the head of adults and children was simulated with an FDTD-derived algorithm, with EM parameters fitted to realistic models of the head of a child and an adult. Microstrip or patch antennae and quarter-wavelength monopole antennae were used at 1850 and 850 MHz. Under similar conditions, the SAR-1 g calculated for children is higher than that for adults. In the model of a child aged 10 yr, SAR values > 60% of those for adults are obtained.
Ioó <i>et al.</i> (2006)	Metal-framed spectacles and implants and SAR in models of the heads of adults and children	The SAR from mobile telephones in the head of adults and children wearing metal-rim spectacles and having metallic implants was calculated by the FDTD method, and compared with the ANSI/IEEE standards and with the EU standard limits for radiation at 900/1800/2100 MHz. A maximum of the SAR in the child's head was found, which in children with metallic implants could be as much as 100% higher than in the adult head. In the case of exposure at 2100 MHz with vertical position of the phone for adults and at 900 MHz for children with metallic implants, the ANSI/IEEE limits were exceeded.
Fujimoto <i>et al.</i> (2006)	Temperature increase and peak SAR in head models for children and adults	The correlation between peak SAR and rise in temperature was studied in head models of adults and children exposed to radiation from a dipole antenna. The maximum rise in temperature can be estimated linearly in terms of peak SAR-1 g or peak SAR-10 g of tissue. No clear difference was observed between adults and children in terms of the slopes correlating the maximum rise in temperature with the peak SAR. The effect of electrical and thermal constants of the tissue on this correlation was marginal.
Beard <i>et al.</i> (2006)	SAR comparison between SAM phantom and human head models	The SAM was used to calculate the SAR of the pinna, separately from that of the head. In this case, the SAR found in the head was higher than that found with anatomically correct head models. The peak SAR-1 g or SAR-10 g was statistically significantly higher in the larger (adult) head than in the smaller (child) head for all conditions of frequency and position.
Lee <i>et al.</i> (2007)	Changes of SAR in head models by age	Four head models, representing different ages, were used to calculate SARs from exposure to three bar-type phones, according to positioning against the ear. Input resistance of the phone antennae in the cheek position increased when head size grew with age, but for the tilt position this value showed a slight decrease. For a fixed input power, the head models by age showed a 15% change in peak SAR-1 g and peak SAR-10 g. For a fixed radiated power, the peak SARs diminished in the smaller head model and were higher in the larger model, compared with those for the fixed input power. A simultaneous change (up to 20–30%) in the conductivity and permittivity of head tissue had no effect on energy absorption.

Table 1.10 (continued)

Reference	Description of the model	Main results and comments
Wiaart <i>et al.</i> (2007, 2008)	RF exposure assessment in children head	RF exposure in the head tissues of children was analysed with phantom models and with a dipole and a generic handset at 900, 1800, 2100 and 2400 MHz. The SAR-10 g in the head was studied in heterogeneous head models (seven for children, six for adults). The maximum SAR-10 g estimated in the head models of adults and children were small compared with the SDs. However, the maximum SAR-1 g in peripheral brain tissues of the child models (age, 5–8 yr) is about twice that in adult models, which is not seen with head models for children older than 8 yr. The differences can be explained by the lower thicknesses of pinna, skin and skull of the models for the younger child.
Christ <i>et al.</i> (2007)	The exposure of the human body to fields from wireless body-mounted or hand-held devices	A generic body model and simulations of anatomical models were used to evaluate the worst-case tissue composition with respect to the absorption of EM energy from wireless body-mounted or hand-held devices. Standing-wave effects and enhanced coupling of reactive near-field components can lead to an increased SAR compared with homogeneous tissue. With respect to compliance testing, the increased SAR may require the introduction of a multiplication factor for the psSAR measured in the liquid-filled phantom to obtain a conservative exposure assessment. The observed tissue heating at the body surface under adiabatic conditions can be significant, whereas the rise in temperature in the inner organs is negligible.
Christ <i>et al.</i> (2010b)	RF absorption in the heads of adult and juvenile mobile-phone users	The external ear (pinna) is the spacer between the top of a phone and the head tissue. Variations of this distance as a function of age, the mechanical force on the pinna, and how it affects the psSAR were investigated among adults and children (age, 6–8 yr) while applying a defined force on the ear. The average distances were 10.5 ± 2.0 (SD) mm for children (age, 6–8 yr) and 9.5 ± 2.0 (SD) mm for adults. The pinnae of three anatomical high-resolution head models (one adult, two child) were transformed accordingly. Numerical exposure analysis showed that the reduced distance due to compression of the pinna can increase the maximum psSAR by approximately 2 dB for adults and children, if the exposure maximum is associated with the upper part of the phone.
Lee & Yun (2011)	Comparison of SARs for the SAM phantom and head models for children	SARs in three head models for children (Korean aged 7 yr; European aged 5 or 9yr) were compared with those of the SAM phantom for exposure at 835 and 1900 MHz. Compression of the pinnae, different positions of the earpiece against the ear entrance canal, different skin and fat properties, and different internal fat and muscle morphologies in the tissue near the phone, were analysed. A phone with a monopole antenna was used for the calculation at each frequency. Results show that a compressed pinna did change the SAR values at 835 MHz, but at 1900 MHz there was an average 25–29% increase in SAR-10 g for pinna-excluded and pinna-included tissue. The peak SAR-10 g was very sensitive to subcutaneous fat and muscle structure when touched by the mobile phone; a muscle-dominant internal head structure led to a higher peak SAR-10 g. The SAM phantom does not seem to provide a conservative estimation of exposure of children's heads at 1900 MHz.

ANSI, American National Standards Institute; EM, electromagnetic; EU, European Union; FDTD, finite-difference time-domain; IEEE, Institute of Electrical and Electronic Engineers; MRI, magnetic resonance imaging; psSAR, peak spatial SAR; RF, radiofrequency; SAM, specific anthropomorphic mannequin; SAR, specific absorption rate; SD, standard deviation; yr, year or years

Table 1.11 Summary of studies on models of whole-body exposure

Reference	Description of the model	Main results and comments
Dimbylow (1997)	Calculation of whole-body-averaged SAR in a voxel model	FDTD calculations were made of the whole-body-averaged SAR in an anatomically realistic voxel model of the human body, which consists of approximately 9 million voxels segmented into 37 tissue types. SAR values are presented for an adult phantom and for scaled models of children aged 10, 5 or 1 yr, grounded and isolated in air from 1 MHz to 1 GHz for plane-wave exposure. External electric-field values corresponding to a whole-body-averaged SAR of 0.4 W/kg are also presented.
Tinniswood <i>et al.</i> (1998)	Calculation of power deposition in the head and the neck for plane-wave exposures	When the human head becomes a resonant structure at certain frequencies, the power absorbed by the head and neck becomes significantly larger than would normally be expected from its shadow cross-section. Resonant frequencies were 207 MHz and 193 MHz for the isolated and grounded conditions, with absorption cross-sections that are respectively 3.27 and 2.62 times the shadow cross-section.
Hurt <i>et al.</i> (2000)	Effects of variation in permittivity on SAR calculations	The authors studied the effect of variation in permittivity values on SAR calculations. Whole-sphere averaged and localized SAR values along the diameter of a 4-cm sphere were calculated for exposures of 1 MHz to 1 GHz. When the sphere is small compared with the wavelength, the whole-sphere averaged SAR is inversely proportional to the permittivity of the material composing the sphere, but the localized SAR values vary greatly depending on the location within the sphere.
Dimbylow (2002)	Calculation of SAR up to 3 GHz	FDTD calculations of whole-body-averaged SAR values were made for frequencies from 100 MHz to 3 GHz at 2-mm resolution without any rescaling to larger cell sizes. The small voxel size allows SAR to be calculated at higher frequencies. In addition, the calculations were extended down to 10 MHz, covering whole-body resonance regions at 4-mm resolution. SAR values were also calculated for scaled versions representing children aged 10, 5 or 1 yr for both grounded and isolated conditions.
Bernardi <i>et al.</i> (2003)	SAR and rise in temperature in the far field of RF sources at 10–900 MHz	The EMF inside an anatomical heterogeneous model of the human body exposed at 10–900 MHz was computed with the FDTD method; the corresponding increase in temperature was also evaluated. The thermal model took account of the thermoregulatory system of the human body. Compared with the whole-body averaged SAR, the SAR-10 g shows an increase of 25-fold in the trunk and 50-fold in the limbs, whereas the peak SAR-1 g shows an increase of 30–60-fold in the trunk, and up to 135-fold in the ankles.
Findlay & Dimbylow (2005)	Effects of posture on SAR	A change in posture can significantly affect the way in which the human body absorbs RF EM radiation. The FDTD method was used to calculate the whole-body-averaged SAR at frequencies from 10 MHz to 300 MHz at a resolution of 4 mm. Raising an arm above the head increased the SAR value at resonance by up to 35% compared with the standard, arms-by-the-side position.
Wang <i>et al.</i> (2006)	Whole-body averaged SAR in adult and child models	Due to the difficulty of measuring SAR in an actual human body exposed to RF-EMF, the incident electric field or power density is often used as a reference. In verifying the validity of the reference level, it is essential to have accurate modelling for humans. A detailed error analysis in the whole-body-averaged SAR calculation was done with the FDTD method in conjunction with the perfectly matched layer (PML) absorbing boundaries. To clarify whole-body-averaged SAR values, a Japanese adult model and a scaled child model were used. The whole-body-averaged SAR under the reference level exceeded the basic safety limit by nearly 30% for the child model, both in the resonance frequency and in the band 2 GHz.
Dimbylow (2007a)	SAR values in voxel models of mother and fetus	An FDTD calculation was conducted of SAR values (20 MHz to 3 GHz) in hybrid voxel-mathematical models of the pregnant female. Models of the developing fetus at 8, 13, 26 and 38 wk of gestation were converted into voxels and combined with the adult female model, at a resolution of 2 mm. Whole-body-averaged SAR in the mother, the average SAR over the fetus, over the fetal brain, and in 10 g of the fetus were calculated.

Table 1.11 (continued)

Reference	Description of the model	Main results and comments
Dimbylow & Bolch (2007b)	Whole-body-averaged SAR in voxel phantoms for a child	Five paediatric phantoms (representing boys aged 9 months, 11 yr and 14 yr, and girls aged 4 and 8 yr) were adapted to calculate the whole-body-averaged SARs in children for plane-wave exposure from 50 MHz to 4 GHz. A comparison was made with previous linearly scaled versions, at a resolution of 2 mm. Further FDTD calculations were performed at resolutions of 1 and 0.7 mm above 900 MHz, to elucidate the effects of variation in grid resolution.
Hirata <i>et al.</i> (2003)	Temperature increase in head and brain	The temperature increase (ΔT) in a human head exposed to EM waves (900 MHz to 2.45 GHz) from a dipole antenna was investigated. The maximum ΔT in the head and brain was compared with values from the literature of 10 °C and 3.5 °C for microwave-induced physiological damage. The SAR in the head model was initially calculated by the FDTD method. The ΔT distribution in the head is largely dependent on the frequency of the EM waves, and the maximum ΔT values in the head and brain are significantly affected by the frequency and polarization of the waves. The maximum ΔT in the head (excluding auricles) and brain are determined through linear extrapolation of the peak average SAR in these regions. The peak SAR-1 g should be approximately 65 W/kg to achieve a maximum ΔT of 10 °C in the head, excluding auricles.
Vermeeren <i>et al.</i> (2008)	Statistical method to calculate absorption of RF energy	Quantifying the absorption of EM radiation in a human body in a realistic, multipath exposure environment requires a statistical approach, because it needs to be determined for several thousands of possible exposures. To avoid having to make this large number of time-consuming calculations with the FDTD method, a fast numerical method was developed to determine the whole-body absorption in a spheroid human-body model in a realistic exposure environment. This method uses field distributions of a limited set of incident plane waves to rapidly calculate whole-body absorption for any single or multiple plane-wave exposure. This fast method has now been extended to realistic heterogeneous human-body models.
Nagaoka <i>et al.</i> (2007)	SAR of a whole-body phantom for pregnant women	A new model for the fetus, including inherent tissues of pregnant women, was constructed on the basis of abdominal MRI data for a woman at week 26 of pregnancy. A whole-body pregnant-woman model was developed by combining the fetus model with a nonpregnant-woman model, developed previously. The model consists of about 7 million cubical voxels (size, 2 mm) and is segmented into 56 tissues and organs. The basic SAR characteristics are presented of the pregnant-woman model exposed to vertically and horizontally polarized EM waves from 10 MHz to 2 GHz.
Nagaoka <i>et al.</i> (2008)	SAR for a phantom model of Japanese children	An existing voxel model of a Japanese adult in combination with 3D deformation was used to develop three voxel models that match the average body proportions of Japanese children at age 3, 5 and 7 yr. The models consist of cubic voxels (size, 2 mm) and are segmented into 51 tissues and organs. Whole-body-averaged SARs and tissue-averaged SARs were calculated for the child models, for exposures to plane waves from 30 MHz to 3 GHz.
Togashi <i>et al.</i> (2008)	SAR for the fetus in a pregnant-woman model	Dosimetry of EM radiation is described in a pregnant woman in the proximity of a mobile-phone terminal by use of the numerical model of a woman in the seventh month of pregnancy. The model is based on the high-resolution whole-body voxel model of a Japanese adult woman. It is composed of 56 organs, which include the intrinsic organs of a pregnant woman.

Table 1.11 (continued)

Reference	Description of the model	Main results and comments
Conil <i>et al.</i> (2008)	SAR for different adult and child models using the FDTD method	Six adult anthropomorphic voxel models were collected and used to build models for children aged 5, 8 and 12 yr, with a morphing method that respects anatomical parameters. FDTD calculations of SARs were performed for frequencies from 20 MHz to 2.4 GHz for isolated models exposed to plane waves. A whole-body-averaged SAR, average SARs on specific tissues such as skin, muscles, fat or bones, and the average SAR on specific parts of the body such as head, legs, arms or torso, were calculated. The SD of the whole-body-averaged SAR of adult models can reach 40%. For adults, compliance with reference levels ensures compliance with basic restrictions. For children, the whole-body-averaged SAR exceeds the fundamental safety limits by up to 40%.
Kühn <i>et al.</i> (2009b)	Assessment of induced EMFs in various human models	The absorption characteristics are given for various anatomies ranging from a child aged 6 yr to a large adult male, by numerical modelling, with exposure to plane waves incident from all six major sides of the humans with two orthogonal polarizations each. Worst-case scattered-field exposure scenarios were constructed to test the implemented procedures of current in situ compliance measurement standards. The results suggest that the reference levels of current EM safety guidelines for demonstrating compliance, as well as some of the current measurement standards, are not consistent with the basic restrictions and need to be revised.
Neubauer <i>et al.</i> (2009)	SAR and EMF intensity for heterogeneous exposure	The relation between the incident EMF strength, the wBSAR, and the local SAR, was investigated for heterogeneous exposure scenarios at mobile communication frequencies. For whole-body exposure at 946 MHz, 12% of all heterogeneous cases examined represent worse exposure conditions than plane-wave exposure. This percentage increases to 15% at 1840 MHz, and to 22% at 2140 MHz. The results indicate the need to extend investigations to numerical simulations with additional human phantoms representing parts of the human population having different anatomy and morphology compared with the phantom used here. This also applies to phantoms of children.
Hirata <i>et al.</i> (2009)	Whole-body-averaged SAR in children	The whole-body-averaged SAR was calculated in an infant model with the FDTD method, and the effect of polarization of incident EM waves on this SAR was investigated. The whole-body-averaged SAR for plane-wave exposure with a vertically aligned electric field is smaller than that with a horizontally aligned electric field for frequencies above 2 GHz. The main reason for this difference is probably the component of the surface area perpendicular to the electric field of the incident wave.
Nagaoka & Watanabe (2009)	Estimation of SAR in different models for children	To estimate individual variability in SAR for children of different age and with different physical features, a large set of 3D body-shape data from actual children aged 3 yr was used to develop several homogeneous models of these children. The variability in SAR of these models of whole-body exposure to RF-EMF in the VHF band was calculated by the FDTD method.
Findlay <i>et al.</i> (2009)	SAR for voxel models for children in different postures	SAR calculations were performed on two voxel models (NORMAN, ETRI) for a child aged 7 yr in different postures (standing with arms down, standing with arms up, sitting) for plane-wave exposure under isolated and grounded conditions between 10 MHz and 3 GHz. There was little difference at each resonant frequency between the whole-body-averaged SARs calculated for the two models for each of the postures studied. However, compared with the arms-down posture, raising the arms increased the SAR by up to 25%.

Table 1.11 (continued)

Reference	Description of the model	Main results and comments
Kühn <i>et al.</i> (2009b)	SAR induced in the human head from mobile phones used with hands-free kits	To determine the extent to which the use of wired and wireless hands-free kits can reduce human exposure, the SARs from these kits were determined experimentally while connected to mobile phones (GSM900/1800, UMTS1950) under maximized current coupling onto the cable and various wire-routing configurations. The maximum psSAR in the head when using wired hands-free kits was more than five times lower than current recommended limits. The SAR in the head depends on the output power of the mobile phone, the coupling between the antenna and cable, external attenuation and potential cable-specific attenuation. In general, a wired hands-free kit considerably reduces the exposure of the entire head region compared with mobile phones operated at the head.
Gosselin <i>et al.</i> (2009)	Human exposure in the close vicinity of mobile-phone base-station antennae	Human exposure in close vicinity of mobile-phone base-station antennae was assessed by means of FDTD simulations. The peak spatial average SAR and the whole-body-averaged SAR were calculated for three different anatomical models (55–101 kg) at distances between 0.5 and 4 m from various antenna types, at frequencies of 450–2140 MHz. The whole-body absorption generally determines the maximum permissible output power for collinear array antennae. In particular for short antennae, the peak spatial average SAR can be more restrictive than the whole-body absorption because they may only expose a fraction of the body.
Vermeeren <i>et al.</i> (2010)	SARs in a phantom for a human male exposed to representative base-station antennae	The variation in whole-body and peak spatially averaged SARs was determined for the heterogeneous “virtual family” male model placed at 30 cm, 1 m, 3 m and 10 m in front of different base-station antennae in a reflective environment. SAR values were also compared with those in the free-space situation. The six base-station antennae operated at 300 MHz, 450 MHz, 900 MHz, 2.1 GHz, 3.5 GHz and 5.0 GHz, respectively. The ratio of the SAR in a reflective environment and the SAR in the free-space environment ranged from –8.7 dB up to 8.0 dB.
Uusitupa <i>et al.</i> (2010)	SAR variation for 15 voxel models including different postures	A study on SARs covering 720 simulations and 15 voxel models (body weight range, 18–105 kg) was performed by applying the parallel FDTD method. The models were irradiated with plane waves (300 MHz to 5 GHz) with various incoming directions and polarizations. For an adult, the effect of incoming direction on wbSAR is larger in the GHz range than at around 300–450 MHz, and the effect is stronger with vertical polarization. For a child (height, ~1.2 m), the effect of incoming direction is similar as for an adult, except at 300 MHz for horizontal polarization. Body posture has little effect on wbSAR in the GHz range, but at around 300–450 MHz, a rise of 2 dB in wbSAR may occur when posture is changed from the standing position. Between 2 and 5 GHz for adults, wbSAR is higher for horizontal than for vertical polarization. In the GHz range, horizontal polarization gives higher wbSAR, especially for irradiation from the lateral direction. A homogenized model underestimates wbSAR, especially at approximately 2 GHz.
Kawai <i>et al.</i> (2010)	Computational dosimetry of SAR in models of embryos of different age	SAR dosimetry is presented in models of pregnant (4 and 8 wk) Japanese women, with a cubic (4 wk) or spheroidal (8 wk) embryo, exposed to plane waves at frequencies of 10 MHz to 1.5 GHz. The averaged SARs were calculated in the embryos exposed to vertically and horizontally polarized plane waves. The maximum average SAR in the exposed embryos is < 0.08 W/kg when the incident power density is at the recommended environmental level for the general public.

3D, three-dimensional; ANSI, American National Standards Institute; EMF, electromagnetic field; EU, European Union; FDTD, finite-difference time-domain; IEEE, Institute of Electrical and Electronic Engineers; mo, month or months; MRI, magnetic resonance imaging; psSAR, peak spatial SAR; PML, perfectly matched layer; RF, radiofrequency; SAM, specific anthropomorphic mannequin; SAR, specific absorption rate; sSAR, spatially averaged SAR; SD, standard deviation; VHF, very high frequency; wbSAR, whole-body SAR; wk, week or weeks; yr, year or years

For a summary of studies with models for partial- or whole-body exposure, see [Tables 1.10](#) and [1.11](#).

1.4 Measurement techniques

1.4.1 Introduction

Assessment of the incident exposure is simple for plane-wave or far-field conditions. Unfortunately, when high exposures are involved, far-field conditions rarely occur, due to the proximity of the source. In addition, the reflecting environments result in fading, producing fields that are highly variable spatially and temporally.

In general, far-field conditions are approximately met locally by changing the amplitude in space at distances larger than the extension of the reactive near-field zone ([CENELEC, 2008](#)):

Thus, for distances meeting the requirements of the equation, only the maximum of the field components must be determined to demonstrate compliance. For any distance smaller than the requirements of the equation, the maximum of both components must be spatially scanned to reliably predict that the maximal induced fields are below a certain limit. Fine volume scanning of transmitting antennae in the very near field yields greater uncertainty, neglects reflection back to the source antenna due to the presence of a lossy body, and is more time-consuming than dosimetric measurements in homogeneous phantoms. Since such near-field assessments are more conservative, they are rarely conducted in the context of exposure assessments.

In summary, reference levels are easy to assess if the plane-wave or far-field conditions are approximately met (see Section 1.3.2) and the resulting SAR and induced current densities are below the corresponding basic restrictions under all circumstances. The reference limits for occupational/controlled and for the general public/uncontrolled exposure are given in [NCRP \(1986\)](#), [ANSI/IEEE \(1991, 2002b\)](#) and [ICNIRP](#)

([1998](#)). However, sometimes only the incident electric field (E-field) or the E-field-based equivalent power density is also reported for cases in which the above equation is not satisfied and therefore may not well represent true exposure. It should also be noted that the maximum value that is often reported is suitable for reporting compliance with guidelines, but may greatly overestimate typical exposures at that location.

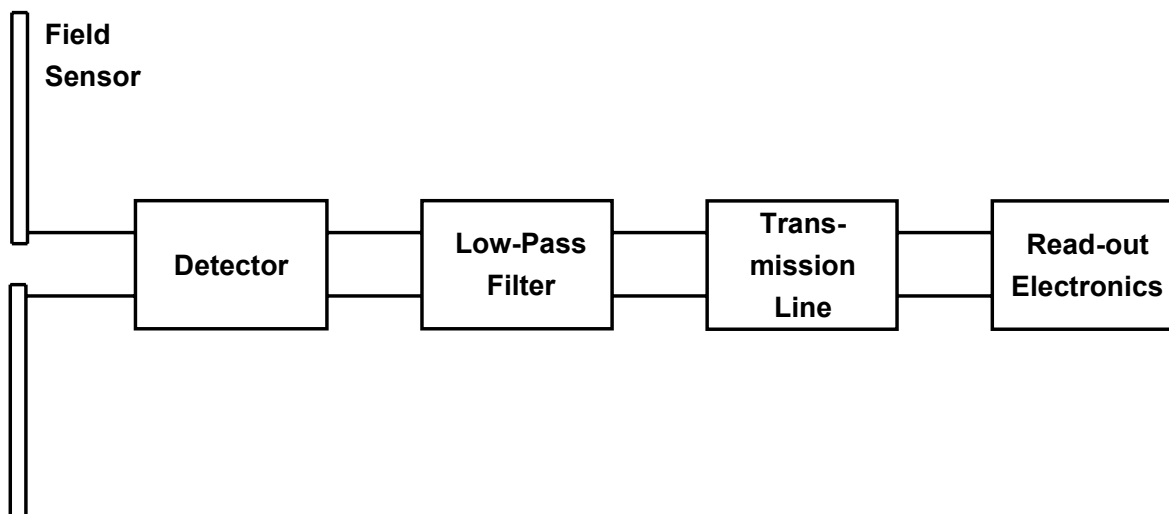
1.4.2 Near-field and dosimetric probes

The first instruments were developed in the early 1970s and they covered the 10 MHz to 10 GHz region of the spectrum. One involved the use of two pairs of thin-film thermo-coupling vacuum-evaporated electrothermic elements that functioned as both antenna and detector ([Aslan, 1970](#)). In another instance, two small diode-loaded dipoles were employed as sensor elements ([Rudge, 1970](#)). In 1975, the first prototype of an isotropic, miniature field probe was introduced ([Bassen et al., 1975](#)). Fibre-optic field probes were proposed as early as the 1970s ([Bassen et al., 1977](#)). Comprehensive overviews of field probes have been published ([Bassen & Smith, 1983](#); [Poković, 1999](#)).

(a) Broadband E-field probes

Diode-based field probes are most commonly used for dosimetric assessments. These instruments consist of field sensors, a detector, transmission lines and readout electronics (Fig. 1.14). The probe is constituted of three mutually orthogonal diode-loaded dipoles with an isotropic receiving pattern. Different orthogonal sensor configurations are available.

An RF detector Schottky-type diode is placed at the centre of the dipole sensor. If the detector diode operates in the square-root law region, the detected voltage is proportional to the RF power. The data-acquisition electronics are connected to the detector diodes by high-resistance transmission lines to minimize incident-field perturbation

Fig. 1.14 Simplified schematic of a broadband-field probe

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and spurious pick-up effects. A detailed investigation of transmissionline design can be found in [Smith \(1981\)](#).

Magnetic-field (H-field) probes are also available; the basic theory on which such probes are designed can be found in [Whiteside & King \(1964\)](#). H-field probes and E-field probes have similar features except that H-field probes employ a small loop element instead of a dipole sensor. Loop-based sensors present the disadvantage of a strong frequency dependence and induction of currents by both H- and E-fields. Different methods for flattening the frequency response of loop probes have been suggested ([Kanda, 1993](#); [Poković, 1999](#)). Lossy covers have been proposed to further suppress the E-field sensitivity of the loop ([Poković, 1999](#)).

A general problem of diode-based probes is their inherent nonlinearity over their dynamic range. Methods to overcome these limitations are presented in [Kühn et al. \(2007b\)](#).

Unlike diode-based sensors, thermocouple probes are true square-law detectors. Such sensors are particularly useful in free-space field surveys ([Narda STS, 2005](#)). These sensors are, however,

impractical for dosimetric and near-field measurements because of their size, generally lower sensitivity and dynamic range.

Thermistors are also small true square-law detectors. They can have a higher resolution than thermocouples, but need more frequent calibration.

The performance of these probes depends strongly on the following parameters:

- Frequency, modulation, and field strength;
- Polarization, direction of propagation, and field gradients;
- Material boundaries near the probe sensors;
- Sources of interference (noise, static and low-frequency fields, vibration, temperature, etc.).

The influence of these parameters must be characterized by individual calibration under well-defined conditions for each probe. A detailed summary of different calibration methods for field probes and a characterization of the most crucial parameters contributing to the measurement of uncertainty are given in [Poković \(1999\)](#). The influence of these parameters must

be included in the resulting uncertainty assessment, since the conditions of actual use of the probes may differ considerably from the conditions under which they are calibrated.

Modern free-space and dosimetric field probes operate in the frequency band from 10 MHz up to 6 GHz. They have an isotropy error smaller than ± 0.5 dB and sensitivities in the range 5–10 $\mu\text{W/g}$. These probes have very small sensor tips (2.5 mm) to allow high spatial resolution and measurements very close to material boundaries. A probe with reduced size (tip diameter, 1.0 mm) has been described for accurate dosimetric measurements at frequencies exceeding 10 GHz ([Poković et al., 2000a](#)). Probes for determining both the electrical- and magnetic-field pseudo-vector information are presented in [Poković et al. \(2000b\)](#).

(b) *Electro-optical sensors*

Modern, electro-optical sensors allow the measurement of the full RF-frequency domain and phase as well as intermediate-frequency time-domain signal information while maintaining a superior electrical isolation through the use of optical fibres for signal transmission.

In general, two sensor concepts are used today: (1) passive optical sensors ([Togo et al., 2007](#)); and (2) active optical sensors [Kramer et al. \(2006\)](#). Modern passive electro-optical sensors typically modulate the information on laser light passing through electro-optically active crystals embedded in a fibre-optic system. Common crystal materials include cadmium telluride (CdTe) and lithium niobate (LiNbO_3), which change their refractive indices depending on the E-field applied across the crystal, or cadmium manganese telluride (CdMnTe), which is sensitive to the magnetic fields applied across the crystal ([Poković, 1999](#)). The sensitivities of modern active optical sensors can be as low as 100 $\mu\text{V/m}$ per Hz^2 , or greater than 0.3 V/m when measuring a signal of width of 5 MHz ([Kramer et al., 2006](#)).

1.4.3 *Measurement antennae*

Different types of broadband-matched antennae are usually applied for the frequency-selective exposure assessment of incident fields. These broadband antennae are matched to 50 ohm to be compatible with standard RF receivers. They have applications in far-field measurement of radiation, e.g. from cellular base stations and broadcast services.

Common broadband RF-measurement antennae such as horn or log-periodic antennae have a certain directivity. This reduces the applicability of these antennae for complex propagation scenarios, particularly at locations where the incident field is not dominated by a direct line-of-sight propagation path, but by multipath propagation ([Kühn, 2009](#)).

Tuned dipole antennae have an isotropic pattern (no directivity) in azimuth, but lack broadband characteristics.

Conical dipole antennae have an isotropic pattern in azimuth and generally good broadband characteristics ([Seibersdorf Research, 2011](#)), which substantially reduces the number of measurements needed.

1.4.4 *Temperature instrumentation*

(a) *Temperature probes*

Local SAR values can also be assessed by temperature measurements (see Section 1.3); however, thermal-diffusion effects must be practically absent. This is only possible if the system is in thermal equilibrium at the beginning of the exposure, or if heat-diffusion processes are known for the assessment period. Heat losses due to radiation and convection during the measurement interval must be negligible, or known and corrected for. If these heat-diffusion processes are unknown, the response time of the thermal measurement equipment must be sufficiently short to avoid underestimation of the exposure ([Schuderer et al., 2004a](#)).

Two types of temperature probes exist: thermistor-based and those based on optical effects. The requirements for temperature probes for SAR assessments are:

- Small size: the probe must be small to resolve high temperature gradients, without disturbing the temperature distribution or the RF field;
- Non-conductive materials: only electrically non-conductive materials prevent heating of the probe by induced currents because they are transparent to EMFs;
- Low noise level: small differences in temperature must be detected accurately, especially for dynamic temperature measurements, e.g. of SAR, and thus the noise level should be much less than 10 mK;
- Short response time: this is essential for SAR measurements as the temperature rise (dT/dt) is proportional to the SAR only in the absence of heat diffusion. A probe suitable for SAR measurements must have reaction times much faster than 100 ms ([Schuderer et al., 2004a](#)).

A novel design for temperature probes for dosimetric assessments, introduced by [Schuderer et al. \(2004a\)](#), provides a spatial resolution of 0.02 mm², a noise level of the temperature of 4 mK, and a sensitivity of 0.5 mK/s with a response time of < 14 ms.

Temperature probes based on thermo-optical effects are applied in high-voltage transformers, industrial microwave ovens and in treatment for hyperthermia. One exploited effect is the decay rate of a phosphorescent layer at the tip of a fibre-optic cable ([Wickersheim & Sun, 1987](#)). These commercially available probes have a noise level of 0.1 K, with reaction times of 250 ms. Another optical effect is the interferometric property of a cavity filled with materials that have highly temperature-dependent refractive indices. These probes reach sensitivities of 2–3 mK/s ([Burkhardt et al., 1996](#)).

(b) *Infrared photography*

The measurement of temperature by black-body-equivalent radiation (infrared photography) is an alternative to invasive measurements using temperature probes. The resolution of infrared thermographs can be very high and the sensitivity of affordable infrared detection systems has improved substantially over the past 30 years. This was also one of the first methods used to measure SAR ([Guy, 1971](#)), as the surface radiation can be recorded quickly with infrared cameras without perturbing the incident field. Infrared cameras were used to measure the temperature increase on a human head exposed to GSM mobile phones ([Taurisano & Vander Vorst, 2000](#)). The technique has several disadvantages:

- Limited sensitivity compared with temperature or dosimetric probes;
- Can be used only for measurements of surface temperature;
- The thermal radiation characteristics of the materials must be determined accurately;
- The background radiation must be homogeneous;
- Evaporation and convection can cause substantial errors and must be controlled;
- Different viewing angles of the camera can yield different results, since surfaces are not isotropic infrared radiators.

(c) *Microcapsulated thermo-chromic liquid crystals*

A novel idea to assess three-dimensional temperature distributions optically and in quasi real-time was proposed by [Baba et al. \(2005\)](#). Microcapsulated thermochromic liquid crystals (MTLC) were suspended uniformly in a gel with the dielectric properties of human muscle tissue. The temperature of the gel is determined by measuring the light scattered from a laser beam

that scans through the liquid. The technique has limited dynamic range and sensitivity.

(d) *Calorimeters*

Calorimetry encompasses methods for measuring heat produced by biological, chemical or physical endothermic or exothermic processes. Calorimetric methods are suitable for determining average *wbSAR*, but they cannot provide information about *SAR* distribution.

Calorimetry can be subdivided into two types:

- Direct calorimetry: the heat is measured directly by use of calorimeters;
- Indirect calorimetry: the quantity of heat is determined by measuring the amount of oxygen consumption and relating it to the oxaloric equivalent of the reaction.

Basically, calorimetric dosimetry analyses the heating and cooling processes of a sample exposed to RF radiation. Typical direct calorimeters used in microwave dosimetry are the Dewar flask and the twin-well calorimeter ([Gajsek et al., 2003](#)).

1.4.5 *Measuring SAR and the near field*

Dosimetric evaluation inside test phantoms such as SAM requires the measurement of *SAR* at several hundreds of points distributed over a complex three-dimensional phantom. The process is divided into: (1) searching for the location of the maximum absorption on a two-dimensional grid; and (2) determining the *psSAR* value on a fine three-dimensional grid. These points must be determined with high accuracy, especially at high frequencies, to achieve low measurement uncertainty despite high attenuation and large variations in spatial-field intensity. Automated systems for dosimetric assessment have been developed to perform these compliance tests. A typical system for dosimetric assessment is a computer-controlled six-axis robotic positioner. It is used to move the dosimetric E-field probe

within a scanning grid, which can be adaptive, e.g. it follows the surface that is being detected during the scanning job and positions the probe axis orthogonal to that surface. The measurement results, i.e. field and *SAR* distributions, as well as 1 g and 10 g spatial average peak *SAR*, are automatically evaluated and visualized. The expanded standard uncertainty ($k = 2$) is less than 20%. It should be noted that this approach provides reliable conservative estimates of the maximum peak spatial *SAR* that might occur in the user population, but offers little information about the exposure of specific tissues or individual exposure ([Kühn, 2009](#)).

In summary, compliance evaluation of body-mounted transceivers provides reliable conservative estimates of maximum *psSAR*-1 g and *psSAR*-10 g anywhere in the body, but these estimates are generally poorly correlated with the maximum exposure of specific tissues (e.g. brain tissue) or typical exposure levels during daily usage of the device (system- and network-dependent). In other words, the information has only limited value for epidemiological studies.

1.4.6 *Incident-field measurements in the far field*

Evaluation of the exposure in the far field of a transmitter is usually conducted for fixed installations such as radio and television broadcast antennae, radar sites, or cellular base stations. Exposure assessments are carried out in areas that are generally accessible or for which access is restricted to qualified working personnel only. Compliance is tested with respect to the reference levels by assuming free-space field impedance for the RF energy, i.e. by E-field evaluation. Only one measurement point is required under real far-field conditions. However, actual environments usually involve nearby reflectors and scatterers, i.e. a scanning procedure is required to find the maximum incident fields ([Kühn, 2009](#)).

Since the transmitters under evaluation do not always operate at maximum power – the transmitted power of base stations being dependent on traffic intensity – broadband instantaneous measurements are often insufficient to determine the highest level of exposure. In such cases, information on the maximum exposure with respect to the measured values must be available and soundly applied to establish exposure in the worst-case scenario. [Table 1.12](#) lists the parameters necessary for extrapolation of exposure in the worst case and to reduce the uncertainty of the actual measurement campaign. It is easier to determine the measurement methods when additional parameters are known. General sources of error are:

- Field perturbation by measurement personnel, e.g. scattering and absorption of EMFs due to the body of the measurement engineer;
- Application of an inappropriate measurement antenna, e.g. disregard for antenna directivity and polarization;
- Application of ineffectively decoupled cables, acting as secondary antennae;
- Application of incorrect measurement settings of the RF receiver for the type of signal to be measured;
- Incorrect selection of the measurement location, e.g. measurement points that are not appropriate for yielding the maximum EMF exposure or measurement points close to bodies that influence the calibration of the measurement antennae ([Kühn, 2009](#)).

Different methods for assessing EMF exposure in the far field have been proposed. One approach is the antenna-sweeping method. This method requires the engineer to slowly move the measurement antenna with varying polarizations and directions through the volume of interest ([Sektion NIS, 2002](#)). Another method is based on the examination of several well defined points in the area of interest. In this case, the antenna is

mounted on a tripod and the different directions and polarizations are examined at the considered points ([ANFR, 2004](#)). The first method is conservative, but sensitive to the position of the operator with respect to the antenna. With the second method, measurements can be performed with the engineer located further away, but the number of measurements in the volume is small. A combination of both methods is presented by [Coray et al. \(2002\)](#), who suggest that the region is first scanned for the field maximum in the area of interest and that an isotropic and frequency-selective measurement is then performed at the location of the maximum.

Often, far-field techniques are employed in the near field of transmitters, e.g. on transmitter towers. Some standards allow a spatial averaging of E-field evaluations ([ANSI/IEEE, 1991](#)), the rationale of which is based on the wbSAR limit. However, this constitutes a relaxation of the safety criteria as it does not consider H-field coupling as the dominant mechanism in the near field nor the limits of psSAR. On the basis of current knowledge, such relaxations do not exclude the possibility of exceeding the basic restrictions or underestimating the local exposure ([Kühn, 2009](#)).

The advantages and limitations of different measurement equipment for assessing the exposure of unknown transmitters are discussed below.

1.4.7 Broadband measurements

Broadband-measurement probes are single-axis or three-axis sensors (dipole or loop) constructed in a similar way to the near-field sensors. No information on the spectral characteristics of the field is provided by these probes. Therefore, if a broadband meter is used for compliance testing, the measured field value must be no higher than the lowest permissible limit defined for the frequency range of the meter. Broadband survey meters are also

Table 1.12 Important parameters of radiofrequency transmitter sites assessed in the far field

Site parameter	Explanation
Location	The location of the transmitter with respect to the measurement point
Line of sight/nonline of sight	Determines if a prevalent propagation path may be expected
Type of site	Single or multiple antenna site
Antenna directivity	Antenna beam characteristics
Antenna radiation direction	The direction of maximum radiation
Antenna power at measurement	The antenna input power at the time the measurement takes place
Maximum antenna input power	Maximum permissible antenna input power
Frequency	Frequencies at which the site transmits
Communication system	Communication system that is used, i.e. which signal modulation characteristics are to be expected
Other sources of radiation	The field at the measurement points when the assessed transmitter is switched off

Adapted from [Kühn \(2009\)](#)

relatively inexpensive and easy to use, and are thus often used for field-survey measurements ([Kühn, 2009](#)).

Fig. 1.15 displays the components of typical broadband field-survey meters. Fig 1.16 shows the frequency response of two broadband probes.

Some broadband probes are designed to match the frequency dependence of the human exposure limits. In all cases, it is advised that the out-of-band response of these instruments is carefully characterized to avoid spurious readings. If a specific transmitter is the dominant source, compliance testing is substantially simplified ([CENELEC, 2005](#)).

The main sources of uncertainty regarding broadband survey meters are: calibration, linearity, frequency response, isotropy, time-domain response, and temperature response; so the accuracy of broadband evaluations is significantly limited, but generally conservative ([Kühn, 2009](#)).

(a) Frequency-selective measurements

Frequency-selective measurement techniques can overcome the difficulties of the unknown spectrum of the field. However, the execution of the measurement is more complicated and requires specialized engineers.

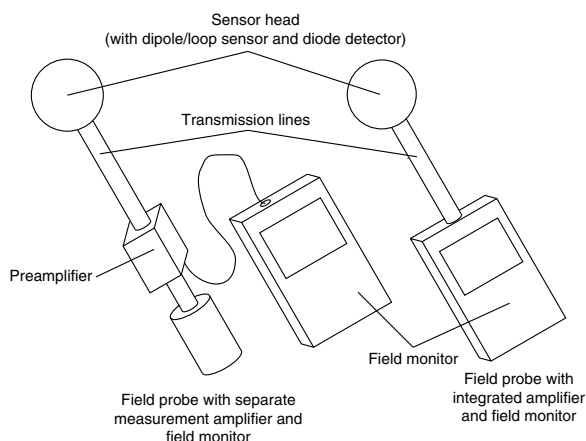
Measurements in the frequency domain are performed with an antenna connected to a spectrum analyser.

Most spectrum analysers provide video filters for additional smoothing of the spectral signal. Optimal parameter settings for the analyser for GSM and UMTS based on a simulation approach have been presented ([Olivier & Martens, 2005, 2006](#)). The application of spectrum analysers is a complex topic. Procedures dealing with frequency-selective measurements should always describe the parameter settings of the spectrum analyser to produce correct, reproducible and comparable results. Nevertheless, the engineer should test the actual applicability of these settings for the particular measurement equipment ([Kühn, 2009](#)).

The main sources of uncertainty regarding frequency-selective measurements are:

- Calibration of the spectrum analyser, cable, and measurement antenna;
- Linearity of the spectrum analyser, cable, and measurement antenna;
- Frequency response of the spectrum analyser, cable, and measurement antenna;
- Demodulation method of the spectrum analyser (detector type);

Fig. 1.15 Schematic of the most common broadband radiofrequency field-survey meters



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- Temperature response of the spectrum analyser, cable, and measurement antenna; and
- Mismatch between measurement equipment.

Although frequency-selective measurement methods overcome most of the problems affecting use of broadband-survey meters, they are not always sufficient to correctly evaluate exposure from different transmitters operating at the same frequency. In this case, measurement receivers should be applied ([Kühn, 2009](#)), as presented below.

(b) Code-selective measurements

Code-selective measurements are specifically necessary when the exposure from a transmitter involves code-division multiple access (CDMA), e.g. when a Universal Mobile Telecommunications System (UMTS) is to be assessed. All UMTS base stations usually transmit in the same frequency band. With a frequency-selective receiver, it is not possible to discriminate between exposures from different base stations, because a single frequency band is used and the channels are multiplexed in the code domain. Code-selective

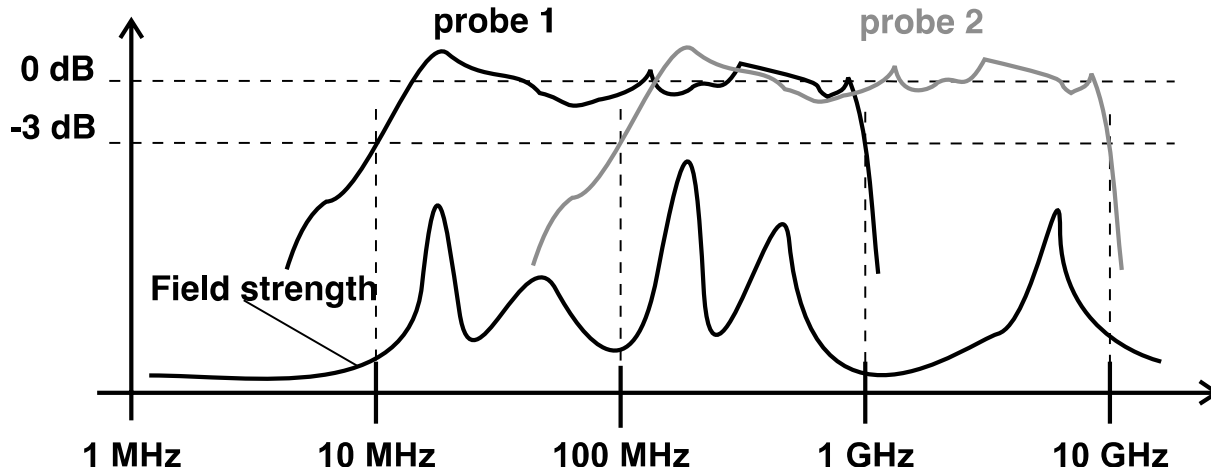
receivers decode the signal received from a base station, i.e. the receiver is able to discriminate between the received field strength from its base station and other noise-like sources. The receiver measures only the field received from its transmitting base station if the particular descrambling code is used for decoding. Basically, the same sources of uncertainty must be considered for code- and frequency-selective measurements. In general, if measurement receivers are applied, the overestimation of the measured field values is expected to be smaller than for frequency-selective and broadband measurements ([Kühn, 2009](#)).

1.4.8 Calibration

Measurement with known uncertainty can only be performed if the measurement equipment is appropriately calibrated. In general, calibration of the measurement equipment is demanding (sensitivity as a function of frequency and modulation, linearity for different modulations, deviation from isotropy, etc.). High-quality calibration documentation is essential to determine the accuracy of the measurements or their uncertainty, respectively.

1.4.9 Uncertainty assessment

Exposure assessments are prone to many uncertainties that must be carefully determined. This is the most difficult aspect of any measurement protocol, because it usually covers many more parameters than only the uncertainties associated with the measurement equipment. For example, in the case of demonstration of compliance with respect to basic restrictions, it includes estimation of the coverage factor for the exposed populations. In the case of uniform incident field, it is necessary to determine the uncertainty of the field measured during the period of measurement with respect to the maximum exposure at this site. In case of non-uniform fields, it needs to

Fig. 1.16 Frequency response of two broadband radiofrequency field-survey meters

The fields in the frequency ranges are summed. If the outputs from probes 1 and 2 are added, then the fields in the overlapping frequency range are counted twice. The field values measured with probe 1 must comply with the lowest limit in the frequency band 10 MHz to 1 GHz, while the readout of probe 2 must comply with the lowest limit between 100 MHz and 10 GHz. The overlapping frequency range is surveyed twice if the exposure values are superimposed to cover the entire frequency range. Republished with permission of Taylor and Francis Group LLC Books, from [Kühn & Kuster \(2007\)](#); permission conveyed through Copyright Clearance Center, Inc.

be demonstrated that the ratio of measured fields to reference levels is conservative with respect to the induced fields.

[The Working Group noted that it should be good measurement practice for the results of any measurement campaign to be presented only when accompanied by an extensive uncertainty assessment.]

1.4.10 Specific measurement problems

(a) Demonstration of compliance with dosimetric safety limits

The objective of compliance demonstration is to determine the exposure conservatively for the range of intended usage of the device or equipment with respect to the entire user group. In general, there is a strong dependence on position, distance, anatomy and posture. This dependence can only be determined by numerical simulations. Given an acceptable uncertainty, several hundred permutations of the most important parameters must be performed. In other words, the parameter space is large and the assessment

must be done with sufficient care and followed by an extensive discussion on the parameters investigated and the resulting uncertainties.

(b) Assessing personal exposure

In its most recent update of *Research Agenda for Radiofrequency Fields*, the World Health Organization (WHO) has recommended improvement of exposure assessment in epidemiological studies as a high-priority research need: “Quantify personal exposures from a range of radiofrequency sources and identify the determinants of exposure in the general population” ([WHO, 2010a](#)).

Associated problems with personal exposure assessment are:

- Compliance tests versus real-life exposure;
- Assessment of incident versus induced fields;
- Appropriate dosimetric quantities;
- Combination of exposure from multiple sources operating at different distances and frequencies;

- Strong temporal, geographical and usage dependence of the exposure, especially in relation to the exposure period relevant to the epidemiological data;
- Technology dependence of exposure and rapid technological changes; and
- Selection and, even more importantly, exclusion of potential exposure proxies.

As mentioned before, the worst-case levels of exposure determined during compliance testing of, e.g. mobile phones or base stations are in many cases not representative of actual real-life and everyday exposure. The protocols for compliance testing are generally optimized to provide a conservative estimate of maximum exposure. However, exposure assessment in epidemiological research aims at categorizing actual personal exposure. Results from compliance testing can, however, be useful to validate propagation models ([Bürge et al., 2008](#)), or to compare potential proxies that can be independently assessed, such as those based on mobile-phone design ([Kühn, 2009](#)).

Assessment and dosimetry of EMF exposure in epidemiological and human studies have been and often still are performed in terms of quantities that are only representative for demonstration of compliance with safety guidelines, e.g. incident-field quantification, or induced wbSAR and psSAR. The dosimetric meaning of the aforementioned quantities is questionable for current studies, which all aim at detecting potential effects for exposures well below established safety levels. In addition, the end-points investigated are typically effects on specific tissues, organs or functional regions of the brain and the quantification of the classical dose evaluations often does not allow a clear distinction between body regions or an accumulation of the dose from various sources. Quantification of exposure in terms of incident fields is especially problematic, since incident fields are often not directly related to induced fields. A common mistake is to combine exposure in terms of incident fields at different

frequencies by applying the root-sum-square (see Glossary) over the individual frequency contributions. Currently, novel dosimetric models are being developed to relate incident to induced EMF ([Djafarzadeh et al., 2009](#)) or to relate SAR-compliance test data measured in homogeneous media to SAR in specific anatomical regions of the human brain ([Gosselin et al., 2011](#)). By expressing exposure directly in terms of induced EMF or SAR in specific regions, the combination of multiple sources also becomes straightforward. Also, it allows a direct assessment of different source contributions according to geographical location or usage.

Variations due to geographical location in the far-field of transmitters should only be addressed with validated propagation models (e.g. [Andersen et al., 2007](#); [Frei et al., 2009a](#)), and not with, e.g. simplistic distance metrics. For near-field exposure, e.g. from mobile phones, the orientation of the source with respect to the body is relatively well defined; however, due to the output power control of modern mobile devices, there can be large variation in exposure depending on geographical location (more than twofold) and, even more importantly, the communication system (a factor of 100 or more). The assessment of these variations is typically addressed in terms of measurements in situ ([Wiart et al., 2000](#); [Kühn, 2009](#); [Kelsh et al., 2010](#)).

(c) *Measurement of the very close near field below 10 MHz*

The assessment of human exposure at frequencies between 30 MHz and 6 GHz is well established. International standards and national guidelines provide detailed assessment methods that are well specified with relatively low uncertainty. The measurement of incident fields at frequencies below 10 MHz is also well established. However, there is comparatively little research on the measurement of induced fields at frequencies below 10 MHz. The problems

associated with induced-field measurement at frequencies below 10 MHz include:

- Strong spatial non-uniformity of the fields, requiring high resolution of measurements;
- Strong temporal variation in the fields, especially from signals with transients, requiring equipment to have a large operating bandwidth;
- Field values measured very close to the source greatly overestimate the induced values, i.e. compliance often needs to be demonstrated by assessment of the induced fields;
- High variation in the permittivity and conductivity of tissues, making human modelling (e.g. development of phantoms) difficult;
- Practical limitations in the use of time-domain numerical electromagnetic solvers at low frequencies, resulting in slow convergence; and
- Limitations in the applicability of certain frequency-domain numerical electromagnetic solvers (e.g. electro-quasistatic solvers) due to assumptions and approximations.

(d) Measurement of signals with complex modulations

Today, most broadband field probes, as well as personal exposure meters, are calibrated with narrow-band (single-frequency) continuous wave signals. However, the measured signals differ greatly from continuous wave signals in terms of variation in time-domain amplitude and signal bandwidth. Variation in the time-domain amplitude of modern communication signals (peak-to-average power ratio [PAPR] of up to 14) places great demands on the linearity of the detectors in broadband probes and exposure meters, and in spectrum analysers. These requirements are often not fulfilled for the detectors and filters in traditional field probes

and exposure meters, such that these respond differently when comparing continuous wave and waveforms applying modern modulation schemes. For compliance testing, field probes can be calibrated with the actual test signals. For measurements in situ, e.g. with exposure meters, such a calibration is not straightforward since the real-life communication-signal characteristics might not always remain constant during measurement. Care should be taken also when using narrow-band receivers, i.e. spectrum analysers, when measuring complex-modulation waveforms. Also, these receivers require modulation-specific measurement settings, e.g. filter, detector, resolution bandwidth, sweep time etc. to perform field measurements with reasonably small uncertainties ([Joseph et al., 2002, 2008](#); [Olivier & Martens, 2005, 2006](#)).

1.5 Interaction of RF-EMF with biological systems

Although numerous experimental studies have been published on the non-thermal biological effects of RF-EMF, multiple computational analyses based on biophysical and thermodynamic considerations have concluded that it is theoretically implausible for physiological effects (except for reactions mediated by free radical pairs) to be induced at exposure intensities that do not cause an increase in tissue temperature ([Foster, 2000](#); [Adair, 2002, 2003](#); [Sheppard et al., 2008](#)).

RF electromagnetic radiation is classed as non-ionizing radiation as it comprises photons that do not have sufficient energy to break chemical bonds or ionize biological molecules ([Stuchly, 1979](#)). The energy of a photon of an electromagnetic wave is given by $E = hf$, where h is Planck's constant (6.626×10^{-34} J·s or 4.136×10^{-15} eV·s) and f is frequency, thus the energy of a photon in the RF spectrum varies from approximately 4.1×10^{-6} eV (6.6×10^{-25} J) at 1 GHz to 1.2×10^{-3} eV

(2.0×10^{-22} J) at 300 GHz. This is thus far less than the minimum amount of energy needed to ionize organic materials or metals, which is approximately 5–10 eV.

When a biological body (animal or human) or tissue is exposed to an RF-EMF, the RF energy is scattered and attenuated as it penetrates body tissues. Energy absorption is largely a function of the radiation frequency and the composition of the exposed tissue. Because of the high dielectric constant of water, the water content of the tissue determines to a large extent the penetration of a frequency-specified electromagnetic wave. The rate of energy absorbed by or deposited per unit mass per unit time is the specific absorption rate (SAR); this value is proportional to the root-mean-square (rms) of the induced electrical field strength $[E]^2$ and to the electrical conductivity (σ) of the tissue per tissue density (ρ):

$$\text{SAR} = [E]^2 \cdot \sigma / \rho$$

The SAR expressed in units of watts per kilogram (or mW/g) can also be estimated from measurements of the rise in temperature caused by RF-energy absorption in tissue:

$$\text{SAR} = C_p \cdot \delta T / \delta t$$

where C_p is the specific heat of the tissue or medium, $\delta T / \delta t$ is the initial rise in temperature over time. Values for the dielectric constant and conductivity vary substantially over the RF range (30 MHz to 300 GHz).

To cause a biological response, the EMF must penetrate the exposed biological system and induce internal EMFs. RF-energy absorption depends on incident field parameters (frequency, intensity, polarization), zone of exposure (near field or far field), characteristics of the exposed object (size, geometry, dielectric permittivity and electric conductivity), and absorption or scattering effects of objects near the exposed body ([Stuchly, 1979](#)).

Based on the relationship between wavelength (λ) and frequency

$$c = f \cdot \lambda$$

where c is the speed of light (3×10^8 m/s), it is obvious that the wavelength of RF radiation varies substantially between 30 kHz (10 km) and 300 GHz (0.1 cm). At the frequencies used for mobile phones (approximately 1–2 GHz), the corresponding wavelengths are 30 and 15 cm. Considering that near-field exposures occur at distances from a radiating antenna within approximately one wavelength of the radiated EMF and that far-field exposures occur at distances that exceed one wavelength of the radiated EMF, it is clear that reactive near-field and far-field exposures may occur, depending on the frequency of the incident field and the distance of the exposed person from the radiating antenna. Both near-field and far-field exposures can occur with the use of wireless telecommunication devices. In the near-field region, the electric and magnetic fields are decoupled and not uniform, wave impedance varies from point to point, power is transferred back and forth between the antenna and the surrounding object, and the energy distribution is a function of both the incident angle and distance from the antenna ([Lin, 2007](#)). Because the electric and magnetic fields are decoupled in the near field, the induced field can be obtained by combining the independent strengths of the electric and magnetic fields, i.e. the electric and the magnetically induced electric fields inside the body ([Lin, 2007](#)).

1.5.1 Thermal effects

The most recognized effect of RF radiation in biological systems is tissue heating. The absorption of RF-EMF energy by biological systems generates an oscillating current that is transferred into molecular motion of charged particles and water molecules, which are strongly dipolar and

are the major component of biological tissues. Polar molecules move to align themselves with the EMF to minimize the potential energy of the dipoles. Absorption and resonant oscillations in polar subgroups of macromolecules (e.g. proteins, DNA) are largely damped by collisions with surrounding water molecules. Damping or friction slows the motion of the oscillator. These collisions disperse the energy of the RF signal into random molecular motion. Tissue heating occurs because the rotational motion of molecular dipoles is hindered by the viscosity of water and interactions with other molecules, i.e. the rotational energy is transferred to the surrounding aqueous environment as heat. The magnitude of motion that results from the interaction of polar substances with electric fields is dependent on the strength and frequency of the field. In addition, the actual increase in temperature is dependent on the ability of the organism to thermoregulate. At high frequencies where the orientation of dipoles cannot keep up with the oscillations of the field, the system behaves like a non-polar substance ([Stuchly, 1979](#)).

As electrical fields penetrate complex biological tissues, the electric field is reduced as a result of dielectric constituents becoming polarized in response to the field. Standards for RF exposure of workers and the general population are based on protection against adverse effects that might occur due to increases in tissue or body temperature of 1 °C (wbSAR, ~4 W/kg) or less (after applying safety factors). Because RF-energy penetration and induced effects are dependent on the frequency of incident-field parameters and the composition of exposed tissues, quantifying SARs in small averaging regions is more relevant for evaluations of human health effects. Estimates of SARs in the head of individuals exposed to RF radiation during use of mobile phones that operate at a power output of 0.25 W indicate that the emitted energy would cause a rise in brain temperature of approximately 0.1 °C ([Van Leeuwen et al., 1999](#); [Wainwright, 2000](#));

therefore, it has been suggested it is unlikely that effects in the brain would be caused by increases in temperature ([Repacholi, 2001](#)). However, it is possible that temperature-sensitive molecular and physiological effects occur already with an increase of the temperature of ≤ 0.1 °C, while temperature changes approaching 1 °C are likely to affect several biological processes ([Foster & Glaser, 2007](#)).

Rates of temperature increase may be important in affecting a physiological change. Indeed, microwave-induced heating has been attributed to a rapid rate of heating 1–10 °C/s, which leads to acoustic waves due to expansion of tissue water. This auditory effect associated with brief pulses (1–10 μ s) at frequencies of 1–10 GHz and peak power-densities of $\sim 10^4$ W/m² (10³ mW/cm²) occurs with only small increases in temperature in the head ([Foster & Glaser, 2007](#)). Low levels of exposure to RF radiation may result in small temperature changes that cause conformational changes in temperature-sensitive proteins and induce the expression of heat-shock proteins; studies on the effects of low intensity RF-EMF exposures on temperature changes and expression of heat-shock proteins are described in Section 4 of this *Monograph*.

1.5.2 Physiological effects

Non-thermal effects (or effects associated with a negligible increase in temperature) are defined as biological changes that occur with body temperature changes that are < 1 °C, below measurable heating, or in the range of thermal noise. Several arguments have been presented against the plausibility of a non-thermal mechanism by which RF radiation could affect physiological changes; these include: (a) damping effects of the water surrounding biological structures are too strong to allow resonances to exist at radiofrequencies ([Adair, 2002](#)); (b) the relaxation time – the time for a molecule to return from an excited state to equilibrium – for excitations produced

by RF fields (e.g. vibrations in molecules), is similar to the relaxation time for thermal noise, and shorter than the lifetime of the absorption and transfer of energy into resonant modes of oscillating elements in biological systems ([Adair, 2003](#)); and (c) the perturbation of the biological structure induced by the applied field must be greater than the effects of random thermal motion and the effects of other dissipative forces, such as viscous damping by the surrounding medium ([Foster, 2000](#)). Random thermal motion of charged components in biological systems (i.e. thermal noise) creates random fluctuating EMFs. [Adair \(2003\)](#) has concluded that it is unlikely that RF radiation with a power density of less than 10 mW/cm^2 (100 W/m^2) could have a significant effect on biological processes by non-thermal mechanisms.

[Sheppard et al. \(2008\)](#) have evaluated several potential mechanisms of interaction of RF radiation with biological systems and concluded that, other than heating and possible effects on reactions mediated by free radical pairs, RF field strengths in excess of system noise (collisions among various molecular oscillators generated largely by thermal agitation) could not alter physiological activities without also causing detectable tissue heating. Some mechanistic considerations addressed by these authors include:

- *Endogenous electric fields* involved in physiological processes (e.g. embryonic development, wound healing, and neuronal activity) have strengths in the range 1–200 V/m. While neuronal circuit oscillations were affected *in vitro* by extremely low-frequency electric fields, no mechanisms for inducing changes in cell-membrane potential at frequencies above ~10 MHz have been demonstrated. Furthermore, the net field effect on such a biological system would be the sum of the endogenous and applied fields. Thus, to alter a biological response such as ion

transport through a membrane channel, the amplitude of the external signal would need to be of the same order of magnitude as the endogenous field ([Adair, 2003](#); [Sheppard et al., 2008](#)).

- *Specialized sensory systems* may be capable of detecting weak EMFs by integrating signals from numerous sensors over space and time. While specific sensory systems have been shown to exist for low-frequency, infrared and visible radiation, there is no evidence for the existence of RF-sensitive receptors in biological systems ([Sheppard et al., 2008](#)). However, some sensory systems may respond to very small increases in temperature ($< 0.1 \text{ }^\circ\text{C}$).
- *Effects of weak RF fields that do not cause heating* would be likely to require frequency-dependent resonant absorption or multiple-photon absorption to induce an amplified signal strong enough to overcome intrinsic molecular noise ([Sheppard et al., 2008](#)). This is because the photon energy of RF radiation is much smaller than thermal energy at body temperature ($k \cdot T$, where k is the Boltzmann constant, $1.38 \times 10^{-23} \text{ J/K}$ ($8.62 \times 10^{-5} \text{ eV/K}$), and T is the absolute temperature), i.e. $27 \times 10^{-3} \text{ eV}$ per oscillating mode at body temperature. However, biological systems appear to absorb RF signals like a broadband receiver rather than eliciting line spectra characteristic of resonant vibrational motion ([Prohofsky, 2004](#); [Sheppard et al., 2008](#)). In addition, RF electric field strengths of up to 200 V/m cannot transfer sufficient energy to organelles or biological molecules to alter biological activities or affect thermal noise (kT) fluctuations, such as the opening of voltage-gated ion channels, spatial arrangements of membrane-associated ions, collision rates of charged ligands with proteins, or enzyme reaction kinetics ([Adair, 2002, 2003](#);

[Sheppard et al., 2008](#)). [Adair \(2002\)](#) suggested that, while coupling of RF-EMF to biological systems may exhibit resonance behaviour, damping of the vibrational motion by interactions with the aqueous environment prevents the absorption of sufficient energy to induce a biological effect. To significantly affect a biological system, the response from the RF signal must be comparable to the effect of thermal noise ([Adair, 2003](#)).

- *RF-EMF may be directed to specific sites* of a biological structure, leading to local areas of enhanced field strength. However, the smallest focal spot of concentrated energy would have a radius of the order of a wavelength, which is much larger than most cells (e.g. at 300 GHz, $\lambda = 1000 \mu\text{m}$). Thus, on a cellular basis, RF-energy absorption is very small. [Fröhlich \(1968\)](#) has suggested that incident RF energy may be captured by a large group of oscillating dipoles and integrated into a single mode of coherent vibrational energy. For this to occur and produce a coherent response, [Sheppard et al. \(2008\)](#) suggested that the energy stored in the coupled oscillators would need to be comparable to thermal energy and protected from damping by water or other molecules. In addition, energy and thermal diffusion prevent the formation of significant temperature differences at the cellular and subcellular levels.
- *In order for RF electric fields to induce small changes in protein structure* that would affect binding of substrates or ligands to enzymes or receptor proteins, extremely high field strengths would be required ($\sim 10^9 \text{ V/m}$) ([Sheppard et al., 2008](#)).

Since living systems are not in thermal equilibrium, mechanistic theories on interactions between RF-EMF and biological tissues must consider the non-equilibrium and nonlinearity

of these systems. [Binhi & Rubin \(2007\)](#) suggest that biochemical effects may be induced by weak EMFs in targeted systems that are in non-equilibrium states in which the time to transition from an intermediate metastable state to a final active or inactive state may be less than the thermalization time of the induced field.

[Prohofsky \(2004\)](#) has suggested that protein conformation might be affected by RF radiation if amplitudes of specific vibrational modes were altered. However, only intermolecular vibrational modes of proteins and the surrounding tissue are possible at RF frequencies, because high-frequency intramolecular resonant vibrational modes exist above several hundred GHz. Further, this author concluded that the biological effects of RF radiation in macromolecules (proteins and DNA) can only be due to temperature changes, because the absorbed energy associated with intermolecular vibrations is rapidly thermalized; the relaxation time for coupling RF waves to surrounding water (i.e. damping) is faster than the speed with which it can be transferred to intramolecular resonant modes. A non-thermal effect might exist if there were a very strong energy coupling between the intermolecular and intramolecular modes. Exceptions to the above-mentioned considerations are proteins such as myoglobin or haemoglobin, in which the haem group can oscillate in the protein pocket at lower frequencies (184 GHz is the lowest mode in myoglobin) ([Prohofsky, 2004](#)).

Any theories on the potential effects on biological systems of RF energy at low field strengths must account for the facts that biological systems do not exist at equilibrium, that the dynamic nature of these systems is controlled by enzyme-mediated reactions, and that primary effects may be amplified by nonlinear biological processes ([Georgiou, 2010](#)). The reproducibility of reported effects may be influenced by exposure characteristics (including SAR or power density, duration of exposure, carrier frequency, type of modulation, polarization, continuous versus

intermittent exposures, pulsed-field variables, and background electromagnetic environment), biological parameters (including cell type, growth phase, cell density, sex, and age) and environmental conditions (including culture medium, aeration, and antioxidant levels) ([Belyaev, 2010](#)).

A biophysical theory on how low-intensity RF-EMF exposures might affect physiological functions involves the alteration of ligand binding to hydrophobic sites in receptor proteins ([Chiabrera et al., 2000](#)). Collisions of the ligand ion in the hydrophobic region of the receptor protein result in loss of its vibrational energy. In order for RF exposures to affect the binding probability of an ion ligand with a membrane protein receptor, basal metabolic energy would have to amplify the effect of the RF field by maintaining the cell in thermodynamic non-equilibrium. Otherwise, the low-intensity exposure would be negligible compared with thermal noise. Other elements of this model that were used to evaluate the effects of low-intensity RF exposures on ligand binding are the extremely fast (“instantaneous”) rearrangement of atoms in the hydrophobic core of protein by the ligand ion, the fact that the endogenous field at the protein boundaries is large enough to exclude water molecules from the hydrophobic core, and that the ion-collision frequency near the hydrophobic binding site is much less than it is in water. The authors of this study noted that thermal noise must be taken into account when evaluating potential biological effects of RF exposures ([Chiabrera et al., 2000](#)).

Demodulation of pulsed RF signals (e.g. GSM pulsed at 217 Hz) might produce low-frequency electric fields ([Challis, 2005](#)). To confirm a biological effect from a low-frequency, amplitude-modulated RF signal, a nonlinear response in the biological sample would be expected ([Balzano & Sheppard, 2003](#)). Except for the case of an incident flux of RF energy at extremely high field-strength pulses that causes mechanical vibrations, most oscillators in a biological system respond linearly to the incident low-energy

photons in the RF spectrum; the dispersion of RF energy into random molecular motion energy occurs without generating harmonics of the incident signal in the energy spectrum of re-radiated photons by the exposed material. However, the authors of this study considered the possibility that demodulation of high-frequency incident RF signals might produce nonlinear interactions with biochemically induced transient oscillators in living tissues (e.g. uncoupled electrons of free radicals) by extracting low-energy signals. If this occurred, then the spectrum of RF-emission energy emitted from the exposed tissue would be altered, producing a second harmonic that would show up as a spectral line at twice the frequency of the incident signal ([Balzano & Sheppard, 2003](#)). Sensitive, frequency-selective instruments are available to detect the presence of frequency-doubling signals produced by nonlinear interactions between amplitude-modulated RF signals and molecular oscillators vibrating in unison in living cells ([Balzano, 2003](#)). Exposure of several different types of cell and tissue to continuous wave fields (input powers of 0.1 or 1 mW) in a double-resonant cavity at the resonant frequency of the loaded cavity for each sample (~880–890 MHz) did not emit second harmonic signals at twice the frequency of the incident signal ([Kowalczyk et al., 2010](#)). SAR values were approximately 11 mW/g for cells and 2.5 mW/g for tissues exposed to 1 mW RF fields. Although these results were inconsistent with the hypothesis that living cells can act as effective radio receivers and demodulate RF energy, a second harmonic response may be elicited by much more intense continuous waves (which would be likely to cause rapid heating) or very short-pulsed RF signals ([Kowalczyk et al., 2010](#)). [Sheppard et al. \(2008\)](#) concluded that it is unlikely that modulated RF fields significantly affect physiological activities of membranes, because non-thermal stimulation of cell membranes has not been observed above approximately 10 MHz and the voltage across a cell membrane from an

amplitude-modulated RF electric field of 100 V/m is much lower than the low-frequency voltage noise associated with membrane voltage fluctuations. Much higher incident field strengths, at levels that would cause significant tissue heating, would be needed to create electric fields comparable with endogenous fields.

Lipid-protein complexes appear to be more sensitive to perturbations from RF radiation at membrane phase-transition temperatures ([Liburdy & Penn, 1984](#); [Allis & Sinha-Robinson, 1987](#)). [Blackman et al. \(1989\)](#) suggested that the chick brain surface is also poised at a phase transition at physiological temperatures, and the long-range order that occurs in such a state would minimize the thermal noise limitations calculated for single-phase systems on signal detection of weak RF radiation. Consistent with this hypothesis, [Blackman et al. \(1991\)](#) observed that RF radiation-induced calcium-ion efflux-changes occurred only within the narrow temperature range of 36–37 °C.

The aggregation of dielectric objects by attractive forces between them is referred to as the pearl-chain effect ([Challis, 2005](#)). RF fields of about 125 V/m and at frequencies of up to about 100 MHz can produce oscillating fields in cells that enhance their attraction. At higher frequencies the induced dipoles might not have sufficient time to reverse direction and, therefore, stronger fields would be needed to produce the same attractive energy.

Electroporation is a process by which short pulses (~100 µs) of strong electric fields (e.g. 10–100 kV/m) are applied to cell membranes to induce transient pores that allow uptake of drugs, DNA, or other membrane-impermeable substances ([Foster, 2000](#); [Sheppard et al., 2008](#)). These changes occur without causing significant tissue heating or thermal damage.

1.5.3 Magnetic-field effects

Low-frequency magnetic fields might produce biological effects if they induce ferromagnetic resonance in tissues that contain high concentrations of iron particles (magnetite) ([Challis, 2005](#)).

Free radicals, which are highly reactive molecules or ions with unpaired electrons, are formed when radical pairs dissociate. By altering the recombination of short-lived radical pairs with antiparallel spins, low-intensity magnetic fields may increase the concentration of free radicals ([Challis, 2005](#); [Georgiou, 2010](#)). The expected increase in radical concentration is 30% or less ([Timmel et al., 1998](#)). The extent to which this increase can produce oxidative stress-induced tissue damage (e.g. membrane-lipid peroxidation or DNA damage) is not known. Furthermore, radicals are also a part of normal cellular physiology, being involved in intracellular signal transduction ([Finkel, 2003](#)). Therefore, even small effects on radical concentration could potentially affect multiple biological functions. By prolonging the lifetime of free radicals, RF fields can increase the probability of free-radical-induced biological damage. To affect DNA recombination and thus the repair of damage caused by radicals, external magnetic fields must act over the times that the radical pairs dissociate ($> 10^{-9}$ s); hence, [Adair \(2003\)](#) concludes that the effect of RF fields on free-radical concentrations would likely be limited to about 10 MHz or less. Resonance phenomena occur below 10 MHz, and may result in biological effects from low-level RF fields at about 1 MHz ([Henbest et al., 2004](#); [Ritz et al., 2009](#)).

[Georgiou \(2010\)](#) cited several studies that provide evidence for the induction of oxidative stress via the free-radical pair mechanism in biological systems exposed to RF radiation; some of the reported effects include increased production of reactive oxygen species, enhancement of oxidative stress-related metabolic processes, an increase in DNA single-strand breaks, increased

lipid peroxidation, and alterations in the activities of enzymes associated with antioxidative defence. Furthermore, many of the changes observed in RF-exposed cells were prevented by (pre)treatment with antioxidants.

1.5.4 Conclusion

In conclusion, tissue heating is the best-established mechanism for RF radiation-induced effects in biological systems. However, there are also numerous reports of specific biological effects from modulated RF-EMF, particularly low-frequency modulated fields (see Section 4). Mechanistic studies will be needed to determine how effects that are reproducible might be occurring, e.g. via the induction of reactive oxygen species, induction of ferromagnetic resonance, demodulation of pulsed RF signals, or alteration of ligand binding to hydrophobic sites in receptor proteins. Although it has been argued that RF radiation cannot induce physiological effects at exposure intensities that do not cause an increase in tissue temperature, it is likely that not all mechanisms of interaction between weak RF-EMF (with the various signal modulations used in wireless communications) and biological structures have been discovered or fully characterized. Biological systems are complex and factors such as metabolic activity, growth phase, cell density, and antioxidant level might alter the potential effects of RF radiation. Alternative mechanisms will need to be considered and explored to explain consistently observed RF-dependent changes in controlled studies of biological exposure (see Section 4 for examples of reported biological effects). While the debate continues on whether or not non-thermal biological effects occur as a result of exposures to low-intensity RF radiation, it may be difficult to specify observed effects as non-thermal because of the high sensitivities of certain physiological responses to small increases in temperature.

1.6 Exposure to RF radiation

Exposure of workers and the general community to RF radiation can occur from many different sources and in a wide variety of circumstances. These exposures can be grouped into three major categories: personal, occupational and environmental.

1.6.1 Personal exposure

The general community can come into contact with several potentially important sources of RF radiation as part of their personal life, involving some degree of choice, including use of a mobile phone, other communication technologies, or household devices (see Section 1.2).

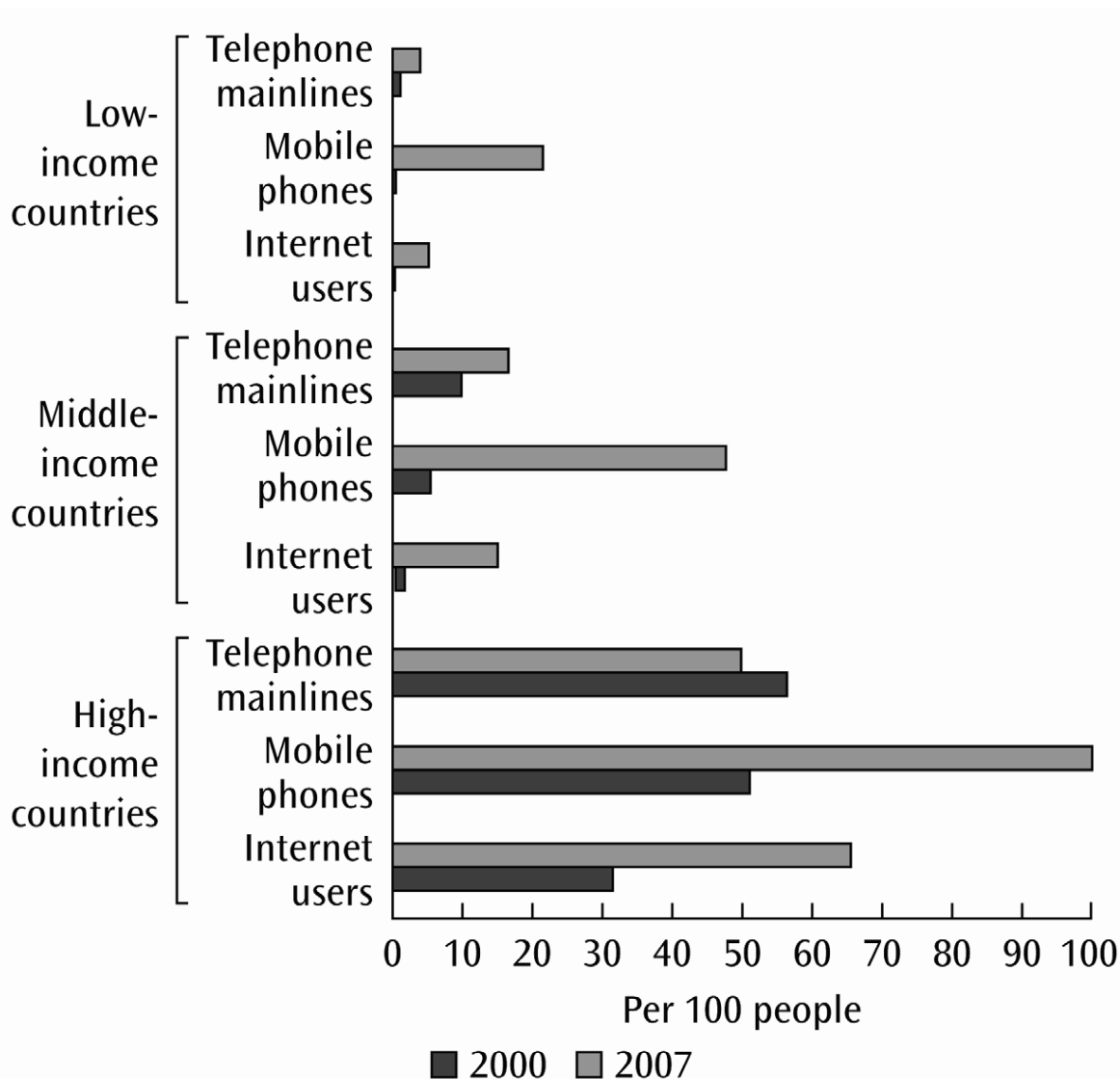
(a) Mobile phones

(i) Increase in mobile-phone subscriptions

Analogue mobile phones were first introduced around 1980 and GSM phones in the mid-1990s. Over the past two decades, the number of people owning a mobile phone has increased rapidly around the world. For example, the number of mobile-phone subscribers in the USA has risen from 0.34 million in 1985 to 109 million in 2000, and 263 million in 2008 ([InfoPlease, 2011](#)). WHO has estimated that at the end of 2009 there were 4.6 billion mobile-phone subscriptions globally ([WHO, 2010b](#)). Fig 1.17 illustrates the rapid rise in mobile-phone subscriptions compared with other types of phone and Internet usage over the past decade, although it should be noted that the number of subscriptions does not equate to number of users, as some people have more than one subscription and a single subscription can be used by more than one person.

This rapid increase in mobile-phone use is not just restricted to the industrialized countries. Fig 1.17 shows the increase in mobile-phone subscriptions from 2000 to 2007 in high-, middle- and low-income countries ([World Bank, 2009](#)). While industrialized countries continue to

Fig. 1.17 Mobile-phone subscriptions per 100 people in high-, middle-, and low-income countries, 2000 and 2007



© World Bank

From [World Bank; International Monetary Fund \(2009\)](#)

have the highest number of subscriptions per 100 people, the percentage increase over this time has been much greater in low- and middle-income countries. In low-income countries, subscription rates in 2000 were negligible, but in 2007 they were 25% of the rate in high-income countries, while in middle-income countries the rise was from about 10 to 50 subscriptions per 100 people, to reach about 50% of the rate in high-income countries in 2007.

There have also been considerable changes in the types of mobile phone used over the past 10 years, which has important implications for RF exposure of the user (see Section 1.2). Earlier mobile phones used analogue technology, which emitted waves of 450–900 MHz. Digital phones, with RF frequencies of up to 2200 MHz, were introduced in the mid-1990s and by the year 2000 had almost completely replaced analogue phones. The largest growth in recent years has been for smartphones, which allow the user access to a wide range of non-voice data applications (taking photographs, Internet access, playing games, music, and recording videos). In the USA, 18% of phones in 2010 were smartphones, up from 13% in 2008 ([Nielsen, 2010](#)).

(iii) Mobile-phone use among children

Within the increasing subscription figures, there have been questions raised about increasing use of mobile phones by children. As was seen in Section 1.3, published dosimetry studies using phantom heads have found that RF absorption can be higher in children than in adults, due to anatomical and physiological differences. A recent study used a modified version of the Interphone questionnaire in 317 children in secondary school (median age, 13 years) in one state of Australia, and found that 80% used a mobile phone ([Redmayne et al., 2010](#)). Data on national use of mobile phones in 2009, collected by the Australian Bureau of Statistics (ABS), has shown that 31% of Australian children had a mobile phone, with the highest ownership being

in the age group 12–14 years (76%) ([ABS, 2009](#)). Similar rates of mobile-phone use by children were found in three major cities in Hungary in 2005, where 76% of children in secondary school owned a mobile phone, 24% used a mobile phone daily to make calls, and an additional 33% used mobile phones to make calls at least several times per week ([Mezei et al., 2007](#)).

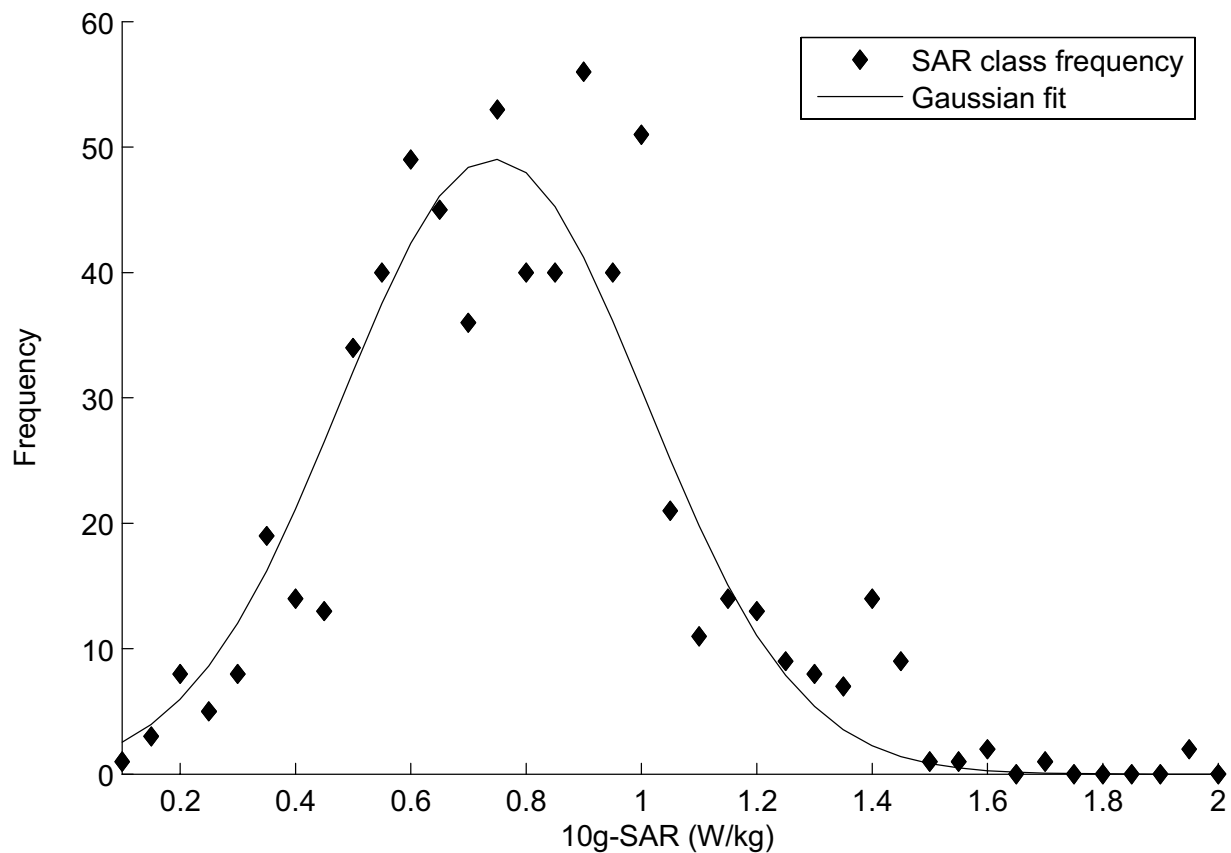
While the increase in mobile-phone subscriptions over the past 15 years is well documented, less is known about changes in call frequency and duration over that time. One study in Finland found that the median duration of calls per month was 186 minutes in 2007, increasing to 221 minutes in 2009, while the average monthly number of calls increased slightly from 52 to 57 calls ([Heinävaara et al., 2011](#)). The daily local RF exposure of the general public has increased by several orders of magnitude with the introduction and proliferation of mobile handsets. This has triggered concern among health agencies and the public, since the tissue with the highest exposure is the brain. Figs 1.18 and 1.19 display the frequency of worst-case SAR from mobile phones, measured according to [IEEE \(2003\)](#) and [CENELEC \(2001\)](#) guidelines.

Fig. 1.18 represents the typical SAR values for Europe (mean psSAR-10 g, 0.74) and Fig. 1.19 for North America (mean psSAR-1 g, 0.96). The different averaging masses are due to different legal regulations in Europe and the USA. These values are a considerable percentage of the limit values (see Section 1.7). A recent statistical analysis of the SAR database of the Federal Communications Commission (FCC) found that the SAR values of newer phones are typically lower than those of older phones, despite the greatly reduced size (see Section 1.3).

(iii) Exposure metrics for epidemiological studies

To develop suitable exposure metrics for use in epidemiological studies on RF exposure from mobile phones and health effects such as cancer, there is a need to access technical data such as

Fig. 1.18 Statistical distribution of maximum psSAR-10 g measured for 668 mobile phones, according to standard EN50361 ([CENELEC, 2001](#))



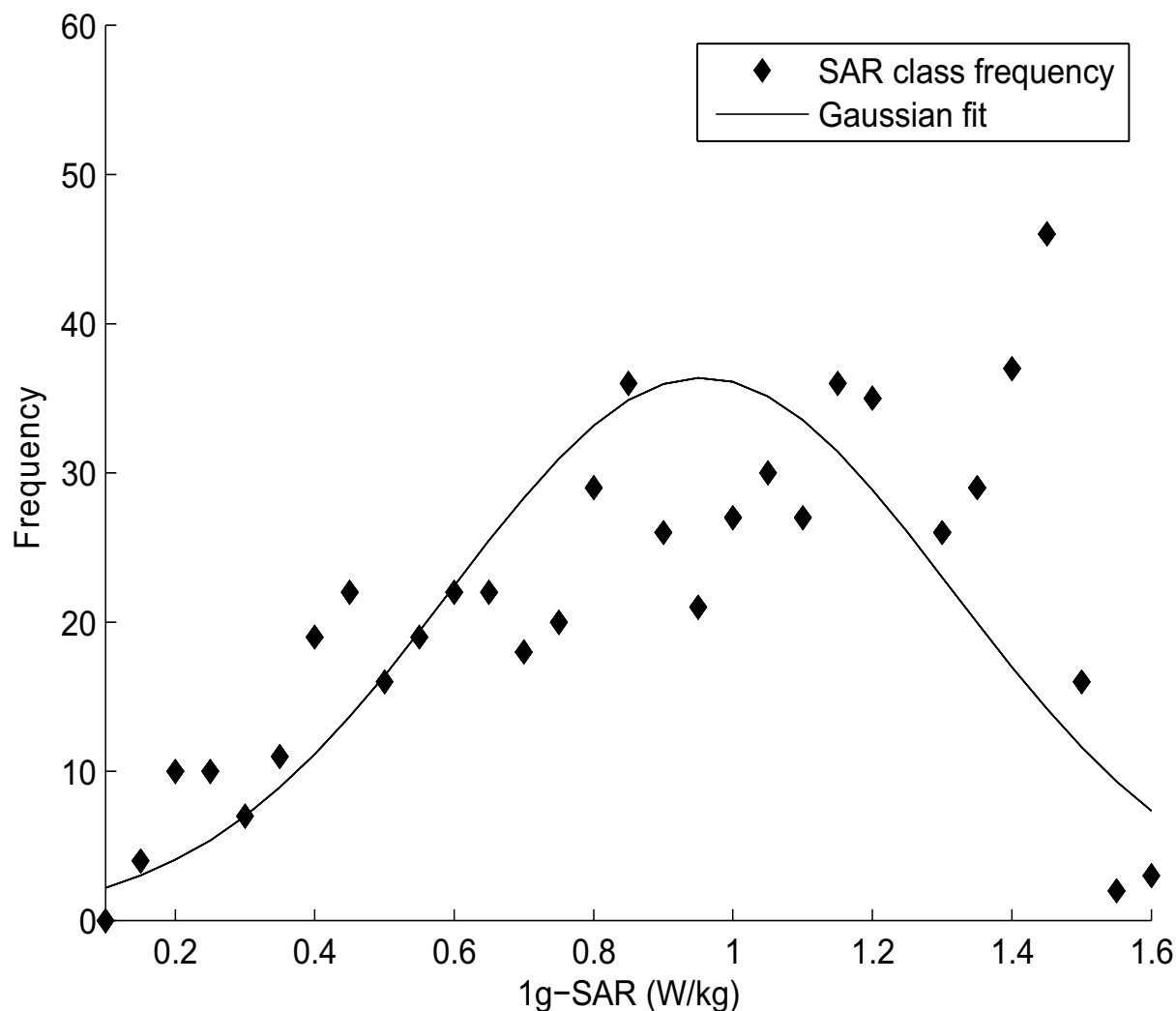
Data from German Federal Office for Radiation Protection, in [Kühn & Kuster \(2007\)](#)

the generation of phone, frequency, modulation and network-related factors that might influence the output power of the phone, as well as reliable information about the pattern of mobile-phone use from each subject. This includes such variables as reported number of calls, duration of calls and laterality, i.e. the side of the head on which the phone is most often placed by the subject when talking on the phone.

As exposure data related to mobile-phone use are usually collected from the subjects themselves, several studies have been conducted to test the validity of this type of self-reported information. Several methods are available to validate self-report, including telephone-company records, software-modified phones

and hardware-modified phones ([Inyang et al., 2008](#)). A study of 59 children in the seventh year of school (age 11–12 years) in Australia used GSM-type software-modified phones to record exposure details (e.g. number and duration of calls) to validate questionnaire data on mobile-phone use. This study found a modest correlation of 0.3 for recall of number of calls, but almost no correlation (0.1) for duration ([Inyang et al., 2009](#)). There was little difference with the main findings for different demographic groups, although for some subgroups, numbers were small. This study was carried out over one week and a possible explanation of the poor correlations is that the change in phone type imposed by the study protocol (from 3G to GSM) may have

Fig. 1.19 Statistical distribution of maximum psSAR-1 g measured for 687 mobile phones, according to standard IEEE-1528 ([CENELEC, 2001](#))



Data from Federal Communications Commission, in [Kühn & Kuster \(2007\)](#)

resulted in a change in phone-use behaviour for many of the children.

Another potential problem is differential recall of mobile-phone use in case-control studies. In the CEFALO case-control study of brain tumours in adolescents, a validation study was undertaken to estimate the effect of both random and systematic errors in 59 cases (26% of all cases who owned a mobile phone) and 91 controls (22% of all controls who owned a mobile phone) for whom phone-use data were available

from the mobile-phone provider ([Aydin *et al.*, 2011](#)). The study found that cases overestimated their number of calls by 9% on average, and controls overestimated by 34% on average. Cases also overestimated the duration of their calls by 52% on average, while controls overestimated by a much greater 163%, suggesting that duration-of-call data from self-reports are less reliable and may be more prone to recall bias than self-reports of number of calls in studies of cancer in children.

Such differential reporting between cases and controls was not such a problem in two validation studies undertaken as part of the Interphone case-control study of brain tumours in adults. A 6-month volunteer study used the Interphone questionnaire and either phone records or software-modified phones in 11 countries and found that, although there was considerable random error, there was fair to moderate agreement for both number and duration of calls, with weighted kappas ranging from 0.20 to 0.60 ([Vrijheid et al., 2006](#)). In addition, there was some systematic error, as heavy users tended to overestimate their use, while lighter users tended to underestimate theirs. There was also some heterogeneity between countries. A subsequent validation study among subjects from five countries in the Interphone study compared reported mobile-phone use against phone records over an average of two years. This substudy found that the extent of underreporting of number of calls (0.8) and of over-reporting of call duration (1.4) was similar in each group. Differential recall was greater with longer periods of recall, although numbers were small for the group with longest recall period ([Vrijheid et al., 2009b](#)). More recently, a pilot study in Finland for the prospective cohort study of mobile-phone users (COSMOS study) validated reported phone use against phone-company records for 418 subjects who had a single operator ([Heinävaara et al., 2011](#)). The authors found that overestimation of reported mobile-phone use was common and there was moderate agreement ($\kappa = 0.60$) for monthly average duration of calls, although there was more overestimation and less agreement as the call duration increased. A further small validation study in 60 engineers and scientists, who are not representative of the wider community, used mobile-phone records to validate self-reporting and found similar agreement; the conclusion was, that reporting monthly use was more reliable than weekly or daily use ([Shum et al., 2011](#)).

Laterality, i.e. against which ear the mobile phone is mainly held during calls, is another important factor that can influence estimations of exposure within the head. Laterality does not always coincide with the subject's dominant hand and may be related to other activities, such as writing. A validation study of self-reported laterality with hardware-modified phones found that agreement between the information from these phones and self-reported laterality was modest, with a kappa of only 0.3 ([Inyang et al., 2010](#)). [Schüz \(2009\)](#) demonstrated that laterality effects are similar across exposure categories and highlighted the problem of possible reporting bias. The Interphone study has addressed this problem in a sensitivity analysis, whereby different allocations of side-of-head were used; this caused only minor reductions in the odds ratios for the highest quintile of exposure, which suggests that the findings are not sensitive to errors in the recall of laterality of phone use ([Cardis et al., 2011a](#)).

Mobile phones are low-powered RF transmitters, operating at frequencies between 450 and 2700 MHz, with peak powers in the range of 0.1 to 2 W, the power being highest during a call. The handset only transmits RF power when it is turned on, but the newer smartphones regularly give short bursts of power to check e-mails and other Internet services. One study has found that mobile-phone output power is usually higher in rural areas where base stations are further apart, whereas the other factors examined in the study (length of call, moving/stationary, indoor/outdoor) were found to be of less importance as predictors of power output from the phone ([Hillert et al., 2006](#)).

Using a mobile phone in areas of good reception (such as in cities where mobile phone-base stations are close together) also decreases exposure as it allows the phone to transmit at reduced power. Conversely, people using a phone in rural areas where mobile-phone reception is poorer may receive higher RF exposure. This was one

factor examined in a study of 512 subjects in 12 countries who were asked to use GSM software-modified phones; the study, monitored date, time and duration of each call, frequency band and power output for a month ([Vrijheid et al., 2009a](#)). The main predictors of power output were the study location, the network, and the duration of the call, with shorter calls being associated with higher power output. The measured power levels in GSM networks were substantially higher than the average levels theoretically achievable, which has important implications for estimating exposure in epidemiological studies. Rural location was only a major factor in Sweden, where subjects were living in very sparsely populated areas; these results are consistent with those of an earlier paper from [Lönn et al. \(2004\)](#) in Sweden, who reported that the highest power level was used about 50% of the time in the rural areas, but only about 25% of the time in urban areas. This highlights the problem of identifying genuinely sparsely populated rural areas where major differences in power output can be found. Another paper from the Interphone study reported an investigation of the effects of parameters that were thought to influence the level of RF SAR in the brain. Total cumulative specific energy was estimated, based on data collected during the Interphone study, to assess the relative importance of the different factors and these results were used to develop an algorithm, which was tested on study subjects in five countries ([Cardis et al., 2011b](#)). This study found that the type of phone with the highest mean total specific cumulative energy (TSCE) was AMPS800 (5165 J/kg), followed by D-AMPS800 (3946 J/kg), GSM800/900 (2452 J/kg), GSM1800 (4675 J/kg), CDMA1900 (1855 J/kg), and CDMA800 (164 J/kg). The main determinants were communication system, frequency band, and number and duration of mobile-phone calls. The study also identified several uncertainties in relation to SAR estimation, including those related to spatial SAR distribution for each phone

class, error in recall of phone use, and laterality and uncertainties about the most biologically relevant dose metric.

A study in the USA examined the impact of phone type and location by use of software-modified phones driven over several pre-determined routes ([Kelsh et al., 2010](#)). This study found that RF levels were highest for the older analogue phones, intermediate for GSM and TDMA phones, and lowest for CDMA phones. The main predictors of RF level were phone technology and, to a lesser extent, degree of urbanization.

Patterns of personal mobile-phone use have been changing as technology has changed and this can have implications for the strength of the RF field experienced by the user. One important development has been the introduction of the short message service (SMS), which was originally designed for GSM to allow sending non-voice text messages ([Herring, 2004](#)). SMS was first introduced in 1993, but use increased rapidly in the mid-2000s. Text messaging using SMS leads to lower RF exposure than voice calls in two ways: the phone is usually held at least 30 cm from the body during the writing and sending of an SMS and the duration of power output is much shorter (about 11 seconds) than the duration of a voice call.

As with SMS, other mobile-phone communication innovations have been developed that result in lower potential for SAR exposure than voice calls. A person using a mobile phone at least 30 cm away from the body, e.g. when accessing the Internet, with a hands-free device for voice calls or “push-to-talk” with the phone held in front of the head, will therefore have a much lower exposure to RF than someone holding the handset against the head during a voice call.

(b) DECT phones

Another important source of personal RF exposure is the home use of DECT phones, which have been replacing traditional handsets in the home. As the DECT base-station is within

the home and at most some tens of metres from the handset, the average power generated by the DECT phone is less than that of a mobile phone, where the base station may be up to some kilometres away. However, the power output of a DECT base station in close proximity to a person may be comparable to that of a 3G phone, so proximity to a DECT phone base-station should be taken into account when estimating RF exposure in epidemiological studies in which sizeable numbers of subjects have used 3G phones. A recent study of Australian schoolchildren found that 87% had a DECT phone at home, and although there was only a weak correlation ($r = 0.38$) between mobile-phone and DECT-phone use, this suggests that DECT-phone use needs to be considered in the assessment of RF exposure ([Redmayne et al., 2010](#)).

(c) *Other communication technologies and domestic sources*

The incident-field exposures from typical devices used in home and office environments have been assessed ([Kühn et al., 2007a](#)). The maximum E-field exposure values for different device categories are summarized in [Table 1.13](#). The incident-field exposure from cellular base stations may be exceeded by the exposure from these devices due to the generally closer distances involved.

Additionally, an incident exposure of 1 V/m translates to a psSAR value in the brain that is approximately 10 000 times lower than the maximum exposure from a handset. Thus, handsets are by far the most dominant source of RF exposure for the general population.

Within homes there are many other potential sources of RF exposure, including baby monitors, microwave ovens, Wi-Fi, Bluetooth, various types of radios and remote-controlled toys. A study of 226 households in lower Austria measured the peak power of emitted bursts of RF exposure from each of these types of devices in bedrooms, where the residents spend the most

Table 1.13 Worst-case E field at distances of 20 cm and 1 m from typical wireless indoor devices

Device class	Frequency range (MHz)	Worst-case E field (V/m)	
		20 cm	1 m
Baby surveillance	40 – 863	8.5	3.2
DECT	1880 – 1900	11.5	2.9
WLAN	2400 – 2484	3.9	1.1
Bluetooth	2402 – 2480	3.1	1.0
PC peripherals	27 – 40	≤ 1.5	≤ 1.5

DECT, digital enhanced cordless telecommunications; PC, personal computer; WLAN, wireless local area network

Adapted from [Kühn et al. \(2007a\)](#)

time in one position. The highest peak RF values were measured for mobile-phone and DECT base stations in the 2400-MHz band ([Tomitsch et al., 2010](#)).

1.6.2 Occupational exposure

There are many occupations involving potential sources of exposure to RF radiation in the workplace, the more important of which involve work with high-frequency dielectric heaters (PVC welding machines) and induction heaters, broadcast sources, high-power pulsed radars, and medical applications including MRI and diathermy.

(a) *High-frequency dielectric heaters and induction heaters*

High-frequency dielectric heaters (PVC welding machines) functioning at 27 MHz have traditionally involved the highest occupational exposures to RF ([Allen, 1999](#)). This is not a large sector of the industrial workforce, although it is estimated that there are about 1000–2000 PVC dielectric welders in Finland, which has a total population of about five million people. The whole-body average SAR for dielectric heater operators has been estimated to vary from 0.12 to

2 W/kg and it is not uncommon for these workers to report heating effects ([Jokela & Puranen, 1999](#)).

(b) Broadcasting sources

The rapid increase in mobile-phone use and other communication technologies worldwide has required increasing numbers of workers to undertake monitoring and maintenance. A study of exposure to RF radiation from two medium-sized antenna towers in Finland was conducted to document worker exposure ([Alanko & Hietanen, 2007](#)). These towers contained transmitting antennae of several different types, mobile-phone networks (GSM900 and GSM1800), radio and digital television substations and other radio systems. Although the measured power density was quite variable, the maximum instantaneous power density at this site was 2.3 W/m², which was recorded during maintenance tasks at the tower with the GSM1800 antennae. For the tower with both GSM900 and GSM1800 antennae, the maximum registered instantaneous power density inside the climbing space was 0.4 W/m². [The Working Group agreed with the authors who concluded that exposures will depend on the different types of antennae located on the towers and that it is usually difficult to predict occupational RF exposures.]

The above approach to assess exposure is based on spot measurements and does not give an estimate of cumulative exposure over working time, which is the approach employed with other types of workplace hazards. Attempts have been made to employ this cumulative dose approach for exposure to RF radiation, but as there are usually many different sources of RF radiation present in a workplace, this is not straightforward. For example, such an approach was used to assess total exposure and estimate an annual dose on fast patrol boats in the Norwegian Navy, which carry high-frequency antennae and radar ([Baste et al., 2010](#)). This study found considerable variation in exposure at different points around the boats, the highest exposures and

annual dose being found in the captain's cabin. These estimates were done for three time periods (1950–79, 1980–94 and 1995+), and relied on recall of transmission characteristics over several decades. The estimated annual doses in the most recent period were about one third of those in the earliest period. The estimated annual doses for the period from 1995 and later ranged between 4.3 and 51 kVh/m.

(c) Other potential sources of radiofrequency radiation in the workplace

Portable radios, short-wave and surgical diathermy are other potential sources of RF radiation in the workplace, whereas base stations, microwave links and microwave ovens have been considered unlikely to give rise to substantial exposures ([Allen, 1999](#)). For example, a study of exposure to RF radiation in police officers operating speed guns (measurements made at the seated ocular and testicular positions) found that almost all of the 986 measurements made for 54 radar units were below the detection limit, the highest power-density reading being 0.034 mW/cm² ([Fink et al., 1999](#)).

1.6.3 Environmental exposure

The most common sources of RF in the general environment are mobile-phone base stations, which tend to be operated at the lowest power possible for reasons of network efficiency (see Section 1.2; [Allen, 1999](#)). The level of RF exposure is usually poorly correlated with proximity to the antenna, although there is considerable variation in output power from site to site (Section 1.2). A study regarding indoor incident-field exposure from cellular base-station sites was conducted by Austrian Research Centers (ARCS) in the city of Salzburg, Austria ([Coray et al., 2002](#)). [Table 1.14](#) shows two cumulative incident-field exposure values (sum of incident-field exposure from multiple transmitters at one site) measured at different distances from several base-station

Table 1.14 Measurement of indoor incident electric-field (E) strength at base stations in Salzburg, Austria^a

Base station	Measurement 1		Measurement 2	
	Distance to base station (m)	Cumulative incident E field (V/m)	Distance to base station (m)	Cumulative incident E field (V/m)
1	196	0.37	347	0.35
2	88	0.51	108	0.89
3	9	0.034	15	0.037
4	16	0.62	8	1.00
5	85	0.94	152	0.75
6	81	1.8	85	1.71
7	4	3.9	25	1.02
8	93	0.19	208	0.19
9	34	0.40	55	0.63
10	39	1.9	76	2.8
11	174	0.59	220	0.45
12	41	0.70	107	0.67
13	2.5	0.25	5.5	0.15

^a For each base station site, two examples of measurements of cumulative incident field exposure (sum of incident field exposure from multiple transmitters at one site) at different distances are shown.

Compiled by the Working Group from BAKOM Report, [Coray et al. \(2002\)](#)

sites. The values are between 0.1 and 1 V/m for distances of up to several hundreds of metres. Values greater than 1 V/m and up to 3.9 V/m were measured for distances of less than 86 m. These data also underline that the distance to the base station site has a poor correlation for the incident exposure. Similar results were reported in a study that also included outdoor measurement points and addressed the time dependence, i.e. traffic dependence of the exposure from cellular base stations. The results showed a substantial time dependence for base stations with multiple traffic channels. In these cases, clearly lower exposure can be expected at night and at week-ends ([Bornkessel et al., 2007](#)).

In an attempt to measure typical exposure to RF radiation over a whole week, volunteers in a Swiss study were asked to wear an RF exposimeter and to complete an activity diary ([Frei et al., 2009b](#)). The main contributions to exposure were found to come from mobile-phone base stations (32.0%), mobile-phone handsets (29.1%) and DECT phones (22.7%).

Breckenkamp *et al.* undertook a validation study of exposure to RF radiation in 1132 households in Germany located within 500 m of at least one mobile-phone base station (average number of base stations, 3.4; average number of antennae, 17) ([Breckenkamp et al., 2008](#)). An exposure model was developed, based on 15 parameters related to the base station and the antennae, from the database of the federal network agency and information about the home from the residents and interviewer. Dosimetric measurements were undertaken in the bedroom of the home in 2006. There was considerable variability across cities (range of kappa values, 0.04–0.49), with higher kappa related to low-density housing with buildings comprising more than three floors. There was greater agreement for households located less than 300 m from the base stations and the authors concluded that the model was only useful where high-precision input data were available.

Little is known about geographical variation in exposures in different settings in the general community, but published data related

to exposures within different forms of transport, homes, offices and outdoors in five European countries were reviewed in a recent study ([Joseph et al., 2010](#)). Power density (mW/m^2) was measured in each microenvironment and highest exposures were measured in transportation, followed by outdoor environments, offices and homes. In the Netherlands, the highest exposures were measured in the office environment. In all studies, the lowest exposures were in the home, with exposures of about 0.1 mW/m^2 recorded in all countries. In transport vehicles, virtually of the exposure was from mobile phones, whereas in offices and homes, the sources were quite variable between countries. [The Working Group suggested that these conclusions should be treated with some caution, as it was not clear how representative the measured microenvironments were.]

In a feasibility study in Germany, the aim of which was to develop reliable exposure metrics for studies of health effects of exposure to RF radiation from mobile-phone base stations, data were collected on distance to base station and spot measurements at the homes of nine controls taking part in a case-control study of cancer. Distance from base station was a poor proxy for the total power density within the home due to the directional characteristics of the base-station beam, scattering, shielding and reflection of the radiated fields and the contribution to power density from other sources ([Schüz & Mann, 2000](#)). [The Working Group noted that use of this metric would be likely to result in considerable exposure misclassification.]

A further study of a random sample of 200 subjects in France used a personal exposure meter to estimate the doses, time patterns and frequencies of RF exposures with measurements of electric-field strength in 12 different bands at regular intervals over 24 hours ([Viel et al., 2009](#)). This allowed differentiation of different sources of RF radiation, including mobile-phone base stations. For each of GSM, DCS and UMTS, more

than 96% of the measurements were below the detection limit and the median of the maximum levels for all three systems ranged between 0.05 and 0.07 V/m . In addition, exposures were found to vary greatly at similar distances from GSM and DCS base stations, although two peaks were observed (at 280 m mainly in urban areas, and 1000 m mainly in periurban areas), although most distances exceeded the 300 m within which the exposure model developed by [Breckenkamp et al. \(2008\)](#) was found to have the highest agreement with measured levels.

In another study by [Frei et al. \(2009a\)](#), the aim was to develop a model to predict personal exposure to RF radiation. One hundred and sixty-six subjects carried a personal dosimeter for one week and completed a diary. Important predictors of exposure were housing characteristics, ownership of communication devices, time spent in public transport, and other behavioural aspects, with about half of the variance being explained by these factors.

A range of personal exposure meters is now available. These are more robust for the purposes of exposure assessment in epidemiological studies, and a considerable step forward compared with the traditional spot-measurement approach, which is usually chosen for compliance purposes and does not result in a representative estimate of personal exposure ([Mann, 2010](#)). [The Working Group noted that care needs to be taken in interpreting the results of personal exposure measurements, because of the low sensitivity and the failure to account for the fact that they respond to TDMA signals, which may lead to an overemphasis of DECT, Wi-Fi and GSM phone signals in average exposure. Because burst powers may have been measured for these signals, rather than average powers, any exposure proportions attributed to source categories in these studies should be treated with caution when assessing exposure for epidemiological studies.]

1.7 Exposure guidelines and standards

Guidelines and standards for limiting human exposure to RF fields have been developed by several organizations, the most prominent being those of the International Commission on Non-Ionizing Radiation (ICNIRP) and the Institute of Electrical and Electronic Engineers (IEEE). ICNIRP published its present RF guidelines in 1998 ([ICNIRP, 1998](#)) and restated them in 2009 ([ICNIRP, 2009a](#)). IEEE published its present guidelines in 2005 ([IEEE, 2005](#)), but its 1999 guidelines are still used in some countries ([IEEE, 1999](#)).

These guidelines contain restrictions on exposure that are intended to assist those with responsibility for the safety of the general public and workers. The guidelines provide clearly defined exposure levels below which the established acute health effects of exposure are avoided. Exposures can be measured or calculated and compared with these values. If exposures are found to be above the guideline values, measures are put in place to reduce exposure. The guidelines apply to all human exposures to EMFs, irrespective of how such exposures arise, and they do not make specific mention of sources.

The guidelines are not mandatory by themselves, but have been adopted by regulatory authorities and governments in many countries/regions of the world in a variety of different ways. Some regulatory regimes focus on limiting exposures of the public and/or workers, while others focus on limiting product emissions (to control exposures) as part of the certification process before placing products on the market. For example, harmonized technical standards have been implemented in Europe that provide a basis for assessing exposures from equipment such as mobile phones and ensuring that exposures are below values taken from the ICNIRP guidelines. The values in the 1999 IEEE guidelines are used

in a similar way in countries such as the USA and Canada.

1.7.1 Scientific basis

Both ICNIRP and IEEE have reviewed the broad base of the scientific evidence in developing their guidelines and arrived at similar conclusions regarding the evidence for health effects. This consensus is well expressed in the following excerpt taken from the ICNIRP 2009 restatement of its guidelines ([ICNIRP, 2009b](#)):

It is the opinion of ICNIRP that the scientific literature published since the 1998 guidelines has provided no evidence of any adverse effects below the basic restrictions and does not necessitate an immediate revision of its guidance on limiting exposure to high frequency EMFs. The biological basis of such guidance remains the avoidance of adverse effects such as ‘work stoppage’ caused by mild whole body heat stress and/or tissue damage caused by excessive localized heating.

Absorption of RF fields in the body tissues leads to the deposition of energy in these tissues and this energy adds to that produced by metabolism. This energy imposes an additional thermoregulatory burden on the organism and the temperature can increase if the energy absorption rises above a certain level (see Section 1.3). Localized temperature increase can occur in response to localized absorption of energy and the core body temperature can go up in response to generalized absorption of energy throughout the body tissues. The ICNIRP guidelines ([ICNIRP, 1998](#)) conclude from the literature that:

Established biological and health effects in the frequency range from 10 MHz to a few GHz are consistent with responses to a body temperature rise of more than 1 °C. This level of temperature increase results from exposure of individuals under moderate environmental conditions to a *wbSAR* of approximately 4 W/kg for about 30 minutes.

Table 1.15 Basic restrictions on SAR (W/kg), as taken from the ICNIRP and IEEE exposure guidelines

Body region	Workers/controlled			General public/uncontrolled		
	ICNIRP (1998)	IEEE (1999)	IEEE (2005)^d	ICNIRP (1998)	IEEE (1999)	IEEE (2005)
Whole body ^a	0.4 (6)	0.4 (6)	0.4	0.08 (6)	0.08 (30)	0.08
Head and trunk ^{a b}	10 (6) [10]	8 (6) [1]	10 (6) [10]	2 (6) [10]	1.6 (30) [1]	2 (30) [10]
Extremities ^{a b c}	20 (6) [10]	20 (6) [10]	20 (6) [10]	4 (6) [10]	4 (30) [10]	4 (30) [10]

^a In round brackets after the SAR value is the averaging time in seconds. The averaging times vary with frequency in the IEEE guidelines and the values given are for the 400 MHz to 2 GHz range typically used for mobile communication.

^b In square brackets (where relevant) is the averaging mass in grams: the averaging mass is specified as a contiguous tissue volume by ICNIRP and as in the shape of a cube by IEEE.

^c Extremities are taken as the hands, wrists, feet and ankles in the context of the [IEEE \(1999\)](#) guidelines, distal from the knees and elbows in the [IEEE \(2005\)](#) guidelines, and as the entire limbs in the context of the ICNIRP guidelines.

^d The restrictions apply over the frequency range 100 kHz to 6 GHz in the case of [IEEE \(1999\)](#).

ICNIRP, International Commission on Non-Ionizing Radiation Protection; IEEE, Institute of Electrical and Electronics Engineers; SAR, specific absorption rate.

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Effects due to whole-body heating are also considered for frequencies below 10 MHz and down to 100 kHz; however, wavelength becomes progressively larger in relation to the body dimensions as frequency decreases to below 10 MHz and coupling to the fields becomes progressively weaker, with the result that less energy is absorbed. Above 10 GHz, absorption of RF fields by the body tissues becomes so strong that the RF fields are considered to be absorbed within a few millimetres of the body surface; hence the guidelines are designed to restrict surface heating.

A further class of thermal effect can be elicited with pulse-modulated RF waveforms, including certain radar signals. This effect is known as the microwave auditory effect and occurs as a result of energy absorption from successive RF pulses, causing pulsed thermal expansion of the head tissues ([ICNIRP, 2009a](#)). [ICNIRP \(1998\)](#) states that repeated or prolonged exposure to microwave auditory effects may be stressful and potentially harmful, and it provides additional guidance for restricting exposures to pulse-modulated fields to avoid this effect.

1.7.2 Basic restrictions

Considering the evidence relating to whole-body heating and localized heating of parts of the body, ICNIRP and IEEE have specified the basic restriction quantities shown in [Table 1.15](#). The information presented here is a summary of the main aspects of the restrictions in the guidelines and serves to provide a simplified comparison for the purposes of this *Monograph*.

From [Table 1.15](#) it is clear that the various sets of guidelines contain similar restriction values and have many common features. Moreover, the 2005 guidelines from IEEE have brought the ICNIRP and IEEE guidelines even closer: the SAR values are now identical and the residual differences now only pertain to averaging times, definition of the extremities, and the shape of the mass used with localized SAR restrictions.

IEEE and ICNIRP both frame their guidelines in terms of two tiers. The first tier includes a wbSAR value that is a factor of 10 lower than the 4 W/kg mentioned above, while the second tier includes restriction values that are five times lower than those in the first tier. In the case of the ICNIRP guidelines, these tiers are presented as restrictions for exposure of workers (tier 1)

Table 1.16 Basic restrictions on induced current density or induced electric field between 30 kHz and 10 MHz, as taken from the ICNIRP and IEEE exposure guidelines

Body region	Workers/controlled				General public/uncontrolled			
	ICNIRP (1998)^a (mA/m ²)	IEEE (1999)^b (mA/m ²)	IEEE (2005)^c (mV/m)	ICNIRP (2010)^d (mV/m)	ICNIRP (1998)^a (mA/m ²)	IEEE (1999)^b (mA/m ²)	IEEE (2005)^c (mV/m)	ICNIRP (2010)^d (mV/m)
Whole body	-	350	-	270	-	157	-	135
CNS	10	-	-	-	2	-	-	-
Brain	-	-	885	-	-	-	294.5	-
Heart	-	-	5647	-	-	-	5647	-
Extremities	-	-	626.9	-	-	-	626.9	-
Other tissues	-	-	626.9	-	-	-	209.3	-

^a Peak rms current density in mA/m², averaged over 1 cm² area perpendicular to the current direction. Applies to the CNS tissues. Applicable frequency range is 1 kHz to 10 MHz.

^b Peak rms current density in mA/m², averaged over any 1 cm² area of tissue in 1 second. Applies anywhere in the body. Applicable frequency range is 3 kHz to 100 kHz.

^c Peak rms internal electric field in mV/m, averaged over a straight-line segment of 5 mm length, oriented in any direction. The averaging time for an rms measurement is 0.2 s. Applicable frequency range is 3.35 kHz to 5 MHz. Values are rounded to four significant digits.

^d Internal electric field in mV/m, averaged over a contiguous tissue volume of 2 × 2 × 2 mm³. The 99th percentile value of the electric field for a specific tissue should be compared with the basic restriction. Applicable frequency range is 3 kHz to 10 MHz.

CNS, central nervous system; rms, root-mean-square value of the electric field strength

Note: All numbers denote the frequency in kHz; all limits in this frequency range increase linearly with frequency. Limits for contact currents also apply (not shown here).

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and the general public (tier 2). ICNIRP explains that the lower basic restrictions for exposure of the general public take into account the fact that their age and health status may differ from those of workers. The first tier in the IEEE guidelines is described as for controlled environments (subject to a RF safety programme as prescribed by IEEE) and the second tier as for uncontrolled environments, as accessible to the general public.

Electrical effects caused by stimulation of the peripheral and central nervous system are also considered below 10 MHz, although the maximum sensitivity to these effects occurs at considerably lower frequencies, in the tens of hertz to a few kilohertz region ([ICNIRP, 2010](#)). The guidelines should be referred to for further information about these effects; however, the restrictions are summarized in [Table 1.16](#) for frequencies between 30 kHz and 10 MHz.

1.7.3 Reference levels

The guidelines also contain reference levels (called maximum permissible exposures, or MPEs, by IEEE) expressed in terms of electric- and magnetic-field strengths, or plane-wave equivalent power-density incident on the body (see Glossary). Measured or calculated values can be compared with these quantities to verify that the basic restrictions on SAR or induced current/electric fields are not exceeded.

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2. CANCER IN HUMANS

This section is a review of the large body of epidemiological evidence from studies of exposure of occupational groups and the general population to radiofrequency (RF) radiation from diverse sources, including from the use of mobile telephones. The results of these studies comprise a large amount of data, which could not be fully reproduced here. The Working Group included studies that assessed specific sources of RF radiation or job titles that were specifically linked to RF radiation. Studies that were excluded used job titles only for classification, or source surrogates only, without specifically addressing RF exposure. The Tables in this section summarize the main findings, but do not uniformly capture the results for all exposure metrics or all subgroups given in the original publications. In the text, the Working Group provides comments on those findings that are of greatest relevance to the evaluation, e.g. risk in the overall exposed group, patterns of change in risk with increasing exposure (such as a monotonic increase in risk with increasing exposure), and changes in risk with duration of exposure or latency.

2.1 Occupational exposure

The occupational environment is one domain in which humans are exposed to RF radiation. Many occupational circumstances entail regular or occasional exposure to RF radiation from fixed or mobile sources. A wide variety of workers are involved, including military and security personnel using walkie-talkie devices, radar operators, radio and television antenna maintenance and repair workers, welders performing dielectric (high-frequency) welding and sealing of plastics, workers using RF radiation for drying or testing operations, and physiotherapists employing medical diathermy equipment. Only a limited number of studies have assessed the risk of cancer in relation to either measured or inferred levels of exposure. There have been

a large number of epidemiological studies of workers who were not evaluated in terms of their exposure to RF radiation, but rather with respect to their exposure to electric or magnetic fields (EMF), extremely low-frequency (ELF) fields, i.e. < 300Hz ([IARC, 2002](#)), or microwaves (MW), and an even larger number of studies in which it might be suspected that some workers were likely to have been exposed to RF radiation. The Working Group did not include these studies in the present review because it was not certain that sizable fractions of the workers in such studies were actually exposed to RF radiation, or at what levels they were exposed. This review is therefore limited to occupational studies in which the investigators made an effort to specifically document or assess exposures to RF radiation in the workers considered to be exposed.

2.1.1 Cancer of the brain

(a) Case-control studies

[Thomas et al. \(1987\)](#) conducted a death-certificate-based case-control study in selected counties of the north-eastern and southern United States of America (USA). The cases were men who had died from tumours of the brain or other parts of the central nervous system (CNS) at age ≥ 30 years between 1978 and 1981. Diagnoses were verified in hospital records. One control decedent, whose cause of death was not brain cancer, epilepsy, stroke, suicide or homicide, was selected for each case, and matched by age and year of death, and usual area of residence. The next-of-kin of the study subjects were interviewed: participation rates were 74% for cases and 63% for controls. For each job held since age 15 years, the job title and a brief description of the work, the industry, the location, the employment dates, and the hours worked per week were obtained. Two methods were used to classify men according to their occupational exposure to MW or RF radiation: one was based on a selection of broad job titles [most of which would have had mixed or predominant exposure to EMF frequencies other than RF], while in the other an industrial hygienist classified each job according to exposure to RF radiation, lead and soldering fumes. Data from 435 cases and 386 controls were analysed. Only results based on the industrial hygienist's classification are reviewed here. [While controls were individually matched to cases, there was a deficit of controls, possibly due to poorer participation, but no mention was made of adjusting for the matching variables in the analysis; thus there may have been uncorrected bias due to study design in the calculated odds ratios (ORs).] Risk of brain tumours was increased in those ever occupationally exposed to RF radiation (OR, 1.7; 95% CI, 1.1–2.7) adjusted for educational level ([Table 2.1](#)); however, the odds ratio decreased when men also exposed to soldering fumes or lead were removed from

the exposed group, and dropped even further when those who might also have had exposure to organic solvents were removed from the exposed group. [This study was one of the few to directly attempt to address possible confounding of occupational exposure to RF radiation with coexposure to soldering fumes, lead and organic solvents. It was limited by the fact that it was based on death certificates (the dead controls were unlikely to accurately represent the population from which the dead cases came) and on an analysis that may not have controlled for bias due to the matched design.]

[Berg et al. \(2006\)](#) analysed data obtained from cases (glioma and meningioma) and controls using a detailed questionnaire on occupational exposure to what the authors described as RF/MW/EMF, which formed part of the data collected in the German component of the INTERPHONE study (as described in Section 2.2.2 in relation to [Schüz et al., 2006a](#)). Participants were asked screening questions about use of industrial heating equipment to process food, to bond, seal, and weld materials, or to melt, dry, and cure materials. Questions were also asked about manufacturing semiconductor chips or microelectronic devices; using radar; maintaining electromagnetic devices used to treat or diagnose diseases; working with or nearby to broadcasting and telecommunications antennae and masts; using different kinds of transmitters; and using amateur (“ham”) radio. When a participant screened positive for one of these activities, further questions were asked to determine whether the occupation entailed exposure to RF/MW/EMF. Each person was classified as having: no exposure (responded negatively to the screening questions, or were positive for some activities thought not to entail exposure); no probable exposure (exposure existed but probably not exposed continuously during working hours in any activity); probable exposure (probably exposed continuously during working hours in at least one activity); or high exposure

Table 2.1 Case-control studies on cancer of the brain and occupational exposure to radiofrequency radiation

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Comments
Thomas et al. (1987) USA, 1979-81	435	386	Death certificates of residents who had died from a cause other than brain tumour, epilepsy, stroke, suicide or homicide, matched to cases by age and year at death, and area of residence	Next-of-kin interviews regarding employment history and other risk factors for brain tumours. Exposure classified according to job title and results of previous studies, and by an industrial hygienist	Brain	<i>Occupational exposure to MW or RF based on assessment by the industrial hygienist</i> Never exposed Ever exposed Ever exposed, excluding those with co-exposure to soldering fumes or lead Ever exposed, excluding those with co-exposure to organic solvents	 2	 1.0 1.7 (1.1-2.7) 1.4 (0.7-3.1) 0.4 (NR)	Restricted to white men aged > 30 yr. Methods of statistical analysis were not described. Covariates: matched by age and year at death, and area of residence, but not included as covariates in the unmatched analysis

Table 2.1 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Comments
Berg et al. (2006) Germany, 2000–03	381 cases of meningioma, 366 cases of glioma	1494, of whom 732 matched to glioma and 762 to meningioma cases	2449 controls frequency-matched on age, sex and centre, were derived from population registries, 63% participated. Subsequently, controls were matched to cases on a 2:1 basis	CAPI mostly in hospital for cases, and at home for controls. Interview included questions about job title and specific occupational activities followed by expert assessment of exposure to RF/MW	Glioma (C71.0–71.9; 9380–9383, 9390–9393, 9400–9401, 9410–9411, 9420–9421, 9440–9442, 9450–9451) and meningioma (C70.0; 9530–9539)	<i>Exposure to RF</i> <i>Glioma</i> Total exposure: No/not probable Probable/high exposure Probable exposure: No exposure Not probable Probable High Duration of high exposure: Not highly exposed Highly exposed for < 10 yr Highly exposed for ≥ 10 yr	328 38 308 20 16 22	1.00 1.04 (0.68–1.61) 1.00 0.84 (0.48–1.46) 0.84 (1.46–1.56) 1.22 (0.69–2.15)	Aged 30–69 yr Covariates: SES, urban or rural, exposure to ionizing radiation, smoking history, age at diagnosis

Table 2.1 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Comments
Berg et al. (2006) (contd.)	<i>Meningioma</i>								
	Total exposure:								
	No/not probable						355	1.00	
	Probable/high exposure						26	1.12 (0.66–1.87)	
	Probable exposure:								
	No exposure						340	1.00	
	Not probable						15	1.11 (0.57–2.15)	
	Probable						15	1.01 (0.52–1.93)	
	High						11	1.34 (0.61–2.96)	
	Duration of high exposure:								
	Not highly exposed						370	1.00	
	Highly exposed for < 10 yr						5	1.14 (0.37–3.48)	
	Highly exposed for ≥ 10 yr						6	1.55 (0.52–4.62)	

Table 2.1 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Comments
Karipidis et al. (2007) Australia, 1987–91	416	422	Population of four major centres as recorded by the electoral rolls for the Australian state of Victoria	Comprehensive job history, including self-reported RF exposure in each job, expert assessment by occupational hygienist and application of a community-based job-exposure matrix, FINJEM	Glioma (ICD-O codes 938–946)	<i>Total cumulative exposure to RF given by FINJEM ($W/m^2 \cdot yr$)</i>	396	1.00	Covariates: age, sex, and year of education
						Unexposed			
						> 0–11			
						> 11–52			
						> 52			
						<i>P</i> trend			
						<i>Self-reported duration of exposure (yr) to RF</i>			
						Unexposed			
						> 0–3			
						> 3–8			
						> 8			
						<i>P</i> trend			
						<i>Expert assessment of duration of exposure (yr) to RF</i>			
						Unexposed			
						> 0–3			
> 3–6									
> 6									
<i>P</i> trend									
							381	1.00	
							10	1.20 (0.48–3.04)	
							12	1.65 (0.66–4.17)	
							11	1.57 (0.62–4.02)	
								0.17	

Table 2.1 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Comments
Baldi <i>et al.</i> (2011) France, 1999–2001	221	442	Population selected from local electoral rolls and individually matched on age, sex and department of residence	Interviewer-administered face-to-face questionnaire, which included a lifetime occupational history documenting for each job held for ≥ 6 mo: job title, industry, begin and end dates, and details of tasks performed. Occupational hygienists assessed probability of exposure to RF and duration for each job.	CNS (70.0–70.9, 71.0–71.9, 72.2–72.9)	Occupational exposure to RF All brain tumours ($n = 221$): Unexposed Exposed Glioma ($n = 105$): Unexposed Exposed Meningioma ($n = 67$): Unexposed Exposed Acoustic neurinoma ($n = 32$): Unexposed Exposed <i>Amateur radio practice</i> All brain tumours ($n = 221$): No Yes	148 7 71 7 61 0 31 1 NR NR	1.00 1.50 (0.48–4.70) 1.00 1.44 (0.50–4.13) 1.00 - 1.00 0.40 (0.05–3.42) 1.00 1.39 (0.67–2.86)	95 males, 126 females. 70% of eligible cases participated and 69% of eligible and contactable controls. Covariates: exposure to pesticides, smoking, educational level

CAP1, computer-assisted personal interview; FINJEM, FIN(nish) job-exposure matrix; mo, month; MW, microwave; NR, not reported; RF, radiofrequency radiation; SES, socioeconomic status; W, watt; yr, year

(certainly exposed continuously during working hours and sometimes at levels $> 0.08\text{W/kg}$ in at least one activity). Analyses included data from proxy interviews, and results were not sensitive to removal of proxy interview data. There was weak evidence that risk of glioma and of meningioma increased with increasing duration of high occupational exposure to RF/MW/EMF. For glioma, the odds ratio for < 10 years of high exposure relative to no exposure was 1.11 (95% CI, 0.48–2.56) and that for ≥ 10 years of high exposure was 1.39 (95% CI, 0.67–2.88); the analysis controlled for centre, sex, age at diagnosis, socioeconomic status, urban or rural area, exposure to ionizing radiation, and smoking history. The corresponding odds ratios for meningioma were 1.14 (95% CI, 0.37–3.48) and 1.55 (95% CI, 0.52–4.62) ([Table 2.1](#)). [The strengths of this study were its large size and evaluation of exposure at the job-activity level. Its main weaknesses included the small numbers of cases with high exposure and lack of associated consideration of other sources of exposure to RF radiation.]

[Karipidis *et al.* \(2007\)](#) reported on risk of glioma in relation to occupational exposure to RF radiation in a case-control study in five major population centres in the Australian state of Victoria. Cases were patients aged 20–70 years with glioma, newly diagnosed between July 1987 and December 1991, who were ascertained by screening the medical records of 14 major Melbourne (capital of Victoria) hospitals that together provided most of the neurosurgical services in the state. Completeness of ascertainment was checked against cancer-registry records of Victoria. Controls were randomly selected from the electoral rolls and frequency-matched to cases by age and sex; the electoral rolls covered about 85% of citizens at that time. Controls were excluded if they had a history of brain tumour, stroke or epilepsy. Participants completed a self-administered work-history questionnaire, which included queries about occupation, employer, industry, main tasks and duties, equipment used,

start and finish dates, number of hours worked per day, number of days worked per week and whether or not they had been exposed to RF radiation, for all jobs undertaken since age 12 years that had lasted ≥ 3 months. Work histories were checked for completeness at a subsequent face-to-face interview. For 44% of cases and 2% of controls, a next-of-kin proxy completed the work history. In addition to the self-report, exposure to RF radiation was assessed from the work history by use of the Finnish National Job-Exposure Matrix (FINJEM; a community-based job-exposure matrix developed by the Finnish Institute of Occupational Health) and by review of the work histories by an expert occupational hygienist. Four categories of cumulative exposure were created for each exposure measure: unexposed, and thirds of the ranked exposure distributions for all exposed subjects. Results were adjusted for age, sex and years of schooling (a surrogate for socioeconomic status). Data on occupational exposure were obtained for 414 cases and 421 controls, i.e. 66% and 65%, respectively, of those eligible and contactable [respective numbers not contacted were not given]. With FINJEM, 18 cases and 17 controls were classified as exposed to RF radiation, 29 and 48 by self-report and 33 and 25 by expert assessment. Only in the case of classification based on expert assessment of exposure was there any consistent indication that risk of glioma increased with exposure to RF radiation: relative to those who were not exposed, odds ratios were 1.20 (95% CI, 0.48–3.04) for > 0 –3 years of exposure, 1.65 (95% CI, 0.66–4.17) for > 3 –6 years and 1.57 (95% CI, 0.62–4.02) for > 6 years ([Table 2.1](#)). Analyses excluding participants with proxy information showed no major differences in results. [The use of multiple measures of occupational exposure to RF radiation, including expert assessment of a comprehensive occupational history, was a strength of the study. It was limited by lack of inclusion of non-contactable subjects when estimating participation rates, by the large proportion of

cases requiring proxy respondents and by the comparatively small number of subjects who were exposed to RF radiation. FINJEM provides a probably incomplete assessment of occupational exposure to RF radiation.]

[Baldi et al. \(2011\)](#) reported on a case-control study of people aged ≥ 16 years, newly diagnosed with cancer of the primary CNS between mid-1999 and mid-2001 in the administrative region of Gironde in south-western France. Patients with neurofibromatosis, Von Hippel-Lindau disease or AIDS were excluded. Controls were selected from local electoral rolls, which automatically register all French subjects, and individually matched to cases by age, sex and department of residence. Participation rates were 70% of eligible cases and 69% of eligible and contactable controls. Occupational exposure to RF radiation was assessed by two occupational hygienists from lifetime histories of jobs that had lasted ≥ 6 months (including job title, industry, dates each job began and ended, details of tasks performed), which were collected by face-to-face interview. Information on use of amateur radio was also collected. The odds ratio for occupational exposure to RF radiation and all tumours of the brain was 1.50 (95% CI, 0.48–4.70), while for use of amateur radio it was 1.39 (95% CI, 0.67–2.86) ([Table 2.1](#)). [The Working Group noted the comparatively small size of the study and the small number of exposed subjects, which appeared to have precluded analysis at multiple exposure levels; the exposure assessment based on a comparatively limited occupational history, and an estimated participation rate for controls that was not based on all potentially eligible participants.]

(b) *Cohort studies*

[Lilienfeld et al. \(1978\)](#) reported on a retrospective cohort study of USA employees and their dependents who had worked or lived at the United States embassy in Moscow during 1953–1976 and, for comparison, employees and their

dependents at other United States embassies in eastern Europe who had not served in Moscow over the same period. There were unusual levels of background exposure to MW in the embassy in Moscow. The maximum measured levels were $5 \mu\text{W}/\text{cm}^2$ for 9 hours per day, $15 \mu\text{W}/\text{cm}^2$ for 18 hours per day, and $< 1 \mu\text{W}/\text{cm}^2$ thereafter for non-overlapping time periods between 1953 and 1975 and between 1975 and 1976. Only background levels of exposure to MW were recorded in other eastern-European embassies. Relevant health information and follow-up data were obtained from the medical records of employees and their dependents (held by the Department of State) and a health-history questionnaire sent to each employee or dependent who could be located. Death certificates were sought for all decedents. The analysis was based only on subjects who could be traced ($> 90\%$): 1719 Moscow employees and 1224 dependents known to have lived with them in the embassy, and 2460 employees at other embassies and 2072 dependents known to have lived with them. For embassy employees, 194 deaths were ascertained; of these, there was sufficient information for 181 for inclusion in the analysis, and death certificates were available for 125. There were no deaths from tumours of the brain or other parts of the CNS in Moscow employees, compared with 0.9 expected on the basis of comparable mortality rates in the USA [standardized mortality ratio, SMR, 0; 95% CI, 0–4.1]. For other embassy employees, there were five deaths from tumours of the brain or other parts of the CNS, with 1.5 expected (SMR, 3.3; 95% CI, 1.1–7.7). For dependents known to have lived in the relevant embassy, $> 90\%$ were traced, 67 deaths were ascertained, 62 death certificates were available. There were no observed deaths from tumours of the brain or other parts of the CNS (0.15 expected) [SMR, 0; 95% CI, 0–24.6] for the Moscow embassy and 1 death was observed (0.19 expected) for the other embassies ([Table 2.2](#)). [This study was available only in a United States government report; it was not published in the

peer-reviewed literature. Its main weaknesses were the small sizes of the two cohorts and the small number of deaths from cancer of the CNS observed. The long and continuous exposure to high background levels of MW in the Moscow Embassy was a strength. Possible confounding factors were not addressed.]

[Milham \(1988a, b\)](#) followed a cohort of people who were licensed as amateur radio-operators between 1 January 1979 and 16 June 1984 (a licence was valid for 5 years) and had addresses in Washington State or California. The full names and dates of birth of male cohort members (67 829 people; there were few females) were matched with deaths in Washington State and California. Only exact matches were accepted. Person-years at risk started on the effective current registration day and ended on the day of death, or on 31 December 1984. There were 232 499 person-years at risk and 2485 deaths; 29 deaths from cancer of the brain (International Classification of Disease Revision 8 [ICD-8] code 191) were observed and 20.8 expected [the death rates used to estimate the expected numbers were not specified], SMR for deaths from cancer of the brain was 1.39 (95% CI, 0.93–2.00) ([Table 2.2](#)). Licensees were further subdivided by licence class, i.e. Novice, Technician, General, Advanced and Extra. Novices were limited in their use of transmitter power and transmission frequencies; these conditions became more liberal as licence class rose. The average age increased with rising licence class; those holding higher-level licences may have generally been amateur radio operators for longer than those holding lower-level licences. Deaths from cancer of the brain were more frequent than expected for each licence class after Novice, but with little evidence of progressive increases as licence class rose ([Table 2.2](#)). [The main strength of this study was its clear and straightforward execution. Its weaknesses included lack of information about erroneous or missed links of cohort members to deaths, lack of consideration of possible migration of cohort

members from Washington State and California, limited validation of licence class as a surrogate for intensity and duration of exposure to RF radiation, and the small number of observed deaths from cancer of the brain. Possible confounding factors were not addressed.]

[Armstrong *et al.* \(1994\)](#) carried out a nested case–control analysis of the association of several cancers, including tumours of the brain, and exposure to pulsed electromagnetic fields (PEMFs; frequency range, 5–20 MHz) in two cohorts of electrical-utility workers in Quebec, Canada (21 749 men; follow-up, 1970–1988), and France (170 000 men; follow-up, 1978–1989), among whom 2679 cases of cancer were identified, 84 malignant tumours of the brain and 25 benign tumours of the brain. Utility-based job-exposure matrices were created with information obtained from surveys of samples of 466 (Quebec) and 829 (France) workers wearing exposure meters in 1991–1992. For malignant tumours, the odds ratios were 0.84 (95% CI, 0.47–1.50) for above-median exposure to PEMFs and 1.90 (95% CI, 0.48–7.58) for exposure at or above the 90th percentile, while for astrocytoma – the most common type of glioma – the odds ratio for exposure at or above the 90th percentile was 6.26 (95% CI, 0.30–132). For benign tumours, the odds ratio was 1.58 (95% CI, 0.52–4.78) for above-median exposure. None of the odds ratios for other subtypes of cancer of the brain were elevated ([Table 2.2](#)).

[Grayson \(1996\)](#) reported on risk of brain cancer related to exposure to equipment producing RF or MW (RF/MW) radiation in a case–control study conducted within a cohort of male members of the United States Air Force in 1970–89 ([Table 2.2](#)). Four matched controls were randomly selected for each case from all cohort members. Controls were not eligible if they had been diagnosed with leukaemia, cancer of the breast or melanoma “...because excess risks of these tumours have been associated with EMF exposures in other studies” [this exclusion was

not appropriate in a nested case-control study as if the excluded tumours were associated with EMF exposure, this could bias exposure in controls downwards, though probably only to a very small degree given the relative rarity of these cancers]. An expert panel assessed each job title-time couplet for probability of exposure to RF/MW radiation, which was recorded as “unexposed,” “possibly exposed” and “probably exposed.” Incident cases of cancer of the brain (ICD-9 code 191) were identified from hospital discharge records of serving personnel; confirmatory data (imaging or histopathology records) were not sought. Conditional logistic regression was used for the analysis; no potential confounders were included as covariates in the models. The odds ratio for cancer of the brain with ever-exposure to RF/MW was 1.39 (95% CI, 1.01–1.90). There was only weak evidence of a trend towards increasing odds ratio with increase in the value of the product of a score for probable intensity of exposure and duration of exposure. [The strengths of this study included its basis within a cohort, the careful design and the probably complete ascertainment of brain cancers occurring within the study period. It is limited by its lack of confirmation of diagnosis through access to diagnostic records, the reliance on occupational title to identify instances of potential exposure to RF/EMF radiation, and the uncertain accuracy of exposure quantification. Any bias due to these weaknesses would probably be towards the null and would weaken a dose-response relationship, if there were one.]

[Szmigielski \(1996\)](#) studied the incidence of cancer in the whole population of career military personnel in Poland from 1971 to 1985, averaging about 127 800 men over these 15 years. [This study appeared to be a cross-sectional study rather than a cohort study ([Table 2.2](#)).] Annual data were obtained on all career servicemen from personnel and health departments, and included numbers of servicemen, types of service posts and exposure to possible

carcinogenic factors during service, while military safety groups provided information on the number of personnel exposed to RF radiation. On average, 3720 men were considered to have been exposed to RF radiation each year. It was estimated that of these, 80–85% were exposed at $< 2 \text{ W/m}^2$ and the remainder at $2\text{--}6 \text{ W/m}^2$, but individual exposure levels could not be assigned. Exposure was largely to pulse-modulated RF radiation at 150–3500 MHz. Annual data on all men newly diagnosed with cancer were collected from records of military hospitals and the military medical board; in addition to type of cancer, they included duration and type of service and exposure to possible carcinogenic factors during service, including whether or not they were exposed to RF radiation. [It was unclear from the text whether information in individual health records may have been used, in addition to information applicable to all servicemen, in allocating a man diagnosed with cancer to the group exposed to RF radiation.]

[It appeared to the Working Group that these data were insufficient to permit calculation of annual age-specific rates of all cancers (in age groups of 10 years) and individual types of cancer in men exposed to RF radiation and men not exposed and thus to calculate ratios of incidence in the exposed group to that in the unexposed group for each year and for the whole period. The methods were described in limited detail and it was not stated how the rates or rate ratios were summarized across age groups and years and, in particular, whether cancer-incidence rate ratios based on all exposed and all unexposed men were age-standardized. The observed numbers of cases of all cancers or individual types of cancer were not presented, but could be approximated from average annual rates of incidence, from which it appeared that two to three cases of cancer of the nervous system and brain (ICD codes not specified) were diagnosed in men exposed to RF radiation over the 15 years, and about 54 cases in men not exposed.]

Table 2.2 Cohort studies of cancer of the brain and occupational exposure to radiofrequency radiation

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Lilienfeld et al. (1978) United States embassies in eastern Europe	7475	1953–76	Worked or lived in the United States embassy in Moscow during study period. The maximum measured levels were 5 $\mu\text{W}/\text{cm}^2$ for 9 h/d, 15 $\mu\text{W}/\text{cm}^2$ for 18 h/d, and < 1 $\mu\text{W}/\text{cm}^2$ thereafter for non-overlapping periods between 1953 and 1975 and between 1975 and 1976	Brain and CNS (ICD-7 code 193)	<i>Role and location in eastern Europe</i> Employed in the United States embassy in Moscow Dependent of a Moscow United States embassy employee Employed in a different United States embassy in eastern Europe	0 0 5	SMR 0 [0–4.1]* 0 [0–24.6]* 3.3 [1.1–7.7]*	Sex, age	SMRs are relative to the corresponding mortality rates in the USA. Contract report is available through the National Technical Information Service of the USA.
Milham (1988a, b) USA	67 829	1979–84	Licensing as an amateur radio operator	Brain (191)	<i>Amateur radio operator licence class</i> Novice Technician General Advanced Extra All	1 4 11 11 2 29	SMR 0.34 1.12 1.75 1.74 1.14 1.39 (0.93–2.00)		

Table 2.2 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Armstrong et al. (1994) Canada and France	191 749	1970–89	Exposure assessed through a job-exposure matrix based on about 1000 person-weeks of measurements from exposure meters worn by workers to derive estimates of short-duration PEMFs, or high-frequency transient fields.	Brain (191)	PEMFs Malignant cancer of the brain: < Median > Median ≥ 90th percentile Astrocytoma: < Median > Median ≥ 90th percentile Glioblastoma: < Median > Median ≥ 90th percentile Other cancers: < Median > Median ≥ 90th percentile Benign tumours of the brain: < Median > Median ≥ 90th percentile	49 35 9 22 12 3 16 13 5 7 6 1 9 16 1	OR 1.00 0.84 (0.47–1.50) 1.90 (0.48–7.58) 1.00 0.89 (0.29–2.67) 6.26 (0.30–132) 1.00 0.49 (0.19–1.28) 0.57 (0.08–3.91) 1.00 2.67 (0.43–16.71) – 1.00 1.58 (0.52–4.78) –	SES	Nested case-control analysis. Controls for each case were selected at random from the cases risk set and matched by utility and year of birth. Exposure was counted only up to the date of diagnosis of the case.

Table 2.2 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Grayson (1996) USA	230 cases, 920 controls	1970–89	Job title-time-exposure matrix. Census of control histories was carried out at time of matched case's diagnosis. Exposure score was the sum of the products of duration of deployment in each job and the assessed probability of RF exposure in that job at that time.	Brain (191)	RF/MW Never exposed Exposed RF/MW exposure score None 2–48 49–127 128–235 236–610	94 136 136 15 29 25 25	OR 1.00 1.39 (1.01–1.90) 1.00 1.26 (0.71–2.24) 1.50 (0.90–2.52) 1.26 (0.71–2.22) 1.51 (0.90–2.51)	Controls exactly matched to case on year of birth, race and presence in the cohort at the time when the case was diagnosed. Matching retained in analysis. Age, race, rank (senior or other) included as covariates.	United States Air Force personnel Nested case-control analysis within cohort study. All male members of United States Air Force. Rank associated with risk; senior officers at increased risk.
Szmigielski (1996) Poland	Average of 127 800 men, yearly during 15 yr	1971–85	Military safety (health and hygiene) groups classified military service posts as having exposure, or not, to RF	Nervous system, including brain	Occupational exposure to RF Not exposed Exposed P value	[54]* [2–3]*	1.00 1.91 (1.08–3.47) < 0.05	None specified	Cross-sectional study. Incidence rate ratio for all cancers, 2.07 (95% CI, 1.12–3.58) suggests possible upward bias in rate ratios.
Tynes et al. (1996) Norway	2619 women	1961–91	Radio and telegraph operators with potential exposure to RF and ELF	Brain (ICD-7 code 193)	Radio and telegraph operators with potential exposure to RF and ELF	5	SIR, 1.0 (0.3–2.3)	Age, time since certification, calendar year, age at first childbirth	Women certified as radio and telegraph operators 1920–80

Table 2.2 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Lagorio et al. (1997) Italy	481 women	1962–92	Occupational history from plant records. RF generated by dielectric heat sealers. Exposure of 10 W/m ² equivalent power-density frequently exceeded.	Brain (191)	Job title			Sex, age, calendar period-specific regional person-year at risk	Mortality analysis restricted to women
					RF-sealer operators	1	10 [0.25–56]*		
					Other workers	0	0 [0–46]*		
					All female workers	1	5 [0.13–28]*		
Morgan et al. (2000) USA	195 775 Motorola employees	1976–96	Job-exposure matrix	Brain/CNS	Cumulative exposure to RF		Rate ratio	Age, sex, and race for external comparisons; and age, sex, and period of hire for internal comparisons	All employees of Motorola; exposure from cellular phones not assessed. Definition of exposure categories unclear.
					None	34	1.00		
					< Median	7	0.97 (0.37–2.16)		
					≥ Median	10	0.91 (0.41–1.86)		
					Usual exposure to RF				
					None	38	1.00		
					Low	7	0.92 (0.43–1.77)		
					Moderate	3	1.18 (0.36–2.92)		
					High	3	1.07 (0.32–2.66)		
					Peak exposure to RF				
					None	34	1.00		
					Low	10	0.98 (0.50–1.80)		
					Moderate	3	0.70 (0.21–1.77)		
					High	4	1.04 (0.36–2.40)		
					Duration of exposure				
					None	44	1.00		
					≤ 5 yr	3	0.74 (0.22–1.84)		
					> 5 yr	4	0.99 (0.35–2.26)		
					Cumulative exposure to RF				
					Males-low	23	1.00		
					Males-high	8	1.11 (0.38–2.78)		
					Females-low	18	1.00		
					Females-high	2	0.58 (0.03–3.30)		

Table 2.2 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Groves et al. (2002) USA	40 581 men (271 women excluded)	1950–97	United States Navy personnel with potential for RF exposure; job classified as entailing low or high exposure	Brain (ICD-9 codes 191.0–191.9)	<i>Job-associated exposure to RF</i> Low High Total cohort Within-cohort comparison: Low exposure High exposure	51 37 88	SMR 1.01 (0.77–1.33) 0.71 (0.51–0.98) 0.86 (0.70–1.06)	Age at cohort entry, attained age	White United States Navy (male) veterans of Korean War (1950–54)
Degraeve et al. (2009) Belgium	Military personnel (4417 men) in battalions equipped with radar, and 2932 controls	1968–2003	Exposure levels on the site where the battalion lived and worked were characterized, individual exposure assessment could not be conducted.	Cancer of eye, brain and nervous system (190–192)	Control cohort Radar-exposed	2 8	1.00 2.71 (0.42–17.49)		Cause of death found for 71% of the men in the radar group and 70% in the control group.

* values calculated/deducted by the Working Group
d, day or days; ELF, extremely low-frequency electric and magnetic field; h, hour or hours; mo, month; MW, microwaves; OR, odds ratio; PEMFs, pulsed electromagnetic fields; RF, radiofrequency radiation; SES, socioeconomic status; SIR, standardized incidence ratio; SMR, standardized mortality ratio; W, watt; yr, year

The incidence rate ratio (IRR) for cancer of the nervous system and brain over the 15 years in those exposed to RF radiation was estimated to be 1.91 (95% CI, 1.08–3.47). The corresponding incidence rate ratio for all cancers was 2.07 (95% CI, 1.12–3.58). [The similarity of these two incidence rate ratios suggested the possibility of consistent upward bias in their estimation. It also appeared that the 95% confidence intervals had not been correctly calculated given their similar width and the large difference in the observed numbers on which they were based: 2–3 cancers of the nervous system and brain and about 32 cancers of all types.] Age-specific incidence rate ratios for all cancers ranged from 2.33 at age 20–29 years to 1.47 at age 50–59 years. [This was somewhat against the hypothesis that failure to standardize by age had increased the incidence rate ratios with exposure to RF radiation. The interpretation of this study was hampered by its cross-sectional design, in which risk of cancer was related only to current exposure to RF radiation; uncertainty about the accuracy of the classification of exposure; lack of a quantitative measure of exposure; lack of information on completeness of ascertainment of cancer incidence; lack of clarity concerning the analytical methods, including whether incidence rate ratios were age-standardized; and probable errors in the statistical analysis. Possible confounding factors were not addressed. The possibility that medical records accessed for men with cancer may have provided information that led them to being classified as exposed to RF radiation may explain the apparently high risks of cancer in men exposed to RF radiation in this study.]

[Tynes et al. \(1996\)](#) examined incidence of cancer in a cohort of 2619 Norwegian women who were certified as radio and telegraph operators between 1920 and 1980 ([Table 2.2](#)); 98% had worked on merchant navy ships. They were followed from 1961 to 1991 via the Norwegian cancer registry; 41 were lost to follow-up. Electric and magnetic fields were measured in the radio

rooms of three older Norwegian ships. They were below detection levels at radio-frequencies at the operators' desks and were considered to be comparable to those found at normal Norwegian workplaces. The age- and calendar period-adjusted standardized incidence ratio (SIR) for cancers of the brain and nervous system (ICD-7 code 193) was 1.0 (95% CI, 0.3–2.3; based on five cases) with reference to the national Norwegian female population. [The strengths of this study were its homogeneous cohort and near-complete follow-up; its principal weaknesses were the small number of cases of brain cancer and the probably low exposure of the cohort to RF radiation. Possible confounding factors were not addressed.]

[Lagorio et al. \(1997\)](#) reported on mortality from all causes and from specific cancers in a group of 201 men and 481 women employed in a plastic-ware manufacturing facility in Grosseto, Italy, from 1962 to 1992 and followed until death, or until the end of 1992 ([Table 2.2](#)). Those lost to follow-up were considered to be alive at the end of 1992. Vital status and cause of death were ascertained from the registry office of the municipality of residence and death. Workers were classified into three groups: RF-sealer operators, other labourers and white-collar workers. RF-sealer operators received the greatest exposure to RF radiation. They were also exposed to vinyl chloride monomer due to its volatilization from polyvinyl chloride (PVC) sheets during sealing. At the end of follow-up, 661 subjects were alive, 16 had died and 5 were lost to follow-up [details of tracing methods were not given]. The mortality analysis was restricted to women, who were mostly employed in the manufacturing department (6772 person-years in RF-sealer operators). There was one death ascribed to a tumour of the brain and 0.2 expected based on mortality rates in the regional population; this single death occurred in an RF-sealer operator (expected, 0.1). [The principal weakness of this study was its

small size. Possible confounding from exposure to vinyl chloride was not addressed.]

[Morgan et al. \(2000\)](#) studied a cohort of all employees of Motorola USA with at least 6 months of cumulative employment, who were employed for at least 1 day between 1976 and 1996, and followed to 31 December 1996. Deaths were ascertained through reference to the Social Security Administration Master Mortality File and the National Death Index. Death certificates were obtained from the state vital statistics offices and company benefits records, and causes of death were coded according to ICD-9. There were 195 775 workers, 2.7 million person-years of follow-up and 6296 deaths, 53 of which were attributed to cancer of the CNS [ICD-9 codes not stated]. No losses to follow-up were reported [it is probable that the 116 700 workers who had retired or whose employment had been terminated were assumed to be alive if no death record was found for them]. Exposure to RF radiation was assessed on the basis of a company-wide job-exposure matrix, developed through expert consultation, that categorized each of 9724 job titles into one of four exposure groups: background, low, moderate, and high, corresponding roughly to < 0.6 W, $0.6 - < 2.0$ W, $2.0 - < 5.0$ W, $5.0 - < 50$ W and ≥ 50 W. About 45 500 employees were thought to have had usual exposures of ≥ 0.6 W, 8900 employees had a high usual exposure (≥ 50 W) and 9000 employees had unknown usual exposure. Relative to mortality in the combined populations of Arizona, Florida, Illinois, and Texas, where most Motorola facilities were located, the SMR for tumours of the CNS was 0.60 (95% CI, 0.45–0.78). Internal comparisons between categories of estimated cumulative, usual and peak exposure to RF radiation; duration of exposure; (cumulative exposure lagged 5, 10 and 20 years) and cumulative exposure in males and females separately, showed no consistent evidence of an increase in risk of tumours of the CNS with increasing estimated exposure to RF radiation ([Table 2.2](#)). [The main

strength of this study was the clear and straightforward execution and comprehensive analyses. Its weaknesses included lack of measured exposure to RF radiation on which to base the exposure classification; inadequate description of the exposure-validation study; lack of detail on how the links between cohort members and death records were established, and therefore uncertainty about completeness and accuracy of death ascertainment; the comparatively small number of observed deaths from tumours of the CNS; and possible conservative bias due to exclusion of mobile-phone use from the estimate of exposure to RF radiation. Possible confounding factors were not addressed.]

[Groves et al. \(2002\)](#) reported on an extended follow-up to death or to the end of 1997 for 40 890 United States Navy personnel originally studied by [Robinette et al. \(1980\)](#). These men were graduates of Class-A Navy technical training schools who had served on ships in the Korean War during 1950–54, and who had potentially been exposed to high-intensity radar. They were divided into two occupational groups considered by a consensus of Navy personnel involved in training and operations to have had high exposure to RF radiation (electronics, fire-control and aviation-electronics technicians: 20 109 men) or low exposure (radiomen, radar men and aviation electricians' mates: 20 781 men). Potential exposure in each job category was evaluated from the records for 435 men who had died and those of a randomly selected 5% of living men as "the sum of all power ratings of all fire control radars aboard the ship or search radars aboard the aircraft to which the technician was assigned multiplied by the number of months of assignment." Ascertainment of death required use of Department of Veterans' Affairs and Social Security Administration records and the National Death Index. [Its completeness was uncertain.] It was necessary to impute moderate proportions of dates of entry into the cohort (1950–54) and dates of birth, because of missing

data. The analysis was limited to 40 581 men and SMRs were calculated with reference to all white men in the USA, standardized for age at entry to the cohort and attained age. Altogether, there were 51 deaths from cancer of the brain (ICD-9 codes 191.0–191.9); there was no evidence of any increase in risk of cancer of the brain associated with high exposure to RF radiation ([Table 2.2](#)). The SMRs for cancer of the brain were 0.86 (95% CI, 0.70–1.06) for the whole cohort, 1.01 (95% CI, 0.77–1.33) for the group with low exposure to RF radiation (usual exposures well below 1 mW/cm²) and 0.71 (95% CI, 0.51–0.98) for the group with high exposure to RF radiation (potential for exposures up to 100 kW/cm², but usually less than 1 mW/cm²). Within the cohort, the relative risk (RR) of death from cancer of the brain in the group with high exposure to RF radiation relative to the group with low exposure was 0.65 (95% CI, 0.43–1.01). [This appeared to have been an initially somewhat poorly documented cohort, for which follow-up was difficult and some missing data, including birth date, had to be imputed. While expert assessment permitted division of the cohort into groups with low and high exposure to RF radiation, no specific measurements of exposure were reported. Assessment of exposure appeared to have been limited to 1950–54. Possible confounding factors, such as occupational exposure to other agents, were not addressed.]

In a cohort of 4417 Belgian male professional military personnel who served in battalions equipped with radar for anti-aircraft defence, cause-specific mortality was compared with that of 2932 Belgian military personnel who served in battalions not equipped with radar ([Degraeve et al., 2009](#)). Administrative archives of the battalions were used to reconstruct a list of personnel serving in each battalion. Lists were matched to those of the Department of Human Resources of the Belgian Army to find the subjects' birthdays, which allowed retrieval of their Belgian national identity number. With this number, mortality

follow-up could be conducted. For military personnel who died before 1979, the registry only recorded month and year of birth, and thus for 35 dead military exact birth-dates were not available, matching was equivocal and the cause of death was not used. The registry was complete until 1997 and from 1998 to 2004, only for the northern, Dutch-speaking part of Belgium. In parallel, for all professional military personnel who died up to 31 December 2004, first-degree family members were sought and a questionnaire sent to enquire about likely cause of death. For the period of follow-up of this study, the Belgian cancer registry was incomplete, but the information on cases of cancer reported to the registry was reliable. Thus the cancer registry was used only for confirmation, but not for identification of cancer cases. The risk ratio for deaths from cancers of the eye, brain and nervous system in the cohort serving in battalions equipped with radar compared with the unexposed cohort was 2.71 (95% CI, 0.42–17.49) ([Table 2.2](#)). [The Working Group noted the difficulties in following-up the study population that may have affected the study results, as well the difficulty of attributing any possible increase in risk ratio to exposure to RF radiation, given possible confounding due to ionizing radiation also emitted by devices producing MW radiation.]

2.1.2 Leukaemia and lymphoma

(a) Case-control studies

No data were available to the Working Group.

(b) Cohort studies

[Lilienfeld et al. \(1978\)](#) reported on a retrospective cohort study of USA employees, and their dependents, who had worked or lived at the United States embassy in Moscow during 1953–76 (see Section 2.1.1 for details). The total risk ratio for leukaemia in the embassy employees was 2.5 (95% CI, 0.3–9.0).

[Milham \(1988a, b\)](#) followed a cohort of people who were licensed as amateur radio operators between 1 January 1979 and 16 June 1984 (see Section 2.1.1 for details). There was a borderline excess risk of death from lymphatic and haematopoietic neoplasms, from acute myeloid leukaemia, and from multiple myeloma and lymphoma ([Table 2.3](#)). There was no evidence for an increase in SMR for these neoplasms with higher license class (see Section 2.1.1. for discussion of the strengths and weaknesses of this study).

[Armstrong et al. \(1994\)](#) conducted a nested case-control study of cancers at different sites within cohorts of electrical workers in Quebec, Canada, and in France (see Section 2.1.1 for details). There were no excess risks for all haematological cancers, non-Hodgkin lymphoma (NHL) or for all leukaemias, or for any of the subtypes of leukaemia, associated with exposure to PEMF ([Table 2.3](#)). [The strengths and weaknesses of this study are described in Section 2.1.1.]

The study by [Szmigielski \(1996\)](#) is described in detail in Section 2.1.1. A significantly increased incidence rate ratio for cancers of the haematological system and lymphatic organs was reported ([Table 2.3](#)). [The results were difficult to interpret, as there were many methodological flaws in the design and analysis of the study. Main issues were that exact data on the age of the subjects in the cohort were missing and that collection of exposure data was potentially differential.]

[Tynes et al. \(1996\)](#) followed a cohort of 2619 Norwegian women who were certified as radio and telegraph operators between 1920 and 1980. There was no elevation in risk of lymphoma or leukaemia for those potentially exposed to RF radiation ([Table 2.3](#)). [The strengths of this study are discussed in Section 2.1.1; its principal weaknesses were the small number of cancer cases and the probably low exposure of the cohort to RF radiation. Possible confounding factors were not addressed.]

[Lagorio et al. \(1997\)](#) reported on mortality from all causes and from specific cancers in a group of plastic sealers in Italy (see Section 2.1.1 for details). There was one death (0.2 expected) ascribed to leukaemia in an RF-sealer operator ([Table 2.3](#)). [The principal weakness of this study was its small size. Possible confounding factors were not addressed.]

[Morgan et al. \(2000\)](#) reported on a 20-year follow-up of 195 775 employees of Motorola USA (described in Section 2.1.1) and considered death from lymphatic and haematopoietic malignancies ([Table 2.3](#)). Of these, there were 87 deaths from leukaemia, 19 from Hodgkin disease and 91 from NHL. Reduced odds ratios for lymphatic and haematopoietic malignancies and subtypes were seen among workers categorized as exposed (compared with non-exposed workers) in most categories of estimated exposure, duration of exposure and cumulative exposure lagged 5, 10 and 20 years. [The Working Group noted the small number of deaths from lymphoma and leukaemia in the exposed cohort, which, together with the other limitations mentioned in Section 2.1.1, complicated the interpretation of these findings.]

[Richter et al. \(2000\)](#) collected data on six patients claiming compensation for their cancer who visited the Unit of Occupational and Environmental Medicine at the Hebrew University-Hatlawah Medical School, Jerusalem in 1992–99. They were judged to have received high RF/MW radiation based on self-reports, information from manuals containing specifications of the equipment they had used and repaired, and results of sporadic measurements from their work and medical records. A study was then conducted of 25 co-workers of one of the patients and of other personnel with self-reported exposure to RF radiation. An increased risk of haematolymphatic malignancies was found (5 cases observed compared with 0.02 cases expected among Jewish men aged 20–54 years). [The Working Group noted that the results of

this study were very difficult to interpret, due to unclear definition of the study population, follow-up and exposure assessment.]

[Groves et al. \(2002\)](#) reported on mortality in a cohort of 40 890 male United States Navy personnel who had served on ships during the Korean War in 1950–54 in an extended follow-up to 1997 (described in more detail in Section 2.1.1). The cohort was divided into two subgroups on the basis of job title, with potential exposure to RF radiation based on expert assessment: 20 109 workers comprising a subcohort with high exposure to RF radiation (potential for exposures up to 100 kW/cm², but usually < 1 mW/cm²) and a subcohort of 20 781 workers with low exposure (usually well below 1 mW/cm²). A total of 182 deaths from lymphoma or multiple myeloma (91 each in the high- and low-exposure subcohorts) and 113 deaths from leukaemia (44 and 69 in the high- and low-exposure subcohorts, respectively) were identified in 1950–97. In both subcohorts, SMRs were not elevated for lymphoma and multiple myeloma, all leukaemias, lymphocytic leukaemia or non-lymphocytic leukaemia ([Table 2.3](#)). An internal comparison of high relative to low exposure to RF radiation elicited RRs of 0.91 (95% CI, 0.68–1.22) for lymphoma and multiple myeloma, 1.48 (95% CI, 1.01–2.17) for all leukaemias, 1.82 (95% CI, 1.05–3.14) for non-lymphocytic leukaemia and 1.87 (95% CI, 0.98–3.58) for acute non-lymphocytic leukaemia. An increased risk of all leukaemias was observed primarily in aviation-electronics technicians (RR, 2.60; 95% CI, 1.53–4.43, based on 23 deaths) and was highest for acute myeloid leukaemia (RR, 3.85; 95% CI, 1.50–9.84, based on 9 deaths). RRs for other job categories with high exposure were close to 1. This was interpreted as indicating a possible association, since aviation-electronics technicians who dealt primarily with mobile radar units may have had more potential to enter the beam path of an operating radar than members of other groups who worked with ship-mounted radars. [The limitations of this study

are discussed in Section 2.1.1, including limitations in the documentation of the cohort definition and difficulties in follow-up. Classification of exposure to RF radiation in the different groups was based on expert assessment. No measurement of RF radiation was provided.]

[Degraeve et al. \(2009\)](#) compared a cohort of 4417 Belgian male professional military personnel who served in battalions equipped with radars for anti-aircraft defence with 2932 Belgian male professional military personnel who served at the same time in the same place in battalions not equipped with radars. Attempts were made to characterize exposure levels on the site where the battalion lived and worked, but individual exposure assessment could not be conducted. Administrative archives of the battalions were used to reconstruct a list of personnel serving in each battalion. These archives only provided first name, family names, and a unique identification number. Lists were matched to those of the Department of Human Resources of the Belgian Army to find the subjects' birthdays, which allowed retrieval of their Belgian national identity number. With this number, mortality follow-up could be conducted. The first source of information on cause of death was the official Belgian death registry, which collects anonymous data. Linkage was conducted using date of birth and date of death as matching variables (cause of death could be found for 71% of persons in the radar group and 70% in the control group). For military personnel who died before 1979, the registry only recorded month and year of birth, and exact birth-dates were not available for 35 of the dead, while matching was equivocal and the cause of death was not used. The registry was complete until 1997 and from 1998 to 2004, only for the Northern, Dutch-speaking part of Belgium. In parallel, for all professional military personnel who died up to 31 December 2004, first-degree family members were sent a questionnaire to enquire about the likely cause of death. For the period of follow-up of this study,

Table 2.3 Cohort studies of leukaemia and occupational exposure to radiofrequency radiation

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Lilienfeld et al. (1978)	7475	1953–76	Worked or lived in the United States embassy in Moscow during study period. The maximum measured levels were 5 $\mu\text{W}/\text{cm}^2$ for 9 h/d, 15 $\mu\text{W}/\text{cm}^2$ for 18 h/d, and < 1 $\mu\text{W}/\text{cm}^2$ thereafter for non-overlapping periods 1953–1975 and 1975–1976	Leukaemia	Embassy employees		2.5 (0.3–9.0)	Sex, age	SMRs are relative to the corresponding mortality rates in the USA. Contract report is available through the National Technical Information Service of the USA.
Milham (1988a, b) USA	67 829	1979–1984	Licensing as an amateur radio operator	Lymphatic and haematopoietic cancers	All leukaemia AML Multiple myeloma and lymphoma All lymphatic and haematopoietic cancers <i>By licence class:</i> Novice Technician General Advanced Extra	89 36 15 43	SMR 1.23 (0.99–1.52) 1.24 (0.87–1.72) 1.76 (1.03–2.85) 1.62 (1.17–2.18)		The death rates used to estimate the expected numbers were not specified

Table 2.3 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Armstrong et al. (1994) Canada and France	191 749	1970–89	Exposures assessed through a job-exposure matrix based on about 1000 person-weeks of measurements from exposure meters worn by workers to derive estimates of short-duration PEMFs, or high-frequency transient fields.	Haematological	PEMFs All haematological cancers: < Median > Median ≥ 90th percentile NHL: < Median > Median ≥ 90th percentile All leukaemias: < Median > Median ≥ 90th percentile	167 135 28 54 56 13 57 38 9	OR 1.00 0.90 (0.65–1.25) 0.96 (0.48–1.90) 1.00 1.41 (0.83–2.38) 1.80 (0.62–5.25) 1.00 0.69 (0.40–1.17) 0.80 (0.19–3.36)	SES	Nested case-control analysis Controls for each case were selected at random from the cases risk set, and matched to the case by utility and year of birth. Exposure was counted only up to the date of diagnosis of the case.
Szmigielski (1996) Poland	Average of 127 800 men, yearly during 15 yr	1971–85	Military safety (health and hygiene) groups classified military service posts as having exposure, or not, to RF	Cancer of the haematopoietic system and lymphatic organs	Occupational exposure to RF Not exposed Exposed	[131]* [24]*	Incidence rate ratios 1.00 6.31 (3.12–14.32)	None specified	Cross-sectional study
Tynes et al. (1996) Norway	2619 women	1961–91	Radio and telegraph operators with potential exposure to RF and ELF fields	Lymphoma (ICD-7 code 201) Leukaemia (ICD-7 code 204)	Potential exposure Lymphoma Leukaemia	5 2	SIR 1.3 (0.4–2.9) 1.1 (0.1–4.1)	Age, time since certification, calendar year, age at first childbirth	Women certified as radio and telegraph operators, 1920–80

Table 2.3 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Lagorio <i>et al.</i> (1997) Italy	481 women	1962–92	Occupational history from plant records. RF generated by dielectric heat sealers. Exposure of 10 W/m ² equivalent power-density frequently exceeded.	Leukaemia (204–208)	<i>Job title</i> RF-sealer operators Other workers All female workers	1 1 2	SMR [5.0 (1.27–27.85)]* [11.1]* 8.0 (1.0–28.8)	Sex, age, calendar period-specific regional person-year at risk	Mortality analysis; restricted to women
Richter <i>et al.</i> (2000) Israel	Co-workers (n = 25) of one of the six patients claiming compensation for their cancer, and other personnel with self-reported exposure to RF	1992–99	Self-reports, information from manuals containing specifications of the equipment they had used and repaired, and results of sporadic measurements from their work and from medical records.	Haemato-lymphatic malignancies	Jewish men aged 20–54 yr	5	SIR [250 (81.17–583.42)]*	None	[The Working Group noted that the results of this study were very difficult to interpret, due to unclear definition of the study population, follow-up, and exposure assessment.]

Table 2.3 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Morgan et al. (2000) USA	195 775	1976–96	Job-exposure matrix; developed through expert consultation that categorized each of 9724 job titles into different RF exposure groups: background, low, moderate, and high, corresponding roughly to < 0.6 W, 0.6 to < 2.0 W, 2.0 to < 5.0 W, 5.0 to < 50 W and ≥ 50 W, respectively	All lymphatic/haematopoietic cancers (n = 203)	Cumulative exposure to RF		Rate ratio	Age, sex, and race for external comparisons; and age, sex, and period of hire for internal comparisons	All employees of Motorola; exposure from cellular telephones not assessed. Definition of exposure categories unclear
					None	148	1.00		
					< Median	21	0.74 (0.39–1.28)		
					> Median	34	0.67 (0.40–1.05)		
					Usual exposure to RF				
					None	152	1.00		
					Low	28	0.94 (0.57–1.47)		
					Moderate	10	0.90 (0.39–1.78)		
					High	8	0.70 (0.27–1.47)		
					Peak exposure to RF				
					None	145	1.00		
					Low	34	0.79 (0.49–1.23)		
					Moderate	11	0.58 (0.25–1.13)		
					High	10	0.60 (0.49–1.23)		
					Duration of exposure				
					None	182	1.00		
					≤ 5 yr	5	0.29 (0.12–0.57)		
					> 5 yr	16	0.89 (0.55–1.35)		
					Cumulative exposure to RF				
					Males-low	109	1.00		
					Males-high	19	0.53 (0.39–0.72)		
					Females-low	60	1.00		
					Females-high	15	1.12 (0.22–3.90)		

Table 2.3 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Groves <i>et al.</i> (2002) USA	40 581 men (271 women excluded)	1950–97	United States Navy personnel with potential exposure to RF; job classified as having low or high exposure by a consensus of Navy personnel involved in training and operation	All haematopoietic cancers (200–208)	<i>Job-associated exposure to RF</i>		SMR	Age at cohort entry and attained age	White (male) United States Navy veterans of Korean War (1950–54) Reference: all white men, USA Group of aviation-electronics technicians: RR 2.60 (1.53–4.43) for all leukaemias; RR 3.85 (1.50–9.84) for acute myeloid leukaemia
					Lymphoma and multiple myeloma:				
					Low	91	0.94 (0.77–1.16)		
					High	91	0.89 (0.72–1.09)		
					All leukaemias:				
					Low	44	0.77 (0.58–1.04)		
					High	69	1.14 (0.90–1.44)		
					Lymphocytic leukaemia:				
					Low	17	1.31 (0.81–2.11)		
					High	16	1.12 (0.69–1.83)		
					Non-lymphocytic leukaemia:				
					Low	20	0.67 (0.43–1.04)		
					High	39	1.24 (0.90–1.69)		

Table 2.3 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Degraeve et al. (2009) Belgium	Military personnel (4417 men) in battalions equipped with radar, and 2932 controls	1968–2003	Exposure levels on the site where the battalion lived and worked were characterized; individual exposure assessment could not be conducted.	Cancer of lymphatic and haematopoietic tissue (200–208)	Control cohort Radar exposed	1 11	1.00 7.22 (1.09–48.9)		Cause of death found for 71% of the men in the radar group and 70% in the control group

* values calculated/deduced by the Working Group
 AML, acute myeloid leukaemia; ELF, extremely low frequency electric and magnetic field; MW, microwaves; NHL, non-Hodgkin lymphoma; OR, odds ratio; PEMFs, pulsed electromagnetic fields; RF, radiofrequency radiation; SES, socioeconomic status; SIR, standardized incidence ratio; SMR, standardized mortality ratio; W, watt; yr, year

the Belgian cancer registry was extremely incomplete, but the information on cases of cancer reported to the registry was reliable. Thus, the cancer registry was used only for confirmation but not for identification of cancer cases. The relative risks were estimated, adjusting for age in 10-year categories with a Poisson regression model. There were 11 deaths from lymphatic and haematopoietic neoplasms in the radar battalion compared with 1 in the unexposed cohort (RR, 7.22; 95% CI, 1.09–48.9) (Table 2.3). [The Working Group noted the difficulties in following-up the study population, which may have affected the study results, as well the difficulty in attributing any possible increase in relative risk to exposure to RF radiation, given possible confounding due to ionizing radiation also emitted by devices producing MW radiation.]

2.1.3 Uveal (ocular) melanoma

Stang *et al.* (2001) conducted population-based and hospital-based case-control studies of uveal melanoma and occupational exposures to different sources of electromagnetic radiation, including RF radiation. For the population-based study, 37 cases were identified by a reference pathologist (response rate, 84%) and 327 controls were sampled and matched from the same region of residence, age and sex (response rate, 48%). For the hospital-based study, the 81 cases were patients treated at the University of Essen (response rate, 88%) and controls ($n = 148$) were patients with benign intra-ocular tumours (response rate, 79%). The results of these studies were pooled. The 118 female and male cases and 475 controls were interviewed by a trained interviewer with a structured questionnaire involving medical history, lifestyle, occupation and occupational exposure to RF radiation. Participants were specifically asked about exposure to radar and to other RF-emitting devices (“Did you use radio sets, mobile phones or similar devices at your workplace for at least several hours per

day?”) and more detail was obtained from those who reported exposure. Additional information provided by exposed participants was used by two of the authors, working independently and unaware of case or control status, to classify them as: exposed only to radio receivers that do not transmit RF radiation and therefore unexposed; exposed to RF radiation from walkie-talkies and radio sets; possibly exposed to RF radiation from mobile phones; and probably or certainly exposed to RF radiation from mobile phones. Few participants reported occupational exposure to radar. The odds ratio for uveal melanoma was 0.4 (95% CI, 0.0–2.6). For exposure to radio sets, the odds ratio was 3.3 (95% CI, 1.2–9.2) (Table 2.4). Adjustment for socioeconomic status or iris/hair colour did not alter these results. The results for reported occupational use of mobile phones are considered in Section 2.3. [This study was weakened by its poor assessment of occupational exposure to RF radiation, particularly the retrospective classification of exposure to other RF-emitting devices, although neither should be a source of positive bias. Confounding of occupational exposure to RF radiation with exposure to ultraviolet light from the sun or other sources was not considered and may have been important if, for example, much of the use of radio sets had entailed use of walkie-talkie radios for communication outdoors.]

2.1.4 Cancer of the testis

(a) Case-control studies

Interpretable results were available from only two case-control studies (Table 2.5). Both were limited by reliance on self-report for exposure classification.

Hayes *et al.* (1990) carried out a case-control study in the USA examining associations of testicular cancer with occupation and occupational exposures. Cases ($n = 271$) were aged 18–42 years and diagnosed between 1976 and 1981 in three medical institutions, two of which

treated military personnel, while the controls ($n = 259$) were men diagnosed in the same centres with a cancer other than of the genital tract. A complete occupational history was taken and participants were also asked about specific exposures, including to radar equipment and to MW radiation, MW ovens or other radio-waves. For all cancers of the testis combined, the odds ratio associated with exposure to MW radiation, MW ovens or other radio-waves was significantly increased, while the odds ratio for exposure to radar equipment was not elevated ([Table 2.5](#)). The participants were further classified by an industrial hygienist as to degree of exposure to MW radiation, MW ovens, and other radio-waves and no indication of an exposure–response relationship was found. [The Working Group noted that the exposure-classification approach was based on self-report and was probably subject to substantial misclassification.]

[Baumgardt-Elms et al. \(2002\)](#) carried out a case–control study examining the association of cancer of the testis with workplace exposures to EMF. The histologically confirmed cases ($n = 269$; including 170 seminomas and 99 non-seminomas) were recruited between 1995 and 1997 from five German regions (response rate, 76%). The controls ($n = 797$) were randomly selected from mandatory registries of residents, with matching on age and region (response rate, 57%). Occupational exposure to EMF was assessed in standardized face-to-face interviews with closed questions. For radar, job descriptions were selected for participants who had reported exposure to radar or had worked in occupations and industries involving such exposures. The participants were classified as to exposure to radar on the basis of expert review and measurements conducted in Germany. There was no excess risk of cancer of the testis associated with being classified as having exposure to radar. [A comparison of self-report of exposure with classification by the expert panel showed substantial misclassification from reliance on self-report.]

(b) Cohort study

[Davis & Mostofi \(1993\)](#) reported six cases of cancer of the testis in a cohort of 340 police officers who used hand-held radar guns in the state of Washington, USA. Only one case was expected, based on national data. [The Working Group noted that the finding of the six cases as a cluster had sparked the investigation. Exposure assessments were not made for the full cohort.]

2.1.5 Other cancers

[Armstrong et al. \(1994\)](#) carried out a nested case–control study of the association between exposure to PEMFs and various cancers, including lung (described in Section 2.1.1). An association was observed between exposure to PEMFs and cancer of the lung ([Table 2.6](#)). The highest excess risk was found in cases first exposed 20 years before diagnosis. [The relevance of the measured EMF parameters to exposure to RF radiation was unclear.]

No association of RF radiation with cancer of the lung has been reported in other studies ([Milham, 1988a](#); [Szmigielski, 1996](#); [Tynes et al., 1996](#); [Lagorio et al., 1997](#); [Morgan et al., 2000](#); [Groves et al., 2002](#); [Degraeve et al., 2009](#); described in Section 2.1.1, and [Table 2.6](#)). A later overview by [Szmigielski et al. \(2001\)](#) reported an incidence rate ratio of 1.06 in the population studied by [Szmigielski \(1996\)](#), based on 724 not-exposed cases and 27 exposed cases.

[Tynes et al. \(1996\)](#) (described in Section 2.1.1) studied the impact of exposure to RF radiation (405 kHz to 25 MHz) in an occupational cohort of Norwegian female radio/telegraph operators who had worked at sea for extended periods. There were increased standardized incidence ratios (SIR) for cancers of the breast and uterus ([Table 2.6](#)). A nested case–control analysis for cancer of the breast was performed within this cohort. To control for the possible confounding effect of reproductive history, the investigators linked the cohort to a unique database from

the Norwegian Central Bureau of Statistics that contained information on the reproductive histories of Norwegian women born between 1935 and 1969. After adjusting for duration of employment, the odds ratio for cancer of the breast was 4.3 (95% CI, 0.7–26.0) in women aged ≥ 50 years who had performed a large amount of shift-work (> 3.1 – 20.7 category–years). Adjustment for shift-work and relevant reproductive history reduced the odds ratio for cancer of the breast to 1.1 (95% CI, 0.2–6.1) in those with the longest duration of employment. [The apparent excess risk of cancer of the breast in this cohort, based on high-quality databases and linkage, was not explained by reproductive history and could be potentially attributed to exposure to light at night.]

2.2 Environmental exposure from fixed-site transmitters

Ecological studies are considered to provide a lower quality of evidence than case–control or cohort studies, as they reflect the possibility of uncontrolled confounding and possible misclassification of exposure. With regard to exposure to RF radiation and its association with cancers of the brain, there appears to be little possibility of confounding by anything other than socio-demographic factors associated with diagnostic opportunity. For other cancer sites, confounding may be of greater concern.

Individual measurements of distance from a transmitter as a proxy for exposure are effectively ecological measures, in which the ecological unit includes everyone living at the same distance, or within a restricted range of distances, from the transmitter. Spot measurements will only be partly correlated with total exposure and even a personal exposure meter provides only an approximation of the dose of radiation absorbed by a specific tissue. Measurement of lifetime exposure is problematic regardless of the study design, particularly when there is a high level of

population mobility and measurements of exposure are not readily available for previous areas of residence.

The crucial issue is to what extent the exposure surrogate is associated with the radiation absorbed, since this modulates the statistical power of the study. Some studies have validated correlations between proxy measures, based either purely on distance or on a more sophisticated propagation model. In some cases the correlation has been estimated at approximately 60%, in others it is $< 10\%$, especially when based upon self-report of exposures ([Schmiedel *et al.*, 2009](#); [Viel *et al.*, 2009](#); [Frei *et al.*, 2010](#)). Hence it is difficult to assume that exposure classification based on distance-based proxy measurements is useful, unless validation measurements are included. Detailed modelling of field propagation shows that several parameters are potentially required.

2.2.1 Cancer of the brain

(a) Ecological studies

In several ecological studies, incidence or mortality rates of brain tumours have been compared between defined populations living close to television or radio broadcast stations or other RF radiation fixed-site transmitters or transmission towers.

[Selvin *et al.* \(1992\)](#) undertook a cross-sectional analysis in which the proportion of people aged < 21 years with cancer of the brain diagnosed between 1973 and 1978 living ≤ 3.5 km or > 3.5 km from a large MW transmission tower (Sutro Tower) in San Francisco, USA ($n = 35$) was compared with corresponding proportions from the 1980 USA census. The odds ratio for cancer of the brain and living ≤ 3.5 km from the tower was 1.16 [95% CI, 0.56–2.39]. [No possible confounding factors were considered, nor were the ambient levels of RF radiation in the compared areas documented.]

Table 2.4 Case-control study of uveal melanoma and occupational exposure to radiofrequency radiation

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Relative risk (95% CI)	Covariates	Comments
Stang <i>et al.</i> (2001) Germany, 1995–98	118 (37/81)	475	Population-based study: 327 controls sampled and matched from the same region of residence, age, and sex. Hospital-based study: controls were 148 patients with benign intraocular tumours.	Interviewer-administered structured questionnaire	Uveal melanoma	Radar units Radio set: Ever exposed Exposed ≥ 5 yr before Exposed for ≥ 3 yr	1 9 9 7	0.4 (0.0–2.6) 3.3 (1.2–9.2) 3.3 (1.2–9.2) 2.5 (0.8–7.7)	Age, sex, region, SES, colour of iris and hair	Results of the population-based study (37 cases) and the hospital-based study (81 cases) were pooled.

SES, socioeconomic status; yr, year

Table 2.5 Case-control studies of cancer of the testis and occupational exposure to radiofrequency radiation

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hayes <i>et al.</i> (1990) USA, 1976–81	271	259	Non-genital cancer, diagnosed in same hospital	Interviews, including a complete occupational history.	Testicular seminoma (<i>n</i> = 60) Other germinal (<i>n</i> = 206) Non-germinal (<i>n</i> = 5)	<i>Radar equipment</i> Seminoma Other Total <i>Other MW/RF</i> Seminoma Other Total Based on job title: None Low Medium High	12 30 NR 7 24 NR 116 10 6 12	1.3 (0.6–2.8) 1.1 (0.6–1.9) 1.1 (0.7–1.9) 2.8 (0.9–8.6) 3.2 (1.4–7.4) 3.1 (1.4–6.9) 1.0 2.3 (0.6–9.4) 1.0 (0.3–3.8) 0.8 (0.3–2.0)	Age	Self-reported exposure Two of the three centres treated military personnel Poor agreement between self-reporting and job title No response rates reported

Table 2.5 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Baumgardt-Elms <i>et al.</i> (2002) Germany, 1995–97	269	797	Controls were randomly selected from mandatory registries of residents, and matched on age and region (response, 57%).	Interviewer-administered standardized questionnaire	Testicular (170 seminoma and 99 non-seminoma)	Radar units	22	1.0 (0.60–1.75)		Exposure to RF weighted by duration and distance from source
						RF emitters	50	0.9 (0.60–1.24)		
						Radar units				
						0	251	1.0		
						> 0 to ≤ 45	7	1.4 (0.55–3.77)		
						> 45 to ≤ 135	4	0.5 (0.17–1.55)		
						> 135 to ≤ 2225	7	0.9 (0.36–2.19)		
						RF emitters				
						0	220	1.0		
						> 0 to ≤ 6	19	1.0 (0.56–1.74)		
						> 6 to ≤ 15	14	0.7 (0.38–1.35)		
						> 15 to ≤ 102	16	0.9 (0.46–1.56)		

MW, microwave; NR, not reported; RF, radiofrequency radiation

Table 2.6 Cohort studies of cancers of the lung and other sites and occupational exposure to radiofrequency radiation

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Milham (1988a, b) USA	67 829	1979–84	Licensing as an amateur radio operator	Cancer of the respiratory system (160–163)	None	209	SMR, 0.66 (0.58–0.76)		The death rates used to estimate the expected numbers were not specified
Armstrong et al. (1994) Canada and France	191 749	1970–89	Exposures assessed through a job-exposure matrix based on about 1000 person-wks of measurements from exposure meters worn by workers to derive estimates of short-duration PEMFs, or high-frequency transient fields.	Lung cancer (162)	PEMFs < Median > Median ≥ 90th percentile First exposed 0–20 yr before diagnosis: < Median > Median ≥ 90th percentile First exposed > 20 yr before diagnosis: < Median > Median ≥ 90th percentile	200 308 84	OR 1.00 1.27 (0.96–1.68) 3.11 (1.60–6.04)	SES	Nested case-control analysis Controls for each case were selected at random from the cases risk set and matched to the case by utility and year of birth. Exposure was counted only up to the date of diagnosis of the case.
Szmigielski (1996) Poland	Average of 127 800 men, yearly during 15 yr	1971–85	Military safety (health and hygiene) groups classified military service posts as having exposure, or not, to RF	Cancer of the larynx and lung	Occupational exposure to RF Not exposed Exposed	[420]* [13]*	Incidence rate ratios 1.00 1.06 (0.72–1.56)	None specified	Cross-sectional study.
Tynes et al. (1996) Norway	2619 women	1961–91	Radio and telegraph operators with potential exposure to RF and ELF fields	Lung (ICD-7 code 162) Breast (ICD-7 code 170) Uterus (ICD-7 code 172)	Potential exposure Lung Breast Uterus	5 50 12	SIR 1.2 (0.4–2.7) 1.5 (1.1–2.0) 1.9 (1.0–3.2)	Age, time since certification, calendar year, age at first childbirth	Women certified as radio and telegraph operators 1920–80

Table 2.6 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Lagorio et al. (1997) Italy	481 women	1962–92	Occupational history from plant records. RF generated by dielectric heat sealers. Exposure of 10 W/m ² equivalent power-density frequently exceeded.	Lung (162)	<i>Job title</i> RF-sealer operators Other workers All female workers	1 0 1	SMR [5 (1.27–27.85)]* - [3.3]*	Sex, age, calendar period-specific regional person-yr at risk	Mortality analysis; restricted to women
Morgan et al. (2000) USA	195 775	1976–96	Job-exposure matrix, developed through expert consultation that categorized each of 9724 job titles into different RF exposure groups: background, low, moderate, and high, corresponding roughly to < 0.6 W, 0.6 to < 2.0 W, 2.0 to < 5.0 W, 5.0 to < 50 W and ≥ 50 W, respectively.	Respiratory system cancer	<i>RF exposure</i> High and moderate	94	SMR [approx. 0.8]*	Age, sex, and race for external comparisons; and age, sex, and period of hire for internal comparisons	All employees of Motorola; exposure from cellular phones not assessed. Definition of exposure categories unclear

Table 2.6 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Groves et al. (2002) USA	40 581 men (271 women excluded)	1950–97	United States Navy personnel with potential for RF exposure; job classified as having low or high exposure by a consensus of Navy personnel involved in training and operation	Trachea, bronchus and lung (162)	<i>Job-associated exposure to RF</i> Low High	 497 400	SMR 0.87 (0.79–0.94) 0.64 (0.58–0.70)	Age at cohort entry and attained age	White United States Navy (male) veterans of Korean War (1950–54) Reference: all white men, USA
Degraeve et al. (2009) Belgium	Military personnel (4417 men) in battalions equipped with radar, and 2932 controls.	1968–2003	Exposure levels on the site where the battalion lived and worked were characterized; individual exposure assessment could not be conducted.	Respiratory and intra-thoracic organs (160–169)	Control cohort Radar exposed	28 45	1.00 1.07 (0.66–1.71)		Cause of death found for 71% of the men in the radar group and 70% in the control group.

* Values calculated/deducted by the Working Group
ELF, extremely low frequency electric and magnetic field; MW, microwaves; OR, odds ratio; PEMFs, pulsed electromagnetic fields; RF, radiofrequency radiation; SES, socioeconomic status; SIR, standardized incidence ratio; SMR, standardized mortality ratio; W, watt; V, volt; wk, week; yr, year

[Hocking et al. \(1996\)](#) studied incidence and mortality attributable to cancer of the brain (ICD-9 code 191) near three television and FM-radio broadcasting antennae located close together in Sydney, Australia. Exposure from these towers was to amplitude modulation (AM) at 100 kW and frequency modulation (FM) at 10 kW for signals at 63–215 MHz. Calculated power densities of RF radiation ranged from 8.0 $\mu\text{W}/\text{cm}^2$ near the tower to 0.2 $\mu\text{W}/\text{cm}^2$ at a distance of 4 km and 0.02 $\mu\text{W}/\text{cm}^2$ at 12 km. For cancer of the brain at all ages in three “inner ring” municipalities relative to six “outer-ring” municipalities, the rate ratio for incidence was 0.89 (95% CI, 0.71–1.11; 740 cases) and the rate ratio for mortality was 0.82 (95% CI, 0.63–1.07; 606 deaths). [Municipality-specific incidence rates were only available in broad, sex-specific age groups: 0–14, 15–69 and ≥ 70 years]. For children aged 0–14 years, the corresponding rate ratios were 1.10 (95% CI, 0.59–2.06; 64 cases) and 0.73 (95% CI, 0.26–2.10; 30 deaths). All municipalities were said to have upper middle-class populations.

Prompted by reported clustering of leukaemia and lymphoma near a high-power television and FM-radio broadcast antenna in the West Midlands, England, [Dolk et al. \(1997a\)](#) studied the incidence of cancer within a radius of 10 km from the antenna. The authors noted that available field-strength measurements generally showed a decrease of the average field strength with increasing distance from the transmitter, although with undulations in predicted field strength up to about 6 km from the transmitter. The maximum total power-density equivalent summed across frequencies at any one measurement point was 0.013 W/m^2 for television, and 0.057 W/m^2 for FM radio. Observed numbers of cases within 0–2 km and 0–10 km of the antenna were compared with “national” incidence rates, adjusted for age, sex, year and deprivation quintile (calculated based on data on unemployment, overcrowding, and social class of head of

household). For all tumours of the brain (ICD-8/ICD-9 codes 191, 192, 225, and ICD-9 codes 237.5, 237.6, 237.9) in persons aged ≥ 15 years, the SIR was 1.29 (95% CI, 0.80–2.06) within 0–2 km and 1.04 (95% CI, 0.94–1.16) within 0–10 km. For malignant tumours of the brain only, these SIRs were 1.31 (95% CI, 0.75–2.29) and 0.98 (95% CI, 0.86–1.11), respectively.

[Dolk et al. \(1997b\)](#) undertook a similar analysis of cancer incidence in proximity to all 20 other high-power radio and television transmitter antennae in the United Kingdom. [With one exception, information about field distribution and strength in proximity to those antennae was not provided.] In this analysis, results for tumours of the brain were reported only for children aged 0–14 years and in proximity to all 21 such antennae (including that studied by [Dolk et al., 1997a](#)). At 0–2 km from the antenna, SIRs were 0.62 (95% CI, 0.17–1.59) for all tumours of the brain and 0.50 (95% CI, 0.10–1.46) for malignant tumours, while at 0–10 km SIRs were 1.06 (95% CI, 0.93–1.20) and 1.03 (95% CI, 0.90–1.18), respectively.

[Ha et al. \(2003\)](#) studied the incidence of cancer between November 1993 and October 1996 in people aged ≥ 10 years in populations of 11 administrative areas of the Republic of Korea within about 2 km of high-power (≥ 100 kW) AM transmitter antennae, 31 such areas within about 2 km of low-power AM transmitter antennae (50 kW), and 4 control areas near, but not within 2 km, of each high-power transmitter antenna (44 control areas in total). Incident cases of cancer were ascertained from medical insurance records [no information was given regarding the completeness and accuracy of these records]. Directly age-standardized incidence rate ratios for cancer of the brain (ICD-9 codes 191–192, and ICD-10 codes C70–C72) comparing people living near high-power transmitter antennae with people living near low-power antennae were 1.8 (0.9–11.1) in males and females combined. Indirectly age-standardized incidence rate

ratios for cancer of the brain comparing people living near high-power transmitter antennae at different levels of power output with those in control areas were 2.27 (95% CI, 1.30–3.67) for a power output of 100 kW, 0.86 (95% CI, 0.41–1.59) for 250 kW, 1.47 (95% CI, 0.84–2.38) for 500 kW, and 2.19 (95% CI, 0.45–6.39) for 1500 kW.

[Park et al. \(2004\)](#) reported the results of a similar study of cancer mortality in 1994–95 in people of all ages in the Republic of Korea. Mortality rates within an area of 2 km surrounding AM broadcasting towers with a power of > 100 kW were compared with those in control areas that had a similar population and were located in the same province as the matched exposed area. Information on deaths due to cancer was identified in Korean death certificates from 1994 to 1995. The resident population at that time was assumed to correspond to that recorded in the nationwide population census of 1990. To control for possible selection bias, four control areas ($n = 40$) were matched to each exposed area ($n = 10$). Based on six age groups, annual age-adjusted world population-standardized mortality rates were calculated per 100 000 residents. Mortality rate ratios (MRR) were calculated comparing 10 areas within about 2 km of high-power antennae with 40 areas situated > 2 km from high-power antennae in the same or neighbouring provinces. The directly standardized MRR for cancer of the CNS, comparing areas near high-power antennae with control areas, was 1.52 (95% CI, 0.61–3.75).

The incidence of cancer in relation to mobile-phone base-station coverage was investigated in 177 428 people living in 48 municipalities in Bavaria, Germany, between 2002 and 2003 ([Meyer et al., 2006](#)). Municipalities were classified on a crude three-level exposure scale based on the operating duration of each base station and the proportion of the population living within 400 m of the base station. Based on 1116 malignant tumours in 242 508 person-years, no indication of an overall increase in the incidence of cancer

was found in the populations of municipalities belonging to the highest exposure class. The Potthoff-Whittinghill test was used to examine the homogeneity of the case distribution among the communities. The following cancers were not found to be heterogeneously distributed: breast ($P = 0.08$); brain and CNS ($P = 0.17$); and thyroid ($P = 0.11$). For leukaemia, there were indications of underreporting and thus the test for homogeneity was not performed. [The exposure assessment in this study was very crude and likely to result in substantial random exposure misclassification. The number of organ-specific tumours was not reported, but is expected to be small given the total number of tumours. Thus, the observed absence of an association may be real, or due exposure misclassification, or to inadequate statistical power.]

(b) Case-control studies

[Schüz et al. \(2006b\)](#) reported on the association of proximity of a DECT (Digital Enhanced Cordless Telecommunications) cordless-phone base station to a person's bed (a proxy for continuous low-level exposure to RF radiation during the night) with the risk of brain glioma and meningioma in a case-control study in Germany that was a component of the INTERPHONE study. This was a subanalysis of the main study in which no association of either brain glioma or meningioma with use of cordless phones had been found ([Schüz et al., 2006a](#)). Cases were newly diagnosed with a histologically confirmed glioma or meningioma in 2000–03, aged 30–69 years, lived in the study region, had a main residence in the study region, and had a knowledge of German sufficient for interview. Proxy interviews were conducted if the cases or controls had died or were too ill for interview. Controls were selected randomly from compulsory population registers in the study regions, were required to meet relevant case-inclusion criteria, and were initially frequency-matched to the cases by age, sex and region. Participation rates were: patients

with glioma, 79.6%; patients with meningioma, 88.4%; and controls, 62.7%. Interview questions about cordless phones addressed the type of phone (DECT or analogue), make and model, the dates on which use started and stopped, and the location of the base station within the residence. Since many subjects could not recall whether their cordless phone was a DECT phone, information on the make, model and price of the phone and its technical features were used to classify phones into “definitely” or “possibly” DECT, or definitely analogue. Participants were considered definitely or possibly exposed if, in addition, the DECT base station was located 3 m or less from the bed (this was the case for 1.6% of participants). Information from proxy interviews (patients with glioma, 10.9%; patients with meningioma, 1.3%; and control participants, 0.4%) was used in the analysis, since most proxies lived with their index subjects and were users of the same cordless phones. For analysis, controls were individually matched 2 : 1 to cases by birth year, sex, region and date of diagnosis (case) or interview (control); 366 cases of glioma and 381 cases of meningioma were analysed. Risk of glioma or meningioma was not increased with definite or possible exposure to DECT base stations; nor was there any consistent trend with time since first exposure ([Table 2.7](#)). [This study was limited by the small proportion of people who were considered to be exposed, difficulty in classifying cordless phones as DECT or analogue, and lack of associated consideration of other sources of exposure to RF radiation.]

[Ha et al. \(2007\)](#) reported on risk of childhood cancers of the brain in relation to residential exposure to RF radiation from AM-radio fixed-site transmitters (power, > 20 kW) in the Republic of Korea. Cases were diagnosed with cancer of the brain (ICD-9 codes 191–192, and ICD-10 codes C70–C72) between 1993 and 1999, and controls were diagnosed over the same period with a respiratory disease (ICD-9 codes 469–519, and ICD-10 codes J20 and J40–J46). Both cases

and controls were identified through the national health insurance system of the Republic of Korea, and individually matched by age, sex and year of first diagnosis. Both were restricted to children diagnosed at one of fourteen large cancer or tertiary-care hospitals. Cancer diagnoses were confirmed by reference to the national cancer registry or hospital medical records [the basis for confirmation was not stated]. Cases were excluded if the diagnosis of cancer could not be confirmed; controls were excluded if they had a history of cancer recorded in the national cancer registry (which was 80% complete in 1998); and both were excluded if they had incomplete addresses (which were obtained from the medical records). The distance from each subject’s residence to the nearest AM-radio transmitter established before diagnosis was evaluated by means of a geographical information system, and total exposure to RF radiation from all AM-radio fixed-site transmitters was estimated with a flat-earth attenuation statistical-prediction model, which took into account features of the receiving point and the propagating pathway [intervening terrain, the output power of the fixed-site transmitters and their distance from the receiving point]. The prediction program was validated by taking measurements of field strength at sites around 11 fixed-site transmitters, and correction coefficients were calculated and applied to the prediction program. Twenty-nine of the thirty-one radio fixed-site transmitters were established between 1980 and 1995, and children in the study were born between 1978 and 1999. Socioeconomic status was classified according to the number of cars owned per 100 people in defined regions and population density in these regions was used as a surrogate for industrialization and environmental pollution. The odds ratio for cancer of the brain was not materially increased in those living closest (≤ 2 km) to a transmitter (OR, 1.42; 95% CI, 0.38–5.28) or in those with greatest estimated exposure (≥ 881 mV/m) to RF radiation (OR, 0.77; 95% CI, 0.54–1.10) ([Table 2.7](#)). [This

Table 2.7 Case-control studies of cancer of the brain and environmental exposure to radiation from transmitters of radiofrequency signals

Reference, study location and period	Total cases	Total controls	Control source (hospital, Population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Comments
Schüz et al. (2006b) Bielefeld, Heidelberg, Mainz, Mannheim, Germany, 2000–03	747	1494	Population	Interviewer-administered questionnaire	Brain (glioma and meningioma)	DECT cordless-phone base station ≤ 3m from bed			Covariates: age, sex, region, educational level
					Glioma				
					Definitely		342	1.00	
					No				
					Yes		3	0.50 (0.14–1.76)	
					Possibly or definitely				
					No		342	1.00	
					Yes		5	0.82 (0.29–2.33)	
					Time since first exposure (possibly or definitely)				
					No, < 1 yr		342	1.00	
					1–4 yr		3	0.95 (0.24–3.70)	
					≥ 5 yr		2	0.68 (0.14–3.40)	
					Meningioma				
					Definitely				
					No		360	1.00	
					Yes		5	1.09 (0.37–3.23)	
					Possibly or definitely				
					No		364	1.00	
					Yes		5	0.83 (0.29–2.36)	
					Time since first exposure (possibly or definitely)				
					No, < 1 yr		364	1.00	
					1–4 yr		1	0.33 (0.04–2.80)	
					≥ 5 yr		4	1.29 (0.37–4.48)	

Table 2.7 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, Population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Comments
Ha et al. (2007) Republic of Korea, 1993–99	956	1020	Hospital-based study. Controls had attended one of 14 large cancer or tertiary care hospitals where the cases had been diagnosed, for management of respiratory disease (ICD-9 469–519; ICD-10 J20 and J40–46). Controls were individually matched to a case by age, sex, and year of first diagnosis.	Based on locations of 31 AM transmitters of ≥ 20 kW and 49 associated antennae and locations of subjects' residences at time of diagnosis.	Brain (ICD-9 code 191–192; ICD-10 codes C70–C72)	Distance from nearest AM radio-transmitter established before subject's year of diagnosis (km) ≤ 2 $> 2-4$ $> 4-6$ $> 6-8$ $> 8-10$ $> 10-20$ > 20 Unknown <i>P</i> (trend)	All brain cancer (age < 15 yr) 10 32 59 90 114 244 400 7 0.76	 1.42 (0.38–5.28) 1.40 (0.77–2.56) 1.02 (0.66–1.57) 1.08 (0.73–1.59) 0.94 (0.67–1.33) 1.01 (0.77–1.34) 1.00 (reference) 4.30 (0.50–36.73) 0.76	Children aged < 15 yr. The use only of large hospitals for ascertainment of controls are not representative of population from which cases were drawn. Covariates: age, sex, residential location, population density, SES
<i>Total exposure to RF (mV/m)</i>									*Quartiles of the distribution
< 532.55*							329	1.00 (reference)	
532.55 – < 622.91							185	0.66 (0.47–0.92)	
622.91 – < 881.07							181	0.72 (0.51–1.01)	
≥ 881.07							254	0.77 (0.54–1.10)	
<i>P</i> (trend)								0.73	

Table 2.7 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, Population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Comments
Haefel et al. (2007) (cont.)	All brain cancer (age < 1 yr)								
	<i>Distance from nearest AM radio-transmitter established before subject's year of diagnosis (km)</i>								
						≤ 10	10	0.41 (0.05–3.10)	
						> 10–20	10	0.49 (0.06–3.80)	
						> 20	12	1.00 (reference)	
						<i>P</i> (trend)		0.78	
						<i>Total exposure to RF (mV/m)</i>			
Oberfeld (2008) Hausmannstätten & Vasoldsberg, Austria, 1984–97						< 485.85*	9	1.00 (ref.)	The status of this study (printed version, German and English online versions) is controversial. It was therefore decided to remove the description of this study from text and tables.
						485.85 – < 632.96	7	3.56 (0.49–25.95)	
						632.96 – < 810.81	7	1.41 (0.12–17.11)	
						≥ 810.81	9	5.13 (0.44–60.26)	
						<i>P</i> (trend)		0.27	

The status of this study (printed version, German and English online versions) is controversial. It was therefore decided to remove the description of this study from text and tables.

Table 2.7 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, Population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Comments
Spinelli et al. (2010) Western Provence-Alpes-Côte d'Azur (PACA), France, 2005	122	122	Hospital-based	Self-administered questionnaire and face-to-face interview, including lifetime job history, job title, dates, tasks	Brain (glioma grades II–IV)	Residence within 500 m of: Cell-phone tower	19	0.49 (0.26–0.92)	Covariates: age, sex
Elliott et al. (2010) United Kingdom, 1999–2001	251	1004	Population-based study. Controls were children aged < 4 yr, individually matched to cases by sex and date of birth.	Location of birth residence relative to nearby macro-cell mobile-phone base stations; distance to nearest base station; total power output of all base-stations within 700 m; modelled power density at birth address	Brain and CNS (ICD-10 codes C71–C72)	Distance from nearest macro-cell mobile-phone base-station Lowest third Intermediate third Highest third For 15th to 85th centile increase (continuous measure) Total mobile-phone frequency power-output (kW) No base station within 700 m Lower half Upper half For 15th to 85th centile increase (continuous measure)	85 85 81 251	1.00 0.95 (0.67–1.34) 0.95 (0.65–1.38) 1.12 (0.91–1.39)	Covariates: percentage of population with education to degree level or higher, Carstairs score (a composite area-deprivation measure), population density, and population mixing (percentage immigration into the area over the previous year).

Table 2.7 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, Population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Comments
Elliott <i>et al.</i> (2010) (contd.)						<i>Modelled mobile-phone frequency power density (dBm)</i>			
						Lowest third	93	1.00	
						Intermediate third	80	0.97 (0.69–1.37)	
						Highest third	78	0.76 (0.51–1.12)	
						For 15th to 85th centile increase (continuous measure)	251	0.82 (0.55–1.22)	

AM, amplitude modulation (radio); CNS, central nervous system; DECT, Digital Enhanced Cordless Telecommunications; kW, kilowatt; NMT, Nordic Mobile Telephone (standard); RF, radiofrequency radiation; SES, socioeconomic status; Yr, year

study was limited by the lack of a clear population base, possible mismatch between the population sampled for cases and that sampled for controls, and the lack of a cumulative measure of exposure to RF radiation that took into account variation in an individual's place of residence between birth and diagnosis of cancer or respiratory disease.]

[[Oberfeld \(2008\)](#): the status of this study (printed version, German and English online versions) is controversial. It was therefore decided to remove the description of this study from text and tables.]

[Spinelli et al. \(2010\)](#) undertook a pilot case-control study of newly diagnosed, histopathologically confirmed malignant primary tumours of the brain (defined as previously untreated glioma, grades II–IV) in people aged ≥ 18 years treated in the two principal referral hospitals for cancer of the brain in the west of the Provence-Alpes-Côte d'Azur (PACA) region in France. Controls were other patients in the neurosurgery department who were hospitalized for reasons other than cancer (mainly herniated intervertebral disc, intracranial aneurysm, trauma, and epidural haematoma) who were individually matched to cases by age, sex and residence in the west of PACA. Participants completed a self-administered questionnaire and were interviewed by an occupational physician at the hospital they attended within 3 months after surgery; the physician also checked their questionnaire. [It was not stated whether the interviewer was blind to the case or control status of participants.] Family members also helped with self-administered questionnaires, more often for cases than controls. Proxy interviews were completed for 2% of cases. Occupational exposures were the principal focus of the questionnaire and interview, but participants were also asked about use of mobile phones and residence in proximity to a mobile-phone tower. Information was obtained from 75.3% of cases [the participation rate of controls was not stated]. Nineteen cases and thirty-three

controls reported a mobile-phone tower within 500 m of their residence (age- and sex-adjusted OR, 0.49; 95% CI, 0.26–0.92) ([Table 2.7](#)). [This study was limited by its small size and because it was hospital-based. The participation rate for controls was not stated and it is likely that people prone to serious injury were over-represented among the controls. The interviewer may not have been blind to the case or control status of participants. Specific questions regarding proximity of residence to mobile-phone towers were not described and may have been highly prone to recall error, and there were few participants with occupational exposure to RF radiation.]

[Elliott et al. \(2010\)](#) undertook a case-control study of early childhood cancer in the United Kingdom based on all cases of cancer in children aged 0–4 years registered in 1999–2001. Of 1926 registered cases, the geographical coordinates of addresses at birth, and exposure based on the birth address were available for 1397 children (73%). Of the latter, 251 had cancers of the brain and CNS (ICD-10 codes C71–C72). For each case, four controls from the national birth register, with complete birth addresses and individually matched to cases by sex and date of birth (5588 controls), were obtained from 6222 originally randomly selected (90%). The four national mobile-phone operators provided detailed data on all 76 890 macro-cell base stations operating in 1996–2001. Three exposure measures for the birth address of each case and control were obtained: the distance from the nearest macro-cell mobile-phone station; the total power output (kW) from summation across all base stations within 700 m; and computed modelled power density (dBm) at each birth address for base stations within 1400 m. Exposures beyond 1400 m were considered to be at background levels. Measurements from field campaigns in a rural and an urban area were used to set parameter values in the power-density model. The models were validated with data from two further surveys and power-density measurements from 620 locations

across the country. Spearman's correlation coefficients between measured power density and the exposure measures were: 0.66 with modelled power density, 0.72 with distance from nearest base station, and 0.66 with total power output. The exposure measures estimated at each birth address were averaged across monthly estimates for the assumed 9 months of the pregnancy in each case. Each exposure measure was divided into thirds of the distribution across all cases and controls except for total power output, which was zero for 58% (no base station within 700 m), with the remaining 42% in two halves of their distribution. Exposure measures were fitted to models as continuous variables as well as in the above categories. Neither unadjusted nor partly or fully adjusted odds ratios suggested that risk of childhood cancer of the brain increased with increasing exposure to RF radiation from nearby macro-cell mobile-phone base stations ([Table 2.7](#)). [This study was limited by the fact that estimation of exposure was confined to the gestational period; application of birth address to the whole of gestation was assumed; and ecologically measured possible confounding variables were used to apply to individual subjects.]

(c) Cohort studies

No data were available to the Working Group.

2.2.2 Leukaemia and lymphoma

(a) Ecological studies

See [Table 2.8](#)

[Hocking et al. \(1996\)](#) published a study comparing incidence of and mortality from leukaemia during 1972–90 in nine municipalities, three of which were located around television towers and six that were more distant. Increased rate ratios for incidence (IRR, 1.24; 95% CI, 1.09–1.40) and mortality (MRR, 1.17; 95% CI, 0.96–1.43) for leukaemia at all ages were obtained and generally higher rate ratios were seen for childhood leukaemia (IRR, 1.58; 95%

CI, 1.07–2.34; MRR, 2.32; 95% CI, 1.35–4.01) than for leukaemia at all ages, comparing the three “inner ring” municipalities with six “outer ring” municipalities. A more marked association was observed between proximity to television towers and mortality (MRR, 2.4; 95% CI, 1.4–3.7) than incidence (IRR, 1.8; 95% CI, 1.2–2.5) from leukaemia. [No individual measurements were undertaken and main analyses could only be adjusted for covariates by group-level (aggregated) data.]

In 1997, [Dolk et al.](#) published two studies on cancer incidence during 1974–86. The first was a study in a small area in response to an unconfirmed report of a cluster of leukaemias and lymphomas near the Sutton Coldfield television and radio-transmitter in the West Midlands, England ([Dolk et al., 1997a](#)). The second, to place in context the findings of the Sutton Coldfield study, was carried out near 20 high-power television and radio-transmitters in the United Kingdom ([Dolk et al., 1997b](#)). In the Sutton Coldfield study, an increased risk of leukaemia in adults was found when the observed and expected numbers of cases (derived from national incidence rates) were compared (observed/expected, 1.83; 95% CI, 1.22–2.74) within 2 km of the transmitter and there was a decline in risk with distance (Stone's P value = 0.001). The latter was tested by use of 10 bands of increasing distance from the transmitter within a circle with a radius of 10 km around it. The findings appeared to be consistent between 1974 and 1980, and 1981 and 1986. For NHL, a suggestion of a decrease in risk was seen within the 2 km area (observed/expected, 0.66; 95% CI, 0.28–1.30) while for the total study area of 0–10 km, risk appeared to be increased (observed/expected, 1.23; 95% CI, 1.11–1.36). In the second study, covering the United Kingdom ([Dolk et al., 1997b](#)), evidence of a decline in risk of leukaemia was found with increasing distance from the transmitter (Stone's P value = 0.05); however, the magnitude (at 0–10 km: observed/expected, 1.03; 95% CI, 1.00–1.07) and the pattern

Table 2.8 Ecological studies of leukaemia and lymphoma and environmental exposure to radiation from transmitters of radiofrequency signals

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Hocking <i>et al.</i> (1996) Australia	585 000	1972–90	Residential proximity to TV towers	Leukaemia (204–208)	Overall rate ratios for incidence and mortality, respectively, inner and outer residential area compared. Inner area <i>vs</i> ref. population Outer area <i>vs</i> ref. population Inner area <i>vs</i> ref. population Outer area <i>vs</i> ref. population	1206/847	Incidence, 1.24 (1.09–1.40) Mortality, 1.17 (0.96–1.43)	Age, sex, calendar period	Reference population: whole of New South Wales
Dolk <i>et al.</i> (1997a) United Kingdom	Around 408 000	1974–86	Distance to Sutton Coldfield radio and TV transmitter	Haemato-poietic and lymphatic cancers (200–202; 203 + 238.6; 204–208)	Distance 0–2 km Distance 0–10 km Stone's <i>P</i> -value	45 935	1.21 (0.91–1.62) 1.04 (0.98–1.11) Unconditional <i>P</i> = 0.153	Region	Observed/expected ratios and Stone's <i>P</i> -value are given for persons aged ≥ 15 yr, stratified by age, sex, year, and SES. Declining risk with increasing distance was seen only for all leukaemias.
				Leukaemia (204–208)	Distance 0–2 km Distance 0–10 km Stone's <i>P</i> -value	23 304	1.83 (1.22–2.74) 1.01 (0.90–1.13) Unconditional <i>P</i> = 0.001 Conditional <i>P</i> = 0.001		
				NHL	Distance 0–2 km Distance 0–10 km Stone's <i>P</i> value	8 357	0.66 (0.28–1.30) 1.23 (1.11–1.36) Unconditional <i>P</i> = 0.005 Conditional <i>P</i> = 0.958		

Table 2.8 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Dolk <i>et al.</i> (1997b) United Kingdom	Around 3 390 000	1974–86	Distance to radio and television transmitters in the United Kingdom (excluding Sutton Coldfield)	Leukaemia (204–208)	Distance 0–2 km Distance 0–10 km Stone's <i>P</i> value	79 3 305	0.97 (0.78–1.21) 1.03 (1.00–1.07) Unconditional <i>P</i> = 0.001 Conditional <i>P</i> = 0.052	Region	Observed/expected ratios and Stone's <i>P</i> value are given for persons aged ≥ 15 yr, stratified by age, sex, year, and SES.
Cooper <i>et al.</i> (2001) United Kingdom	NR	1987–94	Distance to Sutton Coldfield television transmitter	Leukaemia (204–208)	Distance from transmitter: 0–2 km, all ages, all persons 0–10 km, all ages, all persons Stone's <i>P</i> values, all ages, all persons 0–2 km, age 0–14 yr, all persons 0–10 km, age 0–14 yr, all persons Stone's <i>P</i> values, age 0–14 yr, all persons	20 333 1 26	1.32 (0.81–2.05) 1.16 (1.04–1.29) Unconditional <i>P</i> = 0.038 Conditional <i>P</i> = 0.409 1.13 (0.03–6.27) 1.08 (0.71–1.59) Unconditional <i>P</i> = 0.420	-	Observed/expected ratios and Stone's <i>P</i> values (unconditional and conditional) are given. Stratified by age, sex, and social deprivation. Results for other haematopoietic cancers are reported in the manuscript.

Table 2.8 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Michelozzi et al. (2002) Italy	49 656	1987–99	Distance to Vatican radio station, Rome	Leukaemia (204–208)	Distance from radio station (km), (cumulative areas)			Deprivation index	
					Total (age > 14 yr)		SMR		
					0–2	2	1.8 (0.3–5.5)		
					0–4	11	1.5 (0.8–2.6)		
					0–6	23	1.2 (0.8–1.8)		
					0–8	34	1.2 (0.8–1.6)		
					0–10	40	1.1 (0.8–1.4)		
					P, Stone's conditional test		0.14		
					Children (age 0–14 yr)		SIR		
					0–2	1	6.1 (0.4–27.5)		
					0–4	3	2.9 (0.7–7.6)		
					0–6	8	2.2 (1.0–4.1)		
					0–8	8	1.5 (0.7–2.7)		
					0–10	8	1.2 (0.6–2.3)		
					P, Stone's conditional test		0.036		

Table 2.8 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Ha <i>et al.</i> (2003) Republic of Korea	From 3 152 to 126 523 persons per area (total not given)	1993–96	Distance ≤ 2 km from AM transmitter	Leukaemia (204–208)	High-power (≥ 100 kW) vs low-power (50 kW) transmitter sites:		Rate ratio	Age	Rate ratios (RR) and observed/expected (O/E) ratios are given For all cancers combined: RR, 1.2 (95% CI, 1.1–1.4) for high- vs low-power transmitters
					Men	8.3/6.8 per 100 000 person-yr	1.2 (0.5–5.3)		
					Women	8.7/4.6 per 100 000 person-yr	1.9 (0.8–8.7)		
					Total	8.5/5.7 per 100 000 person-yr	1.5 (0.7–6.6)		
					Transmitter power of sites:		O/E		
					100 kW	9	1.20 (0.55–2.28)		
					250 kW	12	2.45 (1.27–4.29)		
					500 kW	10	0.65 (0.31–1.19)		
					1500 kW	4	4.26 (1.16–10.89)		

Table 2.8 (continued)

Reference, study location	Total No. of subjects	Follow- up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	Relative risk (95% CI)	Covariates	Comments
Ha et al. (2003) (contd.)				Malignant lymphoma (200–202)	<i>High-power</i> (≥ 100 kW) vs <i>low-power</i> (50 kW) transmitter sites:		<i>Rate ratio</i>		
					Men	10.5/7.1 per 100 000 person- year	1.5 (0.7–8.6)		
					Women	8.7/7.1 per 100 000 person- year	1.2 (0.6–5.6)		
					Total	9.6/7.1 per 100 000 person- year	1.4 (0.6–7.0)		
					<i>Transmitting power of sites:</i>		<i>O/E</i>		
					100 kW	9	1.10 (0.51–2.10)		
					250 kW	13	1.28 (0.68–2.19)		
					500 kW	16	0.98 (0.56–1.59)		
					1 500 kW	1	0.44 (0.01–2.48)		

Table 2.8 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Park <i>et al.</i> (2004) Republic of Korea	8 115 906 (of whom 1 234 123 in exposed area)	1994–95	Regions including AM-radio broadcasting towers of > 100 kW	Leukaemia (ICD-10 codes C90–95), including multiple myeloma	Total exposed	55	1.70 (0.84–3.45)	Age	Direct standardized MRRs are given
					<i>vs</i> control (unexposed)				
					Males	33	1.89 (0.75–4.75)		
					Females	22	1.55 (0.52–4.68)		
					<i>Age (yr)</i>				
					0–14	11	2.29 (1.05–5.98)		
					15–29	11	2.44 (1.07–5.24)		
					30–44	9	2.16 (0.95–4.04)		
					45–59	5	0.73 (0.48–2.89)		
					60–74	10	0.87 (0.57–2.78)		
					≥ 75	6	3.08 (0.95–6.59)		
					Total exposed	31	1.60 (0.72–3.56)		
					<i>vs</i> control (unexposed)				
					Males	19	1.52 (0.56–4.14)		
					Females	12	1.80 (0.48–6.71)		
					<i>Age (yr)</i>				
					0–14	1	2.46 (0.07–82.66)		
					15–29	2	1.51 (0.15–15.18)		
					30–44	5	1.94 (0.37–10.20)		
					45–59	8	1.76 (0.43–7.15)		
					60–74	13	1.41 (0.47–4.14)		
					≥ 75	2	0.55 (0.05–5.67)		

AM, amplitude modulation (radio); kW, kilowatt; MRR, mortality rate ratio; NR, not reported; O/E, observed/expected; SIR, standardized incidence ratio; SMR, standardized mortality ratio; TV, television; *vs*, versus; yr, year

of risk seen in the Sutton Coldfield study could not be replicated. Most notably, in the second, nationwide study no increase in risk was seen nearest (within 2 km) the transmitters.

In a letter to the editor, [Cooper et al. \(2001\)](#) published updated results on adult and childhood leukaemia (1987–94) near the Sutton Coldfield transmitter. To investigate risk according to distance, the authors defined the study area as a series of 10 concentric circles around the Sutton mast and calculated the expected number of cases (by numbers, child/adult and sex) for each of the circles and for different cancer sites. Most results for childhood cancers gave no evidence of a decline in the ratios of observed-to-expected numbers of cases with distance from the transmitter. There was some support for a decline in risk of childhood leukaemia in males, as indicated by Stone's test. The risk also declined for acute myeloid leukaemia in adult females, for all leukaemias (females and all persons separately), and for haematopoietic and lymphatic cancers in females. The same four groups were at higher risks over the whole study area (0–10 km). An increased risk was found for acute lymphatic leukaemia within 2 km of the transmitter; however this was based on only two cases. Elevated risks were found for leukaemias and NHL (males and females combined and separately) over the whole study area. No increase or decrease in the ratios of observed-to-expected numbers of cases was seen for NHL.

[Michelozzi et al. \(2002\)](#) published a study on incidence and mortality for adult and childhood leukaemia in an area of 10 km around a high-power radio station in Rome. This station had numerous transmitters with different transmission powers (5–600 kW) operating at different frequencies (short–medium wave). An increased incidence of childhood leukaemia (SIR, 2.2; 95% CI, 1.0–4.1) was found up to 6 km from the radio station; there was a decline with increasing distance from the station for mortality in males and for incidence from childhood leukaemia.

[The small number of cases, possible unmeasured confounding and lack of individual or calculated exposure assessment were some limitations of the study.]

[Ha et al. \(2003\)](#) published a study on the incidence of cancer in the Republic of Korea in 1993–96 in areas proximate to 42 AM-radio-transmitters, characterized by transmission power. An increased rate ratio comparing sites exposed to high-power versus low-power transmitters was seen for all cancers combined (rate ratio, 1.2; 95% CI, 1.1–1.4), while confidence intervals by cancer type were wide, e.g. for leukaemia (rate ratio, 1.5; 95% CI, 0.7–6.6) and malignant lymphoma (rate ratio, 1.4; 95% CI, 0.6–7.0). However, at two of eleven high-power sites, more pronounced increases in the incidence of leukaemia were found. [Interpretation was hampered by limitations related to the ecological design, study size, exposure and outcome assessment, and lack of controls for confounding. There was partial overlap in the populations included in [Park et al. \(2004\)](#) and [Ha et al. \(2007\)](#).

[Park et al. \(2004\)](#) published a study that evaluated cancer mortality in the Republic of Korea in relation to exposure to AM-radio-transmitters. Mortality from leukaemia was higher in exposed areas than in control areas (standardized mortality rate ratio, MRR, 1.70; 95% CI, 0.84–3.45), particularly among young adults (MRR, 2.44; 95% CI, 1.07–5.24), but also in children (MMR, 2.29; 95% CI, 1.05–5.98). According to the authors, however, there was no increasing or decreasing trend with respect to broadcasting power. [In this study, exposure assessment was poor (no individual data) and it was also unclear to what extent the mortality records reflected the true address of the subject, which was used as a proxy for exposure. Other limitations were the lack of control for confounding by socioeconomic status, and possible non-differential disease misclassification.]

(b) *Case-control studies*

See [Table 2.9](#)

[Maskarinec et al. \(1994\)](#) published the results of a small case-control study that indicated an increased incidence in childhood leukaemia (SIR, 2.09; 95% CI, 1.08–3.65) near radio towers in Hawaii, USA. The SIR for acute lymphocytic leukaemia was 1.58 (95% CI, 0.63–3.26) and for acute non-lymphocytic leukaemia it was 3.75 (95% CI, 1.20–8.71). Seven cases of leukaemia had been reported during 1982–84, including all five cases of acute non-lymphocytic leukaemia (SIR, 5.34; 95% CI, 2.14–11.0) that were unusual with respect to sex, age, and type of leukaemia. Twelve cases in children aged < 15 years diagnosed with acute leukaemia in 1979–90 and residing in certain census tracts before diagnosis were included in the case-control study, along with 48 (80%) sex- and age-matched controls that lived in the same area at the time of diagnosis. Collection of data was by non-blinded telephone interviews with parents, which included questions on pregnancy, address, and residence history, the child's medical history and exposure of various kinds, including X-rays and smoking. In addition, the occupational history of both parents was recorded, together with potentially relevant exposures. The odds ratio for acute leukaemia among those having lived within 2.6 miles (4.2 km) of the radio towers before diagnosis was increased (OR, 2.0; 95% CI, 0.6–8.3). [The limitations of this study, besides poor assessment of exposure, were its low power to detect an effect (~50% for OR = 5) and the apparent lack of controls for confounding by socioeconomic status.]

[Ha et al. \(2007, 2008\)](#) published the results of a case-control study that was large enough to give moderate statistical power for detecting an effect of exposure to RF radiation on the risk of childhood leukaemia. Patients aged < 15 years with leukaemia and controls with respiratory illnesses were selected from 14 hospitals in the

Republic of Korea and matched on age, sex and year of diagnosis (1993–99). From a total of 1928 cases of leukaemia and matched controls, risks were estimated by means of conditional logistic regression analysis adjusted for residential area, socioeconomic status and community population density. An increased risk of all types of leukaemia was found among children who lived within 2 km of the nearest AM-radio-transmitter (OR, 2.15; 95% CI, 1.00–4.67). For total exposure to RF radiation, most odds ratios decreased with predicted exposure. The authors reported an odds ratio of 1.40 (95% CI, 1.04–1.88) for lymphocytic leukaemia and 0.63 (95% CI, 0.41–0.97) for myelocytic leukaemia in the quartile of highest peak exposure, although no linear trend was evident with regard to the different exposure categories for total or peak exposure to RF radiation. [The main limitations of the study were related to the exposure estimates calculated by the prediction programme, e.g. the existence of buildings or irregular geographical features was not considered, nor was individual cumulative-exposure history assessed. There was partial overlap in the populations included in [Ha et al. \(2003\)](#) and [Park et al. \(2004\)](#).]

A case-control study on RF radiation and childhood leukaemia was conducted in west Germany by [Merzenich et al. \(2008\)](#). Cases (age, 0–14 years) diagnosed during 1984–2003 and registered at the German Childhood Cancer Registry were included, along with three age-, sex- and transmitter-area-matched controls per case that were drawn randomly from population registries. The analysis included 1959 cases and 5848 controls for which individual exposure to RF radiation 1 year before diagnosis was estimated by means of a field-strength prediction program. The study area encompassed municipalities in the vicinity of Germany's strongest transmitters, including 16 AM and 8 FM transmitters with a power of at least 20 kW. Conditional logistic regression analysis for all types of childhood leukaemia yielded no increase in odds ratio (OR,

Table 2.9 Case-control studies of leukaemia and lymphoma and environmental exposure to radiation from transmitters of radiofrequency signals

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comment
Maskarinec et al. (1994) Hawaii, USA, 1984–2003	12	48	Hospital	Having lived within 2.6 miles of low-frequency radio towers. Distances estimated both manually and by use of a geographical software package	Acute leukaemia (7 ALL, 5 ANLL)	Last residence before diagnosis within 2.6 miles [4.2 km] of radio towers. Residence at birth within 2.6 miles of radio towers	8	2.0 (0.6–8.3)		Matched ORs are given; matching variables: age, sex
Ha et al. (2007) Republic of Korea, 1993–99	1928	3082	Hospital	Prediction program incorporating a geographic information system	All leukaemia (204–208)	Distance (km) > 20 ≤ 2 > 2–4 > 4–6 > 6–8 > 8–10 > 10–20 Unknown P for trend	772 36 73 120 218 276 428 5	1.00 2.15 (1.00–4.67) 0.66 (0.44–0.99) 1.07 (0.77–1.49) 1.26 (0.96–1.65) 1.10 (0.85–1.41) 0.80 (0.65–0.99) 0.48 (0.12–1.95) 0.10	Residential location, population density, SES	Conditional logistic regression; matching variables: age, sex, year of diagnosis. Cases aged < 15 yr. Corrected estimates for total RF exposure according to Ha et al. (2008)

Table 2.9 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comment
Ha et al. (2007) (contd.)						Total exposure to RF (mV/m), quartiles (Q)				In category Q4: OR for LL was 1.40 (95% CI, 1.04–1.88), and for ML, 0.63 (95% CI, 0.41–0.97)
						Q1	737	1.00		
						Q2	362	0.75 (0.58–0.97)		
						Q3	330	0.70 (0.55–0.90)		
						Q4	494	0.83 (0.63–1.08)		
						Unknown	5	0.39 (0.10–1.54)		
						P for trend		0.44		
Merzenich et al. (2008) Germany, 1984–2003	1959	5848	Population	Field-strength prediction programme	ICCC Ia, ICCc Ib, ICCc Ic, ICCc Id, ICCc Ie	Quantiles of median exposure (V/m) to RF-EMFs (AM and FM/TV transmitter) one yr before diagnosis of case				Conditional logistic regression; matching variables age, sex, year of diagnosis, study region. Cases were aged 0–14 yr.
						0 to < 90%	1772	1.00		
						90 to < 95%	101	1.02 (0.80–1.31)		
						95 to ≤ 100%	86	0.86 (0.67–1.11)		
						Distance (km), AM or FM/TV transmitter				
						10 to < 15	551	1.00		
						0 to < 2	25	1.04 (0.65–1.67)		
						2 to < 6	172	0.81 (0.66–0.99)		
						6 to < 10	314	0.79 (0.67–0.93)		
						≥ 15	866	1.00 (0.88–1.14)		

Table 2.9 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comment
Elliott et al. (2010) United Kingdom, 1999–2001	527	5588	Population	(a) Distance from nearest mobile phone base station; (b) Total power output from summation across all base stations within 700 m; (c) Modelled power density at each birth address for base stations within 1400 m	Leukaemia and NHL (C91–95, C82–85)	<i>Distance from nearest base station (m)</i> Lowest Intermediate Highest <i>P</i> for trend <i>Total power output (kW)</i> Lowest Intermediate Highest <i>P</i> for trend <i>Modelled power density (dBm)</i> Lowest Intermediate Highest <i>P</i> for trend	182 167 178 305 112 110	1.00 0.99 (0.78–1.27) 1.05 (0.81–1.35) 0.75 1.00 1.08 (0.84–1.38) 1.08 (0.80–1.42) 0.58	Percentage of population with education to degree level or higher, Carstairs deprivation score, population density, population mixing	Conditional logistic regression; matching variables: age, sex. Cases aged 0–4 yr

ALL, acute lymphocytic leukaemia; AM, amplitude modulation; ANLL, acute non-lymphocytic leukaemia; dBm, modelled power density; FM, frequency modulation; ICC, International Classification of Childhood Cancer; kW, kilowatt; LL, lymphocytic leukaemia; ML, myelocytic leukaemia; OR, odds ratio; NHL, non-Hodgkin lymphoma; RF, radiofrequency radiation; SES, socioeconomic status; TV, television

0.86; 95% CI, 0.67–1.11) when the upper and lower quantiles of RF-radiation distribution were compared. In addition, there was no evidence for an association indicating increased or decreased risk by transmitter type or leukaemia subtype. Nor was there any increased risk (OR, 1.04; 95% CI, 0.65–1.67) for children residing within 2 km of the nearest transmitter. [Lack of information on peak and indoor exposure to RF radiation as well as cumulative lifetime exposure to RF radiation from transmitters, and the low number of cases residing within 2 km of the nearest AM transmitters were the main limitations of this study.]

A case–control study by [Elliott *et al.* \(2010\)](#) (described in Section 2.2.1) examined risk of childhood cancers (e.g. leukaemia and NHL) in association with maternal exposure to RF radiation from mobile-phone base stations during pregnancy. No association or trend for different exposure categories was found for leukaemia or NHL with any of the exposure metrics used. Sociodemographic measures as well as mean distance of birth address from nearest FM, television, and very high frequency (VHF) broadcast antennae were similar for cases and controls. [Although this study had strengths in its size, national coverage and sophisticated exposure assessment compared with previous studies, it was carried out during years when mobile-phone use had become fairly common, yet such usage was not accounted for.]

(c) Cohort studies

No data were available to the Working Group.

2.2.3 Other cancers

There have been several small ecological studies, generally of low quality, that have assessed the correlation between all cancers and distance from mobile-phone base stations ([Eger *et al.*, 2004](#); [Wolf & Wolf, 2004](#); [Gavin & Catney, 2006](#); [Eger & Neppe, 2009](#)). However, the

Working Group considered these studies to be uninformative for the reasons listed below.

Three ecological studies considered risk of all cancers in relation to sources of exposure to RF. [Wolf & Wolf \(2004\)](#) studied the incidence of all cancers around one base station located south of Netanya, Israel, which began operating in July 1996. Among the population of 622 people living within 350 m from the antenna, eight cases were identified between July 1997 and June 1998, and the rate of all cancers among these people was compared to the national rates of cancer in Israel (ratio of rates, 4.15; no confidence intervals provided). [The Working Group considered this study to be uninformative for various reasons, including its small size, unclear method of case ascertainment, crude analyses including incidence rate computed without age standardization, and other methodological limitations.]

Prompted by a reported clustering of cancer cases around a communication mast in Cranlome, Northern Ireland, an ecological study of cancer risk was carried out during 2001–02 ([Gavin & Catney, 2006](#)). The mast was erected in 1989, and was taken down in 2002. The Northern Ireland Cancer Registry was the source of case ascertainment. The rates of incidence of groups of cancer in several concentric geographical areas (up to 5 km) were compared with national rates of cancer incidence. The SIR for all cancers was 0.94 (95% CI, 0.88–0.99) for men and 1.00 (95% CI, 0.94–1.06) for women, while the SIR was 101 (95% CI, 79–104) for brain and 99 (95% CI, 74–124) for lymphoma and leukaemia. [The Working Group considered this study to be uninformative due to its small size, the fact that the number of cases was not reported and the absence of evaluation of exposure to RF radiation.]

[Eger *et al.* \(2004\)](#) studied the incidence of all cancers between 1994 and 2003 in areas determined by circles of radius 400 m around two mobile-phone base stations located in Naila, Germany. The first base station became operational in 1993 and the second in 1997. Streets

within and without the area were randomly selected, and the patient databases of general practitioners were searched for cases living the entire period of 10 years at the same address. [The completeness of the ascertainment appeared to be 90%.] The proportion of new cases of cancer was significantly higher among those patients who had lived for the past 10 years at a distance of up to 400 m from the cellular transmitter site, compared with patients living further away. [The Working Group considered this study uninformative due to the small and ill-defined study base and crude statistical methodology.] The same authors investigated the incidence of cancer around a mobile-phone base station in Westphalia, Germany, between 2000 and 2007 ([Eger & Neppe, 2009](#)). Twenty-three cases were identified by door-to-door interviews. The authors compared the incidence of all cancers in the 5 years immediately after installation of the mast to that in later years, and found a statistically significant increase in incidence 5 years after the base station started transmission. [The Working Group considered this study to be uninformative due to its small size and crude statistical methodology.]

Five additional studies ([Dolk et al., 1997a, b](#); [Ha et al., 2003](#); [Park et al., 2004](#); [Meyer et al., 2006](#)) described information on additional cancer sites ([Table 2.10](#), and see Section 2.2.1). [The interpretation of these results was limited by the small numbers and crude exposure classification.]

2.3 Exposure from mobile phones

With continuing changes in technology, use of mobile phones has become widespread over the last two decades. As a result, the population exposed to RF radiation has greatly increased and is still expanding, with more and more children among its number. Over these two decades, there has been rising concern regarding the potential health risks associated with use of mobile phones, particularly the possibility of increased

risk of cancer of the brain. These concerns have stimulated a diverse programme of research, including epidemiological studies carried out to assess the association of mobile-phone use with risk of cancer of the brain and other diseases. The strength of epidemiological studies is obviously the capacity to directly assess the risks associated with use of mobile phones in the general population; however, the observations collected in these studies clearly only address the various exposure scenarios that existed up to the time of observation. Thus the studies carried out to date include few participants who have used mobile phones for > 10–15 years. Any risks that might be associated with lengthier exposure or with a longer interval since first exposure would not be captured by existing studies.

Three types of study design have been applied to address the question whether an increased risk of cancer is associated with RF emitted by mobile phones. These are ecological studies (in particular, observations of time trends in disease rates), case-control studies, and cohort studies. The strengths and limitations of each of these designs in general have been well described. Here, the Working Group focused on the characteristics of these designs as applied to the investigation of the potential risks of mobile-phone use.

Ecological studies provide only indirect evidence on the potential risks associated with mobile-phone use. The general approach involves comparison of time trends in mobile-phone use with time trends in disease indicators, assessing whether the trends are parallel, and allowing for a potential lag in relationships. Over the last few decades, several factors have affected trends in incidence and mortality for cancer of the brain, in particular, the increasing availability of sensitive imaging technology (computed tomography, CT, and magnetic resonance imaging, MRI) for detecting cancers of the brain, which is likely to have had a variable influence on changes in diagnostic practices, depending on country. Consequently, the interpretation of time trends is

Table 2.10 Ecological studies of other cancers and environmental exposure to radiation from transmitters of radiofrequency signals

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95%CI)	Covariates	Comments
Dolk <i>et al.</i> (1997a) United Kingdom	Around 408 000	1974–86	Distance to Sutton Coldfield radio and TV transmitter	All cancers	Distance 0–2 km	703	1.09 (1.01–1.17)	Region	
					km	17 409	1.03 (1.02–1.05)		
					0–10 km		Unconditional		
					Stone's <i>P</i> value		<i>P</i> = 0.001		
							Conditional		
							<i>P</i> = 0.462		
				Skin melanoma	0–2 km	13	1.43 (0.83–2.44)		
				Eye melanoma		0	0 (0–4.22)		
				Male breast		1	1.64 (0.04–9.13)		
				Female breast		107	1.08 (0.90–1.31)		
				Lung		113	1.01 (0.84–1.21)		
				Colorectal		112	1.13 (0.94–1.35)		
				Stomach		33	0.75 (0.54–1.06)		
				Prostate		37	1.13 (0.82–1.55)		
				Bladder		43	1.52 (1.13–2.04)		
				Skin melanoma	0–10 km	189	0.96 (0.83–1.11)	Region	
				Eye melanoma		20	1.16 (0.75–1.80)		
Dolk <i>et al.</i> (1997b) United Kingdom	Around 3 390 000	1974–86	Distance to radio and TV transmitters in United Kingdom (excluding Sutton Coldfield)	Male breast		15	0.99 (0.60–1.64)		
				Female breast		2412	1.05 (1.01–1.10)		
				Lung		3466	1.01 (0.98–1.05)		
				Colorectal		2529	1.03 (0.99–1.07)		
				Stomach		1326	1.06 (1.01–1.12)		
				Prostate		785	1.03 (0.96–1.11)		
				Bladder		788	1.08 (1.01–1.16)		
				Skin melanoma	0–2 km	51	1.11 (0.84–1.46)	Region	
				Bladder		209	1.08 (0.94–1.24)		
				Skin melanoma	0–10 km	1540	0.90 (0.85–0.94)		
				Bladder		8307	1.09 (1.06–1.11)		

Table 2.10 (continued)

Reference, study location	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95%CI)	Covariates	Comments
Ha et al. (2003) Republic of Korea	From 3 152 to 126 523 persons per area (total not given)	1993–96	kW	Breast cancer	High-power (≥ 100 kW) vs low-power (50 kW) transmitter sites	39.7/33.6 per 100 000 person-years	1.2 (0.8–1.7)	Age	
			Transmitter power (kW)	Breast cancer	Sites with transmitter power:				
					100 kW	29	1.29 (0.86–1.86)		
					250 kW	20	0.88 (0.54–1.36)		
					500 kW	41	0.90 (0.64–1.23)		
					1500 kW	3	2.19 (0.45–6.39)		
Park et al. (2004) Republic of Korea	8 115 906	1993–95	Regions including AM-radio broadcasting towers of > 100 kW	All cancer	Total exposed vs control (unexposed)	6191	1.29 (1.12–1.49)	Age	Direct standardized MRRs are given.
				Oral cavity and pharynx		14	1.21 (0.41–3.57)		
				Oesophagus		49	1.20 (0.71–2.03)		
				Stomach		403	1.18 (0.96–1.44)		
				Colorectum including anus		78	1.33 (0.83–2.11)		
				Liver, including intrahepatic duct		271	1.01 (0.80–1.27)		
				Pancreas		74	1.52 (0.97–2.39)		
				Lung, including trachea		232	1.08 (0.84–1.38)		
				Thyroid		7	1.35 (0.22–8.19)		
				Breast		22	1.38 (0.63–3.02)		
				Bone and connective tissue		8	1.05 (0.21–5.22)		
				Urinary bladder		16	1.13 (0.48–2.65)		
				Skin		8	1.72 (0.36–8.21)		

Table 2.10 (continued)

Reference, study location	Total No. of subjects	Follow- up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/ deaths	Relative risk (95%CI)	Covariates	Comments
Meyer <i>et al.</i> (2006) Germany	177 428 persons living in 48 municipalities in Bavaria		Presence of mobile- telephone relay stations, classified into three categories of relay-station coverage	Breast Brain and CNS Thyroid		NR	NR		Incidence of all cancers combined was not found to be elevated in municipalities with mobile- telephone relay stations. Specific cancers not heterogeneously distributed

AM, amplitude modulation; CNS, central nervous system; kW, kilowatt; MRR, mortality rate ratio; NR, not reported; TV, television

complicated. Nonetheless, the ecological studies provide evidence for consideration in the assessment of the coherence of a causal association of mobile-phone use with cancer of the brain.

The critical evidence comes primarily from case-control studies, as only few cohort studies have been carried out. The basic design of most case-control studies reviewed in this section has involved interviews with cases (most studies are of cancer of the brain) and with appropriate controls; the interviews characterize use of mobile phones, exposures to other sources of RF radiation (e.g. cordless phones) in some instances, potential confounding factors, and other information. The critical methodological concerns around interpretation of the findings of case-control studies of mobile-phone use involve the comparability of cases and controls, the potential for selection bias, and information bias, particularly in ascertainment of exposure to RF radiation from mobile-phone use. Confounding is a less serious concern because, apart from age, the only well-established causal factor for cancer of the brain is ionizing radiation, and also because in the general population the distribution of exposures, primarily from diagnostic irradiation, is unlikely to introduce substantial confounding.

Information bias related to exposure assessment has been a principal concern in interpreting the findings of case-control studies. The investigators have developed interview and questionnaire approaches for ascertaining mobile-phone use and exposure characteristics that attempt to capture the full exposure profile. Key exposure metrics have included the duration of use, call frequency, and cumulative use indicators, the types of device used, and various potential modifiers of exposure, such as use of a hands-free device and the laterality of use. With this approach, some degree of non-differential (random) misclassification of exposure to RF radiation is unavoidable. In studies of the association between protracted exposures and risk of cancer, a related concern is that the key exposure

metrics used may not capture the etiologically relevant period of a person's exposure profile (for example, if the effect of a hazard does not persist indefinitely, or appears only after an induction and latency period). Additionally, as in any case-control study, there is the possibility of differential recall according to case status regarding mobile-phone use and other items. Such bias may be in the direction of underreporting, if, for example, cases with tumours of the brain had diminished cognitive function. The bias may be in the direction of over-reporting if, for example, cases were more likely to recall events that might have led to their disease. A validation study carried out with the INTERPHONE Study demonstrated non-differential information bias, as well as the possibility of greater recall of temporally remote use by cases compared with controls ([Vrijheid et al., 2009a, b](#)). There is the additional possibility that the degree of measurement error varies from study to study, depending on the interview approach and other factors. While random misclassification generally reduces associations, differential misclassification may increase or decrease observed associations from the "true" underlying association.

Selection bias may also affect the results. Selection bias from two sources is of potential concern: specifically, differential participation by cases and controls that is determined by factors influencing likelihood of exposure. Additional selection bias can arise from the process used to select cases and controls, such that the association is distorted from that in the underlying population. This bias is of particular concern in case-control studies involving cases selected from hospitals or other medical institutions, as the factors that lead to hospitalization and diagnosis may also be associated with the exposure(s) under investigation. Selection bias may reduce or increase the observed association.

In interpreting the results of the case-control studies, consideration was given to the net consequences of selection bias and information

bias to answer the question as to whether the observed association(s) could reflect bias (at least in part), rather than causation. The judgment of the Working Group as to the potential consequences of bias was critical to the classification of the evidence from humans. The complexities in interpretation of the findings of case-control studies of mobile phones and cancer of the brain have been reviewed recently ([Ahlbom et al., 2009](#); [Saracci & Samet, 2010](#)).

2.3.1 Cancer of the brain

(a) Ecological studies

Multiple ecological studies have been published that compare time trends in use of mobile phones and incidence and mortality rates of various cancers, primarily brain ([Table 2.11](#)). [Because these studies provided only limited and indirect evidence on the risk of cancer potentially associated with mobile-phone use, the Working Group presented a brief synthesis only.] These included two time-trend studies ([Lönn et al., 2004](#); [Deltour et al., 2009](#)) in the combined Nordic countries, two in the United Kingdom ([Nelson et al., 2006](#); [de Vocht et al., 2011a](#)), three in parts of the USA ([Muscat et al., 2006](#); [Propp et al., 2006](#); [Inskip et al., 2010](#)), one each in Japan ([Nomura et al., 2011](#)), New Zealand ([Cook et al., 2003](#)), Switzerland ([Röösli et al., 2007](#)) and Israel ([Czerninski et al., 2011](#)), and one in a set of eleven countries ([Saika & Katanoda, 2011](#)). Most studies provided some data on the temporal pattern of increasing use of mobile phones, based mostly on annual numbers of private subscriptions and, in a few instances, on estimated prevalence of use. The information on use of mobile phones clearly demonstrated the rapid increase between 1985 and 2000; in some countries, the increase started in about 1990, while in others the increase began later in that decade. In some countries, the reported number of subscriptions had approached the total population of the country in 2000. The number of subscriptions is

a surrogate for population exposure to RF radiation, but the number does not reflect temporal changes in patterns of actual usage. Most of these ecological studies had used rates of cancer incidence calculated from data obtained from national or subnational cancer registries, while two studies used mortality rates. In most of these studies, the temporal association between trends in use of mobile phones and cancer incidence was assessed informally and descriptively. [The geographical correlation study carried out in several states of the USA ([Lehrer et al., 2011](#)) failed to adequately account for population size and composition.]

Studies that covered a long period between increasing use of mobile phones among the population under investigation and available data on cancer incidence from high-quality cancer registries were most informative for evaluating time trends. In Scandinavia, the rise in use of the mobile phone occurred relatively early. The reported prevalence of mobile-phone use among men aged 40–59 years was 7% in 1989 and reached 28% in 1993 ([Deltour et al., 2010](#)). No change in trends in cancer incidence was observed between 1993 and 2003 for this age group, which had the highest proportion of people who started using mobile phones at an early stage ([Deltour et al., 2009](#)). In the USA, the use of mobile phones started to increase somewhat later; about 100 million subscribers were registered in 2000, i.e. 36% of the population ([Inskip et al., 2010](#)). According to data collected by the Surveillance, Epidemiology, and End Results (SEER) Program, age- and sex-specific trends and overall temporal trends in rates of incidence of brain cancer in the USA were flat or downward between 1992 and 2006, with the exception of women aged 20–29 years ([Inskip et al., 2010](#)). In this age group, a statistically significant increasing trend was driven by the rising incidence in tumours of the frontal lobe. [It is the temporal lobe that is most heavily exposed to radiation when using a mobile phone at the ear ([Cardis et al., 2008](#)).]

Table 2.11 Time trends in use of mobile phones and cancer occurrence

Reference	Location	Exposure data	Trend in exposure	Organ site	Period of cancer occurrence	Cancer data	Cancer trend	Comments
Cook <i>et al.</i> (2003)	New Zealand	Proportion of mobile-phone subscribers in the New Zealand population	Sharp increase from 1987 (1%) to 1998 (> 30%), particularly since 1993 (5%)	All brain and salivary gland; temporal lobe; parietal lobe	1986–98	Incidence rates from New Zealand Cancer Registry	Flat trends from 1986 to 1998	No apparent impact of mobile-phone use on incidence of brain cancer. This study could only detect a risk if it occurred within 4 yr of first exposure
Hardell <i>et al.</i> (2003)	Sweden	None	Presumably sharp increases between 1980s and 2000	Vestibular schwannoma	1960–98	Incidence rates from Swedish Cancer Registry	Increase from 1960 to 1985, then rather flat	No effect of mobile-phone trends. Too early
Lönn <i>et al.</i> (2004)	Denmark, Finland, Norway, Sweden	Proportion of mobile-phone subscribers per year in each country	Sharp increase from 1987 (1–2%) to 1998 (30–50%) particularly after 1993	All brain and subtypes	1969–98	Incidence rates from Nordic National Cancer Registries	Gradual increase from 1968–1983; flat from 1983–96; slight upticks in 1997 and 1998	No apparent impact of mobile-phone use on incidence of brain cancer. Long-standing, high-quality registries. Increased incidence in late 1970s and early 1980s coincides with improvements in diagnosis. This study could only detect a mobile-phone-related risk if it occurred within about 5 yr of first exposure.
Muscat <i>et al.</i> (2006)	USA (SEER Program); 17 registries; about one quarter of the USA population	Unclear	From 0% to about 50% of the population; “exponential increase”	Neuronal tumours	1973–2002	Incidence rates from SEER	No change in incidence between two periods (1973–85 and 1986–2002)	No apparent impact of mobile-phone use on incidence of neuronal tumours. No data on year-by-year variability. Not clear when the number of users increased, probably in the early to mid-1990s. Neuronal tumours are extremely rare.

Table 2.11 (continued)

Reference	Location	Exposure data	Trend in exposure	Organ site	Period of cancer occurrence	Cancer data	Cancer trend	Comments
Nelson <i>et al.</i> (2006)	England and Wales, United Kingdom	Active mobile-phone subscriptions by year, 1987–2004	Very little before 1993; gradual increase to 1997 (10 million) then sharp annual increase to 2004 (60 million)	Acoustic neuroma	1979–2001	Incidence rates from National Cancer Registry for England and Wales	Gradual increase from 1980 to 1990; sharp increase to 1997; decline to 2000. Rise and decline of acoustic neuroma attributed to changes in diagnosis and registration	No apparent impact of mobile-phone use on incidence of acoustic neuroma. The reason for decline in rates after 1997 is uncertain, but its magnitude illustrates the difficulty of detecting a signal if there is one, against the background noise of statistical variability and methodological challenges. The number of subscriptions, approx. 60 million, is clearly in excess of the number of people with subscriptions in England and Wales.

Table 2.11 (continued)

Reference	Location	Exposure data	Trend in exposure	Organ site	Period of cancer occurrence	Cancer data	Cancer trend	Comments
Propp <i>et al.</i> (2006)	Several centres in the USA	None	NR	Acoustic neuroma (vestibular schwannoma)	Los Angeles 1975–98; other centres 1985–99	Incidence rates from the Central Brain Tumor Registry of the USA, and the Los Angeles County Cancer Surveillance Program	Modest, but discernable gradual increases over the period of observation	No apparent impact of mobile-phone use on incidence of acoustic neuroma. Modest increase in risk over the period of time studied (1970s to 1990s) could be due to improvement in diagnosis and registration or to some environmental factor. While the authors present no data on trends in mobile-phone use, it is likely that use increased in the early to mid-1990s. This study could only detect a mobile-phone-related risk if it occurred within about 5 yr of first exposure.
Röösli <i>et al.</i> (2007)	Switzerland	Prevalence of mobile phone use by year, with mortality rates	None before 1987; slow increase to 1996 (< 10%) and then sharp increase to 2000 (> 60%)	All brain (ICD-8 code 191)	1969–2002	Mortality rates from Swiss Federal Statistical Office	Gradual increase from 1969 to 2002, reaching a plateau after 1997. Smaller increase in rates after 1987 than before. For the whole period, there was a significant increase for men and women in older age groups, but not in younger ones. From 1987 onwards, rather stable rates in all age groups.	No apparent impact of mobile-phone use on incidence of cancer of the brain. High-quality mortality data. Authors quantify difficulty in detecting risk in such an ecological study. Improvements in survival may influence trends in mortality.

Table 2.11 (continued)

Reference	Location	Exposure data	Trend in exposure	Organ site	Period of cancer occurrence	Cancer data	Cancer trend	Comments
Deltour <i>et al.</i> (2009)	Denmark, Finland, Norway, Sweden	Unclear	Use increased from zero in the mid-1980s to 'widespread' in the early 1990s to 'sharply increased' in the mid-1990s.	Glioma and meningioma	1974–2003	Incidence rates from Nordic National Cancer Registries	Very slight increases in incidence from 1974 to 1997; no change after 1998	No apparent impact of mobile-phone use on incidence of cancer of the brain. High-quality registration. Up to 10 yr potential latency
Hardell & Carlberg (2009)	Sweden	None	Presumably sharp increases between 1980s and 2000	Brain, age > 19 yr Acoustic neuroma, age > 19 yr	1970–2007	Incidence rates from Swedish Cancer Registry	Changing annual incidence: 1970–79 (+0.15%) 1980–89 (+1.54%) 1990–99 (–0.25%) 2000–07 (+1.26%) 1970–79 (–1.66%) 1980–89 (+4.86%) 1990–99 (+0.66%) 2000–07 (–7.08%)	No evidence of an impact of mobile-phone use on the risk of acoustic neuroma. No or very weak evidence of an effect of phone use on risk of tumours of the brain. Slightly stronger evidence for increased risk of astrocytoma in the most recent period
Inskip <i>et al.</i> (2010)	USA (SEER Program); nine state or regional population-based cancer registries	Number of mobile-phone subscribers in USA by year	From very few in 1990 to 25 million in 1995; 100 million in 2000 and 200 million in 2005	All brain, excluding meningioma and lymphoma	1977–2006	Incidence rates from SEER	Gradual increase in risks from 1977 to 1985; since 1986 the pattern is flat or slightly decreasing. Some age/sex subgroups show increasing trends in some subtypes	No apparent impact of mobile-phone use on incidence of cancer of the brain. Very large numbers of cases. Up to 10 yr of potential latency

Table 2.11 (continued)

Reference	Location	Exposure data	Trend in exposure	Organ site	Period of cancer occurrence	Cancer data	Cancer trend	Comments
Czerninski et al. (2011)	Israel	None	Exposure trend not shown but presumably sharp increase between mid-1980s and 2006	Parotid gland	1970–2006	Incident numbers of cases from Israel National Cancer Registry	Approximate tripling of number of tumours of the parotid gland, with increase starting around 1977 and picking-up around 1990	Authors state that population growth explains part of the increase, but they do not acknowledge the role of ageing of the population. Rates would be more convincing than numbers. While numbers increased greatly after 1998, there were, nevertheless, important increases in numbers of cases before mobile phones could plausibly have caused large numbers of cases.
de Vocht et al. (2011a)	England	Mobile-phone subscriptions	Sharp increase from 0 in 1985 to 10 million in 1997 to > 50 million in 2003	All brain and each of 11 subsites	1998–2007	Incidence rates from United Kingdom Office of National Statistics	Linear regression for each of 24 sex/site categories. No significant trend for all cancers combined. Significant increase in incidence of tumours of temporal lobe and decreases in tumours of parietal lobe	No apparent impact of mobile-phone use on incidence of cancer of the brain, except for a small but unconvincing increase in incidence of tumours of the temporal lobe. Up to 10 yr of potential latency. The number of subscriptions, approx. 50 million in 2003, is clearly in excess of the number of people with subscriptions in England.

Table 2.11 (continued)

Reference	Location	Exposure data	Trend in exposure	Organ site	Period of cancer occurrence	Cancer data	Cancer trend	Comments
Nomura <i>et al.</i> (2011)	Osaka	None	Presumably sharp increases between 1980s and 2000	Intracranial	1975–2004	Incidence rates from Osaka Cancer Registry	Age 0–1 yr, flat; age 20–74 yr, flat until 1999 then slight decline; age 75 yr, sharp increase to 1983 then flat	No increase in incidence rates in recent years. Increasing rates in early years may have been due to diagnostic improvements.
Saika & Katanoda, (2011)	Study involved 11 countries: Japan, Hong Kong Special Administrative Region, Republic of Korea, USA, Australia, the Russian Federation, United Kingdom, Italy, Spain, France, Germany	None	Presumably sharp increases between 1980s and 2000	Brain and CNS	1990–2005	Mortality rates from WHO database	In most of the 22 country/sex data sets, there was a rather flat or declining rate; only in the Russian Federation and Spain was there an increase among females	No apparent increase in mortality from cancer of the brain. Mortality rates may reflect trends in diagnostic standards and in survival.

NR, not reported; SEER, Surveillance, Epidemiology and End Results; yr, year

In another study, trends in rates of newly diagnosed cases of cancer of the brain in England between 1998 and 2007 were examined ([de Vocht et al., 2011a](#)). Overall rates of incidence of cancer of the brain in males or females, or in any specific age group were not increased. However, the incidence of tumours of the temporal lobe increased between 1998 and 2007. In a subsequent letter, the same authors reported separate time trends for the periods 1979–99 and 2000–08. For men, a linear regression of age-adjusted rates showed an overall annual increase in 2000–08 of 3.3% (95% CI, 1.1–5.4), whereas it was 2.0% (95% CI, 1.4–2.6) for 1979–99 ([de Vocht et al., 2011b](#)). [The linear regression used for this analysis was not an appropriate method and therefore the 95% confidence intervals reported may not be reliable.] For women, corresponding annual increases were 2.8% (95% CI, 0.9–4.9) for 2000–08 and 1.4% (95% CI, 0.7–2.2) for 1979–99.

[The Working Group noted that time-trend analyses did not provide any indication that the rapid increase in use of mobile phones had been followed by a parallel increase in incidence rates of cancer of the brain. Increases in rates of brain tumours in the 1970s and 1980s had paralleled the introduction and distribution of new diagnostic tools, namely CT and MRI. The Working Group further noted that these descriptive analyses would be null if an excess in cancer risk from mobile-phone use became manifest only decades after phone use began, or if an increase affected only a small proportion of the cases by location.]

(b) Cohort studies

An early attempt to conduct a cohort study in the USA on cancer and mobile-phone use was halted by legal action; consequently, the study did not provide useful results ([Dreyer et al., 1999](#)). A retrospective cohort study was conducted in Denmark based on the subscriber lists from the two Danish mobile-phone operating companies, including 420 095 individual (i.e. virtually all non-institutional) subscribers from 1982 to

1995. Using unique identifiers, these subscribers were linked to the Danish Cancer Registry from 1982 onwards. The linkage allowed the identification of all cancers occurring in this cohort, and notably cancers of putative target organs. Expected numbers of cases were based on rates in the Danish population. Two papers appeared, one covering cancer outcomes from 1982 to 1996 ([Johansen et al., 2001](#)) and the second covering outcomes from 1982 to 2002 ([Schüz et al., 2006c](#)). In the latter, more recent, analysis, the expected rates were computed with cohort members excluded from the reference population by subtracting the number of cases of cancer and person-years observed in the cohort from the corresponding figures for the total Danish population. Approximately 85% of the cohort members were males.

There were various sources of misclassification, as acknowledged by the authors. Members of the reference population, apart from cohort members, may well have used mobile phones, either with subscriptions that were not in their names (e.g. corporate accounts), or with subscriptions taken out after 1995. Moreover, a member of the cohort may have been the official subscriber to an account, but not the true user. Using information from a separate case-control study, it was estimated that as many as 39% of cohort members may not have been mobile-phone users before 1996 and as many as 16% of the reference population may have been users. Using information from Statistics Denmark, it appeared that the cohort members represented a somewhat more affluent section of the Danish population. While the investigators had no data on individual patterns of use, they had information on the year of the individual's first subscription, and this was used to compute SIRs by time since first use. The median duration of subscription among subscribers was 8 years and the maximum was 21 years.

For the entire cohort there was a slight deficit of total cancers among males (SIR, 0.93; 95% CI,

0.92–0.95), and a slight excess among females (SIR, 1.03; 95% CI, 0.99–1.07). For the main cancer types of interest, the results were similarly close to the null value, with relatively narrow confidence intervals, as shown in [Table 2.12](#). For subtypes of cancer of the brain, most SIRs were close to the null value.

The SIR for glioma was 1.01 (95% CI, 0.89–1.14; 257 cases). The odds ratios for glioma in the two lobes closest to the ear showed conflicting results, with a SIR of 1.21 (95% CI, 0.91–1.58) for the temporal lobe and a SIR of 0.58 (95% CI, 0.36–0.89) for the parietal lobe. The SIR was lower for all other areas of the brain, although confidence intervals were overlapping. [[Cardis et al. \(2008\)](#) have reported that it is the temporal lobe of the brain that receives the highest percentage of RF radiation deposition (50%).]

The SIR for meningioma was 0.86 (95% CI, 0.67–1.09) and for acoustic neuroma (nerve sheath tumour) it was 0.73 (95% CI, 0.50–1.03). There was no trend in SIR according to years since first subscription, and the subgroup with > 10 years since first subscription had a low SIR for all tumours of the brain and nervous system (SIR, 0.66; 95% CI, 0.44–0.95). [There were few subscribers who began using a mobile phone \geq 10 years before the end of follow-up, and there was no information on individual levels of mobile-phone use.]

The Danish subscriber cohort study was updated for occurrence of acoustic neuroma (vestibular schwannoma) until 2006 ([Schüz et al., 2011](#)). This update and analysis was restricted to a large subset of subscribers and of the Danish population (2.9 million subscribers and non-subscribers) for which independent information was available on each subject's highest level of education, annual disposable income and marital status. Further to the follow-up with data from the Danish cancer registry, a clinical registry of acoustic neuroma was used to achieve completeness of case ascertainment and obtain additional tumour characteristics, such as laterality, and

spread and size of the acoustic neuroma. In this cohort analysis, having a long-term mobile-phone subscription of \geq 11 years was not related to an increased risk of vestibular schwannoma in men (RR, 0.87; 95% CI, 0.52–1.46; adjusted for sociodemographic factors); and no cases of acoustic neuroma occurred among long-term female subscribers versus 1.6 cases expected. Although 53% of Danes reported that they mainly used their phones on the right side, with 35% preferring the left side and 13% having no preferred side, based on data from the launch of a prospective cohort study described in [Schüz et al., 2011](#)), acoustic neuroma in the subscriber cohort occurred equally on both sides (48% of tumours were on the right side, with no change in this proportion over time). Acoustic neuromas in long-term male subscribers were not larger than those in non-subscribers and short-term subscribers (mean diameter, 14.6 versus 15.9 mm).

(c) Case-control studies

There have been many case-control studies of tumours of the brain in relation to use of mobile phones: a series from one group in Sweden (this study also included cordless phones), an IARC-coordinated series from 13 countries known as INTERPHONE (this study included use of cordless phones among the unexposed group), and several others, including three from the USA, and one each from Finland, France, Greece and Japan. Some studies considered all major types of tumours of the brain, while others considered glioma and meningioma, or glioma only, or acoustic neuroma only. The studies are presented below by major tumour type. Most studies were based on interviews with study subjects or proxies, and involved questions on history of mobile-phone use. Various exposure metrics were used in the different studies, including binary indicators of ever versus never use, metrics of duration of use, frequency of use, and time since start of use. In addition, some analyses

Table 2.12 Cohort study of cancer of the brain and use of mobile phones

Reference, study location and period	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Comments
Schüz <i>et al.</i> (2006c) Denmark, 1982–2002	420 095, 357 553 men, 62 542 women	1982–2002	Subscribers to mobile-phone service	All cancers	Ever subscribed Ever subscribed: men Ever subscribed: women	14 291 11 802 2 447	0.95 (0.93–0.97) 0.93 (0.92–0.95) 1.03 (0.99–1.07)	Update of Johansen <i>et al.</i> (2001) . Median time since first subscription, 8 yr. Expected numbers derived from Danish National Cancer Registry after excluding cohort members from the population. Questionable correspondence between mobile-phone subscriptions and use levels.
				Brain, CNS	Ever subscribed	580	0.97 (NR)	
				Brain, CNS	Ever subscribed: men	491	0.96 (0.87–1.05)	
				Brain, CNS	Ever subscribed: women	89	1.03 (0.82–1.26)	
				Brain, CNS	Latency, < 1 yr since start	51	0.90 (0.67–1.18)	
				Brain, CNS	Latency, 1–4 yr since start	266	1.03 (0.91–1.17)	
				Brain, CNS	Latency, 5–9 yr since start	235	0.96 (0.84–1.09)	
				Brain, CNS	Latency, ≥ 10 yr since start	28	0.66 (0.44–0.95)	
				Glioma (191–191.9)	Ever subscribed	257	1.01 (0.89–1.14)	
				Temporal lobe (191.2)	Ever subscribed	54	1.21 (0.91–1.58)	
				Parietal lobe (191.3)	Ever subscribed	21	0.58 (0.36–0.89)	
				Meningioma (192.1)	Ever subscribed	68	0.86 (0.67–1.09)	
				Nerve sheath tumours (192.0) ^a	Ever subscribed	32	0.73 (0.50–1.03)	

^a Includes other rare tumours of the nerve sheath
CI, confidence interval; CNS, central nervous system; NR, not reported; yr, year or years

considered modifiers of exposure, such as laterality of mobile-phone use. The latter was based on the premise that if there were a risk related to mobile-phone use, it should manifest itself in a greater proportion of tumours on the side of the head corresponding to the subject's preferred side of phone use. Some studies analysed exposure in relation to the lobe in which the tumour appeared, based on the premise that some lobes absorb more RF radiation than others.

(i) *Glioma*

See [Table 2.13](#)

A case-control study of cancer of the brain was conducted in five academic medical centres in the north-eastern USA during 1994–1998 ([Muscat et al., 2000](#)). Interviews were conducted with the cases ($n = 469$), mainly patients with glioma, and with controls ($n = 422$) selected from the same medical centres. Analysis of reported histories of mobile-phone use, adjusting for sociodemographic factors, study centre, proxy status, and date of interview, yielded a set of odds-ratio estimates that showed no effect, whether by various exposure metrics, anatomical location of the tumour, or histological subtypes. The only exception was an odds ratio of 2.1 (95% CI, 0.9–4.7) for neuroepitheliomatous tumours (14 exposed cases). [The Working Group noted that the highest prevalence of these tumours occurred in the temporal lobe.] The longest duration of use considered was ≥ 4 years. [The numbers of cases were small, exposure levels were low: of the 422 controls, 346 had never used a mobile phone and 22 had used a mobile phone for ≥ 4 years.]

[Inskip et al. \(2001\)](#) conducted a case-control study of tumours of the brain in three centres between 1994 and 1998. A total of 489 cases of glioma were interviewed, as were 799 controls. Compared with non-users, self-reported regular use of mobile phones was not associated with excess risk of glioma (OR, 0.8; 95% CI, 0.6–1.2). Based on very small numbers, there was no indication of excess risk among people with the

heaviest (cumulative use, > 500 hours) or longest (5 years or more) use of mobile phones, or any relationship between reported laterality of use and laterality of the tumours, or any relationship with neuroepitheliomatous tumours (OR, 0.5; 95% CI, 0.1–2.0; eight exposed cases). [Of the 799 controls, 625 had never or rarely used a hand-held mobile phone and only 50 had used a hand-held mobile phone before 1993.]

In a case-control study in Finland, the researchers enrolled cases of tumours of the brain and salivary gland occurring in 1996, as well as a 5 : 1 control series selected from the general population ([Auvinen et al., 2002](#)). There were 198 cases of glioma. Each subject was linked to a list of all subscribers to the two mobile-phone companies operating in Finland, to establish whether the subject had been a subscriber, for how long, and what type of phone he or she was using (analogue/digital). Linkage of records to the census allowed the investigators to ensure that the case and control series were similar in occupational, socioeconomic and urban/rural characteristics. The odds ratio for glioma was 1.5 (95% CI, 1.0–2.4) for those who had ever had a mobile-phone subscription (about 12% of all subjects), and 1.7 (95% CI, 0.9–3.5) for those who had had a subscription for > 2 years ($< 4\%$ of all subjects). When examined separately, the ever-users of analogue phones had an odds ratio for glioma of 2.1 (95% CI, 1.3–3.4) and ever-users of digital phones had an odds ratio of 1.0 (95% CI, 0.5–2.0). [A strength of this study was the linkage of cancer records, population-register records, and mobile-phone subscription records. It was limited by small numbers, inability to assess impact of use of mobile phones for > 2 years, and uncertainty about the correspondence between subscription to a mobile-phone service and individual use of mobile phones.]

Two hospital-based case-control studies ([Gousias et al., 2009](#); [Spinelli et al., 2010](#)), one in Greece and the other in France, examined associations between glioma and malignant tumours

of the brain, respectively, and mobile-phone use. The results are summarized in [Table 2.13](#). Neither study was informative due to small numbers and unclear methods of exposure assessment.

The INTERPHONE study, a multicentre case-control study on use of mobile phones and various types of tumour of the brain, is the largest study on this topic so far. The study was coordinated by IARC and conducted in 16 study centres in 13 countries with a common core protocol (Australia, Canada, Denmark, Finland, France, Germany, Israel, Italy, Japan, New Zealand, Norway, Sweden, and the United Kingdom). A detailed description of the study design, epidemiological methods and study population can be found in [Cardis et al. \(2007\)](#). In brief, the source population was generally restricted to major metropolitan areas where mobile phones were first introduced and where most of the population was considered to be unlikely to leave the region for diagnosis and treatment. Residents aged between 30 and 59 years were eligible for the study, but somewhat larger age ranges were applied in some of the centres. The study periods also varied somewhat across centres, ranging from 2 to 4 years between 2000 and 2004. Eligible cases were ascertained rapidly through neurological and neurosurgical facilities in the study regions, and completeness of ascertainment was checked with secondary sources ([Cardis et al., 2007](#)). Cases had a histologically confirmed or unequivocal imaging-based diagnosis of a first primary glioma, meningioma or acoustic neuroma. Three centres also included malignant tumours of the parotid gland, and Japan additionally included pituitary tumours. Population controls were randomly selected from population registries (part of Canada, Denmark, Finland, Germany, Italy, Norway, Sweden), electoral lists (Australia, part of Canada, France, New Zealand), patient lists from general practice (United Kingdom) or by random-digit dialling (part of Canada, France, Japan). Controls were individually (part of Canada, France, Japan,

New Zealand, United Kingdom) or frequency-matched (remaining countries) to cases on year of birth (within categories of 5 years), sex and study region. One control was recruited for each patient with a tumour of the brain, two for each patient with acoustic neuroma, and three for each patient with a tumour of the parotid gland.

All consenting subjects were interviewed face-to-face by trained interviewers by use of a computer-assisted personal interview (CAPI) whenever possible. If participants had died or were too ill to be interviewed, a proxy was interviewed. The questionnaire covered demographic factors, potential confounders and risk factors for the diseases of interest, including detailed questions on use of mobile phones and other wireless-communication devices. A regular mobile-phone user was defined as having used a mobile phone for at least one call per week during 6 months or more.

Since the first publications of national results in 2004 ([Christensen et al., 2004](#); [Lönn et al., 2004](#)), numerous papers have presented results from single countries ([Christensen et al., 2005](#); [Lönn et al., 2005](#); [Schoemaker et al., 2005](#); [Hepworth et al., 2006](#); [Schüz et al., 2006a, b](#); [Takebayashi et al., 2006, 2008](#); [Hours et al., 2007](#); [Klaeboe et al., 2007](#); [Schlehofer et al., 2007](#); [Sadetzki et al., 2008](#); [Hartikka et al., 2009](#)) or pooled results from a subset of the INTERPHONE countries, such as the five north European countries: Denmark, Finland, Norway, Sweden, and the United Kingdom ([Schoemaker et al., 2005](#); [Lahkola et al., 2007, 2008](#)). In addition, various papers have addressed methodological issues such as exposure misclassification and selection bias ([Samkange-Zeeb et al., 2004](#); [Berg et al., 2005](#); [Lahkola et al., 2005](#); [Vrijheid et al., 2006a, b, 2009a, b](#)). The results presented here focus on the pooled results from all countries.

The [INTERPHONE Study Group \(2010\)](#) published the pooled analysis of the INTERPHONE study on the risk of glioma and meningioma in relation to use of mobile phones,

Table 2.13 Case-control studies of glioma and use of mobile phones

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell <i>et al.</i> (1999) Sweden, 1994–96	136	Two controls per case	Population	Self-administered standardized questionnaire	48 glioblastoma, 46 astrocytoma, 19 oligodendroglioma, 3 ependymoma, 16 mixed glioma, and 4 other malignant tumours	Never use of mobile phone Ever use	53	1.0 1.0 (0.6–1.5)	Age, sex, SEI, and year of diagnosis	
Muscat <i>et al.</i> (2000) USA, 1994–98	469	422	In-patients from five USA academic medical centres. Controls from the same hospitals as cases, from daily admission rosters	In-person interviews, history of mobile-phone use	Brain cancer (191.0–191.9)	Ever use Cumulative use (h): 0 > 0 to ≤ 8.7 > 8.7 to ≤ 60 > 60 to ≤ 480 > 480	NR 17 12 19 14	0.7 (0.5–1.1) 1.0 1.0 (0.5–2.0) 0.6 (0.3–1.3) 0.9 (0.5–1.8) 0.7 (0.3–1.4)	Age, education, sex, race, study centre, proxy, year of interview	Analyses showed no associations by year of use. Few subjects with long-term heavy exposure. Response rates were 82% for cases and 90% for controls.
	108	422			Temporal lobe	Ever use	108	0.9 (0.5–1.7)		
	60	422			Parietal lobe	Ever use	60	0.8 (0.3–2.0)		
	354	422			Astrocytic	Ever use	41	0.8 (0.5–1.2)		
	35	422			Neuro-epitheliomatous	Ever use	14	2.1 (0.9–4.7)		

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Inskip <i>et al.</i> (2001) USA, 1994–98	489	799	Patients admitted to the same hospitals for a variety of non-malignant conditions.	Computer-assisted, personal interview in the hospital	Glioma	Cumulative use (h): Never or rarely used < 13 13–100 > 100 > 500 Regular use Start of use before 1993	398 26 26 32 11 85 23	1.0 0.8 (0.4–1.4) 0.7 (0.4–1.3) 0.9 (0.5–1.6) 0.5 (0.2–1.3) 0.8 (0.6–1.2) 0.6 (0.3–1.4)	Hospital, age, sex, race or ethnic group, proximity of residence to the hospital	There are results for other exposure metrics: average daily use, duration of use, year in which use began. Also results for acoustic neuroma, and for laterality by tumour type.
Auvinen <i>et al.</i> (2002) Finland, 1996	398 (198 glioma)	1990	Population Registry Centre of Finland	Information on subscriptions obtained from the two mobile-network providers operating in Finland in 1996	Glioma (191)	<i>Analogue:</i> Ever < 1 yr 1–2 yr > 2 yr <i>Digital:</i> Ever < 1 yr 1–2 yr > 2 yr	26 4 11 11 10 3 7 0	2.1 (1.3–3.4) 1.6 (0.5–5.1) 2.4 (1.2–5.1) 2.0 (1.0–4.1) 1.0 (0.5–2.0) 0.8 (0.2–2.6) 1.4 (0.6–3.4) 0	Age, sex	Cases, age 20–69 yr

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell <i>et al.</i> (2002b) Sweden, 1997–2000	588	581	Population	Self- administered standardized questionnaire	415 astrocytomas, 6 medullo- blastomas, 54 oligodendro- gliomas, 11 ependymomas, 65 other/mixed gliomas, and 37 other malignant tumours of the brain	Never use of mobile/ cordless phone Analogue, ever use Digital, ever use Digital, > 1–6 yr latency Digital, > 6 yr latency	79 112 100 12	1.0 (reference) 1.1 (0.8–1.6) 1.1 (0.8–1.5) 1.1 (0.8–1.4) 1.7 (0.7–4.3)	Age, sex, SEI, and year of diagnosis	Ipsilateral use of analogue phone was associated with risk of malignant tumour of the brain (OR, 1.8; 95% CI, 1.2–3.0). Ipsilateral use of digital phone was also associated with risk of malignant tumour of the brain (OR, 1.6; 95% CI, 1.1–2.4).

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell <i>et al.</i> (2006a,c) Sweden, 2000–03	317	1990	Population	Self-administered standardized questionnaire	248 astrocytomas, and 69 other malignant tumours of the brain	Never use of mobile/cordless phone	63	1.0	Age, sex, SEI, and year of diagnosis	
						Ever use, analogue	68	2.6 (1.5–4.3)		Analogue phone: Ipsilateral use: 3.1 (95% CI, 1.6–6.2); contralateral use: 2.6 (95% CI, 1.3–5.4)
						Ever use, digital	198	1.9 (1.3–2.7)		Digital phone: Ipsilateral use: 2.6 (95% CI, 1.6–4.1); contralateral use: 1.3 (95% CI, 0.8–2.2)
						<i>Time since start of use, analogue (yr)</i>				
						> 1–5	0	–		
						> 5–10	20	1.8 (0.9–3.5)		
						> 10	48	3.5 (2.0–6.4)		
						<i>Time since start of use, digital (yr)</i>				
						> 1–5	100	1.6 (1.1–2.4)		
						> 5–10	79	2.2 (1.4–3.4)		
						> 10	19	3.6 (1.7–7.5)		

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell <i>et al.</i> (2006b) Sweden, 1997–2003	905	2162	Population	Self-administered standardized questionnaire	539 high-grade astrocytomas, 124 low-grade astrocytomas, 93 oligodendrogliomas, 78 other/mixed gliomas and 71 other malignant tumours of the brain	Never use of mobile/cordless phone		1.0 (reference)	Sex, age, SEI, and year of diagnosis	Pooled analysis of case-control data for living cases ascertained from 1997–2000 and 2000–03. See also further results of analyses of these data in Hardell <i>et al.</i> (2009)
						Ever use, analogue	178	1.5 (1.1–1.9)		
						Ever use, digital	402	1.3 (1.1–1.6)		
						<i>Time since start of use, analogue (yr)</i>				
						> 1–5	39	1.2 (0.8–1.8)		
						> 5–10	57	1.1 (0.8–1.6)		
						> 10	82	2.4 (1.6–3.4)		
						<i>Time since start of use, digital (yr)</i>				
						> 1–5	265	1.2 (1.0–1.5)		
						> 5–10	118	1.7 (1.2–2.2)		
						> 10	19	2.8 (1.4–5.7)		
						<i>Cumulative call time, analogue (h)</i>				
						1–1000	147	1.3 (1.0–1.7)		
						1000–2000	10	3.0 (1.1–7.7)		
						> 2000	21	5.9 (2.5–14)		
						<i>Cumulative call time, digital (h)</i>				
						1–1000	355	1.3 (1.0–1.6)		
						1001–2000	26	1.8 (1.0–3.1)		
						> 2000	21	3.7 (1.7–7.7)		

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell et al. (2006b) (cont.)	<i>Ipsilateral use, analogue:</i>									
					All malignant		95	2.1 (1.5–2.9)		
					High-grade astrocytoma		62	2.4 (1.6–3.6)		
					Low-grade astrocytoma		10	1.8 (0.8–4.1)		
	<i>Contralateral use, analogue:</i>									
					All malignant		54	1.1 (0.8–1.6)		
					High-grade astrocytoma		37	1.6 (1.0–2.5)		
					Low-grade astrocytoma		4	0.5 (0.2–1.6)		
	<i>Ipsilateral use, digital:</i>									
					All malignant		195	1.8 (1.4–2.4)		
					High-grade astrocytoma		127	2.3 (1.7–3.1)		
					Low-grade astrocytoma		27	1.9 (1.0–3.5)		
	<i>Contralateral use, digital:</i>									
					All malignant		119	1.0 (0.7–1.3)		
					High-grade astrocytoma		69	1.1 (0.8–1.5)		
					Low-grade astrocytoma		16	1.1 (0.5–2.1)		

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Gousias <i>et al.</i> (2009) Greece, 2005–07	41	82	Neuro-surgery patients	In-person interviews, history of mobile phone use	Glioma	Minutes per year of mobile-phone use	NR	1.00 (0.99–1.01)	Age, sex, residence area, smoking, alcohol, head trauma	Not informative because of low power and too finely resolved exposure metric
Hardell <i>et al.</i> (2010) Sweden, 1997–2003	346	343 cancer controls, 276 other controls	Swedish Death Registry	Interviews with relative of decedent	314 gliomas and 32 other malignant tumours of the brain	Never use of mobile/cordless phone Ever use, analogue Ever use, digital <i>Cumulative call time, analogue (h)</i> 1–1000 1001–2000 > 2000 <i>Cumulative call time, digital (h)</i> 1–1000 1001–2000 > 2000	61 83	1.0 1.7 (1.1–2.7) 1.4 (1.0–2.1)	Sex, age, SEI, and year of diagnosis	Analysis of deceased cases (and controls) only
							41 5 15	1.5 (1.0–2.5) 1.1 (0.3–3.3) 5.1 (1.8–14)		
							58 8 17	1.2 (0.8–1.8) 2.6 (0.9–8.0) 3.4 (1.5–8.1)		

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Spinelli et al. (2010) France, 2005	122	122	In-patients from neurosurgery departments of the same hospitals; unrelated to cancer	In-person interviews	Malignant primary tumours of the brain, 72 glioblastomas	Subscription hours/year 0 < 4 4–36 ≥ 36	37 8 58 13	1.0 0.9 (0.3–2.4) 1.4 (0.8–2.8) 1.1 (0.4–2.8)	Sex, age	Unclear criteria for recruitment; small numbers
INTERPHONE Study Group (2010) Australia, Canada, Denmark, Finland, France, Germany, Israel, Italy, Japan, New Zealand, Norway, Sweden, United Kingdom, 2000–04	2708	2972	Population (except United Kingdom: GP patients)	Interviewer-administered standardized questionnaire	Glioma (D33.0, D43.0–43.9, C71.0–71.9)	Never regular use of mobile phone Regular use Time since start of use (yr) 1–5 2–4 5–9 ≥ 10 Cumulative call time with no hands-free devices (h) ≤ 5 5–12.9 13–30.9 31–60.9 61–114.9 115–199.9 200–359.9 360–734.9 735–1639.9 ≥ 1 640	1042 1666 156 644 614 252 141 145 189 144 171 160 158 189 159 210	1.0 (ref.) 0.81 (0.70–0.94) 0.62 (0.46–0.81) 0.84 (0.70–1.00) 0.81 (0.60–0.97) 0.98 (0.76–1.26) 0.70 (0.52–0.94) 0.71 (0.53–0.94) 1.05 (0.79–1.38) 0.74 (0.55–0.98) 0.81 (0.61–1.08) 0.73 (0.54–0.98) 0.76 (0.57–1.01) 0.82 (0.62–1.08) 0.71 (0.53–0.96) 1.40 (1.03–1.89)	Sex, age, study centre, ethnicity (in Israel) and education	OR highest in short-term users (start of mobile phone use, 1–4 yr before reference date) (OR, 3.77; 95% CI, 1.25–11.4, based on eight cases)

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell <i>et al.</i> (2011a) Sweden, 1997–2003	1148	2438	Population	Self-administered standardized questionnaire	Glioma	Never use of mobile/cordless phone Ever use (mobile phone) <i>Time since start of use (yr)</i> > 1–5 > 5–10 > 10 <i>Cumulative call time, mobile phone (h)</i> 1–1000 1001–2000 > 2000	529 250 156 123	1.0 1.3 (1.1–1.6) 1.1 (0.9–1.4) 1.3 (1.0–1.6) 2.5 (1.8–3.3) 1.2 (1.03–1.5) 1.8 (1.2–2.8) 3.2 (2.0–5.1)	Sex, age, SEI, and year of diagnosis	Pooled analysis of case-control data for living cases ascertained from 1997–2000, and 2000–03, as well as case-control data for deceased cases 1997–2003.

Table 2.13 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments						
Cardis et al. (2011) Australia, Canada, France, Israel, Italy, New Zealand, 2000–04	553	1762	Population	Interviewer-administered standardized questionnaire	Glioma (D33.0, D43.0–43.9, C71.0–71.9)	<i>RF TCSE (J/kg)</i> < 76.7 76.7– 284.1– 978.9– 3123.9+	67 68 60 57 103	0.76 (0.53–1.09) 0.94 (0.66–1.35) 0.80 (0.54–1.18) 0.89 (0.61–1.30) 1.35 (0.96–1.90)	Sex, age, study centre, ethnicity (in Israel) and education	Interpretation of OR is most meaningful when compared with the corresponding OR for comparable exposure surrogates of mobile-phone use. When stratified for different time windows of time before diagnosis, the OR tended to increase with increasing TSCE for use ≥ 7 yr in the past. For the highest exposure quintile: OR, 1.91 (95% CI, 1.05–3.47)						
											<i>Case-only analyses:</i> Ever regular user <i>Time since start of use (yr)</i> 1–4 5–9 ≥ 10 <i>Cumulative call time without hands-free devices (h)</i> < 39 39–220 220–520 520–1147 > 1147	30	1.35 (0.64–2.87)	1.37 (0.59–3.19) 0.72 (0.27–1.90) 2.80 (1.13–6.94)	6 4 5 10 5	1.19 (0.40–3.51) 0.93 (0.27–3.14) 1.38 (0.42–4.53) 2.55 (0.94–6.91) 0.99 (0.30–3.27)

Table 2.13 (continued)

[illegible]

GP, general practitioner; h, hour; NR, not reported; OR, odds ratio; RF, radiofrequency radiation; SEI, socioeconomic index; SES, socioeconomic status; TCSE, total cumulative specific energy; yr, year

and included 2708 cases of glioma and 2972 controls. The study included 252 cases of glioma and 232 controls who had first used a mobile phone at least 10 years before the reference date. Participation rates were 64% among cases of glioma and 53% among controls. There was wide variation in participation rates for controls between study centres (42–74%).

For regular users, the odds ratio for glioma was 0.81 (95% CI, 0.70–0.94) (Table 2.13). In most study centres, odds ratios of < 1.0 were also seen for all categories of time since start of use and of cumulative number of calls. [The reason for these low odds ratios was not established. While it is plausible that this may in part reflect selection/participation biases, sensitivity analyses carried out by [Vrijheid et al. \(2009a\)](#) indicated that it was unlikely to fully explain these results.] In terms of cumulative call time, all odds ratios were < 1.0 for all deciles of exposure except the highest (10th) decile (> 1640 hours). For this exposure group, the odds ratio for glioma was 1.40 (95% CI, 1.03–1.89). There were 252 cases and 253 controls who reported start of use ≥ 10 years before the reference date. The odds ratio for the highest exposure decile of cumulative call time dropped from 1.40 to 1.27 when subjects (both controls and cases) who reported use > 5 hours per day were excluded from the analysis. When mobile-phone use was truncated at 5 hours, the odds ratio was 1.38 (95% CI, 1.02–1.87). [There was reasonable doubt about the credibility of such reports and it is possible that the excess of cases in those with unreasonably high values reflected a general tendency for cases to overestimate more than controls, which could contribute to the apparent excess risk in the highest decile. As noted earlier, there is evidence that cases tended to overestimate their past exposure more than controls ([Vrijheid et al., 2009a](#)).] For cases of glioma, the proportion of proxy respondents, the number of imputations for missing values, and the proportion of subjects judged by their interviewer to be non-responsive or having

poor memory were all higher than for controls ([INTERPHONE Study Group, 2010](#)). However, sensitivity analyses showed that these differences by themselves did not explain the results seen in the highest decile of cumulative call time. More information on the various methodological issues and corresponding sensitivity analyses were discussed by the [INTERPHONE Study Group \(2010\)](#).] There was no evidence of heterogeneity in effect across study centres.

More detailed analyses were conducted by the INTERPHONE study team to evaluate the possible association between mobile-phone use and risk of glioma. The odds ratio in the highest exposure decile of cumulative use was larger for tumours in the highly exposed temporal lobe (OR, 1.87; 95% CI, 1.09–3.22) than in the less exposed parietal or frontal lobes (OR, 1.25; 95% CI, 0.81–1.91) or for tumours in other locations (OR, 0.91; 95% CI, 0.33–2.51). This result was consistent with patterns of energy deposition in the brain ([Cardis et al., 2008](#)).

The ratio of the odds ratios for ipsilateral phone use to those for contralateral use increased steadily with increasing cumulative number of calls. [This would be expected if there were an exposure–response association.] However, notwithstanding similar trends in higher exposure categories, the highest ratios of these odds ratios for cumulative call time and for time since start of use were observed in the lowest exposure categories. [While these odds ratios were highly imprecise, this pattern may suggest bias in recall of side of phone use.]

In Appendix 2 of the [INTERPHONE Study Group \(2010\)](#) publication, an additional analysis was reported in which never-regular users were excluded from the analysis and the lowest exposure category was used as the reference category. This analysis was based on the assumption that participation bias was the principal explanation for the decreased odds ratios of the main analysis and that bias was related only to mobile-phone user status and not to extent of use. As a result,

most of the odds ratios for glioma increased above unity. Increased odds ratios were found for people who started to use their phone 2–4 years before diagnosis (OR, 1.7; 95% CI, 1.2–2.4), 5–9 years before diagnosis (OR, 1.5; 95% CI, 1.1–2.2) or > 10 years before diagnosis (OR, 2.2; 95% CI, 1.4–3.3). In terms of cumulative call time, the odds ratio for glioma did not show an upward trend for the first nine deciles of exposure, but the odds ratio for the highest category (> 1640 hours) was increased (OR, 1.8; 95% CI, 1.2–2.9).

Some publications of the results for glioma from national INTERPHONE centres were based on broader eligibility criteria, e.g. extending the age range to 20–70 years ([Christensen et al., 2005](#)). Inclusion of additional cases did not yield markedly different results in these national publications compared with the pooled analysis.

[The strengths of the INTERPHONE study included its large sample size, the common core protocol, comprehensive data collection and in-depth data analyses (including a wide variety of sensitivity and validation analyses), and its use of population-based controls. The exposure assessment was, however, a limitation. As in most other case–control studies, mobile-phone use was estimated from retrospectively collected interview data and thus recall error was an issue. According to a comparison of self-reported mobile-phone use with operator-recorded data in a comparatively small sample of INTERPHONE participants from Australia, Canada and Italy, little differential exposure misclassification between cases and controls was found on average. However, in the highest category of cumulative number of calls, overestimation was more pronounced in cases than in controls ([Vrijheid et al., 2009a](#)). Furthermore, the ratio of self-reported phone use to recorded phone use increased with increasing time before the interview to a greater degree in cases than in controls. Such a pattern could explain an increased risk in the most extreme exposure categories. However, the number of subjects with long-term data was

relatively small and recall could only be assessed for 4–6 years at most.

Another limitation of the INTERPHONE study was the relatively low participation rate, particularly for controls (53%), which was less than that for cases (patients with glioma, 64%; meningioma, 78%; acoustic neuroma, 82%). This offered the potential for differentially selective study participation; and there is evidence that people who had ever used mobile phones regularly were more likely to agree to participate than people who had never used mobile phones regularly ([Lahkola et al., 2005](#); [Vrijheid et al., 2009b](#)). This would produce downwardly biased estimates of relative risk. [The Working Group noted that a strength of this study was its use of population-based controls and the relatively high participation rate of cases.]

In summary, there was no increased risk of glioma associated with having ever been a regular user of mobile phones in the INTERPHONE study. There were suggestions of an increased risk of glioma in the group in the highest decile of exposure, for ipsilateral exposures, and for tumours of the temporal lobe [although chance, bias or confounding may explain this increased risk].

After publication of the pooled data on glioma, additional analyses were undertaken by the INTERPHONE researchers to evaluate the association between mobile-phone use and risk of glioma. They included refined dose estimation, case–case analyses, and case–specular analyses. Each of these analyses has its merits in complementing the overall picture and in evaluating the role of bias, as discussed below.

Refined dose estimation

In principle, a measure of absorbed RF radiation should be a more biologically relevant metric than “use” of mobile phones, if estimated accurately. In an attempt to derive a more biologically relevant metric, data from five INTERPHONE countries (Australia, Canada, France, Israel and

New Zealand) were used to examine the associations of tumours of the brain with RF fields from mobile phones by estimating the total cumulative specific-energy (TCSE) dose for each individual ([Cardis et al., 2011](#)). For each case, the location of the tumour was determined by neuroradiologists and the centre of the tumour was estimated by a computer algorithm (Israel) or by the neuroradiologist (most participants in the other countries). This analogous tumour location was allocated to the controls matched to each case. Matching was done *post hoc* by use of an algorithm that optimized matching on interview time and age within strata defined by sex, region and, in Israel, country of birth. The number of controls per case varied from 1 to 19 (median, 3).

For each study participant, the TCSE was calculated with an algorithm considering the frequency band and communication system of all phones the subject had used, multiplied by call duration. In addition, laterality, use of hands-free devices, network characteristics and urban or rural residence were taken into account (for details, see [Cardis et al., 2011](#)). A census of TCSE was carried out 1 year before the reference date.

For the glioma analysis, the 553 cases of glioma for which localization data and communication-systems information were available (42% of all eligible cases) and their 1762 controls (36% of ascertained controls) were included. Odds ratios for glioma were < 1.0 in the first four quintiles of TCSE. In the highest quintile, the odds ratio for glioma was 1.35 (95% CI, 0.96–1.90). Various sensitivity analyses did not markedly affect this odds ratio. Odds ratios in categories of TCSE were also examined in time windows since first use of a mobile phone. There was a fairly consistent dose–response pattern with an odds ratio of 1.91 (95% CI, 1.05–3.47) in the highest exposure quintile when considering TCSE exposure ≥ 7 years before the reference date. There was little evidence of an association for exposures in more recent time windows. [The Working Group noted that TCSE was highly

correlated with cumulative call time (weighted kappa, 0.68). As this exposure surrogate was mainly determined by self-reported data, recall and selection bias were of concern, as they were for the other INTERPHONE analyses. Results from TCSE analyses were similar to those for cumulative duration of mobile-phone use.]

Case–case analyses

This is a novel approach for studying the effect of radiofrequency fields emitted by mobile phones. As it is based on cases only, differential participation and recall error between cases and controls is not of concern. In both studies presented below, reported preferred side of use was not considered for determining exposed brain areas. While, this should reduce the possible impact of recall bias, it probably also introduces exposure misclassification, which is expected to be random and thus would bias any risk estimates towards unity.

The same database of five countries discussed above ([Cardis et al., 2011](#)) was used to conduct a case–case analysis by comparing the characteristics of mobile-phone use among people with tumours in highly exposed areas of the brain, defined as areas absorbing > 50% of the specific absorption rate (SAR) from use of mobile phones at both sides of the head (i.e. without taking into account laterality), with the corresponding characteristics of people with tumours in other parts of the brain. Comparisons were made with respect to time since first use of a mobile phone and cumulative call time. The odds ratio for presence of the tumour in the most exposed part of the brain for people who had started using a mobile-phone ≥ 10 years previously was 2.80 (95% CI, 1.13–6.94; based on 11 exposed cases), but it was not increased for people who had started using a mobile-phone more recently. There was, in addition, moderate but inconsistent evidence that the odds ratio for presence of a tumour in the most exposed area increased with increasing cumulative call time.

Data from seven INTERPHONE European countries (Denmark, Finland, Germany, Italy, Norway, Sweden, and south-eastern England) were also used to conduct a case–case analysis ([Larjavaara et al., 2011](#)). In total, 888 cases of glioma in people aged between 18 and 69 years were included. For each case, the tumour midpoint on a three-dimensional grid was defined, based on radiological images. The distance to the estimated axis of a mobile phone in use on the same side of the head as the glioma was calculated, irrespective of the patient's reported typical side of phone use. Regression models were then computed to compare distance between the midpoint of the glioma and the mobile-phone axis for various exposure groups of self-reported mobile-phone use. In addition, unconditional logistic regression models were applied for the number of tumours occurring at a distance of ≤ 5 cm from the phone axis.

These analyses did not suggest an association between mobile-phone use and distance of glioma from the mobile-phone axis. For instance, the mean distance between tumour midpoint and the phone axis was similar among never-regular mobile-phone users and regular users (6.19 versus 6.29 cm; $P = 0.39$). In the dichotomized analysis examining the occurrence of tumours at a distance of ≤ 5 cm from the phone axis, odds ratios were below unity for the most exposed groups relative to never-regular users. [A limitation of the study was that exposed areas were defined on the basis of distance from the phone axis only; there were no dosimetric calculations. The results of analyses of the spatial distribution of SAR from more than 100 mobile phones ([Cardis et al., 2008](#)) showed that, although there was some variability, most exposure occurs in areas of the brain closest to the ear. Exposure is not evenly distributed along the phone axis; thus the approach used could result in substantial misclassification of exposure.]

Case–specular analysis

In the case–specular analysis, a hypothetical control location is defined in the head of each patient with glioma. This was done for the data from the seven European countries described above ([Larjavaara et al., 2011](#)) by symmetrically reflecting the location of the actual tumour site across the midpoint of the axial and coronal planes to obtain the mirror-image location as the control location. This counterfactual control site and the location of the actual case site were compared with respect to their distances orthogonal to the mobile-phone axis. An association would be indicated if the odds ratio increased systematically with the amount of exposure; however, this pattern was not observed. The odds ratio was larger for never-regular users than regular users. There was no increasing odds ratio for increasing use of cumulative call time.

[The strength of case–specular analysis is that each subject is his/her own control. Nevertheless, the analysis relies on self-reported use of mobile phones when comparing odds ratio between various strata. Thus exposure misclassification affects the analysis. Never-regular users were, on average, older and more commonly female, and if these factors were to affect the tumour location, bias could be introduced. However, there was little indication for this. A limitation of the study was the small number of long-term users in the case-specular analysis, resulting in wide confidence intervals. As noted above, the absence of dosimetric calculations and use of distance to the phone axis rather than to the most exposed part of the brain was a limitation.]

[Hardell et al. \(1999, 2000, 2001, 2002a, b, 2003, 2006a, b, 2009, 2010, 2011a\)](#) have published a series of papers reporting findings regarding associations between use of mobile phones and tumours of the brain. All these epidemiological analyses have been of the case–control design, with cases identified from records of regional cancer registries in Sweden and controls

identified from the Swedish population register or the Swedish death registry (the latter was used when sampling controls for deceased cases). [While reported in a series of publications, the Working Group noted that this research had involved the ongoing collection of case-control data over an extended period of time using a fixed protocol. The Working Group noted that a strength of these analyses followed from the early, and widespread, use of mobile phones in Sweden, implying a population that has accrued exposures from mobile phones over a relatively long time period (analogue phones have been in use since the early 1980s). The fairly long-term exposure from mobile phones permits consideration of any effect that may appear after a more protracted period of exposure than in other locations. Consequently, *Hardell et al.* could address higher cumulative exposures (when measured in terms of total duration of phone use), and include people using devices designed with early mobile-phone technologies, which tended to have higher power output than those based on later mobile-phone technologies.]

In the latest paper available, [Hardell et al. \(2011a\)](#) reported the findings of a pooled analysis of associations between mobile- and cordless-phone use and glioma. Cases were ascertained from 1 January 1997 to 30 June 2000 from population-based cancer registries in Uppsala-Orebro, Stockholm, Linkoping, and Gothenburg, and from 1 July 2000 to 31 December 2003 in Uppsala-Orebro and Linkoping. Eligible cases were aged 20–80 years at diagnosis. Population controls were selected from the Swedish population registry, which includes all residents; controls were matched to cases based on calendar year of diagnosis as well as age (within 5-year categories), sex and study region. Deceased controls for deceased cases were selected from the death registry. Environmental and occupational exposures were assessed by a self-administered 20-page questionnaire sent out by post. The questionnaire solicited information regarding demographic

characteristics, occupational history, and other potential risk factors for cancer of the brain, and asked detailed questions on use of mobile phones and other wireless communication technologies, including year of first use, type of phone, average number of minutes of daily use, and side of head on which the phone had been used most frequently. A maximum of two reminders was sent if the questionnaire was not completed. A trained interviewer, using a structured protocol, carried out supplementary phone interviews to verify information provided in the questionnaire. Questionnaires were assigned an identification code such that the phone interviews and coding of data from questionnaires were blinded to case-control status. Study participants were asked again as to the side of head on which a phone had been used most frequently. [The Working Group noted that bias could be introduced by such an interview process; [Hardell et al. \(2002a\)](#) provided some information regarding classification of cases and controls with respect mobile-phone use based on the questionnaire, and the participants' classification after supplementary interview.] All study participants using mobile or cordless phones were sent an additional letter to re-solicit information on the side of the head on which the phone had been used most frequently. Details regarding the exposure assessment are reported in [Hardell et al. \(2006a, b\)](#). For deceased participants, an interview with a proxy (relative of the deceased) was conducted. Exposure was defined as reported use of a mobile phone and separately reported use of a cordless phone; exposure in the year immediately before case diagnosis or control selection was not included.

Cumulative lifetime use in hours was dichotomized by use of the median number of hours among controls as a cut-off point; and, lifetime use in hours was categorized into the following groups: 1–1000, 1001–2000, and ≥ 2000 hours. Three categories of time since exposure were considered > 1 –5 years, > 5 –10 years, and

> 10 years. Primary statistical analyses were conducted using unconditional and conditional logistic regression models with adjustment for sex, age, socioeconomic index, and year of diagnosis. Participation rates were 85% among cases and 84% among controls.

The analysis included 1148 cases with a histopathological diagnosis of glioma ([Hardell *et al.*, 2011a](#)). When mobile-phone users were compared with people who reported no use of mobile or cordless phones, or exposure > 1 year before the reference date, the odds ratio for glioma was reported to be 1.3 (95% CI, 1.1–1.6) ([Table 2.13](#)). For study participants who first used a mobile phone ≥ 10 years before the reference date, the odds ratio was 2.5 (95% CI, 1.8–3.3). This study included 123 cases of glioma and 106 controls among those who first used a mobile phone ≥ 10 years before the reference date. In terms of cumulative call time using a mobile phone, odds ratios for glioma increased with increasing categories of lifetime exposure. For the highest exposure group (> 2000 hours), the odds ratio was 3.2 (95% CI, 2.0–5.1). Use of cordless phones was also associated with glioma: the odds ratios for 1–1000 hours, 1001–2000 hours and > 2000 hours of use were 1.2 (95% CI, 0.95–1.4), 2.0 (95% CI, 1.4–3.1), and 2.2 (95% CI, 1.4–3.2), respectively. When considering age at first use, the odds ratio for mobile-phone use for all malignant tumours of the brain was 2.9 (95% CI, 1.3–6.0) for ages < 20 years, 1.3 (95% CI, 1.1–1.6) for ages 20–49 years, and 1.2 (95% CI, 1.0–1.5) for ages ≥ 50 years.

[The Working Group noted that information obtained from next of kin may be less reliable than that from living cases and controls. Analyses reported by Hardell *et al.* that are based solely on information obtained from living cases and controls are not affected by the same concerns about bias arising from information obtained from next of kin.] Excluding deceased cases (and affiliated controls) yielded odds ratios of 1.5 (95% CI, 1.1–1.9) for ever-use of analogue phones, 1.3

(95% CI, 1.1–1.6) for ever-use of digital phones, and 1.3 (95% CI, 1.1–1.6) for ever-use of cordless phones ([Hardell *et al.*, 2006a](#)).

Information on laterality of phone use was collected only from living cases and controls. Pooled case–control analyses were restricted to 905 living cases with malignant tumours of the brain and 2162 controls ([Hardell *et al.*, 2006b](#); [Hardell & Carlberg, 2009](#)). Of the cases, 663 were astrocytomas (grades I–IV), 93 were oligodendrogliomas, and the remainder were other malignant tumours of the brain. Participation rates were 90% among cases with malignant tumours and 89% among controls. For users of analogue and digital mobile phones, an increased odds ratio was seen for all malignant tumours of the brain and high-grade astrocytomas with ipsilateral use of mobile phones and with the tumour on the same side of the head, but no increased risk for contralateral use of mobile phones when compared with people who had not used mobile or cordless phones ([Table 2.13](#)). [The Working Group noted that a strength of this study was its use of population-based controls and the high participation rate of cases and of controls.]

An earlier report by Hardell *et al.* included a different set of cases of tumours of the brain ascertained during 1994–96 in Uppsala and 1995–96 in Stockholm ([Hardell *et al.*, 1999](#)). Participation rates were 90% among cases and 91% among controls. The analyses included 136 cases of malignant tumours of the brain (including 48 cases of glioblastoma, 46 cases of astrocytoma, and 19 cases of oligodendroglioma), with controls matched on sex, age, and region. Of the 425 controls, 161 reported ever having used a mobile phone and 85 reported having used a mobile phone for > 136 hours. Use of a mobile phone was not associated with an increased risk of malignant tumours of the brain (OR, 1.0; 95% CI, 0.7–1.4). [The Working Group noted that a strength of the study was the high participation rates of cases and controls.]

It is useful to consider variation in effect estimates by calendar period. Among cases ascertained during 1997–2000 there were 588 malignant tumours of the brain, including 415 cases of astrocytoma and 54 cases of oligodendroglioma. Ever-use of analogue phones yielded an odds ratio of 1.13 (95% CI, 0.82–1.57), with the odds ratio for ipsilateral use being 1.85 (95% CI, 1.16–2.96) and the odds ratio for contralateral use being 0.62 (95% CI, 0.35–1.11). Ever-use of digital phones yielded an odds ratio of 1.13 (95% CI, 0.86–1.48), with an odds ratio for ipsilateral use of 1.59 (95% CI, 1.05–2.41) and an odds ratio for contralateral use of 0.86 (95% CI, 0.53–1.39) ([Hardell et al., 2002b](#)).

Among cases ascertained in 2000–2003, there were 359 malignant tumours of the brain, including 248 cases of astrocytoma and 69 other malignant tumours. Ever-use of analogue phones yielded an odds ratio of 2.6 (95% CI, 1.5–4.3), with 3.1 (95% CI, 1.6–6.2) for ipsilateral use and 2.6 (95% CI, 1.3–5.4) for contralateral use; and, ever-use of digital phones yielded an odds ratio of 1.9 (95% CI, 1.3–2.7) with 2.6 (95% CI, 1.6–4.1) for ipsilateral use and 1.3 (95% CI, 0.8–2.2) for contralateral use. Estimates of an association tended to be larger for use beginning > 10 years before diagnosis ([Hardell et al., 2006c](#)).

(ii) Meningioma

See [Table 2.14](#)

In the case–control study of [Inskip et al. \(2001\)](#) mentioned above, interviews were conducted with a total of 197 cases of meningioma and 799 controls. Compared with non-users, self-reported regular users of mobile phones did not manifest excess risks of meningioma (OR, 0.8; 95% CI, 0.4–1.3).

The Finnish case–control study mentioned above ([Auvinen et al., 2002](#)) included 129 cases of meningioma. The odds ratio for ever-use was 1.1 (95% CI, 0.5–2.4), with a slightly higher odds ratio for use of analogue phones (OR, 1.5; 95% CI, 0.6–3.5). [This study was limited by the short

time since first use of a mobile phone for most people and by the uncertain mobile-phone use ascertainment from subscription information.]

In the pooled INTERPHONE analysis, 2409 cases of meningioma and 2662 controls were included ([INTERPHONE Study Group, 2010](#)). Participation rates were 78% for cases of meningioma and 53% for controls. For regular users, a reduced odds ratio was seen for cases of meningioma (OR, 0.79; 95% CI, 0.68–0.91) (see [Table 2.14](#)). Odds ratios of < 1.0 were also seen for all categories of time since start of use and for cumulative calls. Study participants who first used a mobile phone at least 10 years before interview did not show an increased risk of meningioma. Regarding cumulative number of calls, the group with highest exposure did not show an increased risk of glioma or meningioma. In terms of cumulative call time, all odds ratios were < 1.0 for all deciles of exposure except the highest (10th) decile of recalled cumulative call time (≥ 1640 hours). For this exposure group, the odds ratio for meningioma was 1.15 (95% CI, 0.81–1.62). Increased risk in the highest exposure decile of cumulative call time was more pronounced in short-term users, who started to use phones 1–4 years before the reference date, than in long-term users (≥ 10 years). Sensitivity analyses had little effect on estimated associations between mobile-phone use and risk of meningioma.

The analysis of TCSE and risk of meningioma in five INTERPHONE countries ([Cardis et al., 2011](#)) was based on 674 cases of meningioma and 1796 controls. In the highest quintile of TCSE, the odds ratio for meningioma was 0.90 (95% CI, 0.66–1.24). An odds ratio of 1.01 (95% CI, 0.75–1.36) was reported for the highest quintile of cumulative call time without hands-free devices. In terms of TCSE exposure ≥ 7 years before the reference date, there was no consistent dose–response pattern, but the odds ratio was elevated in the quintile of highest exposure (OR, 2.01; 95% CI, 1.03–3.93). In case-only analyses, the

Table 2.14 Case-control studies of meningioma and use of mobile phones

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell et al. (1999) Sweden, 1994–96	46	439	Population, matched on sex, age, region, and year of diagnosis	Self-administered standardized questionnaire	Meningioma	Never use of mobile phone Ever use	30 16	1.0 1.0 (0.5–2.3)		
Inskip et al. (2001) Phoenix, Boston, Pittsburgh, 1994–98.	197	799	Patients admitted to the same hospitals for a variety of non-malignant conditions.	Computer-assisted, personal interview in the hospital	Meningioma	Regular use Duration ≥ 5 yr Cumulative use (<i>h</i>) Never or rarely used < 13 13–100 > 100 > 500	32 6 165 8 13 11 6	0.8 (0.4–1.3) 0.9 (0.3–2.7) 1.0 0.7 (0.3–1.9) 1.1 (0.5–2.4) 0.7 (0.3–1.7) 0.7 (0.2–2.4)	Hospital, age, sex, race or ethnic group, proximity of residence to the hospital	There are results for other exposure metrics: average daily use, duration, year use began. Also results for laterality.
Auvainen et al. (2002) Finland, 1996	398 (129 meningiomas)	1990	Population Registry Centre of Finland	Information on subscriptions obtained from the two mobile-network providers operating in Finland in 1996	Meningioma (225.2)	Analogue Ever < 1 yr 1–2 yr > 2 yr Digital Ever	8 3 3 2 3	1.5 (0.6–3.5) 2.3 (0.6–9.2) 1.6 (0.4–6.1) 1.0 (0.2–4.4) 0.7 (0.2–2.6)	Age, sex	Cases aged 20–69 yr

Table 2.14 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell <i>et al.</i> (2006a) Sweden, 1997–2003	916	2162	Population.	Self-administered questionnaire	Meningioma	Never used mobile or cordless phone	455	1.0	Age, sex, SEI, year of diagnosis	Ipsilateral use of analogue and digital phones was associated with meningioma (analogue: OR, 1.3; 95% CI, 0.9–2.0; digital: OR, 1.4; 95% CI, 1.0–1.8), contralateral use was not (OR, 1.2; 95% CI, 0.7–1.8; and OR, 1.1; 95% CI, 0.8–1.5, respectively).
						<i>Cumulative use, analogue (h)</i>				
						1–500	99	1.3 (1.0–1.7)		
						501–1000	8	1.1 (0.5–2.6)		
						> 1000	6	1.4 (0.5–3.8)		
						<i>Cumulative use, digital (h)</i>				
						1–500	268	1.1 (0.9–1.3)		
						501–1000	18	1.0 (0.6–1.8)		
						> 1000	9	0.7 (0.3–1.4)		
						<i>Latency, analogue (yr)</i>				
						> 1–5	32	1.2 (0.8–1.8)		
						> 5–10	47	1.2 (0.8–1.8)		
						> 10	34	1.6 (1.0–2.5)		

Table 2.14 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
INTERPHONE Study Group (2010)	2409	2662	Population (except United Kingdom: GP patients)	Interviewer-administered standardized questionnaire	Meningioma (D32.0, D32.9, D42.0, D42.9, C70.0, C70.9)	Never regular use of mobile phone	1147	1.00	Sex, age, study centre, ethnicity (in Israel), and education	
Australia, Canada, Denmark, Finland, France, Germany, Israel, Italy, Japan, New Zealand, Norway, Sweden, United Kingdom, 2000–04						Ever use	1262	0.79 (0.68–0.91)		
						<i>Time since start of use (yr)</i>				
						1–1.9	178	0.90 (0.68–1.18)		
						2–4	557	0.77 (0.65–0.92)		
						5–9	417	0.76 (0.63–0.93)		
						≥ 10	110	0.83 (0.61–1.14)		
						<i>Cumulative call time with no hands-free devices (h)</i>				OR, 4.80 (95% CI, 1.49–15.4) in short-term users (start of mobile-phone use 1–4 yr before reference date) with cumulative call time ≥ 1640 h (based on 22 cases)
						< 5	160	0.90 (0.69–1.18)		
						5–12.9	142	0.82 (0.61–1.10)		
						13–30.9	144	0.69 (0.52–0.91)		
						31–60.9	122	0.69 (0.51–0.94)		
						61–114.9	129	0.75 (0.55–1.00)		
						115–199.9	96	0.69 (0.50–0.96)		
						200–359.9	108	0.71 (0.51–0.98)		
						360–734.9	123	0.90 (0.66–1.23)		
						735–1639.9	108	0.76 (0.54–1.08)		
						≥ 1 640	130	1.15 (0.81–1.62)		

Table 2.14 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Cardis <i>et al.</i> (2011) Australia, Canada, France, Israel, Italy, New Zealand, 2000–04	674	1796	Population	Interviewer-administered standardized questionnaire	Meningioma	RF TCSE (J/kg)			Sex, age, study centre, ethnicity (in Israel) and education	Interpretation of OR is most meaningful when compared with the corresponding OR for comparable exposure surrogates of mobile-phone use
						Never regular user	294	1.0		Subjects with tumour centre estimated by a neuro-radiologist or by means of a computer algorithm
						< 76.7	103	0.90 (0.67–1.21)		
						76.7–284.1–978.9–3123.9+	71 56 62 88	0.74 (0.53–1.04) 0.56 (0.39–0.80) 0.72 (0.51–1.02) 0.90 (0.66 to 1.24)		Exposure ≥ 7 yr before reference date: OR, 2.01 (95% CI, 1.03–3.93), for highest quintile

h, hour; OR, odds ratio; RF, radiofrequency radiation; SEI, socioeconomic index; TCSE, total cumulative specific energy; yr, year

odds ratio for having the centre of the tumour within the most exposed area was 1.34 (95% CI, 0.55–3.25) in those who reported starting to use a mobile phone ≥ 10 years previously.

[Hardell *et al.* \(2006a\)](#) reported the results of a pooled analysis of case-control studies of benign tumours of the brain and use of mobile and cordless phones that included 1254 cases of benign tumours, of which 916 were meningioma; deceased cases (and controls) were not included in this analysis. An odds ratio of 1.3 (95% CI, 0.99–1.7) was reported for meningioma when users of analogue mobile phones were compared with people who reported no use of mobile or cordless phones, or exposure ≤ 1 year before the reference date. The odds ratio was 1.1 (95% CI, 0.9–1.3) for users of digital mobile phones and 1.1 (95% CI, 0.9–1.4) for users of cordless phones. Study participants who first used an analogue, digital, or cordless phone at least 10 years previously showed increased risks of meningioma, although estimates were imprecise (OR, 1.6; 95% CI, 1.0–2.5; OR, 1.3; 95% CI, 0.5–3.2; OR, 1.6; 95% CI, 0.9–2.8, respectively).

(iii) Acoustic neuroma

See [Table 2.15](#)

[Inskip *et al.* \(2001\)](#) included a total of 96 cases with acoustic neuroma and 799 controls. Compared with non-users, self-reported regular users of mobile phones did not manifest excess risks of acoustic neuroma (OR, 1.0; 95% CI, 0.5–1.9).

A case-control study of 90 cases of acoustic neuroma and 86 controls selected from among other patients was conducted in a hospital in New York ([Muscat *et al.*, 2002](#)). Subjects were interviewed regarding use of mobile phones and other factors. Analysis of reported histories of mobile-phone use, adjusting for sociodemographic factors and date of interview, yielded a set of odds-ratio estimates that were close to the null value for cumulative hours of use and years of use. [The Working Group noted that numbers

were small, exposure levels were low, and time since first use was short.]

[Schoemaker *et al.* \(2005\)](#) reported pooled results on acoustic neuroma from a subset of the INTERPHONE countries (the five north European countries: Denmark, Finland, Norway, Sweden, and the United Kingdom). There was no indication of an increased risk of acoustic neuroma associated with mobile-phone use ([Table 2.15](#)). Similar negative findings were reported by the INTERPHONE groups in France ([Hours *et al.*, 2007](#)) and Germany ([Schlehofer *et al.*, 2007](#)), and from a case-control study in Japan ([Takebayashi *et al.*, 2006](#)).

In Japan, [Sato *et al.* \(2011\)](#) identified a series of cases of acoustic neuroma diagnosed between 2000 and 2006 in 22 participating hospitals with neurosurgery departments (32% of hospitals solicited). Of 1589 cases identified, 816 agreed to respond to a self-administered questionnaire, received by post, focusing on history of mobile-phone use and history of pre-diagnosis symptoms. Two case series were constituted consisting of: (a) 180 cases among mobile-phone users whose symptoms had not appeared 1 year before diagnosis; and (b) 150 cases among mobile-phone users whose symptoms had not yet appeared 5 years before diagnosis. In each series, the investigators then compared laterality of the tumour with laterality of mobile-phone use and, using a formula described by [Inskip *et al.* \(2001\)](#), they derived an estimate of relative risk of acoustic neuroma related to various metrics of mobile-phone use. Overall, there was no excess risk of acoustic neuroma among ever-users of mobile phones. However, among some subgroups, namely those with the highest duration of daily calls, there were estimates of high risk ratios in the range of 2.74 (95% CI, 1.18–7.85) to 3.08 (95% CI, 1.47–7.41). This excess appeared to be restricted to a small group of cases who were persistently among the highest users during the past 5 years. The authors considered various alternative explanations for this finding,

Table 2.15 Case-control studies of acoustic neuroma and use of mobile phones

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Inskip et al. (2001) USA, 1994-98	96	799	Patients admitted to the same hospitals for a variety of non-malignant conditions	Computer-assisted personal interview in the hospital	Acoustic neuroma	Regular use Duration ≥ 5 yr	22 5	1.0 (0.5-1.9) 1.9 (0.6-5.9)	Hospital, age, sex, race or ethnic group, proximity of residence to the hospital	Analyses by cumulative use showed no associations. Analyses of laterality of tumour by laterality of phone use showed no associations. Very few subjects with long-term exposure. Response rates were 92% for cases and 86% for controls. In groups with highest duration of daily calls: RR ranged from 2.74 (95% CI, 1.18-7.85) to 3.08 (95% CI, 1.47-7.41)
Muscat et al. (2002) New York City, 1997-99	90	86	In-patients with non-malignant conditions from the same hospitals	Interviews with structured questionnaire	Acoustic neuroma (225.1)	Cumulative use (h): 0 1-60 > 60 Years of use: 0 1-2 3-6	72 9 9 72 7 11	1.0 0.9 (0.3-3.1) 0.7 (0.2-2.6) 1.0 0.5 (0.2-1.3) 1.7 (0.5-5.1)	Age, education, sex, study centre, occupation categories, and date of interview	Also presented as h/mo, with similar results. In mobile-phone users tumour was most often on contralateral side.

Table 2.15 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Schoemaker et al. (2005) Denmark, Finland, Norway, Sweden, United Kingdom, 1999–2004	678	3553	Population (except United Kingdom: GP patients)	Interviewer-administered standardized questionnaire	Acoustic neuroma (D33.3)	Never regular use of mobile phone Regular use	316 360	1.0 0.9 (0.7–1.1)	Educational level and combinations of interview year and interview lag time	Matched for centre, region, 5-yr age group, sex
						<i>Time since start of use (yr)</i> 1.5–4 5–9 ≥ 10 <i>P</i> for trend	174 139 47	0.8 (0.7–1.0) 0.9 (0.7–1.2) 1.0 (0.7–1.5) 0.9		
						<i>Cumulative use (h)</i> < 116 116–534 > 534 <i>P</i> for trend	168 89 94	0.9 (0.7–1.1) 0.9 (0.7–1.2) 0.9 (0.7–1.2) 0.5		
Takebayashi et al. (2006) Japan, 2000–04	101	339	Population (random-digit dialling)	Interviewer-administered standardized questionnaire	Acoustic neuroma (D33.3)	Never regular use of mobile phone Regular use	46 51	1.0 0.73 (0.43–1.23)	Education, marital status	Matched for age, sex, residency
						<i>Time since start of use (yr)</i> < 4 4–7 ≥ 8 <i>P</i> for trend	26 21 4	0.70 (0.39–1.27) 0.76 (0.38–1.53) 0.79 (0.24–2.65) 0.70		
						<i>Cumulative use (h)</i> < 300 300–900 > 900 <i>P</i> for trend	35 9 7	0.67 (0.38–1.17) 1.37 (0.54–3.50) 0.67 (0.25–1.83) 0.69		

Table 2.15 (continued)

Reference, study location and period	Total cases	Total controls (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Ha et al. (2007) France, 2001–03	109	214	Interviewer-administered standardized questionnaire	Acoustic neuroma (D33.3)	Never regular use of mobile phone Regular use	51 58	1.0 0.92 (0.53–1.59)	SES, tobacco consumption, noise exposure	Sex, age (± 5 yr), place of residence
			<i>Duration of use (mo)</i>		< 16 16–27 27–46 > 46	19 17 8 14	1.21 (0.55–2.69) 1.33 (0.58–3.03) 0.63 (0.26–1.53) 0.66 (0.28–1.57)	OR per 1 year, 0.96 (0.84–1.10)	
			<i>Cumulative use (h)</i>		< 20 20–80 80–260 > 260	14 15 13 16	1.06 (0.48–2.36) 0.87 (0.40–1.91) 0.85 (0.38–1.88) 0.92 (0.41–2.07)	OR per 80 h, 1.0 (0.96–1.03)	
Schlehofer et al. (2007) Germany, 1976–88	97	194	Interviewer-administered standardized questionnaire	Acoustic neuroma (D33.3)	Never regular use of mobile phone Regular use	68 29	1.0 0.67 (0.38–1.19)	SES, urbanity	Matched for centre, age, sex
			<i>Time since start of use (yr)</i>		1–4 5–9 ≥ 10	20 8 0	0.78 (0.40–1.50) 0.53 (0.22–1.27) -		
			<i>Cumulative use (h)</i>		< 44 44–195 > 195	16 7 5	1.04 (0.51–2.16) 0.58 (0.22–1.48) 0.35 (0.12–1.01)		

Table 2.15 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Sato <i>et al.</i> (2011) Japan, 2000–06	787	787 (case–case)	Note: the affected ear is the case side; the opposite ear is regarded as the control.	Mailed questionnaire about history of mobile-phone use	Acoustic neuroma	Overall, for regular mobile-phone use until one yr before diagnosis	180	1.08 (0.93–1.28)	Same patients	The authors interpret these significant results with caution, mentioning detection and recall bias as possibilities.
						Overall, for regular mobile-phone use until 5 yr before diagnosis	150	1.14 (0.96–1.40)		
						Weighted daily average call duration > 20 min, 1 yr before diagnosis	23	2.74 (1.18–7.85)		
						Weighted daily average call duration > 20 min, 5 yr before diagnosis	33	3.08 (1.47–7.41)		

Table 2.15 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
INTERPHONE Study Group (2011)	1105	2145	Population (except United Kingdom; GP patients)	Interviewer-administered standardized questionnaire	Schwannoma of the acoustic nerve (ICD-9 code 225.1 or ICD-10 code D33.3, and ICD-O topography code C72.4 and morphology code 9560/0)	Never regular use of mobile phone	801	1.00	Sex, age, study centre, ethnicity (in Israel), and education	Data are given only for exposure up to 5 yr before reference date (risk estimates were generally smaller when exposure up to 1 yr before reference date was considered)
Australia,						Regular use	304	0.95 (0.77–1.17)		
Canada,						<i>Time since start of use (yr)</i>				
Denmark,						5–9	236	0.99 (0.78–1.24)		
Finland,						≥ 10	68	0.83 (0.58–1.19)		
France,						<i>Cumulative call time (h) with no hands-free devices</i>				
Germany,						< 5	42	1.07 (0.69–1.68)		
Israel, Italy,						5–12.9	30	1.06 (0.60–1.87)		
Japan, New Zealand,						13–30.9	40	1.32 (0.80–2.19)		
Norway,						31–60.9	36	0.86 (0.52–1.41)		
Sweden, United Kingdom,						61–114.9	21	0.63 (0.35–1.13)		
2000–04						115–199.9	22	0.71 (0.39–1.29)		
						200–359.9	49	0.83 (0.48–1.46)		
						360–734.9	26	0.74 (0.42–1.28)		
						735–1639.9	22	0.60 (0.34–1.06)		
						≥ 1640	32	2.79 (1.51–5.16)		

Table 2.15 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell <i>et al.</i> (2006a) Sweden, 2000–03	243		Population	Self-administered standardized questionnaire	Acoustic neuroma	Never use of mobile or cordless phone	88	1.0	Age, sex, SEI, and year of diagnosis	
						Ever use of analogue phone	68	2.9 (2.0–4.3)		
						Ever use of digital phone	105	1.5 (1.1–2.1)		
						Ever use of cordless phone	96	1.5 (1.0–2.0)		
						<i>Cumulative call time, analogue (h)</i>				
						1–500	55	2.8 (1.8–4.2)		
						501–1000	7	3.3 (1.3–8.0)		
						> 1000	6	5.1 (1.9–14)		
						<i>Cumulative call time, digital (h)</i>				
						1–500	83	1.4 (1.0–2.0)		Users of analogue phone (> 10 yr) showed OR, 3.1 (95% CI, 1.7–5.7)
						501–1000	10	1.8 (0.8–3.8)		
						> 1000	12	3.1 (1.5–6.4)		
						<i>Cumulative call time, cordless (h)</i>				
						1–500	60	1.3 (0.9–1.9)		
						501–1000	15	1.6 (0.9–3.0)		
						> 1000	21	2.1 (1.2–3.7)		

GP, general practitioner; h, hour; min, minute; mo, month; OR, odds ratio; SEI, socioeconomic index; SES, socioeconomic status; yr, year

including selection bias and recall bias, and they concluded that it was unclear whether the finding was a consequence of bias.

The pooled INTERPHONE analysis for acoustic neuroma ([INTERPHONE Study Group, 2011](#)) followed in general the same methodology as the analyses for glioma and meningioma described above ([INTERPHONE Study Group, 2010](#)). Patients diagnosed with a schwannoma of the acoustic nerve in the study regions during study periods of 2–4 years between 2000 and 2004 were included in the study. For each case, two age-, sex- and study-region-matched controls were recruited. Controls were either specifically sampled for the cases of acoustic neuroma, taken from the pool of INTERPHONE controls drawn for all tumours together, or obtained with a combination of both approaches. In total, 1105 cases (participation rate, 82%) were included in the analyses, together with 2145 controls (participation rate, 53%). The odds ratio for regular use was 0.85 (95% CI, 0.69–1.04) when recording exposure at 1 year before the reference date and 0.95 (95% CI, 0.77–1.17) when recording exposure at 5 years before the reference date. For cumulative call time, the highest odds ratios were observed in the highest category of use: the odds ratios for ≥ 1640 hours were 1.32 (95% CI, 0.88–1.97) when recording exposure at 1 year and 2.79 (95% CI, 1.51–5.16) when recording exposure at 5 years. There was, however, no consistent trend in the exposure–response relationship in the first nine deciles of exposure. Stratifying the analyses according to time since start of mobile-phone use resulted in an increased odds ratio for heavy users of mobile phones only in long-term users (OR, 1.93; 95% CI, 1.10–3.38, based on 37 cases). This risk estimate was more pronounced with respect to ipsilateral use (OR, 3.74; 95% CI, 1.58–8.83, based on 28 cases) and decreased with respect to contralateral use (OR, 0.48; 95% CI, 0.12–1.94, based on 4 cases). Exclusion of participants with an implausible amount of use (> 5 hours per day) resulted in a decrease in odds ratio for exposure

up to 1 year before the reference date, but had little impact on the results of the analyses of exposure up to 5 years before the reference date. The results for cumulative number of calls were broadly similar, but risk estimates were smaller.

Overall, these results were broadly similar to the results for glioma from the INTERPHONE study. [The same methodological limitations were of concern, mainly selection and recall bias. Diagnostic bias was also of concern: patients with acoustic neuroma who use mobile phones may be diagnosed earlier than non-users, since acoustic neuroma affects hearing capability. However, such an effect would be expected to be most relevant for recent users, but of little relevance for exposure 5 years before diagnosis. On the other hand, prodromal symptoms might discourage cases from becoming mobile-phone users. Again, such an effect would be most relevant in the analysis of most recent use of mobile phones, but not in the analysis of exposure at earlier dates. There is also uncertainty as to how early symptoms may affect the preferred side of use. Regarding confounding, socioeconomic status, ionizing radiation and loud noise were considered, with little effect on the results.]

[Hardell et al. \(2006a\)](#) reported the results of a pooled analysis of associations between use of mobile and cordless phones and risk of benign tumours of the brain that included 243 cases of acoustic neuroma. An increased odds ratio was reported for acoustic neuroma (OR, 2.9; 95% CI, 2.0–4.3) when users of analogue mobile phones were compared with people who reported no use of mobile or cordless phones, or exposure ≤ 1 year before the reference date. The odds ratio was 1.5 (95% CI, 1.1–2.1) for users of digital mobile phones and 1.5 (95% CI, 1.04–2.0) for users of cordless phones. Study participants who first used an analogue phone at least 10 years before the reference date showed increased risks (OR, 3.1; 95% CI, 1.7–5.7), but users of digital or cordless phones did not. For users of analogue mobile phones, an increased odds ratio was

seen for ipsilateral use (OR, 3.0; 95% CI, 1.9–5.0) and contralateral use (OR, 2.4; 95% CI, 1.4–4.2) when compared with people who had not used mobile or cordless phones. For users of digital mobile phones, an increased odds ratio was seen for acoustic neuroma with ipsilateral use (OR, 1.7; 95% CI, 1.1–2.6), but not for contralateral use (OR, 1.3; 95% CI, 0.8–2.0) when compared with people who had not used mobile or cordless phones. Similar associations were found for use of cordless phones (ipsilateral use: OR, 1.7; 95% CI, 1.1–2.6; and contralateral use: OR, 1.1; 95% CI, 0.7–1.7, respectively) ([Schüz et al., 2006c](#)).

(iv) *All cancers of the brain combined*

See [Table 2.16](#)

In several studies already referred to above, analyses were presented for all cancers of the brain combined ([Hardell et al., 2000, 2001, 2011a](#); [Inskip et al., 2001](#); [Auvinen et al., 2002](#)). Only in [Hardell et al. \(2011a\)](#) were risks of cancer significantly elevated with prolonged use of mobile phones. A study in France by [Spinelli et al. \(2010\)](#) found no significant excess risks.

(v) *Other cancers of the brain*

A pooled analysis by [Hardell et al. \(2011a\)](#) included 103 cases with a histopathological diagnosis of malignant tumour of the brain other than glioma. Odds ratios for malignant tumours other than glioma by category of duration of mobile-phone use were 1.0 (95% CI, 0.6–1.6) for 1–1000 hours, 1.4 (95% CI, 0.4–4.8) for 1001–2000 hours, and 1.2 (95% CI, 0.3–4.4) for > 2000 hours.

(vi) *Pituitary tumours*

See [Table 2.17](#)

In a Japanese study, 102 cases of pituitary adenoma were included, together with 161 individually matched controls ([Takebayashi et al., 2008](#)). Neither regular use of mobile phones (OR, 0.90; 95% CI, 0.50–1.61) nor cumulative duration of use in years and cumulative call time in hours was associated with an increased risk of pituitary tumours.

In a population-based case–control study from south-eastern England, 291 cases of pituitary tumour diagnosed between 2001 and 2005 were included, together with 630 controls that were frequency-matched for sex, age, and health-authority of residence ([Schoemaker & Swerdlow, 2009](#)). The participation rate was 63% for cases and 43% for controls. Data were collected with a face-to-face interview at the subject's home or another convenient place. Regular use was not associated with an increased risk (OR, 0.9; 95% CI, 0.7–1.3) nor was any other exposure surrogate. Stratified analyses for analogue or digital mobile-phone user did not indicate consistent exposure–response associations.

(d) *Some reviews, meta-analyses, and other studies*

Various meta-analyses and other comparisons of the accumulating data on mobile-phone use and tumours of the brain have been published ([Hardell et al., 2003, 2007a, 2008](#); [Lahkola et al., 2006](#); [Kan et al., 2008](#); [Ahlbom et al., 2009](#); [Hardell & Carlberg, 2009](#); [Khurana et al., 2009](#); [Myung et al., 2009](#)). Such analyses are potentially useful for characterizing the accumulating evidence and for exploring heterogeneity of findings among studies, along with determinants of any observed heterogeneity. [The Working Group based its conclusions on review of the primary studies.]

2.3.2 Leukaemia and lymphoma

(a) *Leukaemia*

There have been four epidemiological studies on leukaemia and use of mobile phones.

In an early cohort study of 285 561 users of analogue phones, identified based on records from two mobile-phone providers in the USA in 1993, mortality attributable to leukaemia was not elevated among users of hand-held phones relative to users of non-hand-held phones (mostly car phones) ([Dreyer et al., 1999](#); [Table 2.18](#)). [A

Table 2.16 Case-control studies of all cancers of the brain and use of mobile phones

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell <i>et al.</i> (2000, 2001) Uppsala-Orebro region and Stockholm region, Sweden, 1994-96	209 cases of brain tumours diagnosed 1994-96	425	Population register: 1:2 case:control ratio with matching on age and sex, and drawn from the same geographical areas as the cases	Self-administered structured, mailed questionnaire	All malignant tumours of the brain. Benign tumours of the brain included from Stockholm in 1996, as part of feasibility study.	No use of mobile or cordless phone, or exposure ≤ 1 yr before reference date	78	1 0.98 (0.69-1.41)	Sex, age (as a continuous variable). Radiotherapy, diagnostic X-ray, asbestos, solvents, smoking	Participation rate was 90% for cases and 91% for controls. Increased risk for tumour in the temporal or occipital lobe on same side as cell-phone use (OR, 2.62; 95% CI, 1.02-6.71). Contralateral use did not increase the risk (OR, 0.97; 95% CI, 0.36-2.59). Deceased cases were not included. This analysis encompassed the case-control data included in Hardell <i>et al.</i> (2000)
Inskip <i>et al.</i> (2001) USA, 1994-98	782	799	Patients admitted to the same hospitals for a variety of non-malignant conditions.	Computer-assisted in person interview in the hospital, history of mobile-phone use	All brain	No use Regular use Duration ≥ 5 yr	471 139 22	1.0 0.8 (0.6-1.1) 0.9 (0.5-1.6)	Hospital, age, sex, race or ethnic group, proximity of residence to the hospital	Analyses by cumulative use showed no associations. Very few subjects with long-term exposure. Response rates 92% for cases and 86% for controls.

Table 2.16 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Auvinen et al. (2002) Finland, 1996	398	1990	Population Registry Centre of Finland	Information on subscriptions obtained from the two mobile-network providers operating in Finland in 1996	All brain (191 and 225.2)	<i>Analogue</i> Ever < 1 yr 1–2 yr > 2 yr <i>Digital</i> Ever < 1 yr 1–2 yr > 2 yr	40 8 15 17 16 4 11 1	 1.6 (1.1–2.3) 1.6 (0.7–3.6) 1.5 (0.9–2.8) 1.6 (0.9–2.8) 0.9 (0.5–1.5) 0.6 (0.2–1.6) 1.2 (0.6–2.3) 0.6 (0.1–4.5)	Age, sex	Cases aged 20–69 yr
Spinelli et al. (2010) France, 2005	122	122	In-patients from neurosurgery departments of the same hospitals; unrelated to cancer	Face-to-face interviews with standardized questionnaire; and self-administered questionnaire	Malignant primary tumours of the brain	<i>Global cellular-phone use (hours-year)</i> 0 ≤ 4 4–36 ≥ 36	37 8 58 13	 1 0.86 (0.30–2.44) 1.45 (0.75–2.80) 1.07 (0.41–2.82)	Age, sex	

Table 2.16 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell <i>et al.</i> (2011a)	1251 cases of malignant brain tumours diagnosed during 1997–2003 among people aged 20–80 yr at diagnosis	2438 controls	Population register. 1:1 case:control ratio with matching on age and sex, and drawn from the same region as the cases. For deceased cases, controls drawn from death registry. 1:1 matching on year of death, sex, age, and medical region	Self-administered structured, mailed questionnaire. For deceased cases and controls, mailed questionnaire was completed by relative of decedent.	All malignant tumours of the brain	No use of mobile or cordless phone, or exposure ≤ 1 yr before reference date Mobile-phone use: Ever 1–1000 h 1001–2000 h > 2000 h	677 574 466 47 61	1.00 1.3 (1.1–1.5) 1.2 (1.0–1.4) 1.8 (1.1–2.7) 3.0 (1.9–4.8)	Sex, age (as a continuous variable), SEI code, year of diagnosis	Participation rates were 85% for cases and 84% for controls. This analysis encompassed the data presented in earlier papers on pooled case–control studies of malignant tumours of the brain among living cases diagnosed in 1997–2003
					Malignant tumours of the brain other than glioma ($n = 103$)	Mobile-phone use: 1–1000 h 1001–2000 h > 2000 h	39 3 3	1.0 (0.6–1.6) 1.4 (0.4–4.8) 1.2 (0.3–4.4)		

h, hour or hours; SEI, socioeconomic index; yr, year

Table 2.17 Case-control studies of cancers of the pituitary and use of mobile phones

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments	
Takebayashi et al. (2008) Japan, 2000–04	101	161	Population (random-digit dialling)	Interviewer-administered standardized questionnaire	Pituitary adenoma (ICD code not reported)	Never regular use of mobile phone	39	1.0	Education, marital status	Matched for age (5 yr), sex, residency	
						Regular use	62	0.90 (0.50–1.61)			
						<i>Time since start of use (yr)</i>					
						< 2.2	14				0.86 (0.39–1.88)
						2.2–4.69	13				0.75 (0.31–1.81)
						4.7–6.5	22				1.64 (0.74–3.66)
						> 6.5	13				0.75 (0.31–1.82)
						<i>P</i> for trend					0.89
						<i>Cumulative use (h)</i>					
						< 39	15				1.00 (0.46–2.16)
39–190	14	0.97 (0.40–2.32)									
190–560	12	0.72 (0.31–1.70)									
> 560	21	1.33 (0.58–3.09)									
Schoemaker & Sverdlow (2009) United Kingdom, 2001–05	291	630	Population (from GP patient list)	Interviewer-administered standardized questionnaire	Pituitary tumour (C75.1, D35.2, D44.3)	Never regular use of mobile phone	116	1.0	Age, sex, category, geographic area, reference date, and Townsend deprivation score		
						Regular use	175	0.9 (0.7–1.3)			
						<i>Time since start of use (yr)</i>					
						1.5–4	89				1.0 (0.7–15)
						5–9	62				0.8 (0.5–1.2)
						10–17	24				1.0 (0.5–1.9)
						<i>P</i> for trend					0.7
						<i>Cumulative use (h)</i>					
						< 113	79				0.9 (0.6–1.3)
						113–596	44				1.1 (0.7–1.8)
> 596	51	1.1 (0.7–1.7)									
<i>P</i> for trend		0.9									

GP, general practitioner; h, hour or hours; yr, year

limitation of this study was that there were only four deaths due to leukaemia among users of hand-held phones, as the study was truncated – with no access to mortality data beyond 1 year – as a result of a legal proceeding.]

A study of cancer incidence in a cohort of 420 095 users of mobile phones in Denmark found no evidence of an elevated risk of leukaemia in males or females (SIR, 1.05; 95% CI, 0.96–1.15) (Schüz *et al.*, 2006c; Table 2.18). The incidence of leukaemia was not increased in any of the reported time intervals since first subscription. Details concerning the design of the study were discussed above (Section 2.3.1). [The results for leukaemia were not reported separately by subtype.]

A hospital-based case–control study of adult-onset leukaemia in Thailand conducted between 1997 and 2003 (180 cases, 756 hospital controls) reported an odds ratio for all leukaemias combined of 1.5 (95% CI, 1.0–2.4) (Kaufman *et al.*, 2009; Table 2.19). Overall, the duration of mobile-phone use was short (median, 24–26 months). The results were similar for acute myeloid leukaemia, chronic myeloid leukaemia and chronic lymphocytic leukaemia. There were no trends in associations of all leukaemias with duration of ownership, lifetime hours of use, or amount of use per year. The odds ratio was highest for persons reporting exclusive use of GSM (Global System for Mobile Communications) services. Using an categorization ad hoc into “high risk” and “low risk” groups of mobile-phone users based on phone characteristics, the authors reported an odds ratio of 1.8 for high-risk versus low-risk users (95% CI, 1.1–3.2). [It was unclear to the Working Group as to how the “high risk” and “low risk” groups were derived and whether it was done *a priori* or *a posteriori*.]

In a study conducted in the United Kingdom between 2003 and 2009, which included 806 cases and 585 controls who were non-blood relatives, regular use of a mobile phone (defined as at least one call per week for at least 6 months) was

not associated with the incidence of leukaemia (Cooke *et al.*, 2010; Table 2.19). Risk was not significantly associated with years since first use, lifetime years of use, cumulative number of calls, or cumulative hours of use. Among people who reported using a phone for ≥ 15 years since first use, the odds ratio was 1.87 (95% CI, 0.96–3.63; 50 exposed cases); however, there was no apparent trend with years since first use. There also was no apparent trend in risk with cumulative hours of use. Findings were similar for digital and analogue phones. There was no apparent variation in results by subtype of leukaemia and no trend in risk with years since first use, years of use, or cumulative hours of use for any subtype. [Only 50% of potential cases participated, with the usual reasons for non-participation being death or disability related to leukaemia.]

(b) Lymphoma

In a population-based case–control study conducted in Sweden between 1999 and 2002 (910 cases, 1016 controls), neither mobile-phone use nor cordless-phone use was significantly associated with risk of NHL overall, nor for the B-cell subtype in particular (90% of the cases) (Hardell *et al.*, 2005; Table 2.19). High odds ratios were reported for some categories of use of cordless phones for T-cell lymphomas, based on very small numbers. Cases in this study were diagnosed between the ages of 18 and 74 years. Males and females were included, but the main results concerning mobile-phone use were presented for both sexes combined.

A population-based case–control study of NHL conducted in the USA between 1998 and 2000 (551 cases, 462 controls) also reported predominantly null findings (Linnet *et al.*, 2006; Table 2.19). Several exposure metrics of mobile-phone use were presented (latency, duration, amount of exposure), but overall there was no consistent trend in risk. Risk of NHL was not associated with minutes per week of use of mobile telephones, duration of use, cumulative

Table 2.18 Cohort studies of leukaemia, lymphoma, and other cancers, and use of mobile phones

Reference, study location and period	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Dreyer et al. (1999) USA, 1993	285 561	1993	Records of mobile-phone service providers	Leukaemia (204–207)	Hand-held phones < 2 min/d ≥ 2 min/d	2 2	SMR 1.6 4.9	Age, sex, metropolitan area	Mortality study; effect estimate = SMR; SMR for non-hand-held phones (non-exposed), 7.0
Schüz et al. (2006c) Denmark, 1982–2002	420 095	1982–2002	Records of mobile-phone service providers	Leukaemia (204–207)	Latency (yr) < 1 1–4 5–9 ≥ 10	33 151 135 32	SIR 1.09 (0.75–1.52) 1.05 (0.90–1.24) 0.92 (0.77–1.08) 1.08 (0.74–1.52)	Age, sex, calendar period of observation	Incidence study; cannot be certain who the user was; phones used under business accounts not included; details of phone use not available
Schüz et al. (2006c) Denmark, 1982–2002	65 542	1982–2002	Records of cellular service providers	Female breast (174)	Subscriber	711	1.04 (0.97–1.12)	Age, calendar year of observation	Incidence study; cannot be certain who the user was; phones used under business accounts not included; details of phone use not available
Schüz et al. (2006c) Denmark, 1982–2002	420 095	1982–2002	Records of cellular service providers	Eye (190)	Subscriber	38 (males) 6 (females)	0.94 (0.66–1.29) 1.10 (0.40–2.39)	Age, calendar year of observation	Incidence study; cannot be certain who the user was; phones used under business accounts not included; details of phone use not available

Table 2.18 (continued)

Reference, study location and period	Total No. of subjects	Follow-up period	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	Relative risk (95% CI)	Covariates	Comments
Schüz <i>et al.</i> (2006c) Denmark, 1982–2002	357 553	1982–2002	Records of cellular service providers	Testis (186)	Subscriber	522	1.05 (0.96–1.15)	Age, calendar year of observation	Incidence study; cannot be certain who the user was; phones used under business accounts not included; details of phone use not available
Schüz <i>et al.</i> (2006c) Denmark, 1982–2002	420 095	1982–2002	Records of cellular service providers	Salivary gland (142)	Subscriber	26 (males) 0 (females)	0.86 (0.56–1.26) 0.00 (0.00–1.02)	Age, calendar year of observation	Incidence study; cannot be certain who the user was; phones used under business accounts not included; details of phone use not available

d, day; h, hour; SIR, standardized incidence ratio; SMR, standardized mortality ratio; yr, year

lifetime use, nor year of first use. The incidence of NHL was elevated among men who had used cell phones for > 8 years (OR, 2.4; 95% CI, 0.8–7.0, based on 17 cases).

2.3.3 Uveal (ocular) melanoma

In a study of 118 cases and 475 controls, [Stang et al. \(2001\)](#) reported an association between assessed occupational use of mobile phones and risk of uveal melanoma ([Table 2.19](#)). Methods for this study are described in greater detail in Section 2.1.3. [There was no adjustment for exposure to ultraviolet radiation, which may be a relevant confounder. Exposure information was crude, and concerns were raised about possible bias in the self-reported data in this small study ([Johansen et al., 2002](#)).]

The same investigators carried out a much larger case–control study (455 cases; aged 20–74 years) between 2002 and 2004 using a more refined exposure-assessment instrument ([Stang et al., 2009; Table 2.19](#)). Three control series were enrolled. One included 827 population controls selected from census data from local districts and matched to case patients on age (5-year age groups), sex and region of residence. A second control series included 180 ophthalmology patients – recruited from practices of the same ophthalmologists who had referred the case patients with uveal melanoma – who had a newly diagnosed benign disease of the eye. The third control group consisted of 187 siblings of cases. Participation rates were 94% for the case patients, 57% for the population and sibling control subjects, and 52% for the ophthalmologists control subjects. The risk of uveal melanoma was not associated with regular use of mobile phones based on any of the three control series (with population controls: OR, 0.7; 95% CI, 0.5–1.0; with ophthalmologist controls: OR, 1.1; 95% CI, 0.6–2.3; and with sibling controls: OR, 1.2 95% CI, 0.5–2.6). There were no associations with cumulative measures of exposure (years of

use, number of calls) based on any of the control series. [The Working Group noted the higher participation rate for cases than for controls and the attendant possibility of selection bias.]

The incidence of cancer of the eye (histology not specified, but likely to include a high proportion of melanomas) was not increased in a large cohort of Danish mobile-phone subscribers relative to the general population in a study that reported follow-up until 2002 ([Schüz et al., 2006c; Table 2.18](#)).

The substantial increase in use of mobile telephones has not been accompanied by an increase in uveal (ocular) melanoma in the USA up to 2000 ([Inskip et al., 2003, 2004](#)), nor was an increase seen in Denmark up to 1996 ([Johansen et al., 2002](#)). The annual percentage change in the USA was –0.7% for males (95% CI, –2.3–0.9) and –1.2% for females (95% CI, –2.5–0.0) ([Inskip et al., 2003](#)). Narrowing the time window to the 1990s failed to reveal any sign of a recent increase in incidence.

2.3.4 Cancer of the testis

The potential exists for the testes to be exposed to RF radiation if a mobile phone is kept in a trouser pocket while in stand-by mode, or when using a hands-free device. The incidence of cancer of the testis was not increased among 357 533 Danish male mobile-phone subscribers relative to that in the general population, based on an average follow-up of 8 years (maximum, 21 years) (SIR, 1.05; 95% CI, 0.96–1.15) ([Schüz et al., 2006c; Table 2.18](#)).

A case–control study of cell-phone use and testicular cancer in Sweden (542 seminomas, 346 non-seminomas, and 870 controls) gave null results for both histopathological subtypes ([Hardell et al., 2007b; Table 2.18](#)). Cases were diagnosed between 1993 and 1997.

Table 2.19 Case-control studies of leukaemia, lymphoma and other cancers and use of mobile phones

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Kaufman et al. (2009) Thailand, 1997–2003	180	756	Hospital	Interviewer-administered standardized questionnaire	Leukaemia (bone marrow) (204–207)	Ever use Use of GSM services	35 NR	1.5 (1.0–2.4) 2.1 (1.1–4.0)	Matching factors age, sex, area of residence, income, exposure to benzene, solvents, pesticides, or power lines	No association with duration of ownership, lifetime hours of use, or h/yr; short duration of use (median, 24–26 months); also evaluated by subtype of leukaemia
Cooke et al. (2010) United Kingdom, 2003–09	806	585	Non-blood relatives	Interviewer-administered standardized questionnaire	Leukaemia (204–207) exclusive of CLL (204.1)	Never, or non-regular use Regular use Lifetime years of use: 0.5–4 5–9 10–14 ≥ 15 <i>P</i> for trend	132 674 201 309 110 42 0.30	1.00 1.06 (0.76–1.46) 0.97 (0.67–1.39) 1.10 (0.77–1.58) 1.04 (0.67–1.61) 1.63 (0.81–3.28)	Age, sex, SES, area of residence, ethnicity, smoking, interview lag-time and period	No significant associations with year since first use, lifetime years of use, cumulative number of calls, or cumulative hours of use; low participation rate (50%)
Hardell et al. (2005) Sweden, 1999–2002	910	1016	Population	Mail questionnaire + telephone	NHL	<i>Use of analogue and digital phones (yr)</i> > 1 > 5 > 10	130 123 70	1.02 (0.73–1.44) 1.04 (0.73–1.46) 0.91 (0.61–1.36)	Age, sex, year of diagnosis	Ages 18–74 yr; no differences by subtype of NHL

Table 2.19 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Linet <i>et al.</i> (2006) USA, 1998–2000	551	462	Population	Mail + home questionnaire	NHL	Ever use Cumulative use (h): ≤ 78 79–208 ≥ 209	234 35 23 35	1.0 (0.7–1.3) 0.8 (0.4–1.4) 0.8 (0.4–1.5) 1.1 (0.6–2.1)	Age, ethnicity, education, geographic site	Risk also not significantly associated with min/wk duration, or year when use started. Results were null for total NHL, large B-cell and follicular lymphoma
Stang <i>et al.</i> (2001) Germany, 1995–98	118	475	Population, hospital	Interview	Uveal melanoma (190)	<i>Probable/certain mobile-phone use</i> Ever ≥ 5 yr in past	6 3	4.2 (1.2–14.5) 4.9 (0.5–51.0)	Age, sex, geographic area	Crude exposure assessment; low prevalence of exposure; few long-term users
Stang <i>et al.</i> (2009) Germany 2002–04	459	1194	Population, ophthalmology, siblings	Questionnaire	Uveal melanoma (190)	Regular use Cumulative use (yr): ≤ 4 5–9 ≥ 10	30 17 11 2	<i>Relative risk</i> 0.7 (0.5–1.0) 0.8 (0.5–1.2) 0.6 (0.4–1.0) 0.6 (0.3–1.4)	Age, sex, residence	RR estimates based on population controls; low participation rate among controls (57%)

Table 2.19 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Hardell <i>et al.</i> (2007b) Sweden 1993–97	888 (542 seminoma; 346 non-seminoma)	870	Population	Questionnaire	Testicular cancer (178)	Cumulative use of mobile-phone (<i>h</i>) Analogue: 1–127 128–547 > 547 Digital: 1–127 128–547 > 547	102 46 27	1.3 (0.9–1.8) 0.7 (0.5–1.0) 0.8 (0.5–1.4)	Age, year of diagnosis, cryptorchidism	Similar null results for seminoma and non-seminoma, as well as by latency
Auvinen <i>et al.</i> (2002) Finland, 1996	34	170	Population	Mobile-phone subscriber lists	Salivary gland cancer (142)	Ever (analogue and digital) Duration (yr): < 1 1–2 > 2	4 0 3 1	1.3 (0.4–4.7) - 1.7 (0.4–7.5) 2.3 (0.2–25.3)	Age, sex	Small number of cases; limited information on exposure; results shown are for analogue and digital phones combined
Hardell <i>et al.</i> (2004) Sweden, 1994–2000	267	1053	Population	Questionnaire	Malignant and benign salivary-gland tumours (142, 210)	Ever use (analogue) Ever use (digital) Latency (yr): > 5 > 10	31 45 17 6	0.92 (0.58–1.44) 1.01 (0.68–1.50) 0.78 (0.44–1.38) 0.71 (0.29–1.74)	Age, sex	Only living cases included; latency results are for analogue phones. No cases among long-term users of digital phones

Table 2.19 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Lönn <i>et al.</i> (2006) Denmark, Sweden, 2000–02	60	681	Population	Interviewer-administered standardized questionnaire	Malignant parotid gland (ICD codes not reported)	Never regular use of mobile phone	35	1.0	Age, sex, geographic region, education	
						Regular use	25	0.7 (0.4–1.3)		
						<i>Time since start of use (yr)</i>				
						< 5	14	0.7 (0.3–1.3)		
						5–9	8	0.7 (0.3–1.7)		
						≥ 10	2	0.4 (0.1–2.6)		
Lönn <i>et al.</i> (2006) Sweden, 2000–02	112	321	Population	Interviewer-administered standardized questionnaire	Benign pleomorphic adenomas (ICD codes not reported)	<i>Cumulative use (h)</i>			Age, sex, geographic region, education	Risk for ipsilateral use: OR, 1.4 (95% CI, 0.2–2.2)
						< 30	7	0.7 (0.3–1.6)		
						30–449	11	0.7 (0.3–1.4)		
						≥ 450	5	0.6 (0.2–1.8)		
						Never regular use of mobile phone	35	1.0		
						Regular use	77	0.9 (0.5–1.5)		
						<i>Time since start of use (yr)</i>				
						< 5	47	1.0 (0.6–1.8)		
						5–9	23	0.8 (0.4–1.5)		
						≥ 10	7	1.4 (0.5–3.9)		
						<i>Cumulative use (h)</i>				
						< 30	20	1.1 (0.6–2.3)		
						30–449	34	0.9 (0.5–1.6)		
						≥ 450	22	1.0 (0.5–2.1)		

Table 2.19 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments
Sadetzki <i>et al.</i> (2008) Israel, 2001–03	460 (48 malignant; 402 benign)	1266	Population	Personal interview	Malignant and benign salivary-gland tumours (142, 210)	Not regular use of mobile phone < 1 yr	175	1.0	Unadjusted (cigarette smoking was considered but did not change OR)	Separate analyses for benign and malignant tumours, with similar results
						Regular use	285	0.87 (0.68–1.13)	Gender, interview date, age, continent of birth	Age at diagnosis, ≥ 18 yr
						<i>Duration of use (yr)</i>				
						1–4.9	138	0.84 (0.63–1.12)		
						5–9.9	134	0.92 (0.67–1.27)		
						≥ 10	13	1.0 (0.48–2.09)		
						<i>Time since start of use (yr)</i>				
						1–4.9	138	0.82 (0.61–1.10)		
						5–9.9	134	0.95 (0.70–1.30)		
						≥ 10	13	0.86 (0.42–1.77)		
						<i>Cumulative use (h)</i>				OR for ipsilateral use: 1.49 (95% CI, 1.05–2.13)
						≤ 266.3	121	0.82 (0.62–1.09)		
						266.4–1034.9	80	1.03 (0.72–1.47)		
						≥ 1035	83	1.09 (0.75–1.60)		

Table 2.19 (continued)

Reference, study location and period	Total cases	Total controls	Control source (hospital, population)	Exposure assessment	Organ site (ICD code)	Exposure categories	Exposed cases	Odds ratio (95% CI)	Covariates	Comments					
Duan <i>et al.</i> (2011) China 1993–2010	136	2051	Hospital	Personal or telephone interviews	Salivary-gland cancer (142)	Regular use model	91	1.14 (0.72–1.81)	Age, sex, residential area, marital status, education, income, smoking status	Possible over-parameterization; difficult to reconcile overall RR with exposure category-specific RRs					
						<i>No. of calls since first use</i>									
						≤ 24 000	78	1.78 (1.12–2.84)	As above	Implausible RR and CI for highest exposure category (one exposed case)					
						24 001–42 000	12	1.76 (1.01–2.51)							
						> 42 000	1	15.36 (13.34–17.38)							
						<i>Duration of use (yr)</i>									
						0–6	67	1.69 (1.05–2.73)	As above	Preceding comments raise serious questions about analysis					
						7–8	7	3.69 (2.82–4.57)							
						9–10	2	7.70 (6.20–9.20)							
						> 10	15	4.14 (1.76–9.69)							

CLL, chronic lymphocytic leukaemia; GSM, Global System for Mobile Communications; min, minute; NHL, non-Hodgkin lymphoma; NR, not reported; RR, relative risk; SES, socioeconomic status; h, hour; yr, year

2.3.5 Cancers of the parotid gland

The salivary glands are potentially exposed to high doses of RF radiation from mobile phones, particularly the parotid gland on the side of the head on which the phone is used. Five case-control studies and one cohort study have addressed a possible relationship between cancer of the salivary gland and use of mobile phones.

An early case-control study by [Auvinen et al. \(2002\)](#) ([Table 2.19](#)) gave null results, but was quite small (34 cases), included only malignant tumours, and provided limited information about details of phone use. Cases were ascertained from the Finnish Cancer Registry and controls from the nationwide population registry. Personal identifiers were linked with subscription records for two cellular networks in 1996. [This register-based approach precludes selection bias to non-response as well as recall bias in the ascertainment of mobile phone use. Information on the frequency or duration of calls was not available, nor was mobile-phone use under a corporate account.]

A case-control study by [Hardell et al. \(2004\)](#) ([Table 2.19](#)) included 267 cases, considered both benign and malignant tumours of the parotid gland, and provided detailed exposure information. Again, the results were null. [The study included few people who had used mobile phones for > 10 years.]

A case-control study by [Lönn et al. \(2006\)](#) ([Table 2.19](#)), which was part of the INTERPHONE study, included 172 cases (benign and malignant parotid tumours combined), 681 controls (for the 60 malignant cases), and 321 controls (for the 112 benign cases). The study found no association with regular use of mobile phones for either malignant or benign parotid tumours. The surrogate exposure metrics considered included frequency of use, duration of regular use, time since first regular use, cumulative use and cumulative number of calls. For benign tumours, there was a slightly elevated risk associated

with ipsilateral use of mobile phones (OR, 1.4; 95% CI, 0.2–2.2, based on 51 cases) but not for contralateral tumours (OR, 0.7; 95% CI, 0.4–1.1, based on 35 cases). [There may have been bias in reporting of laterality of phone use.]

A case-control study of tumours of the parotid gland was conducted in Israel, where use of mobile phones was reported to be very high ([Sadetzki et al., 2008](#); [Table 2.19](#)). This was the largest study of this type (402 cases with benign tumours, 58 with malignant tumours, and 1266 controls), also conducted as part of the INTERPHONE study. Cases were diagnosed at age 18 years or more during 2001 and 2003. In the main analyses, no increased risk was observed for any of the exposure surrogates examined. Laterality analyses generally indicated increased risk for ipsilateral use and reduced risk for contralateral use, e.g. for > 266 hours of cumulative call time with no hands-free devices, the odds ratio for ipsilateral use was 1.49 (95% CI, 1.05–2.13, based on 115 cases), while the odds ratio for contralateral use was 0.84 (95% CI, 0.55–1.28, based on 48 cases). Stratified analyses according to type of residence produced a somewhat higher odds ratio for rural and mixed rural/urban areas than for poor urban areas. For rural and rural/urban users, exposure-response associations were significant for cumulative call time ($P = 0.04$) and borderline significant for number of calls ($P = 0.06$). When the analyses were restricted to regular users only, taking the lowest category of use as the reference, increased odds ratios were found if time since start of use was > 5 years before diagnosis (OR, 1.40; 95% CI, 1.03–1.90, based on 134 cases) and for the highest exposure category of cumulative number of calls (OR, 1.51; 95% CI, 1.05–2.17, based on 81 cases) and duration of calls (OR, 1.50; 95% CI, 1.04–2.16, based on 83 cases). [The fact that there were increased odds ratios for ipsilateral tumours and decreased odds ratios for contralateral tumours suggested the presence of bias in reporting side of use.]

In a hospital-based case-control study of epithelial cancers of the parotid gland conducted in China between 1993 and 2010 (136 cases, 2051 controls), no overall association of cancer risk with regular use of mobile phones was observed ([Duan et al., 2011](#); [Table 2.19](#)). The authors also evaluated several more detailed exposure metrics and commented that several showed evidence of a dose-response relationship. [This interpretation was made uncertain by aspects of variation in the odds ratios. In several instances, there was no indication of a gradient in risk, but a very large increase in the odds ratio for the highest exposure category. Perhaps more puzzling was the fact that, for many of the exposure variables, odds ratios for all categories of exposure were higher than the overall odds ratio of 1.14. One would expect the overall odds ratio for regular use to be a weighted average of category-specific odds ratios. For number of calls since first use, the authors reported an odds ratio of 15.36 (95% CI, 13.34–17.38) for the highest exposure category, based on one exposed case. This cannot be correct and raises doubt about other analyses. The odds ratio presented may be 1/OR, as 0.7% of cases and 12.6% of controls were in this category.]

The incidence of cancers of the salivary gland was not increased relative to that in the general population in a large cohort of mobile-phone subscribers in Denmark followed up for up to 21 years ([Schüz et al., 2006c](#); [Table 2.19](#)).

A recent descriptive study reported an increase in the occurrence of cancer of the parotid gland (not incidence rate) in Israel, which appeared to begin around 1990 and continue through 2006 ([Czerninski et al., 2011](#)). [Interpretation of these findings was difficult given the increase in population size in Israel, possible improvements over time in the ascertainment of cancers of the parotid gland, a substantial shift in diagnoses over time from the category “major salivary gland cancers, not otherwise specified” to more precisely defined types – the large majority of which were cancers of the parotid gland – and the lack of information about mobile-phone use.]

2.3.6 Other cancers

(a) Cancer of the breast

[There was little information concerning mobile-phone use and risk of breast cancer.] Breast cancer did not occur more often than expected based on incidence rates in the general population in a cohort of 65 542 Danish female mobile-phone subscribers followed from as early as 1982 until 1995 ([Schüz et al., 2006c](#); [Table 2.18](#)).

(b) Cancer of the skin

In a case-control study of cutaneous melanoma in the head and neck region (347 cases, 1184 controls), [Hardell et al. \(2011b\)](#) reported no overall association with use of mobile phones (OR, 1.0; 95% CI, 0.7–1.3, based on 223 cases) or cordless phones (OR, 0.9; 95% CI, 0.6–1.2, based on 138 cases), nor among those with heavier use. Use of cordless phones, but not mobile phones, was associated with an increased risk of melanoma in the temporal region, cheek, and ear for the group with 1–5 year latency among those with heavier use (OR, 2.1; 95% CI, 1.1–3.8 for > 365 cumulative hours, based on 21 cases). [The overall pattern in the data pointed more in the direction of no effect. The odds ratio mentioned in the Abstract for the latency period of 1–5 years did not match that in Table 2 of the published manuscript regarding mobile-phone use.]

[To date, there have been no studies of non-melanoma skin cancer in relation to mobile-phone use.]

(c) Other cancer sites

Subscribers to mobile-phone services in Denmark followed from as early as 1982 until 2002 did not show significantly elevated incidence rates of cancers of the lung, larynx, bladder, buccal cavity, oesophagus, liver, uterine cervix, stomach, kidney, pancreas, prostate or other sites, relative to the incidence rates in the Danish general population ([Schüz et al., 2006c](#)).

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3. CANCER IN EXPERIMENTAL ANIMALS

3.1 Studies of carcinogenicity

See [Table 3.1](#)

3.1.1 Mouse

Groups of 50 male and 50 female B6C3F₁ mice (age, 8–9 weeks) were sham-exposed or received whole-body exposure to GSM (Global System for Mobile communications)-modulated radiofrequency (RF) radiation at 902 MHz, or to DCS (Digital Cellular System)-modulated RF radiation at 1747 MHz, in a Ferris wheel/tube-restrained design for 2 hours per day, 5 days per week, for 24 months. Exposures were performed with a three-phase signal imitating “basic,” “talk” and “environment” GSM signals. Cage controls were run in parallel. The average specific absorption rate (SAR) for each signal phase (0, 0.4, 1.3, and 4.0 mW/g), organ-averaged SARs, and corresponding standard variations were calculated. No increases in tumour incidence at any site were observed in exposed mice compared with sham-exposed mice. Decreases in the incidence of liver adenoma were seen in males exposed to GSM at 4.0 mW/g and in males exposed to DCS at 4.0 mW/g ([Tillmann *et al.*, 2007](#)).

3.1.2 Rat

Groups of 100 male Sprague-Dawley rats (age, 8 weeks) were sham-exposed or exposed to RF radiation as pulsed microwaves at 2450 MHz, at 800 pulses per second (pps) with a pulse

width of 10 μ s (range of SAR values: young rats, 0.4 mW/g; older rats, 0.15 mW/g) for 21.5 hours per day, 7 days per week, for 25 months. The exposure to microwaves had no statistically significant effect on survival (median survival time: sham-exposed rats, 663 days; exposed rats, 688 days) or body weight. No statistically significant increases in the incidence of any benign or malignant tumours were identified at any site in exposed rats compared with sham-exposed controls. An increased incidence of total malignant tumours (all sites) was observed in rats exposed to RF radiation compared with sham-exposed controls ([Chou *et al.*, 1992](#)). [The Working Group considered this finding to be of limited biological significance, since it resulted from pooling of non-significant changes in tumour incidence in several sites.]

Groups of female Sprague-Dawley rats (age, 52–70 days) were sham-exposed or exposed to RF radiation as GSM at 900 MHz, with a pulse of 217 Hz, for more than 23 hours per day, 7 days per week, for up to 37 months. In the four experiments that were carried out, the number of rats per group was 12 in experiments 1 and 2, and 30 in experiments 3 and 4. Rats were group-housed with up to 12 rats per cage. Whole-body averaged SARs (wbSARs) during the studies ranged from 32.5–130 mW/kg in rats weighing 170–200 g, to 15–60 mW/kg in rats weighing ~400 g. In experiment 1, surviving rats were killed and necropsied at 770 days [26.7 months] (mortality, 33%), while in experiment 2, surviving rats were killed and

Table 3.1 Studies of carcinogenicity in experimental animals exposed to radiofrequency radiation

Species, strain (sex)	Dosing regimen	Animals/group at start	Incidence of tumours			Significance	Comments
Duration							
Reference							
Mouse, C3H/HeA (F)	2 450 MHz MW far field: sham, 5 mW/cm ² (SAR, 2–3 mW/g), 15 mW/cm ² (SAR, 6–8 mW/g), confinement stress group, cage control		Power density (mW/cm ²)	Mammary-gland tumours	CDT ₅₀	*P < 0.01	Mammary-gland tumours detected only by palpation
10.5 mo	2 h/d, 6 d/wk			at 8 mo			
Szmigielski et al. (1982)	40/group		0 (sham)	3/40	14/40	322 d	
			5	18/40*	32/40*	261 d	
			15	26/40*	37/40*	219 d	
			Confined	16/40*	31/40*	255 d	
Mouse, <i>Eμ-Pim1</i> (F)	900 MHz (217 Hz [pulse repetition, similar to GSM]; pulse width, 0.6 ms), sham		Lymphoma (sham, RF-EMF): 3/100, 6/101 (lymphoblastic)			P = 0.0002 (non-lymphoblastic lymphoma)	No standardized assessment criteria were defined for deciding which mice would be selected for necropsy.
18 mo	SAR: 0.008–4.2 mW/g, 0.13–1.4 mW/g (average)		19/100, 37/101 (non-lymphoblastic)			P = 0.006 (all lymphomas)	Mice surviving the 18 mo of exposure or sham-exposure were discarded without necropsy.
Repacholi et al. (1997)	2 × 30 min/d, 7 d/wk		22/100, 43/101 (all)				
	100/sham-exposed group, 101/RF radiation-exposed group						
Mouse, C3H/HeJ (F)	435 MHz (420–450 MHz) RF radiation with pulse-wave (pulse width, 1.0 μs; pulse rate, 1.0 kHz), sham		Mammary-gland adenocarcinoma: 77/193 (exposed), 74/190 (sham)			NS	Complete histopathology
21 mo	Incident power density of 1.0 mW/cm ² ; SAR, 0.32 mW/g						
Toler et al. (1997)	22 h/d, 7 d/wk						
	200/group						
Mouse, C3H/HeJ (F)	2 450 MHz MW (SAR, 0.3 mW/g), sham		Mammary-gland carcinoma: 44% (RF radiation), 52% (sham)			NS	Complete histopathology
18 mo	20 h/d, 7 d/wk						
Frei et al. (1998a)	100/group						
Mouse, C3H/HeJ (F)	2 450-MHz MW (SAR: 1.0 mW/g), sham		Mammary-gland carcinoma: 38% (RF radiation), 30% (sham)			NS	Complete histopathology
78 wk	20 h/d, 7 d/wk						
Frei et al. (1998b)	100/group						

Table 3.1 (continued)

Species, strain (sex) Duration Reference	Dosing regimen Animals/group at start	Incidence of tumours	Significance	Comments
Mouse, C3H/HeJ (F) 76 wk lauchem et al. (2001)	UWB (rise time, 176 ps; fall time, 3.5 ns; pulse width, 1.9 ns; peak energy field, 40 kV/m; repetition rate, 1 kHz; SAR, 0.0098 mW/g), sham 2 min/wk, 12 wk 100/group	Mammary-gland carcinoma: 48/100 (UWB), 52/100 (sham)	NS	Complete histopathology
Mouse, <i>Eμ-Pim1</i> and wild-type (C57BL/6NTac) (F) 104 wk Uttridge et al. (2002)	GSM-modulated 898.4 MHz (pulse width, 0.6 ms); SAR, 0.25, 1.0, 2.0, 4.0 mW/g; sham, cage control 1 h/d, 5 d/wk 120/group (wild-type and <i>Eμ-Pim1</i>)	Number of tumour-bearing animals: <i>Lymphoblastic lymphoma</i> Sham-exposed control (wild-type, transgenic): 3, 15; SAR = 0.25: 0, 8*; SAR = 1: 2, 8; SAR = 2: 2, 9; SAR = 4: 0, 15; total: 7, 55 <i>Non-lymphoblastic lymphoma</i> Sham-exposed control (wild-type, transgenic): 35, 74; SAR = 0.25: 40, 80; SAR = 1: 35, 78; SAR = 2: 43, 84; SAR = 4: 36, 84; total: 189, 400 <i>Neurological tumours</i> Sham-exposed control (wild-type, transgenic): 11, 1; SAR = 0.25: 17, 4; SAR = 1: 15, 0; SAR = 2: 10, 2; SAR = 4: 9, 2; total: 62, 9	* <i>P</i> = 0.02 (decrease)	Restrained exposure (Ferris wheel) also for sham, but cage-control group unrestrained. Necropsy was performed on all mice.
Mouse, AKR/J (F) 40 wk Sommer et al. (2004)	GSM 900 MHz (overall max. SAR, 5.9 mW/g; average SAR, 0.4 mW/g [whole body]), sham 24 h/d, 7 d/ wk 160/group	No increase in tumour incidence	NS	Histopathology was performed on spleen, thymus, lymph nodes, liver, kidney, lung and brain.
Mouse, <i>Eμ-Pim1</i> (M, F) 18 mo Oberto et al. (2007)	GSM-modulated 900 MHz (pulse width, 0.577 ms); SAR, 0.5, 1.4, 4.0 mW/g (whole body); sham, cage control 2 × 30 min/d, 7 d/wk 50 M and 50 F/group	Number of tumour-bearing animals: <i>Lymphoma</i> (all): (M) – 8 (cage), 9 (sham), 10 (0.5, 1.4 mW/g), 3 (4 mW/g); (F) – 26 (cage), 22 (sham), 18 (0.5 mW/g), 30 (1.4 mW/g), 20 (4.0 mW/g) <i>Harderian gland adenoma</i> : (M) – 0 (sham), 0 (0.5 mW/g), 2 (1.4 mW/g), 4 (4.0 mW/g)	Harderian-gland adenoma: <i>P</i> = 0.0028 (one-tailed test, trend) (M)	Restrained exposure (Ferris wheel). Mortality was higher (<i>P</i> < 0.05) in all three groups of exposed males and in the females exposed at 0.5 mW/g. GLP

Table 3.1 (continued)

Species, strain (sex) Duration Reference	Dosing regimen Animals/group at start	Incidence of tumours	Significance	Comments
Mouse, B6C3F ₁ (M, F) 24 mo Tillmann et al. (2007)	GSM 902 MHz, DCS 1 747 MHz, sham, cage control; wSAR: 0.4, 1.3, 4 mW/g for each signal. 2 h/d, 5 d/wk 50 M and 50 F/group	<i>All tumours (%)</i> GSM (M/F):sham (68/78), low dose (62/78), medium dose (66/74), high dose (64/78); DCS (M/F): sham (74/74), low dose (60/62), medium dose (50/70), high dose (48/66) DCS: No. of males with tumours: 37 (sham), 30 (low dose), 25 (medium dose)* and 24 (high dose)*; No. of males with benign tumours: 27 (sham), 21 (low dose), 17 (medium dose), 12 (high dose)* <i>Liver adenoma in males (%)</i> DCS: 22 (sham), 4 (high dose)*; GSM: 30 (sham); 12 (high dose)*	*P < 0.05 (decrease)	Restrained exposure (Ferris wheel) Complete histopathology GLP
Mouse, <i>Ptcr</i> ^{+/+} , <i>Ptcr</i> ^{1+/+} (wild-type) (M, F) Lifetime Saran et al. (2007)	GSM-modulated 900 MHz (wSAR, 0.4 mW/g), sham, cage control 2 × 30 min/d, 5 d, starting on PND 2 23–26 heterozygous and 22–29 wild-type mice/sex/group	No increase in tumour incidence	NS	Histopathology was performed on brain, a 5 cm ² piece of skin, and any visible neoplasm.
Mouse, AKR/J (F) 43 wk Sommer et al. (2007)	UMTS (FDD, 1 966 MHz; SAR, 0.4 mW/g), sham, cage control 24 h/d, 7 d/wk 160/sham- or RF radiation-exposed group and 30/cage-control group	<i>Lymphoma</i> RF radiation: 141 (88.1%); sham: 149/150 (93.1%); cage control, 29/30 (96.7%)	NS	Histopathology was performed on spleen, thymus, lymph nodes, liver, kidney, lung and brain.
Mouse, AKR/J (M, F) 42 wk Lee et al. (2011)	CDMA 849 MHz and WCDMA 1950 MHz (combined) with SAR of 2 mW/g for CDMA and WCDMA [variation estimated: 1.59–2.52 mW/g], sham 45 min/d, 5 d/wk 40 M and 40 F/group	<i>Thymic lymphoma</i> Sham: M: 30/40 (75%); F: 32/40 (80%) Combined RF radiation: M: 31/40 (78%); F: 31/40 (78%)	NS	Exposure was performed in a reverberation chamber. Histopathology was performed on spleen, thymus, lymph nodes, liver, kidney, lung and brain.
Rat, Sprague-Dawley (M) 25 mo Chou et al. (1992)	2 450 MHz (800 pps; pulse width, 10 µs; pulse modulation, 8 Hz), sham; SAR: 0.15 (800 g bw) and 0.4 mW/g (200 g bw) 21.5 h/d, 7 d/wk 100/group	<i>Malignant neoplasms (all sites)</i> Sham, 5/100; exposed, 18/100 <i>Adrenal gland, pheochromocytoma</i> Sham, 1/100; exposed, 7/100 <i>Adrenal gland, cortical carcinoma</i> Sham, 0/100; exposed, 3/100	P = 0.005, χ^2 NS	Complete histopathology No increase in tumour incidence at any site.

Table 3.1 (continued)

Species, strain (sex) Duration Reference	Dosing regimen Animals/group at start	Incidence of tumours	Significance	Comments
Rat, F344 (M, F) 24 mo La Regina et al. (2003)	FDMA 835.6 MHz, CDMA 847.7 MHz, sham; SAR (brain): 0.85 ± 0.34 mW/g (time-averaged) SAR, 1.3 \pm 0.5 mW/g (nominal time-averaged brain SAR) 4 h/d, 5 d/wk 80 M and 80 F/group	<i>Total No. of tumours</i> (Sham/FDMA/CDMA): 76/79/78 (F), 163/162/148 (M) Mixed glioma (%): 0/1/0 (F) Astrocytoma (%): 0/0/1 (F), 0/1/0 (M) Granular cell tumour (%): 1/0/0 (F), 0/1/0 (M)	NS	Restrained exposure (Ferris wheel). Over the 2-yr time-course study, the brain SAR varied from 0.5 to 2.5 mW/g. Complete histopathology, 20–30 brain sections were examined per rat.
Rat, F344 (M, F) 24 mo Anderson et al. (2004)	1 620 MHz Dams and pups: far-field exposure from GD 19 until weaning (pups aged 23 days). Pups (age, 36 days): near-field exposure of ~2 yr. Brain SAR: 0.16 mW/g (fetuses, far field), 0.16 or 1.6 mW/g (offspring, near-field), sham, cage control. 2 h/d, 7 d/wk (far-field), 2 h/d, 5 d/wk (near field) 80–90 M and 80–90 F pups/group: near-field (2 groups), sham (1 group), cage control (1 group)	No increase in tumour incidence	NS	Restrained exposure (Ferris wheel) for near-field exposure Complete histopathology The incidence of brain neoplasms was within the range for historical controls
Rat, Wistar (M, F) 24 mo Smith et al. (2007)	902 MHz (GSM), 1 747 MHz (DCS), sham, cage control; time-averaged wbSAR: 0.44, 1.33, 4.0 mW/g for each signal. 2 h/d, 5 d/wk 50 M and 50 F/group	No increase in tumour incidence	NS	Restrained exposure (Ferris wheel) Due to increase in bw, the wbSAR of the group at the highest dose (4.0 mW/g targeted) was reduced to 3.0 mW/g after about 2 years; on average, a wbSAR of 3.7 mW/g was obtained; detailed SAR values and uncertainty were estimated for many organs. Complete histopathology GLP

bw, body weight; B[a]P, benzo[a]pyrene; CDMA, code-division multiple access; CDT₅₀, cancer-development time 50 (i.e. time in which 50% of the mice developed skin carcinoma); d, day; DCS, digital personal communications system; FDD, frequency-division duplexing; FDMA, frequency-division multiple access; GD, day of gestation; GLP, good laboratory practice; GSM, Global System for Mobile Communication; min, minute; mo, month; MW, microwave; NS, not significant; PND, postnatal day; RF-EMF, radiofrequency electromagnetic field; SAR, specific absorption rate; UWB, ultra-wide band; WCDMA, wide-band code division multiple access; wk, week; yr, year

necropsied at 580 days [19.3 months] (mortality, 50%). In experiment 1, histopathological evaluations were performed on the main organs, while only gross pathology was performed in experiment 2. In experiments 3 and 4, the rats were followed until natural death and histopathology was done on macroscopically detected changes only. In experiments 1 and 2, fewer pituitary tumours were detected in rats exposed to RF radiation (42% and 33% in experiments 1 and 2, respectively) than in sham-exposed controls (75% and 50% in experiments 1 and 2, respectively). Decreased incidences of mammary tumours (possibly associated with significantly shortened median survival times) were seen in experiments 3 and 4. No evidence of increased incidence of cancer in any tissue was reported in exposed rats compared with sham-exposed controls in any of the four experiments performed ([Bartsch et al., 2010](#)). [The Working Group considered that the value of these experiments was limited by the lack of reproducibility in survival times in experiments 1 and 2 (performed with identical protocols), by the small group sizes in all experiments, and by the poor reporting of tumour data from all experiments. Because complete pathology results were not reported, this study cannot be regarded as a comprehensive carcinogenicity bioassay, and it was not considered in the evaluation.]

Groups of 80 female and 80 male F344 rats (age, 6 weeks) were sham-exposed or exposed to RF radiation in the FDMA mode (frequency-division multiple access) 835.6 MHz, or in the CDMA mode (code-division multiple access) at 847.7 MHz, for 4 hours per day, 5 days per week, for 24 months. Rats were tube-restrained during exposure; time-averaged SAR in the brain was 1.3 mW/g for both signals. There were no significant differences in survival, body weight or tumour incidence at any site in exposed males or females when compared with sex-matched sham-exposed controls ([La Regina et al., 2003](#)).

Groups of pregnant Fischer 344 rats were exposed to far-field RF radiation at 1620 MHz

for 2 hours per day, 7 days per week, from day 19 of gestation until weaning. At age 36 days, groups of 90 male and 90 female offspring were sham-exposed or exposed in tubes to near-field RF radiation at 1620 MHz (head mostly) for 2 hours per day, 5 days per week, for 24 months. Sham-exposure and near-field exposure were performed using a Ferris wheel/tube-restrained design at two targeted levels (brain SAR, 0.16 and 1.6 mW/g). No statistically significant differences between exposed and control groups were observed in number of live pups per litter, survival index, or weaning weights. There were no statistically significant effects of exposure on mean body weight of surviving rats. The percentage of rats surviving at study termination did not differ among groups. Incidences of tumours were similar in all groups ([Anderson et al., 2004](#)).

Groups of 50 male and 50 female Wistar rats (age, approximately 6 weeks) were sham-exposed or received whole-body exposure to GSM-modulated RF radiation at 902 MHz, or to DCS-modulated RF radiation at 1747 MHz, in a Ferris wheel/tube-restrained design for 2 hours per day, 5 days per week, for 24 months. Exposures were performed with a three-phase signal imitating “basic,” “talk” and “environment.” Cage controls were run in parallel. Time-averaged *wbSARs* in the three exposure groups were 0.44, 1.33, and 4.0 mW/g for each signal phase. Body weight and survival were not statistically different between exposed and sham-exposed groups. No significant differences in the incidences of benign or malignant neoplasms at any site were observed between the exposed and sham-exposed groups ([Smith et al., 2007](#)).

3.1.3 Transgenic and tumour-prone models

(a) *Eμ-pim1* transgenic mouse

The *Eμ-Pim1* transgenic mouse strain has been reported to spontaneously develop lymphoma and to show an increased incidence of lymphoma

in response to exposure to chemical carcinogens ([Breuer et al., 1989](#); [van Kreijl et al., 1998](#)).

Groups of 100–101 female heterozygous *Eμ-Pim1* mice (age, 6–8 weeks) were sham-exposed or exposed to RF radiation as “GSM basic” at 900 MHz for up to 18 months. Mean SAR values in exposed mice were 0.13–1.4 mW/g. At study termination, mice that were clinically healthy were counted as survivors and discarded without further investigation. Exposure to RF radiation had no statistically significant effect on body weight [survival data were not reported]. The authors reported a twofold increase in the incidence of lymphoma in *Eμ-Pim1* mice exposed to GSM RF radiation ($P = 0.006$ versus the sham-exposed group) ([Repacholi et al., 1997](#)). [The Working Group considered the complete lack of pathology data to be a major limitation in the design of this study.]

Groups of 120 female heterozygous *Eμ-Pim1* mice and 120 female wild-type mice (C57BL/6NTac) (age, 7.5–9.5 weeks) were sham-exposed or exposed to GSM-modulated RF radiation at 898.4 MHz in a Ferris-wheel/restrained design at four different exposure levels (SAR: 0.25, 1.0, 2.0 or 4.0 mW/g) for 1 hour per day, 5 days per week, for 104 weeks. An unrestrained cage-control group was also included in the study. No significant differences in survival or body weight were observed between exposed and sham-exposed mice of either strain. Survival of the transgenic mice was significantly lower than that of wild-type mice ($P < 0.001$). No significant increases in the incidence of lymphoblastic or non-lymphoblastic lymphoma were seen in exposed mice compared with sham-exposed mice at any exposure level ([Utteridge et al., 2002](#)).

Groups of 50 male and 50 female *Eμ-Pim1* mice (age, 9 weeks) were sham-exposed or exposed to RF radiation as “GSM basic” phase signal at 900 MHz, with a pulse of 217 Hz, and pulse width of 0.5 ms, at wbSARs of 0, 0.5, 1.4, or 4.0 mW/g. Exposures were for 1 hour per day, split into two sessions of 30 minutes (morning and afternoon),

7 days per week, for up to 18 months. Cage controls were run in parallel. As in the study by [Utteridge et al. \(2002\)](#), the mice were restrained in tubes during exposure or sham exposure. Compared with sham-exposed mice, survival until termination of the study was shorter in male mice in all groups exposed to RF radiation and in female mice exposed at 0.5 mW/g. Compared with sham-exposed groups, there was no significant difference in the mean body weight of either females or males. No statistically significant differences were seen in the incidences of malignant lymphoma (lymphoblastic and non-lymphoblastic) in sham-exposed or exposed males or females. The incidences of tumours of the Harderian gland were significantly higher in male mice exposed to RF radiation than in controls, with a dose-dependent trend ($P = 0.0028$, one-tailed test); this resulted in a significant positive trend in the overall incidence of benign tumours ($P < 0.01$). For females, no dose-related trends related to exposure to RF radiation were seen in the overall incidence of benign or malignant tumours, or of tumours regardless of type ([Oberto et al., 2007](#)). [The Working Group noted that in the study of [Repacholi et al. \(1997\)](#), 22% of the sham-exposed female mice had lymphomas, whereas in this study, 44% of the sham-exposed and 52% of the cage-control female mice developed lymphomas. The incidence of lymphoma in the exposed group was 43% in the study of [Repacholi et al. \(1997\)](#), a value similar to that for the control groups in this study.]

(b) *Patched1*^{+/-} mouse

[Saran et al. \(2007\)](#) used newborn *Patched1* heterozygous knockout mice (*Ptc1*^{+/-}), a mouse model characterized by predisposition to tumours of the brain and other tissues, and by hypersensitivity to ionizing radiation. Groups of 23–36 male and 23–36 female *Ptc1*^{+/-} mice, and groups of 22–29 male and 22–29 female wild-type mice were exposed to RF radiation at 900 MHz (wbSAR, 4 mW/g) from postnatal days 2 to

6 (30 minutes, twice per day), the time window of extreme susceptibility to induction of medulloblastoma by ionizing radiation in this strain. Mice were monitored throughout their lifespan for the onset of brain tumours or any other visible neoplasm. No significant differences between exposed and sham-exposed groups were observed in the incidence or size of medulloblastoma, or in the incidence of any other neoplasms in either *Ptc1*^{+/-} mice or wild-type mice. [The Working Group noted that tumour data were not reported by sex. The very short duration of exposure, its timing during the immediate post-parturition period, and the lack of exposure of older juvenile or adult animals may limit the value of this study for hazard identification.]

(c) *AKR mouse*

The AKR mouse strain is known to develop lymphomas and other haematopoietic malignancies within the first year of life.

Groups of 160 unrestrained female AKR/J mice were sham-exposed or exposed to GSM-like RF radiation at 900 MHz for 24 hours per day, 7 days per week, for 40 weeks, at an average wbSAR of 0.4 mW/g. Exposure had a significant effect on body weight gain, with higher values in exposed than in sham-exposed mice. Survival and incidence of lymphoma did not differ between exposed and sham-exposed mice ([Sommer et al., 2004](#)). [The Working Group noted that in the absence of any difference in survival, the ability of the study design to detect any effect on tumour incidence between groups was small.]

Groups of 160 female AKR/J mice (age, 8 weeks) were sham-exposed or exposed to RF radiation as Universal Mobile Telecommunications System (UMTS) at 1966 MHz (SAR, 0.4 mW/g) for 24 hours per day, for 248 days (43 weeks). The 30 mice in the cage-control group gained significantly less weight than did the exposed and sham-exposed animals. No statistically significant differences in total body weight, survival, or incidence of neoplasms were observed between

exposed and sham-exposed mice. The incidence of lymphoma in all three groups was above 88% (RF-radiation exposed, 88.1%; sham-exposed, 93.1%; and cage controls, 96.7%) ([Sommer et al., 2007](#)). [The Working Group noted that in the absence of any difference in survival, the ability of the study design to detect any effect on tumour incidence between groups was small.]

Groups of 40 female and 40 male AKR/J mice (age, 5 weeks) were exposed simultaneously to RF radiation at 849 MHz (SAR, 2 mW/g) and 1950 MHz (SAR, 2 mW/g), for 45 minutes per day, 5 days per week, for 42 weeks. Sham exposures were performed in parallel. No differences in body weight, survival or tumour incidence were observed. The incidence of lymphomas in all groups was greater than 75% ([Lee et al., 2011](#)). [The Working Group noted the short daily exposure period. Furthermore, in the absence of any difference in survival, the ability of the study design to detect any effect on tumour incidence between groups was small.]

(d) *C3H mouse*

The C3H tumour-prone mouse carries a milk-borne virus that induces tumours of the mammary gland.

Groups of 40 female C3H/HeA mice were exposed to RF radiation at 2450 MHz as continuous microwaves from ages 6 weeks to 12 months. Five experimental groups (SAR, 0 [sham-exposed control], 2–3 mW/g, or 6–8 mW/g, confinement-stress group, cage-control group) were used. Mammary-gland tumours were detected by palpation. A more rapid appearance of mammary-gland tumours and a statistically significant increase in the incidence of mammary-gland tumours in both groups of mice exposed to microwave radiation was reported, compared with controls ([Szmigielski et al., 1982](#)). [The Working Group noted that no histopathology was performed.]

Groups of 200 female C3H/HeJ mice were sham-exposed or received whole-body exposure

to a horizontally polarized pulse wave at 435 MHz (pulse width, 1.0 ps; pulse rate, 1.0 kHz; wbSAR, 0.32 mW/g) for 22 hours per day, 7 days per week, for 21 months. No statistically significant differences in survival or body weight, or in the incidence, latency or growth rate of mammary-gland tumours were seen between exposed and sham-exposed groups ([Toler et al., 1997](#)).

Groups of 100 female C3H/HeJ mice (age, 3–4 weeks) were sham-exposed or exposed to continuous microwave radiation at 2450 MHz for 20 hours per day, 7 days per week, for 18 months. The average wbSAR was 0.3 mW/g. No significant differences in survival or body weight, or in the incidence, latency or growth rate of mammary-gland tumours were seen ([Frei et al., 1998a](#)).

A study with a similar design was performed at a higher SAR (1.0 mW/g). Groups of 100 female C3H/HeJ mice (age, 3–4 weeks) were sham-exposed or exposed to continuous microwave radiation at 2450 MHz, for 20 hours per day, 7 days per week, for 78 weeks. No differences in survival or body weight or in the incidence, latency or growth rate of mammary-gland tumours were observed ([Frei et al., 1998b](#)).

Groups of 100 female C3H/HeJ mice (age, 3–4 weeks) were exposed to pulses composed of an ultra-wide band (UWB) of frequencies with a rise time of 176 ps and a peak-energy field of 40 kV/m (SAR, 0.0098 mW/g). The mice were exposed for 2 minutes per week for 12 weeks, followed by a post-exposure period of 64 weeks. No significant differences between groups with respect to body weight, incidence of palpated mammary-gland tumours, latency to onset of mammary-gland tumour development, rate of mammary-gland tumour growth, or survival were found. Histopathological evaluations revealed no significant differences in tumour incidences between the two groups for all tissues studied ([Jauchem et al., 2001](#)). [The Working Group considered that the exposure was very limited.]

(e) *OF1 mouse*

The Ico:OF1 mouse strain is known to develop spontaneous tumours of the lymphoid tissue. Groups of 20 female mice were sham-exposed or exposed to RF radiation at 800 MHz for 1 hour per week, for 4 months, and followed for up to 18 months ([Anghileri et al., 2005](#)). Compared with controls, the exposure caused an earlier onset of general lymphocyte infiltration, formation of lymphoblastic ascites, and development of extranodal tumours of different histological types. [The Working Group considered that the inadequate description of the exposure level and dosimetry, the lack of histopathology, and the small group size did not permit a proper evaluation of this study.]

3.2 Initiation–promotion studies

See [Table 3.2](#)

The effect of exposure to RF radiation on tumours initiated by a chemical or physical carcinogen has been tested in various rodent models.

3.2.1 *Skin-tumour model*

Four groups of female ICR mice (age, 10 weeks) were given a single application of 100 µg of 7,12-dimethylbenz[*a*]anthracene (DMBA) on pre-shaved dorsal skin. Exposure to RF radiation started 1 week later and was continued for 19 weeks. Group 1 (48 mice) was exposed to a TDMA (time-division multiple access) signal at 1.49 GHz (50 pps, near-field), for 90 minutes per day, 5 days per week, at a skin local peak SAR of 2.0 mW/g. Group 2 (48 mice) was sham-exposed. Group 3 (30 mice) was exposed weekly and topically to 4.0 µg of 12-*O*-tetradecanoylphorbol-13-acetate (TPA) per mouse. Group 4 (30 mice) received no further treatment. The incidences of skin papilloma or carcinoma (combined) were 0 out of 48, 0 out of 48, 29 out of 30, and 1 out of 30, respectively ([Imaida et al., 2001](#)).

In a comparable experiment, groups of 20 male ICR mice (age, 7 weeks) received the same single skin application (100 µg of DMBA per mouse). Exposure to RF radiation started 1 week later and was continued for 19 weeks. Group 1 was exposed topically to 4 µg of TPA per mouse, twice per week. Group 2 was sham-exposed. Group 3 was exposed to RF radiation at 849 MHz (45 minutes, twice per day, with an interval of 15 minutes between exposures, for 5 days per week). Group 4 was exposed to RF radiation at 1763 MHz (with a schedule similar to that for group 3). A CDMA signal was used with a wbSAR of 0.4 mW/g. Skin tumours [not further specified] were detected only in the DMBA/TPA-treated positive control group ([Huang et al., 2005](#)). [The Working Group noted the short duration of daily exposures and the use of only one exposure level per experiment.]

Groups of 10 male Swiss albino mice (age, 8 weeks) received a single skin application of 100 µg of DMBA (initiated groups) or were left untreated. Exposure to RF radiation or to croton oil (the positive control) started 2 weeks later. Group 1 was not initiated and was sham-exposed. Group 2 was exposed to DMBA only (cage control). Group 3 was exposed to DMBA plus amplitude-modulated (AM) RF radiation at 112 MHz, with a SAR of 0.75 mW/g, for 2 hours per day, 3 days per week, for 16 weeks. Group 4 was exposed to DMBA plus RF radiation at 2450 MHz with a SAR of 0.1 mW/g for 2 hours per day, 3 days per week, for 16 weeks. Group 5 was exposed to AM RF radiation at 112 MHz only. Group 6 was exposed to RF radiation at 2450 MHz only. Group 7 was exposed to DMBA plus a topical application of croton oil at 1% in 100 µL of acetone per mouse, twice per week, for 16 weeks. At study termination, skin tumours were detected only in the positive-control group (DMBA plus croton oil) ([Paulraj & Behari, 2011](#)). [The Working Group noted that the study was limited by the small group size and the relatively short duration of exposure.]

The promoting activity of RF radiation at 94 GHz was tested in groups of 27–55 female SENCAR mice previously initiated by dorsal application of DMBA at 10 nmol (2.56 µg). In a first experiment, 2 weeks after initiation, restrained mice were dorsally exposed once for 10 seconds to RF-EMF as follows: group 1 was exposed to millimetre wavelength (MMW) continuous wave far-field RF radiation (94 GHz, 1.0 W/cm²) and group 2 was exposed to infrared radiation at 1.5 W/cm². Both exposures led to similar skin heating (13–15 °C). Mice in group 3 were sham-exposed. In the positive-control group, initiated mice received the promoter TPA. After 23 weeks, the incidence and multiplicity of skin tumours was found to be similar in mice exposed to RF radiation, infrared radiation or sham-irradiated. TPA significantly increased both incidence and multiplicity of skin tumours compared with the other groups. [The Working Group noted that the importance of these findings was diminished by the very limited exposure to RF radiation.] In a second experiment, the effects of repeated exposure to RF radiation (333 mW/cm²) or infrared radiation (600 mW/cm²) for 10 seconds, twice per week, for 12 weeks, on skin-cancer promotion or co-promotion together with TPA were investigated. Groups of 50 female SENCAR mice were initiated with DMBA as above, and promotion treatment was started 2 weeks later. Group 1 was exposed to DMBA and sham-exposed; group 2 was exposed to DMBA + TPA and sham-exposed; group 3 was not initiated, exposed to TPA, and sham-exposed; group 4 was exposed to DMBA plus RF radiation at 333 mW/cm²; group 5 was exposed to DMBA plus RF radiation at 333 mW/cm² plus TPA; group 6 was exposed to DMBA plus infrared radiation at 600 mW/cm²; group 7 was exposed to DMBA plus infrared radiation at 600 mW/cm² plus TPA; and group 8 was sham-exposed only. The study was terminated 25 weeks after initiation. TPA promotion increased the incidence and multiplicity of skin tumours. Exposure to RF or infrared radiation did not

Table 3.2 Initiation–promotion studies in experimental animals exposed to radiofrequency radiation

Species, strain (sex) Tumour initiator Duration Reference	Dosing regimen Animals/group at start	Incidence of tumours	Significance	Comments
Mouse, CBA/S (F) X-ray ionizing radiation, 4–6 MV (4 Gy as three 1.33 Gy fractions at 1-wk intervals) on wk 1 78 wk Heikkinen et al. (2001)	902.5 MHz (continuous NMT900), SAR, 1.5 mW/g; 902.4 MHz (pulsed GSM, 217 Hz), SAR, 0.35 mW/g; sham; and cage controls Time averaged input power: 6.1 ± 0.8 W for continuous RF group and 1.3 ± 0.1 W for pulsed RF group 1.5 h/d, 5 d/wk, 78 wk 50/group	Lymphoma (cage, sham, NMT and GSM): 0/50, 12/50, 12/50, 10/50	NS	Restrained exposure Full histopathology
Mouse, ICR (F) DMBA (dermal): 100 µg/100 µL acetone/mouse on wk 1 20 wk Imaida et al. (2001)	1.49 GHz TDMA signal (50 pulses/s), near-field 90 min/d, 5 d/wk, 19 wk (on wk 2) SAR (skin): 2.0 mW/g, SAR (wb, av): 0.084 mW/g Group 1: DMBA + RF-EMF ($n = 48$) Group 2: DMBA + sham ($n = 48$) Group 3: DMBA + TPA ($n = 30$) Group 4: DMBA + cage control ($n = 30$)	Skin squamous cell papilloma or carcinoma (combined), tumour multiplicity 0/48, 0 0/48, 0 29/30*, $18.8 \pm 13.4^*$ 1/30, 0.1 ± 0.5	NS NS * $P < 0.001$ -	
Mouse, SENCAR (F) DMBA (dermal): 10 nmol/200 µL acetone on wk 1 Exp. 1 and Exp. 2: 25 wk Mason et al. (2001)	Exp. 1: 94 GHz MMW CW far-field or IR heating, single skin exposure of 10 s (on wk 3); TPA, $2 \times$ /wk for 23 wk Group 1: DMBA + 1.0 W/cm ² MMW ($n = 55$) Group 2: DMBA + 1.5 W/cm ² IR for 10 s ($n = 55$) Group 3: DMBA + sham ($n = 55$) Group 4: DMBA + TPA ($n = 27$) Exp. 2: MMW or IR, skin exposure of 10 s, $2 \times$ /wk, 12 wk; TPA, $2 \times$ /wk for 23 wk Group 1: DMBA + sham Group 2: DMBA + sham + TPA Group 3: sham + TPA Group 4: DMBA + 333 mW/cm ² MMW Group 5: DMBA + MMW + TPA Group 6: DMBA + 600 mW/cm ² IR Group 7: DMBA + IR + TPA Group 8: sham 50/group	Exp. 1: Comparable skin tumour incidence and multiplicity in sham-, MMW- or IR-exposed groups. TPA increased incidence and multiplicity of skin tumours. Exp. 2: TPA promotion led to increased incidence and multiplicity of DMBA-induced skin tumours; exposure to MMW or IR did not further increase the incidence or multiplicity of DMBA + sham or DMBA + TPA + sham-induced skin tumours.	No SAR given	

Table 3.2 (continued)

Species, strain (sex) Tumour initiator Duration Reference	Dosing regimen Animals/group at start	Incidence of tumours	Significance	Comments
Mouse, ICR (M) DMBA (dermal): 100 µg/100 µL acetone/mouse on wk 1 20 wk Huang et al. (2005)	849 MHz CMDA signal or 1 763 MHz CMDA signal 2 × 45 min/d (15-minute interval between exposures), 5 d/wk, 19 wk (on wk 2) SAR (wb, av): 0.4 mW/g Group 1: DMBA + TPA Group 2: DMBA + sham Group 3: DMBA + 849 MHz Group 4: DMBA + 1 763 MHz 20/group	No skin tumours in RF-EMF-exposed groups. Skin tumours (95%) in DMBA + TPA group only.	NS	Free-moving mice were exposed in a reverberation chamber. The short duration of daily RF- EMF-exposure is questionable.
Mouse, Swiss (M) DMBA (dermal): 100 µg on wk 1 18 wk Paulraj & Behari (2011)	112 MHz, AM at 16 Hz (1.0 mW/cm ² ; SAR, 0.75 mW/g) or 2 450 MHz (0.34 mW/cm ² ; SAR, 0.1 mW/g) 2 h/d, 3 d/wk, 16 wk (on wk 3) Group 1: control Group 2: DMBA Group 3: DMBA + 112 MHz Group 4: DMBA + 2 450 MHz Group 5: 112 MHz Group 6: 2 450 MHz Group 7: DMBA + croton oil 18/group	No skin tumours in any group, except in group 7		Small group size and short duration of exposure
Rat, F344 (M, F) ENU <i>in utero</i> , 4 mg/kg bw iv on GD 18 24 mo Adey et al. (1999)	836.55 MHz, NADC-modulated Far-field: GD 19–PND 21 (weaning) 2 h/d, 7 d/wk Near-field: starting on PND 33/35 2 h/d (8 × 7.5 min field on/off), 4 d/wk, 22 months SAR (brain): 1.1–1.6 mW/g SAR (wb, av): 1.8–2.3 mW/g	CNS tumours Total [%] Group 1: sham/sham, n = 30 M + 30 F Group 2: sham/RF, n = 30 M + 30 F Group 3: ENU/sham, n = 30 M + 30 F Group 4: ENU/RF, n = 30 M + 26 F	Brain [%] 8.3 3.3 15.0 3.6 - NS - NS	Ferris wheel/restrained exposure 25 (M) or 20 (F) sections per brain

Table 3.2 (continued)

Species, strain (sex) Tumour initiator Duration Reference	Dosing regimen Animals/group at start	Incidence of tumours	Significance	Comments	
Rat, Sprague-Dawley (F) B[a]P, 2 mg s.c. 160 d Chagnaud et al. (1999)	900 MHz, GSM-modulated, far-field (55 or 200 μW/cm ²)	Small palpable tumours (sarcomas) detectable from d 90–100 onwards.		Poor and confusing description of experiment and results. The authors stated that tumour onset was “slightly different” (<i>p</i> = 0.05) in the sham-exposed group and one of the exposed groups (55 μW/cm ² , 40 days).	
	SAR (wb, av): 75 or 270 mW/kg	Tumour incidence not reported. No consistent pattern of differences in time to tumour or survival.	NS		
	Exposure started on d 20, 40 or 75 after B[a]P initiation				
	2 h/d, 5 d/wk, 2 wk				
	Group	RF (μW/cm ²)	No. of rats		No. of days after B[a]P initiation
	1, 2	55	17/17		20
	3, 4	55	18/18		40
	5, 6	55	14/17		75
	7, 8	200	8/9		40
	9	cage control	6		-
Rat, F344 (M, F) ENU <i>in utero</i> , 4 mg/kg bw i.v. on GD 18 24 mo Adex et al. (2000)	836.55 MHz, NADC-modulated Far-field: GD 19–PND 21 (weaning) 2.6 ± 0.50 mW/cm ² Near-field: PND 31–731/734 2 h/d (8 × 7.5 min field on/off), 4 d/wk SAR (brain): 1.1–1.6 mW/g SAR (wb, av): 1.8–2.3 mW/g	Primary CNS tumours Total [%] 1.1 4.4 22.2 17.8 14.4 Group 1: sham/sham, <i>n</i> = 45 M + 45 F Group 2: sham/RF, <i>n</i> = 45 M + 45 F Group 3: ENU/sham, <i>n</i> = 45 M + 45 F Group 4: ENU/RF, <i>n</i> = 38 M + 52 F Group 5: ENU/cage control, <i>n</i> = 45 M + 45 F Group 6: cage control, <i>n</i> = 45 M + 45 F	- NS - NS	Ferris wheel/restrained exposure 25 (M) or 20 (F) sections per brain	

Table 3.2 (continued)

Species, strain (sex) Tumour initiator Duration Reference	Dosing regimen Animals/group at start	Incidence of tumours	Significance	Comments
Rat, Sprague-Dawley (M, F) ENU <i>in utero</i> , on GD 15, 0.2.5 or 10 mg/kg bw i.v. 24 mo Zook & Simmons (2001)	860 MHz, PW or CW, near-field 6 h/d, 5 d/wk, 22 months (starting on PND 57) SAR (brain, av): 1.0 ± 0.2 mW/g SAR (wb, av): 0.27–0.42 mW/g 15 groups, 30 M + 30 F/group Group ENU RF (mg/kg bw) 1 0 PW 2 0 Sham 9 0 CW 10 0 Sham 13 0 Cage control 5 2.5 PW 6 2.5 Sham 7 2.5 CW 8 2.5 Sham 11 2.5 CW 12 2.5 Sham 14 2.5 Cage control 3 10.0 PW 4 10.0 Sham 15 10.0 Cage control	No evidence that PW or CW increased the incidence of tumours in any studied tissues or promoted cranial or spinal or spinal nerve-cord tumours initiated by ENU. No. of rats with brain tumours (M + F combined) All Multiple Astrocytoma Oligodendroglioma Mixed glioma 5 0 5 0 0 NS 3 0 3 0 0 - 3 0 2 1 0 NS 5 0 5 0 0 - 6 0 5 1 0 0 7 2 4 2 4 NS 9 1 3 2 5 - 9 0 2 6 1 NS 10 1 5 5 1 - 3 0 0 3 3 NS 6 0 3 1 3 - 36 15 0 26 32 NS 35 12 3 19 26 - 41 9 2 24 25	NS (overall)	Restrained exposure of the head in tube (Ferris wheel) GLP Tissues studied histologically included brain (18–26 step sections [1 mm/brain), spinal cord, trigeminal nerves, lungs, liver, heart, kidneys, spleen, adrenal, pituitary and thyroid glands, and any gross lesions, including all neoplasms.
Rat, Sprague-Dawley (F) DMBA, 50 mg/kg bw by gavage 259–334 d Bartsch et al. (2002)	900 MHz GSM signal (217 Hz pulsed; pulse width, 577 µs) Far-field, 100 µW/cm² ± 3 dB; SAR (wb, av), 17.5–70 mW/kg 23 h/d, 7 d/wk Two groups each in three similar experiments performed over 3 years, 60/ group Exp. 1: Sham Exp. 1: Exposed Exp. 2: Sham Exp. 2: Exposed Exp. 3: Sham Exp. 3: Exposed	Mammary-gland tumours Median tumour latency (d) Malignant Benign 145 316 278 310 95 > 265 95 221 216 293 195 321	Cumulative incidence of tumours [%] Last day of observation Malignant Benign 79 90 82 91 84 38 94 60 91 89 81 92	Malignant tumours were adenocarcinomas Benign - NS P = 0.009 (retardation) - NS - NS NS NS

Table 3.2 (continued)

Species, strain (sex) Tumour initiator Duration Reference	Dosing regimen Animals/group at start	Incidence of tumours	Significance	Comments
Rat, F344 (M, F) ENU <i>in utero</i> , 4 mg/kg bw i.v. on GD 18 24 mo Shirai et al. (2005)	1 439 MHz TDMA signal (near-field) 90 min/d, 5 d/wk, wk 5–109 (offspring) SAR (brain): 0.67, 2.0 mW/g SAR (wb, av): < 0.4 mW/g	Pituitary tumours lower in ENU/high group	$P < 0.01$	Tube exposure/restrained Full histopathology ~10 sections/brain
		CNS tumours [%]		
		Brain		
		M/F		Spinal cord
		0/0		M/F
	Group 1: cage control	18/18		0/0
	Group 2: ENU control	24/30		2/6
	Group 3: ENU/sham	30/18	-	2/4
	Group 4: ENU/low (0.67 mW/g)	22/16	NS	0/6
	Group 5: ENU/high (2.0 mW/g)		NS	4/4
Rat, Sprague-Dawley (F) DMBA, 35 mg/kg bw by gavage on d 1 26 wk Yu et al. (2006)	900 MHz GSM; SAR (wb, av): 0.44, 1.33, 4.0 mW/g 4 h/d, 5 d/wk, 26 wk (starting on d 2)	No significant differences in incidence, latency, multiplicity or size of mammary-gland tumours between sham- and RF-exposed groups		
		Mammary-gland tumours (all) (%)		
		Mammary-gland adenocarcinoma (%)		
		Mammary-gland tumours (%)		
		Benign mammary-gland tumours (%)		
		Mammary-gland hyperplasia (%)		
	Group 1: cage control	60*		14
	Group 2: sham	45		29
	Group 3: 0.44 mW/g	38		24
	Group 4: 1.33 mW/g	41		15
	Group 5: 4.0 mW/g	43		24
Rat, F344 (M, F) ENU <i>in utero</i> , 4 mg/kg bw i.v. on GD 18 24 mo Shirai et al. (2007)	1.95 GHz WCDMA signals for IMT-2000 cellular system (near-field) 90 min/d, 5 d/wk, wk 5–109 (offspring) SAR (brain): 0.67, 2.0 mW/g SAR (wb, av): ≤ 0.464 mW/g	Skin fibroma and large granular lymphocytic leukaemia incidences lower in ENU/high group	$P < 0.05$	Tube exposure/restrained Full histopathology ~10 sections/brain
		CNS tumours [%]		
		Brain		
		M/F		Spinal cord
		2/2		M/F
	Group 1: cage control	16/14		0/0
	Group 2: ENU/cage control	8/10		2/0
	Group 3: ENU/sham	16/10	-	2/0
	Group 4: ENU/low (0.67 mW/g)	16/22	NS	0/0
	Group 5: ENU/high (2.0 mW/g)		NS	2/0

Table 3.2 (continued)

increase tumour incidence or multiplicity when compared with DMBA-treated sham-exposed controls. Exposure to TPA and RF radiation or TPA and infrared radiation did not increase the incidence of tumours or tumour multiplicity when compared with TPA controls. The authors concluded that MMW did not promote or co-promote skin tumorigenesis ([Mason et al., 2001](#)).

Starting at 20, 40, or 75 days after treatment with a single subcutaneous dose of 2 mg of benzo[a]pyrene, groups of 8–18 female Sprague-Dawley rats were exposed to GSM RF radiation at a wbSAR of 75 mW/kg for 2 hours per day, 5 days per week, for 2 weeks. An additional group was exposed to the GSM signal at a wbSAR of 270 mW/kg starting 40 days after the treatment with benzo[a]pyrene. For each GSM-exposed group, a sham-exposure group was included, resulting in a total of eight groups. The study was terminated at approximately 160 days after the treatment with benzo[a]pyrene. Small palpable tumours (sarcomas) were detectable from days 90 to 100. No consistent pattern of differences in time to tumour development or survival was observed between groups ([Chagnaud et al., 1999](#)). [The Working Group noted that the value of this study was diminished by the very limited exposure, the small group sizes, and the absence of histopathology.]

3.2.2 Lymphoma model

CBA/S mice are prone to develop lymphomas after exposure to ionizing radiation. In this study, groups of 50 female CBA/S mice (age, 3–5 weeks) (except the cage-control group) received whole-body exposure to ionizing radiation (X-rays, 4–6 MV, 4 Gy, delivered as three equal fractions of 1.33 Gy at intervals of 1 week) at the beginning of the study, followed by exposure to RF radiation for 1.5 hours per day, 5 days per week, for 78 weeks. A first “X-ray plus RF” group was exposed to continuous NMT900 (Nordic Mobile Telephony

900)-type frequency-modulated RF radiation at a frequency of 902.5 MHz and a nominal average SAR of 1.5 mW/g. A second “X-ray plus RF” group was exposed to pulsed GSM-type RF radiation (carrier-wave frequency, 902.4 MHz; pulse frequency, 217 Hz) at a nominal average SAR of 0.35 mW/g. An X-ray-exposed control group received sham exposure to RF radiation. Exposure to RF radiation did not significantly increase the incidence of tumours compared with the sham-exposed group ([Heikkinen et al., 2001](#)).

3.2.3 Mammary-gland tumour model

Groups of 60 female Sprague-Dawley rats (Hsd:SD) (age, 51 days) were given DMBA as a single intragastric dose of 50 mg/kg bw. On the same day, the rats were sham-exposed or exposed to RF radiation as a GSM signal at 900 MHz (pulse, 217 Hz) for 23 hours per day, 7 days per week, for 259–334 days. Over 3 years, three identical experiments with group-housed rats were performed. At the beginning of each experiment, wbSARs ranged from 32.5 to 130 mW/kg; wbSARs at 11 months ranged from 15 to 60 mW/kg; on average, wbSARs ranged from 17.5 to 70 mW/kg. Rats were killed when mammary-gland tumours reached 1–2 cm in diameter, and tumours were evaluated histopathologically. The incidence of benign or malignant mammary-gland tumours did not differ between sham-exposed and exposed groups. A statistically significant delay in the appearance of mammary-gland adenocarcinoma was seen in RF-exposed rats in the first experiment; this effect was not confirmed in the second or third experiment ([Bartsch et al., 2002](#)). [The Working Group noted the lack of reproducibility in tumour response between the three experiments.]

Two experiments were performed with the same GSM signal at 900 MHz as mentioned above, but with different intensities. In both experiments, groups of 16 female Sprague-Dawley rats were

sham-exposed or exposed to GSM RF radiation for 9 weeks, starting 10 days after administration by gastric intubation of 10 mg of DMBA at the age of 55 days, and were observed for an additional 3 weeks. In the first experiment, groups were exposed at wbSAR 0 (sham), 1.4, 2.2, or 3.5 mW/g and the authors reported that mammary-gland tumours developed more rapidly in rats exposed to signals at wbSAR 1.4 and 2.2 mW/g compared with controls. In the second experiment, groups were exposed at wbSAR 0 (sham), 0.1, 0.7, or 1.4 mW/g, and a decreased incidence of malignant mammary-gland tumours was seen in the group exposed to the signal at a wbSAR of 1.4 mW/g. Overall, no differences in the latency, multiplicity, or volume of mammary-gland tumours were observed ([Anane et al., 2003](#)). [The Working Group noted that the value of this study was reduced by the lack of reproducibility between exposure to RF radiation and mammary-gland tumour responses.]

Groups of 99–100 female Sprague-Dawley rats (age, 48 days) were given DMBA as a single oral dose of 35 mg/kg bw, followed by sham-exposure or exposure to RF radiation as a GSM signal at 900 MHz, for 4 hours per day, 5 days per week, for 26 weeks, in a Ferris wheel/tube-restrained system. Values for wbSAR were 0 (sham), 0.44, 1.33, and 4.0 mW/g. A cage-control group was also included. No differences in body weight, or in the incidence, latency to onset, multiplicity, or size of mammary-gland tumours were seen in this study ([Yu et al., 2006](#)).

In an experiment with a design very similar to that of [Yu et al. \(2006\)](#), groups of 100 female Sprague-Dawley rats (age, 46–48 days) were given DMBA as a single oral dose of 17 mg/kg bw, followed 1–2 days later by sham-exposure or exposure to RF radiation as a GSM signal at 900 MHz (pulse, 217 Hz), for 4 hours per day, 5 days per week, for 6 months, in a Ferris wheel/tube-restrained system. Values for wbSAR were 0 (sham), 0.4, 1.3, and 4.0 mW/g. A cage-control group was also included. Exposure to RF radiation

had no effect on survival or body weight. When compared with the sham-exposed control group, the group at 4.0 mW/g demonstrated a statistically significant increase in the number of rats with malignant mammary-gland tumours (mainly adenocarcinomas) and a significant decrease in the number of rats with benign mammary-gland tumours ([Hruby et al., 2008](#)). [The Working Group noted that incidences of mammary-gland cancer were similar in the group at 4.0 mW/g and in the cage-control group.]

3.2.4 Brain-tumour model

Groups of pregnant F344 rats received *N*-ethyl-*N*-nitrosourea (ENU) as a single intravenous dose at 4 mg/kg bw on day 18 of gestation. From day 19 of gestation to postnatal day 21, the pregnant dams and offspring were exposed to far-field RF radiation as an NDAC (North American Digital Cellular)-modulated signal at 836.55 MHz for 2 hours per day, 7 days per week. On postnatal day 33/35, the offspring were sham-exposed or exposed to intermittent near-field RF radiation, 2 hours (8 × 7.5 minutes field on/off) per day, 4 days per week. The total duration of the near-field plus far-field exposure was 24 months. The head region of each rat was exposed to near-field RF radiation in a Ferris wheel/tube-restrained system. Calculated SAR values in the brain ranged from 1.1–1.6 mW/g. The study included four groups: group 1 was sham-exposed (30 males, 30 females); group 2 was exposed to RF radiation (30 males, 30 females); group 3 was ENU/sham-exposed (30 males, 30 females); and group 4 was ENU/RF-exposed (30 males, 26 females). No statistically significant differences in the incidence of tumours of the brain or spinal cord were observed in the sham-exposed and RF-exposed groups ([Adey et al., 1999](#)).

The same laboratory performed a second study with a similar design in pregnant F344 rats exposed to ENU, the offspring of which then became treatment cohorts in six groups. Group

1 was sham-exposed (45 males and 45 females per group); group 2 was RF-exposed (45 males and 45 females per group); group 3 was ENU/sham-exposed (45 males and 45 females per group); group 4 was ENU/RF-exposed (38 males, 52 females); group 5 was exposed to ENU and served as cage control (45 males and 45 females per group); and group 6 served as cage control (45 males and 45 females per group). Treatment with ENU on day 18 of gestation and exposure to far-field RF radiation ($2.6 \pm 0.50 \text{ W/cm}^2$) from day 19 of gestation to postnatal day 21 was identical to that described in [Adey et al. \(1999\)](#). Sham exposure or exposure to near-field RF radiation (836.55 MHz, “balanced speech” modulation; brain SAR, 1.1–1.6 mW/g) for 2 hours per day, 4 days per week, began on postnatal day 31. The total duration of the near-field plus far-field exposure was 24 months. No statistically significant differences were identified in the incidence or histological type of tumours of the brain and spinal cord in the RF-exposed and sham-exposed groups ([Adey et al., 2000](#)).

Pregnant F344 rats received ENU as a single intravenous dose at 4 mg/kg bw on day 18 of gestation. Offspring were randomized into groups of 50 males and 50 females as follows: group 1 was a cage-control group; group 2 was exposed to ENU only; group 3 was exposed to ENU and sham-exposed to RF radiation; group 4 was exposed to ENU and exposed to RF radiation at a low level (brain averaged SAR, 0.67 mW/g); and group 5 was exposed to ENU and exposed to RF radiation at a high level (brain averaged SAR, 2.0 mW/g). Offspring received “head-only” exposure to near-field RF radiation (1439 MHz, TDMA signal), 90 minutes per day, 5 days per week, until age 104 weeks. Exposure to TDMA-modulated RF radiation had no effect on the survival or body weight of rats treated with ENU. Comparisons of the incidences of tumours of the brain and spinal cord in rats treated with ENU did not reveal any statistically significant effects

of exposure to TDMA RF radiation ([Shirai et al., 2005](#)).

A second study was performed by the same laboratory, with a design that was essentially identical to that described in [Shirai et al. \(2005\)](#). Pregnant F344 rats received ENU as a single intravenous dose at 4 mg/kg bw on day 18 of gestation. Offspring were randomized into groups of 50 males and 50 females as follows: group 1 was a cage-control group; group 2 was exposed to ENU only; group 3 was exposed to ENU and sham-exposed; group 4 was exposed to ENU and exposed to RF radiation at a low level (brain averaged SAR, 0.67 mW/g); and group 5 was exposed to ENU and exposed to RF radiation at a high level (brain averaged SAR, 2.0 mW/g). Offspring received “head-only” exposure to near-field RF radiation as a WCDMA (wide-band code-division multiple access) signal at 1.95 GHz from cell phones for IMT-2000 (International Mobile Telecommunication) cellular systems, for 90 minutes per day, 5 days per week, until age 104 weeks. Exposure to RF radiation had no effect on the survival or body weight of rats treated with ENU. Comparisons of the incidences of tumours of the brain and spinal cord in rats treated with ENU did not reveal any statistically significant effects of exposure to WCDMA RF radiation ([Shirai et al., 2007](#)).

Pregnant Sprague-Dawley rats received ENU by intravenous injection at a dose of 0, 2.5 or 10 mg/kg bw on day 15 of gestation. Beginning on postnatal day 57, groups of offspring were sham-exposed or exposed to RF radiation in a Ferris wheel/tube-restrained system for 6 hours per day, 4 days per week for 22 months. RF metrics tested included a pulsed-wave signal (PW) at 860 MHz and a continuous-wave signal (CW) at 860 MHz. Average brain SAR was 1.0 mW/g for both. Including cage controls, the entire experiment consisted of 15 groups of 30 males and 30 females. Key groups included ENU plus sham-exposure; ENU plus PW exposure; and ENU plus CW exposure. Detailed data regarding

treatment and tumour incidences are tabulated in [Table 3.2](#). The results of this study provided no statistically significant evidence that exposure to PW or to CW increased the incidence of tumours in any of the tissues studied, or that it promoted the induction of cranial or spinal-cord tumours initiated by ENU ([Zook & Simmens, 2001](#)).

In a follow-up study by the same authors, pregnant female Sprague-Dawley rats received ENU as a single intravenous dose at 6.25 or 10.0 mg/kg bw on day 15 of gestation. Offspring were randomized by ENU-dose into groups of 90 males and 90 females and then, as in the previous study, exposed to RF radiation from postnatal day 52 ± 3 as a PW signal at 860 MHz, in a Ferris wheel/tube-restrained system, 6 hours per day, 5 days per week, at an average brain SAR of 1.0 mW/g. The study was terminated when the offspring were aged 24 months. Each group included three cohorts and the study was conducted in three phases, each containing six groups. Groups 1, 2, and 3 received ENU at 6.25 mg/kg bw plus: (i) sham exposure to RF radiation (group 1); or (ii) exposure to RF radiation (group 2); or (iii) no treatment (cage control; group 3); while groups 4, 5 and 6 received ENU at 10.0 mg/kg bw plus: (i) sham exposure to RF radiation (group 4); or (ii) exposure to RF radiation (group 5); or (iii) no treatment (cage control; group 6). Necropsies were performed monthly on selected rats from each group, beginning at age 171 days. Exposure to RF radiation had no statistically significant effects on the survival or body weight of rats treated with ENU. Histopathological evaluation of tissues from the nervous system provided no evidence that exposure to the RF signal affected the incidence, multiplicity or latency of any type of neurogenic tumour ([Zook & Simmens, 2006](#)).

3.3 Co-carcinogenesis

See [Table 3.3](#)

To evaluate the possible effects of RF-radiation exposure on colon carcinogenesis, three groups

of 26–32 male and 26–32 female BALB/c mice (age, 4–5 weeks) were given dimethylhydrazine (DMH) as a subcutaneous injection at a dose of 15 mg/kg bw every week for 14 weeks, and subsequently at a dose of 20 mg/kg bw for 8 weeks. Starting 3 weeks after the first injection of DMH, the mice were either sham-exposed, exposed to RF radiation at 2450 MHz (SAR, 10–12 mW/g) for 3 hours per day, 6 days per week, for 5 months, or given weekly intraperitoneal injections of TPA at 2 µg per mouse for 10 weeks. Mice in the control group were given a subcutaneous injection of saline solution only. The incidences of tumours of the colon were similar in all groups treated with DMH ([Wu et al., 1994](#)).

The possible effects of exposure to RF radiation on tumorigenesis were investigated in the offspring of pregnant female B6C3F₁ mice treated with ENU. Exposure of pregnant mice to RF radiation as a UMTS signal at 1966 MHz was initiated on day 6 of gestation, and was continued throughout pregnancy and for 2 years post-parturition. Pregnant mice were also intraperitoneally injected with ENU at a dose of 40 mg/kg bw on day 14 of gestation. Groups of 54–60 offspring were exposed to UMTS RF radiation at an intensity of 0, 4.8, or 48 W/m² for 20 hours per day, 7 days per week. Group 1 served as a cage control; group 2 was exposed to ENU only; group 3 was sham-exposed only; group 4 was exposed to ENU plus UMTS RF radiation at 4.8 W/m²; and group 5 was exposed to UMTS RF radiation at 48 W/m². Comparable incidences of tumours were seen in the groups that were not exposed to ENU. In groups exposed to ENU, UMTS RF radiation increased the incidence of bronchiolo-alveolar carcinoma and hepatocellular adenoma ([Tillmann et al., 2010](#)). [The Working Group noted that this experimental model had not been used previously in other studies of hazard identification, and its concordance with the human carcinogenic response is unknown.]

Three groups of 45–49 transgenic female K2 mice overexpressing the human ornithine

decarboxylase (*ODC*) gene and their wild-type littermates were exposed to a combination of ultraviolet (UV) radiation and pulsed RF radiation. The UV dose was 240 J/m² delivered three times per week for 52 weeks. The mice were sham-exposed or exposed to RF radiation for 1.5 hours per day, 5 days per week, for 52 weeks. One group of mice was exposed to D-AMPS (digital advanced mobile phone system)-modulated RF radiation at 849 MHz; a second group was exposed to GSM RF radiation at 902.4 MHz; and a third group was sham-exposed. Nominal average SAR for both exposed groups was 0.5 mW/g. A cage-control group of 20 mice was included. There were no differences in the cumulative survival or body weight in groups exposed to UV, regardless of exposure to RF radiation. UV exposure induced macroscopic tumours of the skin in 12% of the non-transgenic mice and in 37% of the transgenic mice. Exposure to RF radiation had no effect on the induction of squamous cell carcinoma of the skin in either transgenic or wild-type mice ([Heikkinen et al., 2003](#)).

A study evaluated the possible effects of exposure to RF radiation on tumorigenesis induced by the mutagen 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5*H*)-furanone (MX), a by-product of water disinfection. Groups of 72 female Wistar rats (age, 7 weeks) were given drinking-water containing MX at a daily average dose of 0 (cage-control) or 1.7 mg/kg bw for 104 weeks, and were then sham-exposed or exposed to pulsed RF radiation at 900 MHz (wbSARs of 0 [sham control], 0.3 or 0.9 mW/g) in restrainers for 2 hours per day, 5 days per week, for 104 weeks. Exposure to RF radiation had no statistically significant effects on mortality or body weight of rats treated with MX. Compared with the MX-treated sham-exposed control group [but not the cage control group], a statistically significant increase in the incidence of combined vascular tumours (haemangiomas, haemangiosarcomas and lymphangiomas combined) was

observed in the mesenteric lymph nodes of the group treated with MX and RF radiation at a high intensity (wbSAR, 0.9 mW/g). Exposure to RF radiation had no significant effect on the incidence of tumours in any other tissue ([Heikkinen et al., 2006](#)). [The Working Group noted that this experimental model had not been used previously in other hazard-identification studies, and its concordance with human carcinogenic response is unknown.]

Groups of 40 male BALB/c mice received 10 µL of a 5% solution of benzo[*a*]pyrene by skin painting on alternate days for 5 months, and were exposed to RF radiation as microwaves at 2450 MHz for 2 hours per day, 6 days per week, in an anechoic chamber, according to two different protocols. In the pre-exposure protocol, the mice were exposed to microwave radiation at SARs of 0 mW/g (sham) or 2–3 mW/g for 1 or 3 months before application of benzo[*a*]pyrene. In the simultaneous-exposure protocol, groups of mice were exposed to RF radiation at SARs of 0 mW/g, 2–3 mW/g, or 6–8 mW/g concurrently with administration of benzo[*a*]pyrene. Pre-exposure or simultaneous exposure to microwave radiation at either SAR value accelerated the development of benzo[*a*]pyrene-induced skin cancer. A comparable acceleration of skin tumorigenesis was reported in benzo[*a*]pyrene-treated mice undergoing confinement stress for 1 or 3 months ([Szmigielski et al., 1982](#)). [The Working Group noted that the study design and experimental data from this paper were poorly presented and difficult to interpret.]

In a second study performed by the same group, six groups of 100 adult male BALB/c mice were painted with 10 µL of 1% benzo[*a*]pyrene on the interscapular region of the skin on alternate days for 6 months. Two different schedules of exposure to microwave radiation at 2450 MHz were used. In the first experiment, three groups were exposed to microwave radiation (mean wbSAR, 4 mW/g) for 2 hours per day, 6 days per week, for 1, 2 or 3 months before the start of

Table 3.3 Co-carcinogenicity studies in experimental animals exposed to radiofrequency radiation

Species, strain (sex) Carcinogen Duration Reference	Dosing regimen Animals/group at start	Incidence of tumours	Significance	Comments
Mouse, K2 (ODC-transgenic and wild-type) (F) UV radiation: 240 J/m ² , 3 × /wk, 52 wk 52 wk Heikkinen et al. (2003)	DAMPS-type (849 MHz) or GSM-type (902.4 MHz) RF, SAR: 0.5 mW/g 1.5 h/d, 5 d/wk, 52 wk Sham (ODC-transgenic + wild-type): 19 + 26; D-AMPS: 20 + 26; GSM: 22 + 27; and cage control: 12 + 8	Squamous-cell carcinoma of the skin: ODC-transgenic (sham): 6/19 ODC-transgenic (GSM): 5/21 ODC-transgenic (DAMPS): 8/20 Wild-type (sham): 2/26 Wild-type (GSM): 4/27 Wild-type (DAMPS): 4/26	NS	Restrained exposure Histopathological evaluation of all skin lesions
Rat, Wistar (F) 3-Chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone [MX], 1.7 mg/kg bw, drinking-water, 104 wk 104 wk Heikkinen et al. (2006)	GSM (900 MHz) PW (whole body) with wBSARs of 0.3 mW/g (low RF) or 0.9 mW/g (high RF), sham, cage control 2 h/d, 5 d/wk, 104 wk 72/group	Combined vascular tumours (haemangioma, haemangiosarcoma and lymphangioma) in the mesenteric lymph nodes (cage control, sham, low RF, high RF): 10/72 (14%), 3/72 (4%), 1/72 (1%), 11/72 (15%)	$P < 0.05$ (Fisher test), high RF vs sham	Complete histopathology No significant difference between high RF-radiation-exposed group and cage-control group
Mouse, BALB/c (M, F) DMH, 15 mg/kg bw per wk for 14 wk followed by 20 mg/kg bw per wk for 8 wk, i.p. 25 wk Wu et al. (1994)	2 450 MHz MW, 3 h/d, 6 d/wk, 5 mo, 10 mW/cm ² (SAR, 10–12 mW/g); sham exposure; or TPA promotion (i.p., 2 µg/wk, 10 wk) 3 wk after 1 st DMH injection A control group was i.p. injected with saline only 26–32/group	Colon tumours: DMH + sham: 13/28 (46%) DMH + MW: 13/26 (50%) DMH + TPA: 17/32 (53%) Control (saline only): 0/29 Protuberant or invasive colon tumours: DMH + sham: 13.9% DMH + MW: 16.3% DMH + TPA: 44.1%	- NS NS - NS $P < 0.05$	Single housing in small plexiglass cages during MW exposure Tumour bearing mice with total tumour areas > 5 mm ² : 71% in DMH + TPA-treated group ($P < 0.05$) vs 31% in DMH- and 31% in DMH + MW-treated groups.
Mouse, B6C3F ₁ (F) ENU, 40 mg/kg bw i.p., on GD 14 106 wk Tillmann et al. (2010)	1966 MHz, UMTS signal 20 h/d, 7 d/wk, 106 wk (starting on GD 6) 0 (sham), 4.8, 48 W/m ² SAR: variable Group 1: cage control Group 2: ENU Group 3: sham exposure Group 4: ENU + UMTS (4.8 W/m ²) Group 5: UMTS (48 W/m ²) 54–60/group	Bronchiolo-alveolar adenoma: group 4 (36/58, 62%) vs group 2 (27/60, 45%) Bronchiolo-alveolar carcinoma: group 4 (45/58, 78%) vs group 2 (33/60, 55%) Hepatocellular adenoma: group 4 (49/58, 85%) vs group 2 (30/60, 50%) Hepatocellular carcinoma: group 4 (30/58, 52%) vs group 2 (31/60, 52%) Comparable incidences of tumours in group 1, 3 and 5	NS $P < 0.05$ $P < 0.001$ NS	Tissues that were histopathologically evaluated included brain, lungs, liver, spleen, kidneys, mesenteric lymph nodes, and any gross lesions detected.

Significance Comments

[illegible]

benzo[a]pyrene application. In the second experiment, three groups were exposed to RF radiation (wbSAR, 0 mW/g [sham-exposed control], 2 mW/g, or 6 mW/g), 2 hours per day, 6 days per week, for 6 months, concurrently with exposure to benzo[a]pyrene. Irradiation by either schedule resulted in an acceleration in the development of benzo[a]pyrene-induced skin carcinoma and decreased the lifespan of the animals ([Szudziński et al., 1982](#)). [The Working Group noted that the study design and experimental data were poorly presented and difficult to interpret. Survival and tumour data from groups receiving pre-exposure to microwave radiation may be invalid due to the lack of concurrent sham-exposed controls.]

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4. OTHER RELEVANT DATA

Data on specific absorption rate (SAR) and distribution of radiofrequency (RF) radiation inside tissues and organs and at the subcellular level are presented elsewhere in this Volume (Section 1.3, Dosimetry).

4.1 Genetic and related effects

4.1.1 Humans

During the past decades, extensive research efforts have focused on determining the extent of DNA damage in eukaryotic and prokaryotic cells exposed to RF radiation. Several published reviews concluded that: (i) the existing data are not sufficiently strong to suggest that RF radiation is directly genotoxic; (ii) exposure to RF radiation probably does not enhance the damage induced by known genotoxic agents; and (iii) some of the reported “adverse effects” may be attributed to hyperthermia induced by RF radiation ([Brusick et al., 1998](#); [Verschaeve & Maes, 1998](#); [Moulder et al., 1999, 2005](#); [Heynick et al., 2003](#); [Meltz, 2003](#); [Vijayalaxmi & Obe, 2004](#); [Verschaeve, 2005](#); [Krewski et al., 2007](#); [Lai, 2007](#); [Vijayalaxmi & Prihoda, 2008](#); [Phillips et al., 2009](#); [Rüdiger, 2009a](#); [Verschaeve, 2009](#); [Verschaeve et al., 2010](#)). International organizations and expert scientific advisory groups in several countries, including Canada, France, the Netherlands, Sweden, the United Kingdom and the USA, have reached similar conclusions ([ICNIRP, 2009](#)).

This Section of the *Monograph* deals with studies on primary DNA damage in humans exposed occupationally or as mobile-phone users;

in these studies DNA damage was measured in peripheral blood lymphocytes and buccal cells by means of the alkaline or neutral single-cell gel electrophoresis assay (comet assay), which reveals alkali-labile lesions and single- and double-strand breaks in DNA, or by use of cytogenetic tests for chromosomal aberrations, micronucleus formation and sister-chromatid exchange (SCE). The studies reviewed below are summarized in [Table 4.1](#) and [Table 4.2](#) (with details of the exposure conditions).

(a) Peripheral blood lymphocytes

(i) Occupational exposure

[Garaj-Vrhovac et al. \(1990a\)](#) were the first to report an increased frequency of chromosomal aberrations in the form of chromatid and chromosome breaks, acentric fragments, dicentrics, rings and polycentric chromosomes, as well as micronuclei in 10 individuals employed in a radar service-station facility. The frequency of cells with chromosomal aberrations and micronuclei ranged from 1.6% to 31.5% and from 1.6% to 27.9%, respectively, in exposed subjects, while the corresponding values in controls were 1.8% and 1.5% [no range given].

In a study in Australia, [Garson et al. \(1991\)](#) collected lymphocytes from 38 radio linesmen,

Table 4.1 Genetic and related effects of radiofrequency radiation in peripheral blood lymphocytes of occupationally exposed individuals

End-point	No. of subjects	Occupation	Frequency	SAR or power density	Duration	Results	Reference
Aneuploidy	18	Air-traffic controllers; engineers	100 kHz to 300 GHz	-	10–27 yr	+	Othman et al. (2001)
CA	10	Radar maintenance workers	0.2 MHz to 26 GHz	0.010–50 mW/cm ²	8–25 yr	+	Garaj-Vrhovac et al. (1990a)
CA	38	Radio linesmen	400 kHz to 20 GHz	614 V/m	5 yr	-	Garson et al. (1991)
CA	6	Air-traffic radar-repairmen	1250–1350 MHz	0.01–20 mW/cm ²	16 yr	+ [after a 30-wk follow-up, total aberrations had decreased]	Garaj-Vrhovac et al. (1993)
CA	6	Transmission-antenna maintenance workers	450–900 MHz	NR	1 yr	-	Maes et al. (1995)
CA	20	Workers in telecommunication and radio-relay stations	8 GHz	1 mW/cm ²	6 yr (12 h/d)	+	Lalić et al. (2001)
CA	50	Air-traffic controllers, engineers	100 kHz to 300 GHz	NR	8–27 yr	+	Aly et al. (2002)
CA	49	Radio engineers	450–900 MHz	NR	2.3 yr (> 1 h/d)	-	Maes et al. (2006)
CA	10	Radar maintenance workers	1250–1350 MHz	0.010–20 mW/cm ²	7–29 yr	+	Garaj-Vrhovac & Orescanin (2009)
MN	10	Radar maintenance workers	0.2 MHz to 26 GHz	0.010–50 mW/cm ²	8–25 yr	+	Garaj-Vrhovac et al. (1990a)
MN	NR	Multiple occupations	1250–1350 MHz	0.01–20 mW/cm ²	15 yr	+	Fucić et al. (1992)
MN	12	Radar maintenance workers	1250–1350 MHz	0.01–20 mW/cm ²	13 yr	+	Garaj-Vrhovac (1999)
SB	40	Flight crew	NR	NR	5–18 yr	-	Cavallo et al. (2002)
SB	49	Radio engineers	450–900 MHz	NR	2.3 yr (> 1 h/d)	-	Maes et al. (2006)
SB	10	Radar maintenance workers	1250–1350 MHz	0.010–20 mW/cm ²	7–29 yr	+	Garaj-Vrhovac & Orescanin (2009)
SCE	50	Air-traffic controllers	100 kHz to 300 GHz	NR	8–27 yr	-	Aly et al. (2002)
SCE	49	Radio engineers	450–900 MHz	NR	2.3 yr (> 1 h/d)	-	Maes et al. (2006)

+ increase; -, no effect; CA, chromosomal aberration; d, day; h, hour; MN, micronucleus formation; NR, not reported; SAR, specific absorption rate; SB, DNA single- and double-strand breaks; SCE, sister-chromatid exchange; wk, week; yr, year

Table 4.2 Genetic and related effects of radiofrequency radiation in peripheral blood lymphocytes and buccal cells of mobile-phone users

End-point	No. of subjects	Frequency	SAR	Duration	Results	Reference
<i>Peripheral blood lymphocytes</i>						
CA	24	890–960 MHz	NR	2 yr	+	Gadhia et al. (2003)
CA	25	NR	0.1–1.9 W/kg	3–5 yr	+	Gandhi & Singh (2005)
MN	24	800–2000 MHz	0.6–1.6 W/kg	1–5 yr	+	Gandhi & Anita (2005)
SB	24	800–2000 MHz	0.6–1.6 W/kg	1–5 yr	+	Gandhi & Anita (2005)
SCE	24	890–960 MHz	NR	2 yr	+	Gadhia et al. (2003)
<i>Buccal cells</i>						
MN	25	NR	0.1–1.9 W/kg	3–5 yr	+	Gandhi & Singh (2005)
MN	85	NR	0.3–1.0 W/kg	2.3 yr (1 h/d)	+	Yadav & Sharma (2008)
MN	112	NR	NR	5–10 yr (3 h/wk)	–	Hintzsche & Stopper (2010)

+, increase; –, no effect; CA, chromosomal aberration; d, day; h, hour; MN, micronucleus formation; NR, not reported; SAR, specific absorption rate; SB, DNA single- and double-strand breaks; SCE, sister-chromatid exchange; wk, week; yr, year

who erected and maintained broadcasting, telecommunication and satellite RF-transmission towers, and found no increase in the frequency of chromosomal aberrations compared with the frequency in 38 controls working as clerical staff. In this study, exposure to RF radiation was at or below occupational limits for Australia.

[Fucić et al. \(1992\)](#) measured the surface area of micronuclei in lymphocytes of workers in multiple occupations exposed to pulsed microwaves, X-rays (< 25 mSv during the previous 2 years) and vinyl-chloride monomer (VCM; average concentration, 50 ppm). The sample size in each category was not mentioned in the paper. There were increased numbers of smaller micronuclei in individuals exposed to X-rays and VCM, indicating a clastogenic effect. Increased numbers of smaller as well as larger micronuclei were found in individuals exposed to microwaves, suggesting a dual role of this type of radiation, as clastogen and aneugen.

In a regular 30-week follow-up investigation of six individuals who were acutely exposed to pulsed-wave RF radiation of high power density at an air-traffic radar-repair station,

[Garaj-Vrhovac et al. \(1993\)](#) observed a decline in the total number of chromosomal aberrations.

A preliminary study conducted by [Maes et al. \(1995\)](#) involved six workers in charge of maintaining transmission antennae linked to a mobile-phone network, and six matched controls. No increase in the frequency of chromosomal aberrations was observed in the maintenance workers. The authors mentioned the limited size of the study and the fact that exposure to RF radiation was intermittent. They then extended the study to 49 professionally employed radio engineers working in the field, and 11 administrative staff. Some of these had participated in the earlier study. No differences between exposed and controls were observed with the alkaline comet assay, the assay for chromosomal aberration, or the test for SCE ([Maes et al., 2006](#)).

[Garaj-Vrhovac \(1999\)](#) examined 12 subjects employed in repair services for radar equipment and antennae, and reported frequencies of 8–23 micronuclei per 500 cells in exposed workers compared with 2–7 per 500 cells in control subjects; this difference was statistically significant.

[Lalić et al. \(2001\)](#) investigated 20 workers in telecommunication and radio-relay stations who were exposed to non-ionizing electromagnetic fields, and 25 subjects employed as X-ray technicians, nurses and engineers in radiology, exposed to ionizing radiation. The analysis indicated an increased frequency of chromosomal aberration in both groups. The incidence of dicentric chromosomes was higher in the group exposed to non-ionizing radiation than in the group exposed to ionizing radiation.

[Othman et al. \(2001\)](#) studied professional air-traffic controllers and engineers exposed to RF radiation emitted by different pieces of equipment at the workplace. In a first study, blood lymphocytes were collected from 18 workers and 5 unexposed controls (all males), and cultured for 72 hours. Fluorescence *in situ* hybridization (FISH) with repetitive α -satellite probes for chromosomes 7, 12, 17, and the heterochromatic region of the Y-chromosome, was used to determine the number of aneuploid cells. The results showed increased frequencies of monosomic cells containing a single copy of chromosome 7 (6.6%) or 17 (6.1%), and of cells lacking the Y-chromosome (8.4%); the corresponding values for the controls were 3.2%, 3.7% and 4.5%, respectively.

In a further study by the same group, [Aly et al. \(2002\)](#) examined lymphocytes from 26 air-traffic controllers, 24 engineers and 10 controls. Conventional cytogenetic techniques revealed an increase in the frequency of structural aberrations (2.7–5.3%) and numerical aberrations (8.9–9.3%) in exposed individuals relative to controls (0.8% and 3.2%, respectively). In subjects exposed to RF radiation, 90% of the cells were hypodiploid, i.e. showed loss of chromosomes. The frequency of SCE was also increased, but this increase did not reach statistical significance. [The Working Group noted that conventional cytogenetic techniques may be less reliable than the FISH technique for counting numerical aberrations.]

[Cavallo et al. \(2002\)](#) studied 40 airline pilots and flight technicians exposed to cosmic rays, electromagnetic fields from radar equipment, pollutants from jet-propulsion fluid etc. and 40 non-exposed individuals working on the ground. In the comet assay, visual examination of the results revealed a small increase in the frequency of DNA strand breaks in exposed individuals compared with ground staff, but this increase was not statistically significant.

[Garaj-Vrhovac & Orescanin \(2009\)](#) used the comet assay to measure DNA strand breaks and the test for sensitivity to bleomycin described by [Michalska et al. \(1998\)](#) to investigate genomic instability in 10 individuals working in radar-equipment and antenna-system services, and in 10 control subjects. In the latter method, the cells were treated with bleomycin (a drug used in clinical treatment of cancer) during the last 5 hours before harvesting after a culture period of 72 hours, to assess the incidence of chromosomal aberrations in the form of chromatid breaks. The results of the comet assay revealed increased DNA damage (tail length, 17.1 μm , and tail moment, 14.4, in the exposed individuals compared with 14.2 μm and 11.7, respectively, in the controls). The test for sensitivity to bleomycin showed a higher number of chromatid breaks (1.7 per cell in the exposed, compared with 0.5 per cell in the controls). All these differences were statistically significant.

[The Working Group noted the following limitations in the above-mentioned studies. Exposure assessment was poor or was not mentioned in many reports. The sample size in terms of number of individuals or number of cells analysed was not sufficient to allow robust statistical analysis. Except in one study, “blind” evaluation of microscope slides, and inclusion of positive controls (subjects or cells) while culturing the lymphocytes *in vitro*, was either not performed or not reported. Several investigations were conducted with blood samples collected from workers in one radar-service

facility in Croatia; it was unclear whether the same individuals had been included in more than one of these studies.]

[Although the reports from Australia ([Garson et al., 1991](#)) and Belgium ([Maes et al., 1995](#)) indicated no effect on the frequency of chromosomal aberrations from exposure to RF radiation, the Working Group noted that situations and exposure conditions in those countries may not have been comparable to those in other countries. Chromosomal changes are highly variable during carcinogenesis and are generally grouped into two categories: (i) reciprocal and balanced structural rearrangements resulting in translocations; and (ii) unbalanced and nonreciprocal structural or numerical changes in which genetic material may be lost or added: the latter can range from a single base pair to the entire chromosome. In the studies reviewed above, reciprocal and balanced structural rearrangements were either not observed or not reported in individuals exposed to RF radiation.]

(ii) *Personal exposure from mobile phones*

[Gadhia et al. \(2003\)](#) collected samples of peripheral blood from 24 users of digital mobile phones and 24 matched controls. Both groups comprised 12 nonsmokers/nondrinkers and 12 smokers/alcoholics [smokers consumed 10–15 cigarettes per day; data on alcohol consumption were not given]. Cytogenetic analysis of lymphocytes cultured for 72 hours indicated a significantly increased incidence ($P < 0.05$) of chromatid gaps and dicentric chromosomes among mobile-phone users who smoked and drank alcohol, but not in nonsmokers/nondrinkers. A significantly increased frequency ($P < 0.05$) of SCE was seen in mobile-phone users of both categories.

[Gandhi & Singh \(2005\)](#) studied G-banded chromosomes in lymphocytes (cultured for 72 hours) from 25 users of mobile phones and 25 non-users. There was a statistically significant increase in the frequency of aberrant metaphases,

including triploid chromosomes, acrocentric associations and centromere separation in lymphocytes of mobile-phone users (31.3%) compared with non-users (10.7%). In a subsequent study, [Gandhi & Anita \(2005\)](#) investigated DNA strand breaks by use of the comet assay in lymphocytes from 24 mobile-phone users and 10 controls. Unstimulated lymphocytes were also examined to record the frequency of micronuclei in 20 of those users and 8 non-users. In mobile-phone users, the frequency of damaged cells was 40%, with an average comet-tail length of 27 μm (determined by visual examination with a micrometer), while these values were lower in non-users, at 10% and 8 μm , respectively; both differences were highly significant. The total number of micronuclei was 100 in 40 000 cells in users, and 8 in 16 000 cells in non-users, *i.e.* 2.5% in the former and 0.5% in the latter ($P < 0.05$). [The Working Group noted that the observations reported by [Gandhi & Singh \(2005\)](#) and [Gandhi & Anita \(2005\)](#) were questioned by others ([Vijayalaxmi et al., 2007](#)), pointing out several inconsistencies and weaknesses in laboratory methods, data collection, exposure assessment, etc. in both publications.]

(b) *Buccal cells: personal exposure from mobile phones*

The oral cavity is within the range of RF emissions from mobile phones used at the ear. Hence, examination of the cells in this region is relevant to evaluation of genotoxicity. The oral mucosa has a rich blood supply and is relatively permeable. It has an outer layer of stratified squamous epithelium that is approximately 40–50 cell-layers thick. These exfoliating cells can easily be obtained by non-invasive procedures (oral swabs) from adults, adolescents and children. The turnover of these cells is estimated at 1–3 weeks ([Harris & Robinson, 1992](#)).

The frequency of micronuclei in exfoliated buccal cells has been investigated in mobile-phone users. [Gandhi & Singh \(2005\)](#) collected

buccal cells from 25 mobile-phone users and 25 non-users. The average frequencies of micronuclei (in %) were 0.82 ± 0.09 in users and 0.06 ± 0.003 in non-users ($P < 0.05$). [The Working Group noted the same limitations for this study as those mentioned above.]

[Yadav & Sharma \(2008\)](#) collected buccal cells from 85 mobile-phone users and 24 controls. In a total of 1000 cells from each donor, the frequency of micronuclei was determined, along with other indications of degeneration, i.e. karyolysis, karyorrhexis, “broken egg” effect, and binucleate cells. The mean frequency in users (10.7 per 1000 cells) was significantly higher than that in non-users (4.0 per 1000 cells). The changes in incidence of other end-points were not statistically significant. There was also a positive, albeit non-significant, correlation of the total number of micronuclei with increased duration of mobile-phone use.

[Hintzsche & Stopper \(2010\)](#) determined the frequency of micronuclei in buccal cells from 112 mobile-phone users and 13 non-users. Four patients receiving radiotherapy were included as positive controls, along with four healthy controls. The average frequency of micronucleus formation in users was not different from that in non-users. Also, there was no difference when the users were subdivided according to the number of hours of use per week and duration of use of up to 10 years. In contrast, the frequency of micronucleated cells in patients receiving radiotherapy was 131 ± 29.1 per 1000 cells. The authors mentioned that the larger number of individuals studied, the use of DNA-specific staining, and the genotypic variation in the study populations may have contributed to the discrepancy between their results and those of [Yadav & Sharma \(2008\)](#).

[The Working Group noted that counting of 2000 differentiated cells and 200 basal cells is recommended for studies using buccal cells, ([Thomas & Fenech, 2011](#)); this was not accomplished in the studies discussed above. Known confounding factors such as tobacco smoking

and alcohol consumption were mentioned in some of the studies, but in view of the limited sample size the influence of such factors on the observed abnormalities is difficult to determine.]

[The Working Group further noted that studies of genotoxicity in humans exposed to RF radiation have been carried out by a limited number of research groups; that methodological weaknesses were found in many studies; and that confounding factors were generally not addressed. Overall, although there were studies with positive results for genotoxicity associated with occupational exposure to RF radiation or with the use of mobile phones, the Working Group concluded that the available evidence was not strong enough to draw firm conclusions.]

4.1.2 Experimental systems: in vivo

The studies on experimental animals exposed to RF radiation were not uniformly clear in describing the rationale for choosing a specific dose.

(a) *Drosophila melanogaster*

Adult male fruit flies (*Drosophila melanogaster*) were exposed to RF radiation at either 146.34 MHz produced by a transmitter of 20 W, or 29.00 MHz produced by a transmitter of 300 W, for 12 hours ([Mittler, 1976](#)). Loss of the X or Y chromosomes, nondisjunction, and the induction of sex-linked recessive lethal mutations were investigated. There was no significant difference between exposed and non-exposed flies for any of these end-points.

In a subsequent study ([Mittler, 1977](#)), *D. melanogaster* were exposed to RF radiation at 98.5 MHz (field strength, 0.3 V/m) for 32 weeks. No significant difference in the incidence of sex-linked recessive lethal mutations was observed in the exposed group compared with the controls.

[Hamnerius et al. \(1979\)](#) examined the effect of exposure to RF radiation on somatic mutation of genes involved in eye pigmentation in

D. melanogaster. When embryos were exposed to continuous-wave RF radiation at 2450 MHz (average SAR, 100 W/kg) for 6 hours, no evidence of mutagenicity was found. The same investigators used the same test system to examine mutation frequency in *D. melanogaster* under different conditions of exposure for 6 hours: continuous-wave radiation at 2.45 GHz, pulsed-wave radiation at 3.1 GHz, and continuous-wave magnetic or electric fields at 27.12 MHz. Under none of these conditions was a change in mutation frequency observed ([Hamnerius et al., 1985](#)).

[Marec et al. \(1985\)](#) investigated the effect of repeated exposures to RF radiation on sex-linked recessive lethal mutations in *D. melanogaster* exposed to continuous-wave RF radiation at 2375 MHz (SAR values: 15 W/cm² for 60 minutes per day; or 20 W/cm² for 10 minutes per day; or 25 W/cm² for 5 minutes per day) for five consecutive days. The mutation frequency in the groups exposed to RF radiation was not significantly different from that in the control group.

In a series of studies from Greece, adverse effects were reported on the reproduction of *D. melanogaster* after exposure to RF radiation at non-thermal mobile-phone frequencies (900 or 1800 MHz). In these experiments commercially available mobile phones were used as exposure devices. The exposures were conducted with the mobile-phone antenna outside the glass vials containing the flies, either in contact with or at a certain distance from the glass wall. The daily duration of exposure varied from 1 to 20 minutes, depending on the experiment. Exposure always started on the day of eclosion and lasted for a total of 5 or 6 days. The temperature within the vials during exposure was monitored with a mercury thermometer with an accuracy of 0.05 °C. The authors explained the decreased reproductive ability as the result of RF radiation-induced DNA fragmentation in the gonads ([Panagopoulos, 2011](#); [Panagopoulos & Margaritis, 2008, 2010a, b](#); [Panagopoulos et al., 2004, 2007, 2010](#)).

[In reviewing these studies with *Drosophila*, the Working Group noted several shortcomings related to the methods of exposure assessment and temperature control, which could have influenced the results.]

(b) *Mouse*

See [Table 4.3](#)

(i) 900 MHz

[Sykes et al. \(2001\)](#) studied somatic intra-chromosomal recombination in the spleen of transgenic pKZ1 mice exposed to pulsed-wave RF radiation at 900 MHz (SAR, 4 W/kg) for 30 minutes per day, for 1, 5, or 25 days. There was a significant reduction in inversions below the spontaneous frequency in the group exposed for 25 days, whereas no effect was found in mice exposed for 1 or 5 days. The authors indicated that the number of mice in each treatment group in this study was small, and that repetition of this study with a larger number of mice was therefore required to confirm these observations.

[Aitken et al. \(2005\)](#) found a significant genotoxic effect on the epididymal spermatozoa of CD1 Swiss mice exposed to low-level RF radiation at 900 MHz (SAR, 0.09 W/kg) for 12 hours per day, for 7 days. No impact on male germ-cell development was observed. [The Working Group noted that insufficient information on dosimetry was provided in this study, which prevented a complete evaluation.]

Two cytogenetic studies were conducted with mice exposed to RF radiation from a mobile phone, with or without coexposure to X-rays or ultraviolet (UV) light. In the first study, female CBA/S mice were exposed for 78 weeks (1.5 hours per day, 5 days per week) either to continuous-wave RF radiation at 902.5 MHz (whole-body SAR, 1.5 W/kg) similar to that emitted by analogue NMT (Nordic Mobile Telephony) phones, or to a pulsed-wave signal at 902.4 MHz (SAR, 0.35 W/kg) similar to that emitted by digital GSM phones. All mice, except

the cage controls, were also exposed to X-rays (3×1.33 Gy; interval, 1 week) for the first 3 weeks of this experiment. In the second study, female transgenic mice (line K2) and their non-transgenic littermates were exposed to one of two digital mobile-phone signals at a frequency of 849 MHz GSM or 902 MHz DAMPS (Digital Advanced Mobile Phone System), with a SAR of 0.5 W/kg, for 1.5 hours per day, 5 days per week, for 52 weeks. All mice in the second study, except the cage controls, were also exposed to UV radiation mimicking the solar spectrum at 1.2 times the human minimal erythema dose (MED, 200 J/m²), three times per week. The results did not show any effects of RF fields on frequency of micronuclei in polychromatic erythrocytes or normochromatic erythrocytes, either alone or in combination with X-rays or UV radiation. The results were consistent in the two mouse strains (and in a transgenic variant of the second strain), after 52 or 78 weeks of exposure, at three SAR levels relevant to human exposure from mobile phones, and for three different mobile signals ([Juutilainen et al., 2007](#)).

(ii) 900 and 1800 MHz

In a study in B6C3F₁ mice exposed to RF radiation at 900 MHz or 1800 MHz (2 hours per day, for 1 week or 6 weeks) at different intensities (with SARs up to 33.2 W/kg in the 1-week experiment, and 24.9 W/kg in the 6-week experiment), the frequency of micronuclei was not increased in erythrocytes of peripheral blood or bone marrow, in keratinocytes or in spleen lymphocytes of the exposed animals compared with controls ([Görlitz et al., 2005](#)).

In a long-term study, micronucleus formation was measured in erythrocytes of B6C3F₁/CrIBR mice exposed to RF radiation at 902 MHz GSM or 1747 MHz (DCS, Digital Cellular System), at SARs of 0.4, 1.3 or 4.0 W/kg, for 2 hours per day, 5 days per week, for 2 years. No differences were found in the frequencies of micronuclei in exposed, sham-exposed or cage-control mice ([Ziemann et al., 2009](#)).

(iii) 1500 MHz

Male Big Blue mice, which are transgenic for the *lacI* marker gene, were locally exposed (in the head region) to near-field RF radiation at 1500 MHz with SARs of 0.67 or 2.0 W/kg, for 90 minutes per day, 5 days per week, for 4 weeks. There was no significant difference between exposed and control mice in the frequency of mutation in the *lacI* transgene in the brain ([Takahashi et al., 2002](#)).

(iv) 450 MHz

[Sarkar et al. \(1994\)](#) found significant alterations in the length of a DNA microsatellite sequence in the brain and testes of Swiss albino mice exposed to RF radiation at 2450 MHz (power level, 1 mW/cm²; SAR, 1.18 W/kg) for 2 hours per day, for 120, 150 or 200 days. The authors hypothesized that a DNA fragment (7.7 kb) – generated by the restriction enzyme *HinfI* – that was found after exposure could represent a hypermutable locus and that exposure to these microwaves may have led to amplification of tandem sequences, generating more copies of 5'-GACA-3' sequences in this particular region. The authors also indicated that the radiation dose applied in the study was close to the prescribed safe limit for population exposure, according to Guidelines of the International Radiation Protection Association at the time ([IRPA, 1988](#)).

C3H/HeJ mice were exposed continuous-wave RF radiation at 2450 MHz in circularly polarized wave-guides (average whole-body SAR, 1.0 W/kg) for 20 hours per day, 7 days per week, for 18 months. Peripheral-blood and bone-marrow smears were examined for the presence of micronuclei in polychromatic erythrocytes. The initial publication reported no difference in micronucleus formation between exposed and sham-exposed mice, but a subsequent correction indicated that there was a slight but significant increase in the incidence of micronucleated cells in peripheral-blood and bone-marrow smears

of mice receiving long-term exposure to this RF radiation ([Vijayalaxmi et al., 1997a, 1998](#)).

Pregnant *lacZ*-transgenic mice (MutaTMMouse) were exposed (16 hours per day) to intermittent (10 seconds on, 50 seconds off) RF radiation at 2450 MHz with an average whole-body SAR of 0.71 W/kg (4.3 W/kg during the exposure periods of 10 seconds), daily between day 0 and day 15 of gestation. Offspring were examined at age 10 weeks. Mutation frequencies at the *LacZ* gene in the spleen, liver, brain, and testis were similar to those observed in offspring of sham-exposed mice ([Ono et al., 2004](#)).

(v) 42 GHz (millimetre waves)

Adult male BALB/c mice were exposed (30 minutes per day) in the nasal region to RF radiation at 42 GHz (incident power density, 31.5 mW/cm²; peak SAR, 622 W/kg), on three consecutive days. The frequency of micronuclei in peripheral blood and in bone marrow was not increased in exposed mice compared with sham-exposed controls. One group of mice received a single injection of cyclophosphamide (15 mg/kg bw) immediately after the exposure to RF radiation on day 2. The micronucleus frequency in this group was not different from that in mice treated with cyclophosphamide only ([Vijayalaxmi et al., 2004](#)).

(vi) Ultra-wide band EMF

Male CF1 mice were exposed for 15 minutes to ultra-wide band (UWB) electromagnetic fields (600 pulses per second) at an estimated whole-body average SAR of 37 mW/kg. The mice were killed at 18 hours or 24 hours after exposure, and peripheral blood and bone marrow were collected and examined for the presence of micronuclei in polychromatic erythrocytes. Under the experimental conditions of this study, there was no evidence of cytogenetic effects in blood or bone marrow of the exposed mice ([Vijayalaxmi et al., 1999](#)).

(c) Rat

See [Table 4.3](#)

(i) 834 MHz

Micronucleus formation was investigated in the offspring of rats exposed to RF radiation. Wistar rats were placed in experimental cages on the first day of pregnancy and exposed (8.5 hours per day) to RF radiation at 834 MHz (26.8–40 V/m; vertical polarization; peak power, 600 mW; calculated SAR, 0.55–1.23 W/kg) from an analogue mobile telephone that was placed close to the plexiglass cage. Exposure was continued throughout gestation. Newborn pups (age, 2 days) showed a statistically significant increase ($P < 0.003$) in micronucleus frequency in erythrocytes (1.23 ± 0.17 per 1000 cells) compared with controls (0.5 ± 0.1 per 1000 cells). Oxidative parameters measured in blood plasma or liver were not different between exposed and control rats ([Ferreira et al., 2006](#)).

(ii) 900–915 MHz

Wistar rats were exposed to RF radiation at 910 MHz (maximum SAR, 0.42 W/kg) for 2 hours per day on 30 consecutive days. Compared with non-exposed control rats, an almost threefold increase in the frequency of micronuclei was found in polychromatic erythrocytes of males and females ($P < 0.001$ and $P < 0.01$, respectively); the induction was significantly lower in females than in males ($P < 0.001$). An increase in micronucleus frequency was also observed in polymorphonuclear cells ([Demsia et al., 2004](#)).

Genotoxic effects of coexposure to RF radiation at 900 MHz with 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX, a by-product of water chlorination; 19 µg/ml in drinking-water) were investigated in female Wistar rats ([Verschaeve et al., 2006](#)). The rats were exposed to RF radiation for 2 hours per day, 5 days per week, for 2 years, at an average SAR of 0.3 or 0.9 W/kg; exposure to MX was continuous. Blood samples were collected at 3, 6 and 24

Table 4.3 Genetic and related effects of radiofrequency radiation, alone or in combination with chemical/physical mutagens: studies in experimental animals *in vivo*

End-point	Frequency	SAR	Duration	Chemical/physical mutagen	Results	Comments	References
<i>Mouse</i>							
MN formation in peripheral blood and bone-marrow cells in tumour-prone C3H/HeJ mice	2450 MHz, CW	1.0 W/kg	20 h/d, 7 d/wk, for 1.5 yr	None	+	Corrected statistical analysis in 1998 paper	Vijayalaxmi <i>et al.</i> (1997a, 1998)
MN formation in PCEs from peripheral blood and bone marrow of CF1 mice	Ultra-wide band radiation	0.037 W/kg	15 min	None	-		Vijayalaxmi <i>et al.</i> (1999)
MN formation in peripheral-blood and bone-marrow cells of male BALB/c mice	42 200 MHz	622 ± 100 W/kg	30 min/d for 3 consecutive days	Coexposure with cyclophosphamide	-	No effect of RF radiation alone; no effect on MN induced by cyclophosphamide	Vijayalaxmi <i>et al.</i> (2004)
MN formation in erythrocytes of blood or bone marrow, in keratinocytes and in spleen lymphocytes of B6C3F ₁ mice	900 MHz (GSM) and 1800 MHz (DCS); AM	3.7, 11 and 33.2 W/kg (1-wk study); and 2.8, 8.3 and 24.9 W/kg (6-wk study)	2 h/d during 1 or 6 wk	None	-		Görlitz <i>et al.</i> (2005)
MN formation in erythrocytes of female inbred CBA/S mice (taken from study by Heikkinen <i>et al.</i> , 2001)	902.5 MHz (NMT), CW or 902.5 MHz (GSM), PW	1.5 W/kg or 0.35 W/kg	1.5 h/d, 5 d/wk, for 78 wk	Also exposed to X-rays (3 × 1.33 Gy, during first 3 wk)	-	No effect of RF radiation alone; no effect on MN induced by X-rays	Juutilainen <i>et al.</i> (2007)
MN formation in erythrocytes of female K2 transgenic and non-transgenic mice (taken from Heikkinen <i>et al.</i> , 2003)	Digital mobile-phone signals, GSM at 849 MHz and DAMPS at 902 MHz	0.5 W/kg	1.5 h/d, 5 d/wk, for 52 wk	Also exposed to UV radiation (1.2 MED), 3×/wk	-	No effect of RF radiation alone; no effect on MN induced by UV	Juutilainen <i>et al.</i> (2007)
MN formation in erythrocytes of B6C3F ₁ /CrIBR male and female mice	GSM (902 MHz) or DCS (1747 MHz)	0.4, 1.3 or 4.0 W/kg	2 h/d, 5 d/wk, for 2 yr	None	-	No difference in MN frequency in exposed, sham-exposed or cage-control mice	Ziemann <i>et al.</i> (2009)
Mutation assay (<i>lacI</i> transgene) in brain tissue of Big Blue mice	1500 MHz	0, 0.67, or 2 W/kg	90 min/d, 5 d/wk, for 4 wk	None	-		Takahashi <i>et al.</i> (2002)

Table 4.3 (continued)

End-point	Frequency	SAR	Duration	Chemical/physical mutagen	Results	Comments	References
Mutation frequency of the <i>lacZ</i> gene in cells from the spleen, liver, brain and testes of the offspring of <i>lacZ</i> -transgenic mice	2450 MHz (intermittent, 10 s on, 50 s off)	0.71 W/kg (average); 4.3 W/kg (for 10 s exposures)	Exposure <i>in utero</i> for 16 h/d on days 0–15 of gestation	None	–	Offspring was analysed at age 10 wk	Ono et al. (2004)
DNA microsatellite analysis with synthetic oligonucleotide probes in cells of brain and testis of Swiss albino mice	2450 MHz, CW	1.2 W/kg	2 h/d, for 120, 150, 200 d	None	+	Change in length of a microsatellite sequence	Sarkar et al. (1994)
DNA damage assessed by quantitative PCR (Q-PCR), alkaline- and pulsed-field electrophoresis in caudal epididymal spermatozoa of CD1 Swiss mice	900 MHz	0.09 W/kg	12 h/d, for 7 d	None	+	No effect on male germ-cell development; Q-PCR showed damage in mitochondrial genome and in nuclear β -globin locus	Aitken et al. (2005)
Somatic intrachromosomal recombination in spleen cells of pKZ1 transgenic mice	900 MHz, PW	4 W/kg	30 min/d for 1, 5, 25 d	None	–	Reduction in inversions below the spontaneous frequency in the group exposed for 25 d	Sykes et al. (2001)
Rat							
MN formation in peripheral-blood and bone-marrow cells of male Sprague-Dawley rats	2450 MHz, CW	12 W/kg	24 h	None	–		Vijayalaxmi et al. (2001a)
MN formation in peripheral blood cells of male Wistar rats	2450 MHz, CW	1 and 2 W/kg	2 h/d for up to 30 d	None	+	Only after 8 (not 2, 15, or 30) exposures of 2 h each	Trosic et al. (2002)
MN formation in PCEs in bone marrow and peripheral blood of Wistar rats	2450 MHz	Whole-body SAR, 1.25 W/kg	2 h/d, 7 d/wk, 30 d	None	+	Increased MN frequency in PCEs in bone marrow on day 15, and in the peripheral blood on day 8	Trosic & Busljeta (2006)
MN formation in bone-marrow cells of male and female Wistar rats	910 MHz	Peak SAR, 0.42 W/kg	2 h/d for 30 consecutive days	None	+		Demsia et al. (2004)

Table 4.3 (continued)

End-point	Frequency	SAR	Duration	Chemical/physical mutagen	Results	Comments	References
MN formation in bone-marrow cells of male Wistar rats	2450 MHz, CW	1.25 W/kg	2 h/d for up to 30 days (total exposure 4, 16, 30 or 60 h)	None	+	Increase in PCE in bone marrow on day 15 (exposure, 30 h). Transient effect on proliferation and maturation of erythropoietic cells	Trosic et al. (2004) ; Busijeta et al. (2004)
MN formation in blood from adult pregnant Wistar rats	834 MHz, mobile-phone antenna, 26.8–40 V/m	0.55–1.23 W/kg	From day 1 of gestation, for 8.5 h/d, until birth of offspring	None	+	Significant increase of MN frequency in erythrocytes of newborn pups exposed <i>in utero</i>	Ferreira et al. (2006)
MN formation in blood of female Wistar rats	900 MHz, AM	0.3 and 0.9 W/kg	2 h/d, 5 d/wk, for 2 yr	Coexposure with MX in drinking-water	–	No increase in MN after coexposure to MX and RF radiation compared with MX [no group exposed to RF only]	Verschaeve et al. (2006)
MN formation in blood cells of Wistar rats	10 000 MHz 50 000 MHz	0.04 W/kg 0.0008 W/kg	2 h/d for 45 d	None	+	Also significant increase of ROS in serum	Kumar et al. (2010)
DNA breaks (SSB, DSB) measured with comet assay in brain cells of male Sprague-Dawley rats	2450 MHz, PW or CW	0.6 and 1.2 W/kg	2 h	None	+	Significant and SAR-dependent increase in SB immediately and at 4 h after exposure to CW; only at 4 h after exposure to PW	Lai & Singh (1995)
DNA breaks (SSB, DSB) measured with comet assay in brain cells of male Sprague-Dawley rats	2450 MHz, PW or CW	1.2 W/kg	2 h	None	+	Significant increase in SB at 4 h after exposure to either PW or CW	Lai & Singh (1996)
DNA breaks (SSB, DSB) measured with comet assay in brain cells of male Sprague-Dawley rats	2450 MHz, PW	1.2 W/kg	2 h	Melatonin or N-tert-butyl- α -phenylnitron (free-radical scavengers)	+	Significant increase in SB at 4 h after exposure. Treatment with radical scavengers before and after exposure to RF prevented/reversed induction of SB	Lai & Singh (1997)

Table 4.3 (continued)

End-point	Frequency	SAR	Duration	Chemical/physical mutagen	Results	Comments	References
DNA breaks (SSB) measured with comet assay in brain cells of male Sprague-Dawley rats	2450 MHz, CW	1.2 W/kg	2 h	None	-		Malyapa et al. (1998)
DNA breaks (SSB) measured with alkaline comet assay (with or without proteinase K) in brain cells of male Sprague-Dawley rats	2450 MHz, PW	1.2 W/kg	2 h	None	-		Lagroye et al. (2004a)
DNA breaks (SSB, DSB) measured with comet assay in brain cells of male Sprague-Dawley rats	2450 MHz, CW, circular polarization	0.6 W/kg	2 h	None	+	Significant increase in SB at 4 h after exposure	Lai & Singh (2005)
DNA breaks (DSB) measured with pulsed-field electrophoresis. Changes in chromatin conformation detected with AVTD assay in brain cells from Wistar rats	915 MHz (GSM)	0.4 W/kg	2 h	None	-	Changes in gene expression were detected	Belyaev et al. (2006)
DNA breaks (SSB) measured with alkaline comet assay in brain cells of male and female Wistar rats	2450 MHz or 16 500 MHz	1.0 W/kg or 2.01 W/kg	2 h/d, for 35 d	None	+	DNA breakage was observed at both frequencies	Paulraj & Behari (2006)
DNA breaks (SSB) measured with alkaline comet assay in blood, liver and brain of female Wistar rats	900 MHz, AM	0.3 or 0.9 W/kg	2 h/d, 5 d/wk for 2 yr	Co-exposure with MX in drinking-water	-	No increase in SB after co-exposure to MX and RF radiation compared with MX [no group exposed to RF only]	Verschaeve et al. (2006)
DNA breaks (DSB) measured with neutral comet assay in brain of male Wistar rats	2450 MHz, from MW oven	0.11 W/kg (whole-body)	2 h/d, 35 d	None	+	Highly significant decrease in anti-oxidant enzymes and increase in catalase were also seen ($P < 0.006$)	Kesari et al. (2010)

Table 4.3 (continued)

End-point	Frequency	SAR	Duration	Chemical/physical mutagen	Results	Comments	References
<i>Rabbit</i>							
Oxidative DNA damage (8-OHdG) in liver of pregnant and non-pregnant New Zealand White rabbits	1800 MHz (GSM-like)	NR	15 min/d for 1 wk (for pregnant rabbits; days 15–22 of gestation)	None	–	No difference in 8-OHdG/10 ⁶ dG between exposed and sham-exposed non-pregnant or pregnant rabbits, or between newborns exposed <i>in utero</i> and sham-exposed newborns	Tomruk et al. (2010)
<i>Cow</i>							
MN formation in erythrocytes of Latvian Brown cows living in the Skrunda radio-station area	154–162 MHz, PW	NR	Cows had been living in the area for at least 2 yr	None	+	Significant increase in MN compared with cows in a control area. Frequencies of MN were low in all cases	Balode (1996)

+, increase; –, no effect; AVTD, anomalous viscosity time-dependence; CW, continuous wave; d, day; DAMPS, Digital Advanced Mobile Phone System, DCS, Digital Cellular System; DSB, DNA double-strand breaks; GSM, Global System for Mobile Communications; h, hour; MED, minimal erythema dose; min, minute; MN, micronuclei; MW, microwave; MX, 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone; NMT, Nordic Mobile Telephone; NR, not reported; PCE, polychromatic erythrocytes; PW, pulsed wave; s, second; SAR, specific absorption rate; SB, DNA strand breaks, SSB, DNA single-strand breaks; wk, week; yr, year

months and brain and liver samples were taken at the end of the study (24 months). The extent of DNA strand breaks in blood, liver and brain cells was determined by means of the alkaline comet assay; the frequency of micronuclei was measured in erythrocytes. Coexposure to MX and RF radiation did not significantly change the effects in blood, liver and brain cells compared with those seen with MX only [the Working Group noted that this study did not include a treatment group exposed to RF radiation only].

Induction of DNA double-strand breaks was measured by means of pulsed-field gel electrophoresis, and changes in chromatin conformation were assessed by use of the anomalous viscosity time-dependence (AVTD) assay in brain tissue of Fisher rats exposed to RF radiation at 915 MHz (GSM; SAR, 0.4 W/kg) for 2 hours. No effects of exposure to RF radiation were found. Analysis of gene-expression profiles in the cerebellum of exposed rats revealed changes in genes associated with neurotransmitter regulation, melatonin production and regulation of the blood-brain barrier ([Belyaev et al., 2006](#)).

(iii) 1600 MHz

Timed-pregnant Fischer 344 rats were exposed from day 19 of gestation, and their nursing offspring until weaning at 3 weeks of age, to far-field RF radiation at 1600 MHz (iridium wireless-communication signal) for 2 hours per day, 7 days per week. The whole-body average SAR was 0.036–0.077 W/kg (0.10–0.22 W/kg in the brain). This first exposure was followed by long-term, head-only exposures of male and female offspring (starting at age 35 days) to a near-field 1600 MHz signal, with a SAR of 0.16 or 1.6 W/kg in the brain, for 2 hours per day, 5 days per week, for 2 years. The micronucleus frequency in polychromatic erythrocytes of the bone marrow was not significantly different between exposed, sham-exposed and cage-control rats ([Vijayalaxmi et al., 2003](#)).

(iv) 2450 MHz

In several publications from the same laboratory it was reported that brain cells of male Sprague-Dawley rats exposed for 2 hours to low-intensity pulsed-wave or continuous-wave RF radiation at 2450 MHz (SAR, 0.6 or 1.2 W/kg) showed an increased number of DNA single- and double-strand breaks – measured by the neutral and alkaline comet assays – at 4 hours after exposure. The authors suggested that this could be due either to a direct effect on DNA or to an effect on DNA repair ([Lai & Singh, 1995, 1996](#)). In subsequent experiments, treatment of the rats with free-radical scavengers appeared to block this effect of RF exposure, suggesting that free radicals may be involved in RF-radiation-induced DNA damage in the rat brain ([Lai & Singh, 1997](#)).

Male Sprague-Dawley rats were exposed to continuous-wave RF radiation at 2450 MHz (SAR of 1.2 W/kg) for 2 hours, which did not cause a rise in the core body-temperature of the rats. One group of rats was killed by carbon dioxide (CO₂) asphyxia, another by decapitation. DNA breakage was assessed by means of the alkaline comet assay. No significant differences were observed in the comet length or the normalized comet moment of cells isolated from either the cerebral cortex or the hippocampus of irradiated rats and those from sham-exposed rats. This was independent of the method by which the rats were killed. However, there was more intrinsic DNA damage and more experiment-to-experiment variation in cells from the asphyxiated rats than from rats killed by decapitation. Therefore, the latter method appeared to be the most appropriate in this type of study ([Malyapa et al., 1998](#)). [The Working Group noted that this study was not a valid replication of the [Lai & Singh \(1995\)](#) study, contrary to the authors' intention, but it provided independent evidence contrary to those results. The Working Group also noted that the increased number of DNA strand breaks after

exposure to RF radiation *in vivo* was particularly protocol-dependent, specifically with respect to the method of killing the animals and the treatment of tissue samples between exposure of the animals and analysis of the tissues.]

[Vijayalaxmi et al. \(2001a\)](#) found no evidence for the induction of micronuclei in peripheral-blood and bone-marrow cells of Wistar rats exposed continuously to continuous-wave RF radiation at 2450 MHz, with an average whole-body SAR of 12 W/kg, for 24 hours.

[Lagroye et al. \(2004a\)](#) investigated the induction of DNA damage in brain cells of Sprague-Dawley rats exposed to pulsed-wave RF radiation at 2450 MHz, with a SAR of 1.2 W/kg, for 2 hours. The rats were decapitated 4 hours after exposure. No DNA damage was detected in separate samples of the same brain-cell preparation from exposed rats, assessed by two variants of the alkaline comet assay.

Wistar rats were exposed to non-thermal RF radiation at 2450 MHz for 2 hours per day on 7 days per week, for up to 30 days. The power-density range was 5–10 mW/cm², which corresponded to an approximate SAR of 1–2 W/kg. Erythrocyte counts, haemoglobin concentrations and haematocrit values were significantly increased in peripheral blood on days 8 and 15, and anuclear cells and erythropoietic precursor cells in bone marrow were significantly decreased. The frequency of micronucleated cells in the bone marrow was significantly increased on day 15, not on days 2, 8, and 30 ([Busljeta et al., 2004](#)).

Adult male Wistar rats were exposed to continuous-wave RF radiation at 2450 MHz for 2 hours per day, 7 days per week, for up to 30 days. The power-density range was 5–10 mW/cm², which corresponded to an approximate SAR of 1–2 W/kg. The frequency of micronuclei in polychromatic erythrocytes was significantly increased in the group that had received 8 irradiation treatments of 2 hours each, but not in the groups that received 2, 15 or 30 treatments, in comparison with the sham-exposed group.

These results would be in line with an adaptive or recovery mechanism that was triggered in this experimental model during treatment ([Trosic et al., 2002, 2004](#)). Similar results were presented in a later publication ([Trosic & Busljeta, 2006](#)).

[Paulraj & Behari \(2006\)](#) reported a significantly increased ($P < 0.001$) level of DNA breakage – measured by means of the alkaline comet assay – in brain cells of rats exposed to RF radiation at 2450 MHz or 16.5 GHz (SAR, 1.0 or 2.01 W/kg) for 2 hours per day, for 35 days.

Wistar rats were exposed to RF radiation at 2450 MHz (power density, 0.34 mW/cm²) for 2 hours per day, for 35 days. The whole-body SAR was estimated to be 0.11 W/kg. After exposure, rats were killed and whole-brain tissue was dissected and used for analysis of DNA double-strand breaks by means of the neutral comet assay. A significant increase was observed in various comet parameters in exposed brain cells compared with controls. Statistically significant changes were also observed in the levels of different antioxidant enzymes, *i.e.* a decrease in glutathione peroxidase, superoxide dismutase and histone kinase, and an increase in catalase ([Kesari et al., 2010](#)).

(v) 10–50 GHz

Wistar rats were exposed continuously to RF radiation at 10 GHz or 50 GHz (SAR, 0.014 W/kg and 0.0008 W/kg, respectively) for 2 hours per day, for 45 days. In both cases, significant increases ($P < 0.05$) in the frequency of micronuclei – deduced from a reduced polychromatic/normochromatic erythrocyte ratio – and in concentrations of reactive oxygen species (ROS) were found in blood cells and serum, respectively ([Kumar et al., 2010](#)).

(d) Rabbit

A study was performed with non-pregnant and pregnant New Zealand White rabbits. The rabbits were exposed (whole-body) to 1800 MHz RF radiation (GSM) for 15 minutes per day,

for 1 week. For the pregnant rats, this exposure period was between day 15 and day 22 of gestation. Control groups of non-pregnant and pregnant rabbits were sham-exposed. No difference was found in the level of 8-hydroxy-2'-deoxyguanosine (an indicator of oxidative DNA damage; expressed as 8-OHdG/ 10^6 dG) in DNA from liver tissue of exposed and sham-exposed rabbits (pregnant or non-pregnant). Changes in malondialdehyde concentration and ferrous oxidation in xylenol orange in the liver of exposed non-pregnant and pregnant rabbits indicated an effect on lipid peroxidation. In pups exposed *in utero*, a reduction in ferrous oxidation in xylenol orange was seen in the liver, but no change was observed in malondialdehyde concentration. These results supported the notion that 1800 MHz GSM-like RF radiation may induce oxidative stress in exposed tissues ([Tomruk et al., 2010](#)).

(e) Cow

Blood samples were obtained from 67 female Latvian Brown cows living on a farm in the vicinity of the Skrunda radio-location station (Latvia), and from 100 cows in a control area, which was selected on the basis of the similarity to the exposed area with regards to many factors except exposure. Frequencies of micronuclei were scored in the erythrocytes and found to be low but statistically significantly increased in the exposed cows compared with those in the controls (0.6/1000 cells compared with 0.1/1000 cells; $P < 0.01$) ([Balode, 1996](#)).

4.1.3 Experimental systems: in vitro

(a) Humans: peripheral blood lymphocytes

The most widely used cell type for investigations *in vitro* is the peripheral blood lymphocyte. Some details on the exposure conditions to RF radiation and a short conclusion of the publications discussed below are presented in [Table 4.4](#).

(i) Studies with a single end-point

DNA-damage induction and repair

The effects of exposure to RF radiation at frequencies ranging from approximately 800 to 8000 MHz were examined by several investigators, who reported no significant effect on induction of DNA strand breaks ([Baohong et al., 2005, 2007](#); [Chemeris et al., 2006](#); [Sannino et al., 2006](#)).

[Vijayalaxmi et al. \(2000\)](#) assessed DNA strand breaks in human lymphocytes and also the capacity of these cells to repair such damage after exposure to RF radiation at 2450 MHz, and observed no effect on either parameter. [Zhijian et al. \(2009\)](#) also reported no effect of exposure to RF radiation at 1800 MHz, not only on induction of DNA strand breaks but also on the repair kinetics of X-irradiation-induced DNA strand breaks. [Tiwari et al. \(2008\)](#) exposed human lymphocytes to RF radiation at 835 MHz (SAR, 1.17 W/kg) and subsequently incubated the cells in the presence of aphidicolin (APC; an inhibitor of DNA repair) at a dose of 0.02 or 2 $\mu\text{g/ml}$. There was no effect on DNA strand breakage from exposure to RF radiation alone. APC (2 $\mu\text{g/ml}$) and combinations of RF radiation with APC (0.02 and 2 $\mu\text{g/ml}$) enhanced the number of DNA strand breaks; this damage is repairable (see Section 4.1.3c).

Chromosomal aberrations

[Maes et al. \(1995\)](#) found an increase in the frequency of chromosomal aberrations in the form of dicentrics and acentric fragments in human lymphocytes exposed to pulsed-wave RF radiation at 954 MHz, while using a cooled box to maintain the temperature at $17 \pm 1^\circ\text{C}$. [Manti et al. \(2008\)](#) carried out FISH analysis with molecular probes specific for whole chromosomes 1 and 2 in lymphocytes from four donors. The cells were exposed to RF radiation at 1950 MHz (UMTS, Universal Mobile Telecommunications System) at SAR 0.5 or 2.0 W/kg, for 24 hours. There was no effect on the fraction of aberrant cells at a

Table 4.4 Genetic and related effects in human peripheral blood lymphocytes exposed to radiofrequency radiation *in vitro*

End-point	Frequency	SAR or power density	Duration	Results	Comments	Reference
Aneuploidy	830 MHz, CW	2.0–8.2 W/kg	72 h	+ (chromosome 17)	Temperature kept at 33.5–37.5 °C. In control without RF, no aneuploidy was seen up to 38.5 °C	Mashevich et al. (2003)
Aneuploidy	100 GHz, CW	0.31 mW/cm ²	1–24 h	+ (chromosomes 11, 17) – (chromosomes 1, 10)	Direct effect questionable. High values in control cells.	Korenstein-Ilan et al. (2008)
Aneuploidy	800 MHz, CW	2.9, 4.1 W/kg	24 h	+ (chromosomes 11, 17) at SAR of 2.9 W/kg + (chromosomes 1, 10) at SAR of 4 W/kg	High values in control cells. In control without RF, no aneuploidy was seen up to 40 °C	Mazor et al. (2008)
Chromosomal aberration	7700 MHz, CW	0.5, 10, 30 mW/cm ²	10, 30, 60 min	+	Aberrations increased at 10 and 30 mW/cm ² at all time-points	Garaj-Vrhovac et al. (1992)
Chromosomal aberration	2450 MHz, PW	75 W/kg	30 min, 2 h	+	MW output was adjusted with a thermistor to keep cells at 36.1 °C	Maes et al. (1993)
Chromosomal aberration	954 MHz, PW; GSM	1.5 W/kg	2 h	±	Questionable dosimetry (pylon from GSM base-station connected to indoor antenna); no statistics provided	Maes et al. (1995)
Chromosomal aberration	440, 900, 1800 MHz, PW; GSM	1.5 W/kg	30–72 h	–		Eberle et al. (1996)
Chromosomal aberration	935.2 MHz, PW; GSM	0.3–0.4 W/kg	2 h	–		Maes et al. (1997)
Chromosomal aberration	2450 MHz, CW	12.5 W/kg	90 min or 3 × 30 min	–		Vijayalaxmi et al. (1997b)
Chromosomal aberration	455.7 MHz, PW	6.5 W/kg	2 h	–	Cells were placed 5 cm from a car phone	Maes et al. (2000)
Chromosomal aberration	900 MHz, PW; CDMA	0.4–10 W/kg	2 h	–		Maes et al. (2001)
Chromosomal aberration	835.62 MHz, CW; FDMA	4.4, 5.0 W/kg	24 h	–		Vijayalaxmi et al. (2001a)
Chromosomal aberration	847.74 MHz, CW; CDMA	4.9, 5.5 W/kg	24 h	–		Vijayalaxmi et al. (2001b)
Chromosomal aberration	2500 MHz 10 500 MHz	627 W/kg 0.25 W/kg	40 s 5 min	– –	MW oven at 3 W	Figueiredo et al. (2004)

Table 4.4 (continued)

End-point	Frequency	SAR or power density	Duration	Results	Comments	Reference
Chromosomal aberration	900 MHz, PW; GSM	0.3, 1 W/kg	2 h	-		Zeni et al. (2005)
Chromosomal aberration	935 MHz, PW; GSM	1, 2 W/kg	24 h	-		Stronati et al. (2006)
Chromosomal aberration	2450, 8200 MHz, PW	2.1, 21 W/kg	2 h	-		Vijayalaxmi et al. (2006)
Chromosomal aberration	1950 MHz, PW; UMTS	0.5, 2 W/kg	24 h	- at SAR of 0.5 W/kg + at SAR of 2 W/kg	Frequency of aberrations/cell was increased at higher SAR; FISH technique was used	Manti et al. (2008)
Chromosomal aberration	18 000 MHz, CW 16 500 MHz, PW	1 mW/cm ² 10 mW/cm ²	53 h	-		Hansteen et al. (2009a)
Chromosomal aberration	2300 MHz, CW, PW	1 mW/cm ²	53 h	-		Hansteen et al. (2009b)
Micronucleus formation	7700 MHz, CW	0.5, 10, 30 mW/cm ²	10, 30, 60 min	+	MN frequency increased at 30 mW/cm ² , after 30 and 60 min of exposure	Garaj-Vrhovac et al. (1992)
Micronucleus formation	2450 MHz, PW	75 W/kg	30 min, 2 h	+	MW output was adjusted with a thermistor to keep cells at 36.1 °C	Maes et al. (1993)
Micronucleus formation	9000 MHz, CW, PW	90 W/kg	10 min	+ with PW - with CW	Temperature during exposure was 30–35 °C. Control cultures were kept at 37 °C	d'Ambrosio et al. (1995)
Micronucleus formation	440, 900, 1800 MHz, PW; GSM	1.5 W/kg	30–72 h	-		Eberle et al. (1996)
Micronucleus formation	2450 MHz, CW	12.5 W/kg	3 × 30 min	-		Vijayalaxmi et al. (1997b)
Micronucleus formation	2450, 7700 MHz, CW	10, 20, 30 mW/cm ²	15, 30, 60 min	+	Experiment carried out at 20–22 °C. Temperature-control measurements were made in water	Zotti-Martelli et al. (2000)
Micronucleus formation	835.62 MHz, CW; FDMA	4.4, 5.0 W/kg	24 h	-		Vijayalaxmi et al. (2001a)
Micronucleus formation	847.74 MHz, CW; CDMA	4.9, 5.5 W/kg	24 h	-		Vijayalaxmi et al. (2001b)
Micronucleus formation	1748 MHz, CW, PW; GSM	5 W/kg	15 min	+ with PW - with CW	Temperature during exposure was 30–35 °C. Control cultures were kept at 37 °C	d'Ambrosio et al. (2002)
Micronucleus formation	1900 MHz, CW, PW	0.1–10 W/kg	2 h	-		McNamee et al. (2002a)

Table 4.4 (continued)

End-point	Frequency	SAR or power density	Duration	Results	Comments	Reference
Micronucleus formation	2450 MHz, PW	5 mW/cm ²	2 h	-		Zhang et al. (2002)
Micronucleus formation	837, 1909 MHz, CW, PW; CDMA, TDMA	1.0, 2.5, 5.0, 10.0 W/kg	3 h, 24 h	+ after 24 h, at SARs of 5 or 10 W/kg	Some exposures were from mobile telephones. Temperature variations were $\pm 0.3^{\circ}\text{C}$ and $\pm 0.5^{\circ}\text{C}$ at 3 h and 24 h, respectively. EMS was included as a positive control	Tice et al. (2002)
Micronucleus formation	1900 MHz, CW, PW	0.1–10 W/kg	24 h	-		McNamee et al. (2003)
Micronucleus formation	120, 130 GHz PW	1 and 0.6 mW average power	20 min	-		Scarfi et al. (2003)
Micronucleus formation	900/925 MHz, CW, PW(i); GSM	1.6 W/kg 0.2 W/kg	14 \times (6 min on, 3 h off) at 1.6 W/kg; 1 h/d for 3 d at 0.2 W/kg	-		Zeni et al. (2003)
Micronucleus formation	1800 MHz, CW	5, 10, 20 mW/cm ²	1, 2, 3 h	+	Large variation between individuals and repeat experiments	Zotti-Martelli et al. (2005)
Micronucleus formation	900 MHz, PW; GSM	0.1–10 W/kg	24 h	-	Concordant results between two research groups in interlaboratory study	Scarfi et al. (2006)
Micronucleus formation	935 MHz, PW; GSM	1, 2 W/kg	24 h	-		Stronati et al. (2006)
Micronucleus formation	2450, 8200 MHz, PW	2.1, 21 W/kg	2 h	-		Vijayalaxmi et al. (2006)
Micronucleus formation	1950 MHz, PW (c, i); UMTS	0.05–2 W/kg	4–48 h	-	Controversial data	Schwarz et al. (2008)
Micronucleus formation	1950 MHz, PW (c, i); UMTS	2.2 W/kg	24–68 h	-		Zeni et al. (2008)
Micronucleus formation	900 MHz, PW; GSM	1.25 W/kg	20 h	-	No effect of RF radiation alone. Reduction of MMC-induced micronucleus frequency. Data indicative of an adaptive response	Sannino et al. (2009a)
Sister-chromatid exchange	2450 MHz, PW	75 W/kg	30 min, 2 h	-	MW output was adjusted with a thermistor to keep cells at 36.1 $^{\circ}\text{C}$	Maes et al. (1993)
Sister-chromatid exchange	954 MHz, PW; GSM	1.5 W/kg	2 h	-		Maes et al. (1996)

Table 4.4 (continued)

End-point	Frequency	SAR or power density	Duration	Results	Comments	Reference
Sister-chromatid exchange	380, 900, 1800 MHz, PW; TETRA, DCS, GSM	0.08–1.7 W/kg	72 h	-		Antonopoulos et al. (1997)
Sister-chromatid exchange	440, 900, 1800 MHz, PW; GSM	1.5 W/kg	30–72 h	-		Eberle et al. (1996)
Sister-chromatid exchange	935.2 MHz, PW; GSM	0.3–0.4 W/kg	2 h	-		Maes et al. (1997)
Sister-chromatid exchange	455.7 MHz, PW; car phone	6.5 W/kg	2 h	-		Maes et al. (2000)
Sister-chromatid exchange	900 MHz, PW; GSM	0.4–10 W/kg	2 h	-		Maes et al. (2001)
Sister-chromatid exchange	900 MHz, PW; GSM	0.3, 1 W/kg	2 h	-		Zeni et al. (2005)
Sister-chromatid exchange	400–900 MHz, PW	-	-	-		Maes et al. (2006)
Sister-chromatid exchange	935 MHz, PW; GSM	1, 2 W/kg	24 h	-		Stronati et al. (2006)
DNA single- and double-strand breaks	935.2 MHz, PW; GSM	0.3–0.4 W/kg	2 h	-		Maes et al. (1997)
DNA single- and double-strand breaks	2450 MHz, PW	2.1 W/kg	2 h	-	No effect, immediately or 4 h after exposure	Vijayalaxmi et al. (2000)
DNA single- and double-strand breaks	1900 MHz, CW, PW	0.1–10 W/kg	2 h	-		McNamee et al. (2002a)
DNA single- and double-strand breaks	2450 MHz, PW	5 mW/cm ²	2 h	-		Zhang et al. (2002)
DNA single- and double-strand breaks	837, 1909 MHz, CW, PW; CDM, TDM	1.0, 2.5, 5.0, 10.0 W/kg	3 h, 24 h	-	Some exposures were from mobile telephones. Temperature variations were ± 0.3 °C and ± 0.5 °C at 3 h and 24 h, respectively. EMS was included as a positive control.	Tice et al. (2002)
DNA single- and double-strand breaks	1900 MHz, CW, PW	0.1–10 W/kg	24 h	-		McNamee et al. (2003)

Table 4.4 (continued)

End-point	Frequency	SAR or power density	Duration	Results	Comments	Reference
DNA single- and double-strand breaks	1800 MHz, PW; GSM	3 W/kg	2 h	-		Baohong et al. (2005)
DNA single- and double-strand breaks	900 MHz, PW; GSM	0.3, 1 W/kg	2 h	-		Zeni et al. (2005)
DNA single- and double-strand breaks	8800 MHz, PW	1.6 kW/kg	40 min	-		Chemeris et al. (2006)
DNA single- and double-strand breaks	1950 MHz, PW; UMTS	0.5, 2 W/kg	24 h	-		Sannino et al. (2006)
DNA single- and double-strand breaks	935 MHz, PW; GSM	1, 2 W/kg	24 h	-		Stronati et al. (2006)
DNA single- and double-strand breaks	1800 MHz, PW; GSM	3 W/kg	1.5, 4 h	-		Baohong et al. (2007)
DNA single- and double-strand breaks	120 000, 130 000 MHz, PW; THz	0.2–2 W/kg	20 min	-		Zeni et al. (2007a)
DNA single- and double-strand breaks	1950 MHz, PW(c, i); UMTS	0.05–2 W/kg	4–48 h	-	Controversial data	Schwarz et al. (2008)
DNA single- and double-strand breaks	835 MHz, PW; CDMA	1.17 W/kg	1 h	-	RF radiation induced repairable DNA damage in the presence of aphidicolin	Tiwari et al. (2008)
DNA single- and double-strand breaks	1950 MHz, PW(c, i); UMTS	2 W/kg	24–68 h	-		Zeni et al. (2008)
DNA single- and double-strand breaks	1800 MHz, PW(i); GSM	2 W/kg	24 h	-	No effect of RF-radiation on repair of X-ray-induced DNA damage	Zhijian et al. (2009)
Mutation at <i>HPRT</i> locus	440, 900, 1800 MHz, PW; GSM	1.5 W/kg	30–72 h	-		Eberle et al. (1996)

Table 4.4 (continued)

End-point	Frequency	SAR or power density	Duration	Results	Comments	Reference
Foci	915 MHz, PW; GSM	37 mW/kg	2 h	+	Decrease in 53BP1-foci (measured by immuno-staining); enhanced chromatin condensation (measured by AVTD)	Belyaev et al. (2005)
Foci	905, 915 MHz, PW; GSM	37 mW/kg	1 h	+ at 915 MHz - at 905 MHz	Decrease in 53BP1- and γ -H2AX-foci (measured by immunostaining) and enhanced chromatin condensation (measured by AVTD)	Marková et al. (2005)
Foci	905, 915, 1947 MHz, PW; GSM, UMTS	0.015–0.145 W/kg	1 h	+ at 915 MHz - at 905 MHz + t 1947 MHz	Decrease in 53BP1- and γ -H2AX-foci (measured by immunostaining) and enhanced chromatin condensation (measured by AVTD). Strongest effect at 1947 MHz	Belyaev et al. (2009)

+, increase; ±, equivocal; -, no effect; APC, aphidicholin (inhibitor of DNA repair); AVTD, anomalous viscosity time-dependence; (c, i); continuous or intermittent exposure; CA, chromosomal aberration; CDMA, code-division multiple access; CW, continuous wave; d, day; DCS, Digital Communication System; EMS, ethylmethane sulfonate; FDMA, frequency-division multiple access; FISH, fluorescence *in situ* hybridization; GSM, Global System for Mobile Communication; h, hour; HPRT, hypoxanthine(guanine)phosphoribosyl transferase; min, minute; MMC, mitomycin C; MW, microwave; PW, pulsed wave; s, second; TDMA, time-division multiple access; TETRA, Trans European Trunked Radio; THz, teraHertz; UMTS, Universal Mobile Telecommunication System

SAR of 0.5 W/kg, while there was a small but statistically significant increase in the frequency of aberrations per cell at 2 W/kg. [Figueiredo et al. \(2004\)](#) and [Hansteen et al. \(2009a, b\)](#) carried out conventional analyses of chromosomal aberrations on Giemsa-stained slides prepared with lymphocytes exposed to RF radiation at 1800–10 500 MHz, and observed no effect.

Micronucleus formation

[Zotti-Martelli et al. \(2000\)](#) exposed whole blood from two volunteers to continuous-wave RF radiation at 2450 MHz or 7700 MHz, with power densities of 10, 20 and 30 mW/cm² for 15, 30, and 60 minutes, and reported an increased micronucleus frequency in exposed cells at 30 mW/cm². In a subsequent study, [Zotti-Martelli et al. \(2005\)](#) observed an increase in the frequency of micronuclei in lymphocytes from nine different donors after exposure to RF radiation at 1800 MHz. This experiment was repeated after 3 months; there was significant variation between experiments. [The Working Group noted that temperature variation in the first study was not measured in blood samples during exposure, and the increased frequency of micronucleus formation may have been related to heating of the blood samples. Also, there were discrepancies between the data on micronuclei given in the text, figures, and tables]. [d'Ambrosio et al. \(1995, 2002\)](#) reported an increase in the formation of micronuclei in lymphocytes exposed to pulsed-wave RF radiation at 1748 MHz or 9000 MHz for 15 and 10 minutes, respectively, while no such increase was observed in cells exposed to continuous-wave RF at the same frequencies. [Zeni et al. \(2003\)](#) observed no significant effect on micronucleus formation in lymphocytes exposed to continuous or pulsed-wave RF radiation at 900 MHz (GSM). [Scarfi et al. \(2003\)](#) reported no micronucleus induction in lymphocytes exposed to continuous-wave RF radiation at 120–130 GHz. [Sannino et al. \(2009a\)](#) reported that a 20-hour pre-exposure of peripheral blood lymphocytes in the S-phase of the cell

cycle to pulsed-wave RF radiation at 900 MHz decreased the micronucleus frequency induced by mitomycin C (MMC), suggesting the existence of an adaptive response (see [Table 4.4](#) for details).

Sister-chromatid exchange (SCE)

[Maes et al. \(1996\)](#) did not find an effect on SCE in lymphocytes exposed to pulsed-wave RF radiation at 954 MHz, with a SAR of 1.5 W/kg, for 2 hours. Likewise, [Antonopoulos et al. \(1997\)](#) did not find an effect on SCE in lymphocytes exposed to RF radiation at 380–1800 MHz, with a SAR of 0.08–1.7 W/kg, for 72 hours.

Phosphorylation of histone protein H2AX and TP53-binding protein 53BP1

Over the past decade, several studies have demonstrated that two cellular check-point proteins, H2AX and TP53-binding protein 53BP1 are rapidly phosphorylated after induction of DNA damage in the form of double-strand breaks. These proteins then congregate to provide a scaffold structure to the repair sites ([Paull et al., 2000](#); [Schultz et al., 2000](#); [DiTullio et al., 2002](#); [Fernandez-Capetillo et al., 2002, 2004](#); [Sedelnikova et al., 2002](#); [Ismail et al., 2007](#)). By use of specific antibodies with fluorescent tags, γ -H2AX – the phosphorylated form of H2AX – and 53BP1 can be visualized as discrete foci, which can be counted directly with a fluorescence microscope.

The AVTD assay is used to detect stress-induced changes in chromatin conformation. [Shckorbatov et al. \(1998, 2009\)](#) and [Sarimov et al. \(2004\)](#) have reported changes in chromatin condensation in human lymphocytes exposed to RF radiation at 42.2 GHz, 35 GHz or 895–915 MHz, respectively, which prevented access of proteins involved in repair of DNA double-strand breaks. [Belyaev et al. \(2005\)](#) exposed human lymphocytes for 2 hours to pulsed-wave RF radiation at 915 MHz (GSM), with a SAR of 37 mW/kg, and reported significant

effects on chromatin condensation and a distinct reduction in the number of 53BP1-foci in samples from all individuals; these results were similar to those found after heat-shock treatment. The overall data suggested a reduced accessibility of 53BP1 to repair DNA double-strand breaks due to chromatin condensation. [Markovà et al. \(2005\)](#) exposed human lymphocytes to pulsed-wave RF radiation at 905 MHz or 915 MHz (GSM), with a SAR of 37 mW/kg, for 1 hour. Chromatin condensation and decreased numbers of 53BP1- and γ -H2AX-foci were observed in cells after exposure at 915 MHz, but not at 905 MHz. The response was similar in healthy subjects and in subjects hypersensitive to RF radiation. [Belyaev et al. \(2009\)](#) exposed lymphocytes to pulsed-wave RF radiation at 905 MHz or 915 MHz (GSM), or 1947 MHz (UMTS), with a SAR of 15–145 mW/kg, for 1 hour. Chromatin condensation and reduction in numbers of 53BP1- and γ -H2AX-foci were much more pronounced in cells after exposure at 1947 MHz than at 915 MHz; there were no such effects after exposure at 905 MHz. The decrease in number of foci persisted for up to 72 hours after exposure, suggesting that not only the formation of double-strand breaks was affected, but also their repair. [Markovà et al. \(2010\)](#) used VH10 primary fibroblasts established from human foreskin and mesenchymal stem cells isolated from adipose tissue of two healthy persons. These cells were exposed to pulsed-wave RF radiation at 905 MHz or 915 MHz (GSM; SAR, 37 mW/kg), or at 1947 MHz (UMTS; SAR, 39 mW/kg), as a single exposure for 1, 2 or 3 hours, or as repeated exposures for 1 hour per day, 5 days per week, for 2 weeks. The decrease in the number of 53BP1-foci was more pronounced in stem cells than in foreskin fibroblasts, and the stem cells did not adapt to long-term exposure to RF radiation.

Aneuploidy

Peripheral blood lymphocytes from five individuals were stimulated with phytohaemagglutinin (PHA) and exposed for 72 hours

to continuous-wave RF radiation at 830 MHz (SAR, 1.6–8.8 W/kg), in an incubator set at temperatures between 33.5 °C (at the highest SAR value) and 37.5 °C. The incidence of aneuploidy of chromosome 17 was determined by use of a probe for α -satellite DNA repeat-sequences present in its centromeric region. The data indicated a linear and SAR-dependent increase in aneuploidy in cells exposed to RF radiation at SAR 2.0–8.2 W/kg (6–9%) compared with control cells (4–5%). Control experiments without RF radiation were conducted at 34.5–41 °C, showing no change in aneuploidy at temperatures up to 38.5 °C. This indicates that the effect of RF radiation was produced via a non-thermal pathway ([Mashevich et al., 2003](#)).

Peripheral blood lymphocytes from nine donors were stimulated with PHA for 1–6 hours, then exposed to continuous-wave RF radiation at 100 GHz (power density, 0.031 mW/cm²) for 1, 2 or 24 hours in an incubator in which CO₂ levels were not controlled. After exposure, the cells were incubated for a total culture period of 69–72 hours, with CO₂ levels at 5%. The cells were harvested and changes in chromosomes 1, 10, 11 and 17 were analysed by means of the FISH technique. For chromosomes 11 and 17, a 30% increase in aneuploidy was found after exposure for 2 or 24 hours, while chromosomes 1 and 10 were not affected. Asynchronous replication of centromeres 1, 11, and 17 was increased by 40% after 2 hours of exposure, while that of all four centromeres had increased by 50% after 24 hours of exposure. During the experiments, fibre-optic sensors were used to measure differences in temperature between exposed and sham-exposed samples; the difference never exceeded 0.3 °C ([Korenstein-Ilan et al., 2008](#)).

[Mazor et al. \(2008\)](#) exposed PHA-stimulated lymphocytes from 10 individuals to continuous-wave RF radiation at 800 MHz (SAR, 2.9 or 4.1 W/kg) for 72 hours, with the incubator set at 33.5 °C to maintain the sample temperature at 36–37 °C, in particular at the high SAR value.

Aneuploidy was scored for chromosomes 1, 10, 11, and 17 by use of the FISH technique. An increased frequency of cells aneuploid for chromosomes 11 and 17 was observed at the lower SAR of 2.9 W/kg, and for chromosomes 1 and 10 at the higher SAR of 4.1 W/kg. Multisomy (chromosomal gain) was the primary contributor to the increase in aneuploidy. Control experiments – without exposure to RF radiation – were conducted in the temperature range 33.5–41 °C; there was no change in aneuploidy.

Spindle disturbance (experiments with human-hamster hybrid cells)

The well established human–hamster hybrid (A_L) cell line, containing a single copy of human chromosome 11, was exposed to pulsed-wave RF radiation at 835 MHz, with increasing electric field strengths from 5 to 90 V/m, for 30 minutes ([Schmid & Schrader, 2007](#)). The results indicated a field strength-dependent increase in the frequency of spindle disturbances during anaphase/telophase of cell division. [The Working Group noted the absence of negative and positive controls.] [Schrader et al. \(2008\)](#) reported similar increases in spindle disturbances in A_L cells exposed for 30 minutes or 2 hours to RF radiation at 835 MHz (90 V/m) compared with non-exposed controls. [Schrader et al. \(2011\)](#) exposed A_L cells to RF radiation at 900 MHz (amplitude-modulated and unmodulated), at electric field strengths of 45 or 90 V/m, and with a SAR of 11.5 W/kg, for 30 minutes. The experiments were conducted with separate electric (E field) and magnetic (H field) components of RF radiation, at 20–22 °C. A significant increase in the frequency of spindle disturbances was observed in cells exposed to the E component, while no effect was seen in cells exposed to the H component (compared with non-exposed control cells). [Hintzsche et al. \(2011\)](#) also reported an increase in spindle disturbance during the anaphase/telophase of cell division in the same A_L cell line exposed to continuous-wave RF radiation at

106 GHz (power densities, 0.043–4.3 mW/cm²) for 30 minutes.

(ii) *Studies with two or more end-points*

[Tice et al. \(2002\)](#) reported a significant and reproducible increase in micronucleus formation in human lymphocytes exposed for 24 hours to RF radiation at 837 or 1909.8 MHz, with an average SAR of 5.0 or 10.0 W/kg. There was no increase in the number of DNA strand breaks in leukocytes, as measured with the alkaline comet assay. [McNamee et al. \(2002a, 2003\)](#) reported no effects on DNA strand-break induction or micronucleus formation in cells exposed to continuous- or pulsed-wave RF radiation at 1900 MHz, with SARs of up to 10 W/kg, for 2 or 24 hours. [Zhang et al. \(2002\)](#) observed no induction of DNA strand breaks or formation of micronuclei in human lymphocytes exposed to pulsed-wave RF radiation at 2450 MHz compared with controls. [Zeni et al. \(2008\)](#) reported no increase in DNA strand breaks or micronucleus formation in human lymphocytes exposed to intermittent (6 minutes on, 2 hours off) RF radiation at 1900 MHz (SAR, 2.2 W/kg) for 24–68 hours. Likewise, [Schwarz et al. \(2008\)](#), reported no increase in DNA strand-break induction or micronucleus formation in PHA-stimulated or non-stimulated human lymphocytes exposed for 16 hours to intermittent (5 minutes on, 10 minutes off) RF radiation at 1950 MHz (SAR, 0.1 W/kg).

[Garaj-Vrhovac et al. \(1992\)](#) reported significantly increased frequencies of chromosomal aberrations and micronuclei in human peripheral blood lymphocytes exposed for up to 60 minutes to continuous-wave RF radiation at 7700 MHz, with power densities up to 30 mW/cm².

In a series of studies from one laboratory, no increase in the frequency of chromosomal aberrations or micronuclei was reported in human lymphocytes exposed to RF radiation at 2450 MHz for 90 minutes, to continuous-wave RF radiation at 835 or 847 MHz for 24 hours, or

to RF radiation at 2450 or 8200 MHz for 2 hours ([Vijayalaxmi et al., 1997b](#), [2001b](#), [c](#), [2006](#)).

[Maes et al. \(1993\)](#) found a time-dependent increase in the frequencies of chromosomal aberrations and micronuclei in peripheral blood lymphocytes exposed to pulsed-wave RF radiation at 2450 MHz (SAR, 75 W/kg) for 30 or 120 minutes. Both effects were statistically significant for the exposure of 120 minutes. No induction of SCE was found. In this study, the microwave output was adjusted by use of a thermistor thermometer to maintain the temperature of the cells at 36.1 °C. In subsequent experiments, [Maes et al. \(2000, 2001\)](#) examined human lymphocytes exposed to pulsed-wave RF radiation at 455.7 MHz (SAR, 6.5 W/kg) or 900 MHz (SAR, 0.4–10 W/kg) for 2 hours; no increase in chromosomal aberrations or SCE was observed.

[Stronati et al. \(2006\)](#) did not report significant changes in DNA strand-break induction, chromosomal aberrations, micronucleus formation or SCE in blood cells exposed to pulsed-wave RF radiation at 935 MHz (SAR, 1 or 2 W/kg). [Eberle et al. \(1996\)](#) measured chromosomal aberrations, micronucleus formation, SCE, and mutations at the *HPRT* locus in human lymphocytes exposed to RF radiation at 440, 900, or 1800 MHz (SAR, 1.5 W/kg). Exposure times varied (39, 50, 70 hours), depending on the experiment. No significant effects were observed for any of these end-points in RF-exposed cells compared with controls.

(b) Humans: other primary and continuously growing cultured cells

Some details on the exposure conditions to RF radiation and a short conclusion for each publication are presented in [Table 4.5](#).

(i) Amniotic cells

Human amniotic cells were exposed to RF radiation at 900 MHz (GSM; SAR, 0.25 W/kg) for 24 hours. Chromosomes were stained by use of the R-banding method and examined to determine

the incidence of structural and numerical aberrations. Exposure to RF radiation had no effect ([Bourthoumieu et al., 2010](#)). [The Working Group noted that R-banding is not recommended for analysis of chromosomal aberrations.] In a subsequent study by the same authors, amniotic cells were collected during amniocentesis from three separate donors. The cells were cultured for 15 days before being exposed to RF radiation at 900 MHz (GSM, pulsed-wave; pulse duration, 0.577 ms; pulse-repetition rate, 217 Hz; SAR, 0.25, 1, 2 or 4 W/kg) for 24 hours in a wire-patch cell at exposure temperatures of 36.3 ± 0.4 °C, 37.0 ± 0.2 °C, 37.5 ± 0.4 °C and 39.7 ± 0.8 °C, respectively, for the four SAR levels. The cells were processed for analysis by two-colour FISH with centromeric α -satellite repetitive probes for chromosomes 11 and 17 in interphase cells. No significant differences were observed between exposed and sham-exposed cells in the percentages of monosomic, trisomic cells or the total number of cells aneuploid for chromosomes 11 or 17 ([Bourthoumieu et al., 2011](#)).

(ii) Glioblastoma and neuroblastoma cells

No effects on DNA strand-break induction were observed in human U87MG glioblastoma cells exposed for up to 24 hours to continuous-wave or pulsed-wave RF radiation at 835, 847, or 2450 MHz (SAR, 0.6 W/kg at 835/847 MHz, and 0.7 or 1.9 W/kg at 2450 MHz) ([Malyapa et al. \(1997a, b\)](#)).

[Miyakoshi et al. \(2002\)](#) did not find an effect on DNA strand-break induction in human MO54 glial cells – derived from a patient with a brain tumour – exposed to RF radiation at 2450 MHz (average SAR, 50 or 100 W/kg) for 2 hours. Likewise, [Sakuma et al. \(2006\)](#) reported no effect on DNA strand-break induction in human A172 glioblastoma cells exposed to pulsed-wave RF radiation at 2142.5 MHz (SAR, up to 800 mW/kg) for 2 or 24 hours, and [Luukkonen et al. \(2009, 2010\)](#) found no effects on DNA strand-break induction in cultured human SH-SY5Y

Table 4.5 Genetic and related effects in human cells (other than lymphocytes) exposed to radiofrequency radiation *in vitro*

End-point	Cells	Frequency	SAR or power density	Duration	Results	Comments	Reference
Aneuploidy	HAC	900 MHz, PW; GSM	0.25, 1, 2, 4 W/kg	24 h	-	Chromosomes 11 and 17 were included in this study	Bourthoumieu et al. (2011)
Chromosomal aberration	HAC	900 MHz, PW; GSM	0.25 W/kg	24 h	-		Bourthoumieu et al. (2010)
Micronucleus formation	BUC	PW; mobile phone	NR	1 h/d for 2.3 yr	+		Yadav & Sharma (2008)
Micronucleus formation	BUC	PW; mobile phone	NR	3 h/wk for 5–10 yr	-		Hintzsche & Stopper (2010)
Micronucleus formation	HSF	1800 MHz, CW, PW(i); GSM	2 W/kg	1, 4, 24 h	-	Replication study. Previous results not confirmed.	Speit et al. (2007)
Micronucleus formation	SHF	1950 MHz, PW(c-i); UMTS	0.05–2 W/kg	4–48 h	+ after 12 h exposure	Controversial data	Schwarz et al. (2008)
Micronucleus formation	SHF	900 MHz, PW; GSM	1 W/kg	24 h	-		Sannino et al. (2009b)
DNA single- and double-strand breaks	GLB	2450 MHz, CW	0.7 W/kg	2–24 h	-		Malyapa et al. (1997a)
DNA single- and double-strand breaks	GLB	835, 847 MHz, CW, PW; FMCW, CDMA	0.6 W/kg	2–24 h	-		Malyapa et al. (1997b)
DNA single- and double-strand breaks	GLB	2450 MHz	13–100 W/kg	2 h	-		Miyakoshi et al. (2002)
DNA single- and double-strand breaks	GLB	2000 MHz, PW; CW, IMT	0.08, 0.25, 0.80 W/kg	2 h, 24 h	-		Sakuma et al. (2006)
DNA single- and double-strand breaks	HSF	1800 MHz, CW, PW(i); GSM	2 W/kg	1, 4, 24 h	-	Replication study. Previous results not confirmed.	Speit et al. (2007)
DNA single- and double-strand breaks	HTR	1817 MHz, PW; GSM	2 W/kg	1 h	-		Valbonesi et al. (2008)

Table 4.5 (continued)

End-point	Cells	Frequency	SAR or power density	Duration	Results	Comments	Reference
DNA single- and double-strand breaks	HTR	1800 MHz, CW, PW(i); GSM	2 W/kg	4–24 h	– with CW – with PW at 4 h + with PW at 16 h and 24 h	Differential response between CW and PW and exposure duration	Franzellitti et al. (2010)
DNA single- and double-strand breaks	LEP	1800 MHz, PW; GSM	1, 2, 3 W/kg	2 h	– at 1 and 2 W/kg + at 3 W/kg		Lixia et al. (2006)
DNA single- and double-strand breaks	LEP	1800 MHz, PW(i); GSM	1, 2, 3, 4 W/kg	2 h	– at 1 and 2 W/kg + at 3 and 4 W/kg		Yao et al. (2008)
DNA single- and double-strand breaks	LUF	2000 MHz, PW, CW; IMT	0.08 W/kg	2, 24 h	–		Sakuma et al. (2006)
DNA single- and double-strand breaks	LYB	813, 836 MHz, PW; iDEN, TDMA	2.4–26 mW/kg	2–21 h	±	Inconsistent results	Phillips et al. (1998)
DNA single- and double-strand breaks	LYB	813, 836, 835, 847 MHz, CW, PW; iDEN, TDMA, FDMA, CDMA	0.0024–0.026 W/kg, 3.2 W/kg	2–21 h	–		Hook et al. (2004a)
DNA single- and double-strand breaks	LYB	1800 MHz, PW; GSM	2 W/kg	6–24 h	–		Zhijian et al. (2010)
DNA single- and double-strand breaks	NUB	872 MHz, CW, PW; GSM	5 W/kg	1 h	–	Temperature-controlled conditions	Luukkonen et al. (2009)
DNA single- and double-strand breaks	NUB	872 MHz, CW, PW; GSM	5 W/kg	3 h	–	Temperature-controlled conditions	Luukkonen et al. (2010)
DNA single- and double-strand breaks	SHF	1800 MHz, PW (c, i)	2 W/kg	4–24 h	+	Controversial data	Diem et al. (2005)
DNA single- and double-strand breaks	SHF	1950 MHz, PW(c-i); UMTS	0.05–2 W/kg	4–48 h	+	Controversial data	Schwarz et al. (2008)

Table 4.5 (continued)

End-point	Cells	Frequency	SAR or power density	Duration	Results	Comments	Reference
DNA single- and double-strand breaks	SHF	900 MHz, PW; GSM	1 W/kg	24 h	-		Sannino et al. (2009b)
Foci	HFB, MST	905, 915, 1947 MHz, PW; GSM, UMTS	0.037, 0.039 W/kg	1–3 h	+ at 915 MHz - at 905 MHz + at 1947 MHz	Decrease in 53BP1-foci, measured by immunostaining	Marková et al. (2010)
Spindle disturbance	HHH	835 MHz, PW; GSM	5–90 V/m	30 min	+	Mitotic cell fraction was scored on slides stained with 2% acetic orcein.	Schmid & Schrader (2007)
Spindle disturbance	HHH	835 MHz, PW	62.5 mW/kg	10 min to 2 h	+	Mitotic cell fraction was scored on slides stained with 2% acetic orcein.	Schrader et al. (2008)
Spindle disturbance	HHH	1060 MHz, CW	0.043–4.3 mW/cm ²	30 min	+	Mitotic cell fraction was scored on slides stained with 2% acetic orcein.	Hintzsche et al. (2011)
Spindle disturbance	HHH	900 MHz, CW, PW	0.0115 W/kg	30 min	+	Mitotic cell fraction was scored on slides stained with 2% acetic orcein.	Schrader et al. (2011)
8-OHdG, oxidative damage in DNA	SPR	1800 MHz	0.4–27.5 W/kg	16 h	+	Temperature controlled at 21 °C; maximum increase 0.4 °C during exposure.	De Iuliis et al. (2009)

+, increase; ±, equivocal; -, no effect; 8-OHdG, 8-hydroxy-2'-deoxyguanosine; BUC, human buccal cells; (c, i), continuous and intermittent exposure; d, day; FDMA, frequency-division multiple access; FMCW, frequency-modulated continuous wave; GLB, glioblastoma cells; h, hour; HAC, human amniotic cells; HFB, human foreskin fibroblasts; HHH, hamster-human hybrid cells; HTR, trophoblast cells; iDEN, Integrated Digital Enhanced Network; IMT, International Mobile Telecommunication; LEP, lens epithelial cells; LUF, human fibroblasts from fetal lung; LYB, lymphoblastoid cells; min, minute; MST, mesenchymal stem cells; NUB, neuroblastoma cells; NR, not reported; SHF, skin human fibroblasts; SPR, sperm cells; TDAM, time-division multiple access; yr, year

neuroblastoma cells exposed to continuous- or pulsed-wave RF radiation at 872 MHz, with a SAR of 5 W/kg. In the studies mentioned above the alkaline comet assay was used to measure strand breakage in DNA.

(iii) *Lens epithelial cells*

Immortalized SRA01/04 human lens epithelial cells were exposed to pulsed-wave RF radiation at 1800 MHz (SAR, 1, 2 or 3 W/kg) for 2 hours to investigate induction of DNA breakage, which was measured by means of the alkaline comet assay. DNA-damage repair was evaluated by further incubation of the exposed cells for 30, 60, 120 or 240 minutes. There was a significant increase ($P < 0.05$) in DNA strand-breaks at a SAR of 3 W/kg immediately after exposure, which had decreased at 30 minutes, and had diminished to control levels at later time-points. At SARs of 1 and 2 W/kg, there were no significant differences between exposed cells and sham-exposed controls ([Lixia et al., 2006](#)).

In a similar study, DNA strand breaks were measured in SRA01/04 human lens epithelial cells exposed to intermittent (5 minutes on, 10 minutes off) pulsed-wave RF radiation at 1800 MHz (SAR, 1, 2, 3, or 4 W/kg) for 2 hours. There was no effect on DNA single-strand breaks – measured with the alkaline comet assay – at SARs of 1 or 2 W/kg, but a significant increase at SARs of 3 or 4 W/kg ($P < 0.001$). At these two higher SAR values, there was no difference in the induction of DNA double-strand breaks, measured with the γ H2AX-focus formation assay ([Yao et al., 2008](#)).

(iv) *Lung fibroblasts*

[Sakuma et al. \(2006\)](#) exposed human IMR-90 fetal lung fibroblasts to pulsed-wave RF radiation at 2000 MHz (SAR, 80 mW/kg) for 24 hours, and observed no effect on induction of DNA strand breaks.

(v) *Lymphoblastoid cells*

[Phillips et al. \(1998\)](#) studied DNA strand-break induction in human Molt-4 lymphoblastoid cells exposed for 2, 3 or 21 hours to RF radiation at 813 or 835 MHz as iDEN (Integrated Digital Enhanced Network) and TDMA (time-division multiple access) signals, with very low SARs of 2.4, 24, 2.6 and 26 mW/kg. There was a general decrease in the number of strand breaks at lower SARs at 2 and 21 hours (but not at 3 hours), and inconsistent results at higher SARs, depending on the type of RF signal, power intensity and duration of exposure. [Hook et al. \(2004a\)](#) examined DNA strand-break induction in Molt-4 cells exposed to the same and additional signals (813.56–847.74 MHz) at the same and higher SARs (2.4 mW/kg–3.4 W/kg) than in the study by [Phillips et al. \(1998\)](#). No effect on DNA strand-break induction was noted. [Zhijian et al. \(2010\)](#) did not find any effect on DNA strand-break induction when human HMy2.CIR lymphoblastoid B-cells were exposed to pulsed-wave RF radiation at 1800 MHz (SAR, 2 W/kg), for 6–24 hours.

(vi) *Skin fibroblasts*

[Diem et al. \(2005\)](#) exposed human ES-1 skin fibroblasts to continuous or intermittent (5 minutes on, 10 minutes off) RF radiation at 1800 MHz (SAR, 1.2 or 2 W/kg) for 4–24 hours. The cells were examined visually and subjectively to evaluate DNA single- and double-strand breaks by use of the alkaline and neutral comet assays. A “tail factor” was devised to express the results. The authors concluded that: there was a significant increase in tail factor after a 16-hour exposure, with no further increase after 24 hours; and that intermittent exposure produced a stronger effect than continuous exposure.

In a study from the same group, [Schwarz et al. \(2008\)](#) used ES-1 cells exposed for 4–48 hours to continuous and intermittent RF radiation at 1950 MHz (UMTS), with a range of SAR values (0.05–2 W/kg). The results from the analyses of

DNA strand breaks by means of the comet assay indicated a significant increase in tail factor ($P < 0.02$) at SAR 0.05 W/kg. In addition, there was a significant increase ($P < 0.02$, at SAR 0.05 W/kg) in the frequency of micronuclei, which turned out be centromere-negative (suggesting a clastogenic effect). In a similar experiment in peripheral blood lymphocytes, there was no effect on the comet tail factor or micronucleus formation (see above). Several discussions about the mode of data acquisition in these two studies subsequently appeared in scientific journals ([Rüdiger et al., 2006](#); [Vijayalaxmi et al., 2006](#); [Tuffs, 2008](#); [Vogel, 2008](#); [Wolf, 2008](#); [Balzano, 2008](#); [Kuster, 2008](#); [Drexler & Schaller, 2009](#); [Rüdiger, 2009b, c](#); [Lerchl & Wilhelm, 2010](#); [Baan, 2009](#)).

[Speit et al. \(2007\)](#) performed independent experiments to replicate and confirm the results of the two studies mentioned above, by use of the same ES-1 skin fibroblasts, the same exposure system supplied by the same company, and the same laboratory protocols. The comet tail factor as well as computerized image-analysis were used to quantify the DNA strand breaks. The experiments were also performed in Chinese hamster V79 cells. The results showed no effect on DNA breakage either by the alkaline comet assay or by the micronucleus test, in either fibroblasts or V79 cells.

Skin fibroblasts established from healthy individuals or from subjects with Turner syndrome were exposed to 900 MHz pulsed-wave RF radiation (SAR, 1 W/kg). It was suggested that cells from patients with Turner syndrome were sensitive to the effects of weakly genotoxic agents ([Scarfi et al., 1997a, b](#)). No effects on DNA strand-break induction or micronucleus formation were observed in either cell line ([Sannino et al., 2009b](#)).

[Marková et al. \(2010\)](#) observed no effect on 53BP1 foci in skin fibroblasts exposed to RF radiation at 905 MHz (SAR, 37 mW/kg). In contrast, a decrease was seen when the same cells were exposed at 915 or 1947 MHz at similar SAR

levels [the Working Group noted that this technique assesses repair foci of DNA double-strand breaks; it is different from the comet assay used for analysis of DNA strand breaks in the other studies with skin cells discussed above].

(vii) *Mesenchymal stem cells*

[Marková et al. \(2010\)](#) observed a decrease in the number of 53BP1 foci in mesenchymal stem cells exposed to RF radiation at 915 or 1947 MHz (GSM; SAR 37 and 39 mW/kg, respectively) for 1, 2, or 3 hours; no effect was noted after exposure at 905 MHz (SAR, 37 mW/kg).

(viii) *Sperm cells*

[De Iuliis et al. \(2009\)](#) studied purified human spermatozoa exposed to RF radiation at 1800 MHz (SAR, 0.4–27.5 W/kg) for 16 hours at 21 °C. With increasing SAR values, motility and vitality of the sperm cells were significantly reduced, while mitochondrial production of reactive oxygen species was significantly increased ($P < 0.001$). There was also a significant increase ($P < 0.05$) in formation of 8-OHdG adducts (measured immunochemically) and DNA fragmentation (measured with the TUNEL – terminal deoxynucleotidyl transferase dUTP nick end labelling – assay) at SARs of 2.8 W/kg and higher. The temperature during these experiments was kept at 21 °C; the highest observed exposure-induced temperature increase was +0.4 °C, at a SAR of 27.5 W/kg.

(ix) *Trophoblast cells*

[Valbonesi et al. \(2008\)](#) observed no effects on DNA strand-break induction in human HTR-8/SVneo trophoblast cells exposed to pulse-modulated RF radiation at 1817 MHz (SAR, 2 W/kg) for 1 hour.

[Franzellitti et al. \(2010\)](#) observed no effects on DNA strand-break induction in HTR-8/SVneo trophoblast cells exposed to continuous-wave RF radiation at 1800 MHz (SAR, 2 W/kg) for 4, 16 or 24 hours. Exposure to this radiation as pulsed-wave amplitude-modulated signals (GSM-217 Hz

and GSM-Talk), caused a significant increase in DNA strand breakage after all three treatment periods when the results of the comet assay were expressed as “% DNA in tail.” The number of DNA strand breaks decreased rapidly during the 2 hours after exposure.

(c) *Humans: interaction of RF radiation with known genotoxic agents*

Some details on the exposure conditions to RF radiation and a short conclusion for each publication are presented in [Table 4.6](#). Unless otherwise mentioned, the results discussed below refer to those observed in human peripheral blood lymphocytes exposed to RF radiation before, during or after exposure to a genotoxic agent.

(i) *Chemotherapeutic drugs*

[Gadhia et al. \(2003\)](#) reported a synergistic increase in chromosomal aberrations (rings, dicentrics) and SCE in lymphocytes collected from mobile-phone users and treated with mitomycin C (MMC) *in vitro*, compared with cells from controls (non-phone users) treated with MMC. This effect was stronger in mobile-phone users who smoked and consumed alcohol.

[Maes et al. \(2006\)](#) found no effect of treatment with MMC on induction of DNA strand breaks, chromosomal aberrations or SCE in lymphocytes obtained from workers at a mobile-phone company. In a series of experiments *in vitro*, the same authors reported a highly reproducible synergistic effect ([Maes et al., 1996](#)), a weak synergistic effect ([Maes et al., 1997](#)), an inconsistent synergistic effect ([Maes et al., 2000](#)), or no synergistic effect ([Maes et al., 2001](#)) of exposure to RF radiation on MMC-induced SCE. [The Working Group noted that the authors made several suggestions regarding possible mechanistic explanations for their findings, which were not pursued in detail. The authors also mentioned the possibility of a thermal effect, and indicated

the incomplete characterization of the exposure conditions in their studies.]

[Zhang et al. \(2002\)](#) investigated a possible synergistic effect in human lymphocytes exposed to RF radiation at 2450 MHz (5 mW/cm²; 2 hours) followed by treatment with MMC (0.0125–0.1 µg/ml; 24 hours). While RF radiation had no effect by itself, it significantly increased the effect of the higher doses of MMC on DNA strand-break induction and micronucleus formation. Since the temperature increase during the 2-hour exposure was less than 0.5 °C, the synergy was not likely to be due to thermal effects.

[Baohong et al. \(2005\)](#) exposed human lymphocytes to pulsed-wave RF radiation at 1800 MHz (SAR, 3 W/kg) for 2 hours, before, together with, or after incubation for 3 hours with four different chemicals. After these treatments, the cells were washed and processed for measurement of DNA strand-break induction at once or after further incubation for 21 hours. Exposure to RF radiation alone had no effect. All combinations of MMC or 4-nitroquinoline-1-oxide (4NQO) with RF radiation showed a significant increase in DNA breakage, compared with the results after incubation with the chemical alone. No such effect was observed when exposure to RF radiation was combined with treatment with bleomycin or methylmethane sulfonate (MMS), suggesting that interaction between RF radiation and different chemical mutagens could vary.

[Hansteen et al. \(2009a\)](#) found no effect on MMC-induced chromosomal aberrations after exposure of human lymphocytes to pulsed-wave RF radiation at 16.5 GHz (power density, 10 W/m²) or 18 GHz continuous-wave RF radiation (power density 1 W/m²) for 53 hours, with MMC added after 30 hours. Similar results were reported by the same authors for exposures to continuous-wave or pulsed-wave RF radiation at 2.3 GHz (power density, 10 W/m²) in combination with MMC ([Hansteen et al., 2009b](#)).

[Sannino et al. \(2009a\)](#) reported that pre-exposure of human lymphocytes to pulsed-wave RF

Table 4.6 Interaction between radiofrequency radiation and known genotoxic agents in human cells *in vitro*

End-point	Cells	Genotoxic agent	Frequency (MHz)	SAR or power density	Duration	Results	Comments	Reference
Chromosomal aberration	PBL	MMC or X-rays	900 MHz, PW; GSM	0.4–10 W/kg	RF radiation for 2 h, followed by X-rays for 1 min (1 Gy) or MMC for 72 h	-	No effect of RF radiation; no synergistic effects of RF radiation and MMC or X-rays	Maes et al. (2001)
Chromosomal aberration	PBL from phone users	MMC	890–960 MHz, PW; GSM	NR	Phone use for 1–3 h/d for 2 yr, MMC for 48 h	+	Increased gaps/dicentric after RF radiation; synergistic effect of RF radiation with MMC	Gadhia et al. (2003)
Chromosomal aberration	PBL	Gamma-rays	2500 MHz 10 500 MHz, PW	627 W/kg 0.25 W/kg	40 s 5 min	-	MW oven used as 2.5 GHz source. No effect of RF radiation; no synergistic effect with gamma-rays	Figueiredo et al. (2004)
Chromosomal aberration	PBL	MMC	400–900 MHz, PW	NR	2.3 yr (> 1h/d) MMC for 72 h	-	Lymphocytes from exposed workers. No synergistic effect with MMC	Maes et al. (2006)
Chromosomal aberration	PBL	X-rays	935 MHz, PW; GSM	1 or 2 W/kg	1 min (1 Gy) X-rays, 24 h RF radiation	-	No effect of RF radiation; no synergistic effect with X-rays	Stronati et al. (2006)
Chromosomal aberration	PBL	X-rays	1950 MHz, PW, UMTS	0.5, 2 W/kg	X-rays 5 min, RF radiation for 24 h	+ at 2 W/kg - at 0.5 W/kg	No effect of RF radiation; synergistic effect of RF radiation with X-rays (4 Gy) at the higher SAR	Manti et al. (2008)
Chromosomal aberration	PBL	MMC	1800, 1650 MHz, CW, PW	0.1, 1 mW/cm ²	53 h; MMC added at 30 h	-	No effect of RF radiation; no synergistic effect with MMC	Hansteen et al. (2009a)
Chromosomal aberration	PBL	MMC	2300 MHz, CW, PW	1 mW/cm ²	53 h; MMC added at 30 h	-	No effect of RF radiation; no synergistic effect with MMC	Hansteen et al. (2009b)
Micronucleus formation	PBL	MMC	2450 MHz, PW	5 mW/cm ²	2 h, then MMC for 24 h	+	No effect of RF radiation; synergistic effect with MMC	Zhang et al. (2002)
Micronucleus formation	PBL	X-rays	935 MHz, PW; GSM	1 or 2 W/kg	1 min (1 Gy) X-rays, 24 h RF	-	No effect of RF radiation; no synergistic effect with X-rays	Stronati et al. (2006)
Micronucleus formation	PBL	MMC	900 MHz, PW; GSM	1.25 W/kg	20 h; MMC for 24 h	+	Reduction of MMC-induced MN frequency (adaptive response?) in lymphocytes from 4 out of 5 donors	Sannino et al. (2009a)

Table 4.6 (continued)

End-point	Cells	Genotoxic agent	Frequency (MHz)	SAR or power density	Duration	Results	Comments	Reference
DNA single- and double-strand breaks	PBL	MMC	2450 MHz, PW	5 mW/cm ²	2 h, then MMC for 24 h	+	No effect of RF radiation; synergistic effect with MMC	Zhang et al. (2002)
DNA single- and double-strand breaks	PBL	MMC, 4NQO	1800 MHz, PW; GSM	3 W/kg	2 h RF irradiation, 3 h with the chemical	+	No effect of RF radiation. Exposure to chemicals before, during or after RF irradiation showed a synergistic effect with MMC and 4NQO	Baohong et al. (2005)
DNA single- and double-strand breaks	PBL	BLM, MMS	1800 MHz, PW, GSM	3 W/kg	2 h RF radiation 3 h with the chemical	-	No effect of RF radiation. Exposure to chemicals before, during or after RF irradiation showed no synergistic effect with BLM and MMS	Baohong et al. (2005)
DNA single- and double-strand breaks	PBL	MMC	400–900 MHz, PW	NR	2.3 yr (> 1 h/d) MMC for 72 h	-	Lymphocytes from exposed workers. No synergistic effect with MMC	Maes et al. (2006)
DNA single- and double-strand breaks	PBL	X-rays	935 MHz, PW; GSM	1 or 2 W/kg	1 min (1 Gy) X-rays, 24 h RF	-	No effect of RF radiation; no synergistic effect with X-rays	Stronati et al. (2006)
DNA single- and double-strand breaks	PBL	UV	1800 MHz, PW; GSM	3 W/kg	1.5 or 4 h; just after UVC at 0.25–2.0 J/m ²	+ at 4 h + at 1.5 h	Effect with UV depended on exposure duration: decrease at 1.5 h, increase at 4 h	Baohong et al. (2007)
DNA single- and double-strand breaks	PBL	APC	835 MHz, PW; CDMA	1.2 W/kg	1 h RF irradiation and APC at 0.2 or 2 µg/ml	+	No effect of RF radiation; synergistic RF effect on aphidicolin-induced repairable DNA damage.	Tiwari et al. (2008)
DNA single- and double-strand breaks	NUB	Menadione	872 MHz, CW, PW; GSM	5 W/kg	1 h RF and 50 µM menadione	+ with CW - with PW	Differential effect of CW and PW with menadione	Luukkonen et al. (2009)
DNA single- and double-strand breaks	HSF	MX	900 MHz, PW; GSM	1 W/kg	24 h RF, 1 h MX at 25 µM	-	No synergistic effect on MX-induced SB	Sannino et al. (2009b)
DNA single- and double-strand breaks	PBL	X-rays	1800 MHz, PW(i); GSM	2 W/kg	24 h (on/off for 5/10 min) then 0.25–2 Gy of X-rays	-	No effect of RF radiation; no synergistic effect with X-rays on SB induction or repair	Zhijian et al. (2009)

Table 4.6 (continued)

End-point	Cells	Genotoxic agent	Frequency (MHz)	SAR or power density	Duration	Results	Comments	Reference
DNA single- and double-strand breaks	NUB	FeCl ₂ + DEM	872 MHz, CW; PW; GSM	5 W/kg	1 h or 3 h RF; 1 h FeCl ₂ ± DEM	-	No effect of RF radiation; no synergistic effect with free radical-inducing chemicals	Luukkainen et al. (2010)
DNA single- and double-strand breaks	LYB	DOX	1800 MHz, PW; GSM	2 W/kg	6–24 h RF; 2 h DOX	-	No effect of RF radiation; no synergistic effect with doxorubicin on induction of single- or double-strand breaks; effect on repair (?)	Zhijian et al. (2010)
Sister-chromatid exchange	PBL	MMC	954 MHz, PW; GSM	1.5 W/kg	2 h RF radiation; 72 h MMC	+	No effect of RF radiation; highly reproducible synergistic effect with MMC	Maes et al. (1996)
Sister-chromatid exchange	PBL	MMC	935.2 MHz, PW; GSM	0.3–0.4 W/kg	2 h RF radiation; 72 h MMC	+	No effect of RF radiation; weak synergistic effect with MMC	Maes et al. (1997)
Sister-chromatid exchange	PBL	MMC	455.7 MHz, PW; car phone	6.5 W/kg	2 h (72 h MMC)	±	No effect of RF radiation; inconsistent synergistic effect with MMC	Maes et al. (2000)
Sister-chromatid exchange	PBL	MMC, X-rays	900 MHz, PW; GSM	0.4–10 W/kg	2 h (72 h MMC)	-	No effect of RF radiation; synergistic effect with MMC or with X-rays	Maes et al. (2001)
Sister-chromatid exchange	PBL from phone users	MMC	890–960 MHz, PW; GSM	NR	1–3 h/d for 2 yr; 48 h MMC	+	Increased SCE after RF radiation; synergistic effect with MMC	Gadhia et al. (2003)
Sister-chromatid exchange	PBL	MMC	400–900 MHz, PW	NR	72 h	-	No effect of RF radiation; synergistic effect with MMC	Maes et al. (2006)
Sister-chromatid exchange	PBL	X-rays	935 MHz, PW; GSM	1, 2 W/kg	24 h	-	No effect of RF radiation; synergistic effect with X-rays	Stronati et al. (2006)

+, increase; ±, equivocal; -, no effect; 4NQO, 4-nitroquinoline-1-oxide; APC, aphidicolin; BLM, bleomycin; CW, continuous wave; d, day; DEM, diethyl maleate; FeCl₂, ferrous chloride; h, hour; HSF, human skin fibroblasts; (i), intermittent exposure; LYB, lymphoblastoid cells; min, minute; MMC, mitomycin C; MMS, methylmethane sulfonate; MX, 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone; NUB, neuroblastoma cells; NR, not reported; PBL, peripheral blood lymphocytes; PW, pulsed wave

radiation at 900 MHz (peak SAR, 10 W/kg) for 20 hours reduced the incidence of MMC-induced micronucleus formation, suggesting that non-ionizing radiation is capable of inducing an “adaptive response” similar to that observed in several studies of ionizing radiation.

[Zhijian et al. \(2010\)](#) treated cultured human lymphoblastoid cells with doxorubicin (DOX) for 2 hours before, during and after exposure to pulsed-wave RF radiation at 1800 MHz (SAR, 2 W/kg). No significant effects on DOX-induced DNA strand-break formation were found.

(ii) Genotoxic chemicals

[Tiwari et al. \(2008\)](#) exposed human peripheral blood lymphocytes to RF radiation at 835 MHz (SAR, 1.17 W/kg) for 1 hour, with and without treatment with aphidicolin (APC; 0.2 or 2 µg/ml), an inhibitor of DNA repair. There was no effect on DNA strand-break induction of RF radiation by itself, or of the low dose of APC alone. There was a significant increase in DNA breakage after combined exposure of the cells to RF radiation with the low ($P = 0.025$) and the high dose ($P = 0.002$) of APC. [Sannino et al. \(2009b\)](#) found no effect on the number of DNA strand breaks induced by 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) in skin fibroblasts from healthy individuals or from subjects with Turner syndrome exposed to pulsed-wave RF radiation at 900 MHz (SAR, 1 W/kg) for 24 hours, followed by treatment with MX for 1 hour.

[Luukkonen et al. \(2009\)](#) found a significant increase ($P < 0.01$) in the number of menadione-induced DNA strand breaks in cultured human SH-SY5Y neuroblastoma cells exposed to continuous-wave RF radiation at 872 MHz (SAR, 5 W/kg) and menadione (25 µM) for 1 hour, but not in cells exposed to pulsed-wave RF radiation (GSM) and menadione. In a subsequent study with the same cell type, the same authors did not observe an increase in the number of DNA strand breaks after exposure to continuous- or pulsed-wave RF radiation at the same frequency

and SAR (872 MHz; 5 W/kg), with or without ferrous chloride and diethyl maleate (the latter compound was added to enhance the free-radical production induced by the former) ([Luukkonen et al., 2010](#)).

(iii) Ionizing radiation

[Figueiredo et al. \(2004\)](#) reported no effects of RF radiation on the induction of chromosomal aberration by gamma radiation in human lymphocytes exposed to pulsed-wave RF radiation at 2.5 or 10.5 GHz (SAR, 627 and 0.25 W/kg, respectively) for 40 seconds or 5 minutes, respectively, followed 2 hours later by exposure to 1.5 Gy gamma radiation from a cobalt-60 source. No effect was observed after exposure to RF radiation alone. [Stronati et al. \(2006\)](#) found no effect of RF radiation on X-ray-induced DNA strand breaks, chromosomal aberrations, micronucleus formation or SCE in human peripheral blood lymphocytes exposed to pulsed-wave RF radiation at 935 MHz (SAR 1 or 2 W/kg) for 24 hours, combined with 1.0 Gy of 250 kVp X-rays, given for 1 minute immediately before or after exposure to RF radiation. In the FISH assay used by [Manti et al. \(2008\)](#), there was no effect of RF radiation on X-ray-induced chromosomal aberrations in human lymphocytes exposed to 4 Gy of X-rays immediately before exposure to pulsed-wave RF radiation at 1950 MHz (SAR, 0.5 W/kg), while a small but statistically significant increase ($P = 0.036$) was observed at a SAR of 2 W/kg. [Zhijian et al. \(2009\)](#) did not find an effect of RF radiation on DNA strand breaks induced by X-rays, or their repair, in lymphocytes exposed to intermittent (5 minutes on, 10 minutes off) pulsed-wave RF radiation at 1800 MHz (SAR, 2 W/kg) for 24 hours, followed by exposure to X-rays (0.25–2.0 Gy).

(iv) Ultraviolet radiation

[Baohong et al. \(2007\)](#) exposed peripheral blood lymphocytes to 254 nm ultraviolet radiation (UVC) at 0.25–2.0 J/m², followed by RF

radiation at 1800 MHz (SAR, 3 W/kg), for 1.5 or 4 hours. The number of UV-induced DNA strand breaks decreased after exposure to RF radiation for 1.5 hours, and increased after exposure for 4 hours.

(d) *Mammalian cells (non-human)*

See [Table 4.7](#)

(i) *800–1800 MHz*

Mouse C3H 10T½ fibroblast cells (both exponentially growing and in plateau phase) were exposed to RF radiation at 835.62 MHz as a frequency-modulated continuous-wave (FMCW) signal, or to RF radiation as a code-division multiple access (CDMA) signal at 847.74 MHz (SAR, 0.6 W/kg), for up to 24 hours. The alkaline comet assay was used to measure induction of DNA strand breaks. No significant differences were observed between the results obtained with FMCW or CDMA radiation and the sham-exposed negative controls ([Malyapa et al., 1997b](#)). The same authors did not find any effects in a similar experiment with continuous-wave RF radiation at 2450 MHz (SAR, 0.7 or 1.9 W/kg) ([Malyapa et al., 1997a](#)).

C3H 10T½ fibroblast cultures (exponentially growing or in the plateau phase) were exposed to RF radiation at 847.74 MHz as a CDMA signal, or to RF radiation at 835.62 MHz as a FDMA signal (SAR, 3.2–5.1 W/kg) for 2, 4, or 24 hours. The alkaline comet assay was used to measure induction of DNA strand breaks. No statistically significant change was found in tail moment or tail length for cells that had been exposed to RF radiation (CDMA or FDMA), compared with sham-exposed controls. Furthermore, in cells exposed for 2 hours to RF radiation, a post-incubation of 4 hours did not result in significant changes in tail moment or tail length ([Li et al., 2001](#)).

Exponentially growing or plateau-phase C3H 10T½ cells – derived from mouse-embryo fibroblasts – were exposed to RF radiation at

835.62 MHz, as CDMA (SAR, 3.2 or 4.8 W/kg) signal, or at 847.74 MHz as frequency-division multiple access (FDMA) signal (SAR, 3.2 or 5.1 W/kg), for 3, 8, 16 or 24 hours. No significant exposure-related differences in micronucleus formation were found for either plateau-phase cells or exponentially growing cells ([Bisht et al., 2002](#)).

[Diem et al. \(2005\)](#) reported the results of an alkaline comet assay with SV40-transformed rat granulosa cells exposed to continuous or intermittent (5 minutes on, 10 minutes off) RF radiation at 1800 MHz (SAR, 1.2 or 2 W/kg) for 4–24 hours. Both continuous and intermittent exposures induced DNA single- and double-strand breaks, with the greatest effect found with intermittent exposure. [Speit et al. \(2007\)](#) independently repeated some of the experiments with V79 Chinese hamster cells, using the same equipment and exposure conditions (1800 MHz; 2 W/kg SAR; continuous wave with intermittent exposure). No effects of exposure to RF radiation were found in assays for DNA strand-break induction and micronucleus formation.

Chinese hamster lung cells exposed to intermittent (5 minutes on, 10 minutes off) RF radiation at 1800 MHz (SAR, 3 W/kg) for 24 hours contained an increased number of γ -H2AX foci – a measure of DNA double-strand breaks – compared with sham-exposed cells. There was no effect after a 1-hour exposure to RF radiation ([Zhang et al., 2006](#)).

Because auditory cells could be exposed to RF radiation at frequencies at which mobile phones operate, [Huang et al. \(2008\)](#) used HEI-OC1 immortalized mouse auditory hair cells to characterize their response to exposure to RF radiation at 1763 MHz (SAR, 20 W/kg), in a CDMA exposure chamber for 24 or 48 hours. No changes were found in the phase-distribution of the cell cycle, DNA strand-break induction, stress response, or gene-expression profiles in the exposed cells, compared with sham-exposed controls.

Table 4.7 Genetic and related effects of exposure to radiofrequency radiation in experimental systems in vitro

Test system, end-point	Exposure conditions	Genotoxic agent	Results and comments	Reference
pBluescript SK(+) plasmid, DNA strand breaks (DNA degradation <i>in vitro</i>)	835 MHz, CW; SAR, 4 W/kg; 48 h	-	Exposure to RF radiation did not change the rate of degradation of plasmid pBluescript SK(+) exposed to H ₂ O ₂ (Fenton-type reaction) as an indicator.	Chang et al. (2005)
<i>Escherichia coli</i> WP2 <i>uvrA</i> , reverse mutation	835 MHz, CW; SAR, 4 W/kg; 48 h	4-NQO, for 48 h during exposure to RF radiation	RF radiation increased 4-NQO-induced mutation rate in <i>Escherichia coli</i> WP2.	Chang et al. (2005)
<i>Escherichia coli</i> WP2 <i>uvrA</i> , reverse mutation	2450 MHz; SAR, 5–200 W/kg; 30 min	-	No effect on mutagenicity	Koyama et al. (2007)
<i>Salmonella typhimurium</i> TA98, TA100, TA102 and TA1535, reverse mutation	835 MHz, CW; SAR, 4 W/kg; 48 h	4-NQO, cumene hydroperoxide (CHP) sodium azide (SA) for 48 h during exposure to RF radiation	RF radiation increased CHP-induced mutation rate in TA102, had no effect on SA-induced revertants in TA100, and reduced SA-induced mutation rate in TA1535	Chang et al. (2005)
<i>Salmonella typhimurium</i> TA98, TA100, TA1535 and TA1537; reverse mutation	2450 MHz; SAR, 5–200 W/kg; 30 min	-	No effect on mutagenicity	Koyama et al. (2007)
<i>Saccharomyces cerevisiae</i> , gene-specific forward mutation at <i>CAN1</i>	900 MHz, GSM pulsed-wave; SAR, 0.13; 1.3 W/kg	MMS	No significant effect on mutation rates at <i>CAN1</i> ± MMS	Gos et al. (2000)
<i>Saccharomyces cerevisiae</i> , induction of respiration-deficient (<i>petite</i>) clones (loss of mitochondrial function)	900 MHz, GSM pulsed-wave; SAR, 0.13; 1.3 W/kg	MMS	No significant effect on the frequency of <i>petite</i> colony formation ±MMS	Gos et al. (2000)
<i>Saccharomyces cerevisiae</i> , intrachromosomal deletion-formation assay	900 MHz, GSM pulsed-wave; SAR, 0.13; 1.3 W/kg	MMS	No significant effect on formation of intrachromosomal deletions ±MMS	Gos et al. (2000)
<i>Saccharomyces cerevisiae</i> , intra-genic recombination assay in the <i>ADE2</i> gene	900 MHz, GSM pulsed-wave; SAR, 0.13; 1.3 W/kg	MMS	No significant effect on rates of intragenic recombination ± MMS	Gos et al. (2000)
<i>Xenopus laevis</i> ; DNA SSB measured in erythrocytes sampled immediately after exposure	8800 MHz pulsed-wave; peak power, 65 kW; SAR, 1.6 W/kg; 40 min	-	No indication of non-thermal effects. Observed DNA damage probably due to temperature rise	Chemeris et al. (2004)
C3H10T ^{1/2} mouse; DNA SSB measured in fibroblasts, sampled immediately and up to 4 h after exposure	2450 MHz CW; SAR, 0.7 and 1.9 W/kg; 2, 4 and 24 h	-	No effect	Malayapa et al. (1997a)

Table 4.7 (continued)

Test system, end-point	Exposure conditions	Genotoxic agent	Results and comments	Reference
C3H10T½ mouse; DNA SSB measured in fibroblasts, sampled immediately after exposure	835.62 MHz FMCW and 847.7 MHz, CDMA CW; SAR, 0.6 W/kg; 2, 4, 24 h	-	No effect	Malyapa et al. (1997b)
C3H10T½ mouse; DNA SSB measured in fibroblasts, sampled immediately and 4 h after exposure	835.6 MHz FDMA and 847.7 MHz FDMA; SAR, 3.2 and 5.1 W/kg; 2, 4 and 24 h exposure	-	No effect	Li et al. (2001)
C3H10T½ mouse; DNA SB, DNA-DNA and DNA-protein cross-links measured in fibroblasts	2450 MHz CW; SAR, 1.9 W/kg; 2 h exposure followed by 4 Gy gamma-rays	Gamma radiation	No effect of RF radiation on SB. No reduction by RF radiation of DNA migration induced by gamma-rays. No induction of DNA-protein crosslinks or changes in amount of DNA-associated protein by RF radiation	Lagroye et al. (2004b)
HEL-OC1 (immortalized mouse auditory hair) cells; DNA damage, stress response and gene expression	1763 MHz; SAR, 20 W/kg; CDMA; continuous exposure for 24 or 48 h		No effect on cellular responses, including cell-cycle distribution, DNA damage, stress response or gene expression	Huang et al. (2008)
L5178Y <i>Tk</i> ^{+/−} mouse lymphoma cells (DNA damage) and Chinese hamster lung fibroblasts (chromosomal aberrations, CA)	835 MHz; SAR, 4 W/kg; exposure for 48 h, alone or combined with chemicals	CPA, 4-NQO	No effect on DNA damage or CA. No effect on EMS-induced CA. Significant increase in CPA- and 4NQO-induced DNA damage.	Kim et al. (2008a)
Chinese hamster V79 cells; DNA damage (SSB, DSB)	1800 MHz; CW or pulsed-wave; continuous or intermittent (5 min on, 10 min off); SAR, 2 W/kg; exposure 1–24 h	-	No induction of DNA damage found in independent repeat experiments.	Speit et al. (2007)
Rat granulosa cells; DNA damage (SSB, DSB), sampled immediately after exposure	1800 MHz; CW or pulsed-wave; continuous and intermittent (5 min on, 10 min off); SAR, 2 W/kg; exposure 1–24 h	-	Induction of DNA SSB and DSB after 16 h intermittent exposure, at different mobile-phone modulations. Objections were raised to the analysis of the data	Diem et al. (2005)
Chinese hamster V79 cells; DNA synthesis, incorporation of [³ H] thymidine	7700 MHz; 300 mW/cm ² ; 15, 30, 60 min	-	Cells are blocked in entering S-phase	Garaj-Vrhovac et al. (1990b)

Table 4.7 (continued)

Test system, end-point	Exposure conditions	Genotoxic agent	Results and comments	Reference
Chinese hamster lung cells; DNA damage, gamma-H2AX focus formation	1800 MHz; intermittent (5 min on, 10 min off); SAR, 3.0 W/kg; for 1 or 24 h	-	RF radiation (24 h exposure, not 1 h) caused gamma-H2AX focus formation. A cell was classified positive when it contained more than five foci.	Zhang et al. (2006)
L5178Y <i>Tk</i> ^{+/+} mouse lymphoma cells; gene mutation	2450 MHz, pulsed-wave; power density, 488 W/m ² ; SAR, 30 or 40 W/kg; 4 h, together with MMC (at lower SAR) of proflavin (at higher SAR).	-	No effect of RF radiation alone. No influence by RF radiation on cell-growth inhibition or on MMC- or proflavin-induced mutagenesis	Meltz et al. (1989, 1990)
Chinese hamster ovary CHO K1 cells; gene mutation <i>Hprt</i> locus	2450 MHz; SAR, 5–200 W/kg; 2 h	Bleomycin, for 1 h before irradiation	RF radiation (200 W/kg) increased <i>Hprt</i> mutation frequency by itself, and increased bleomycin-induced <i>Hprt</i> mutations (100 and 200 W/kg). Effects may be due to hyperthermia	Koyama et al. (2007)
Chinese hamster ovary cells; SCE	2450 MHz, pulsed-wave; 490 W/m ² ; SAR, 33.8 W/kg; 2 h	Simultaneous exposure to adriamycin	No effect of RF radiation alone. No effect on adriamycin-induced SCE	Ciaravino et al. (1991)
C3H10T½ mouse fibroblasts; micronucleus formation	835.6 MHz CW; FDMA and 847.7 MHz CW, CDMA; SAR, 3.2 and 5.1 W/kg	-	No increase in frequency of micronucleus formation	Bisht et al. (2002)
Chinese hamster V79 cells; micronucleus formation	7700 MHz; 30 mW/cm ² ; 15, 30, 60 min	-	Increased micronucleus formation	Garaj-Vrhovac et al. (1991)
Chinese hamster ovary CHO-K1 cells; micronucleus formation	2450 MHz; SAR, 13, 39, 50, 78, 100 W/kg; 18 h	Bleomycin	Increased micronucleus frequency after RF radiation, and potentiation by RF radiation of bleomycin-induced micronucleus formation, both at SARs ≥ 78 W/kg	Koyama et al. (2003)
Chinese hamster ovary CHO-K1 cells; micronucleus formation	2450 MHz; SAR, 5, 10, 20, 50, 100, 200 W/kg; 2 h	Bleomycin	Increased micronucleus formation at SARs of 100 and 200 W/kg. No combined effect of RF and bleomycin	Koyama et al. (2004)
Chinese hamster V79 cells; micronucleus formation	1800 MHz; CW or pulse-wave; continuous and intermittent (5 min on, 10 min off), 1–24 h; SAR, 2 W/kg	-	No effect found. This study was aimed at replicating earlier findings.	Speit et al. (2007)
Bovine lymphocytes; micronucleus formation	9000 MHz, 70 W/kg CW; 10 min	MMC	Increased micronucleus frequency after RF radiation; significant increase by RF radiation of MMC-induced micronuclei.	Scarfi et al. (1996)

Table 4.7 (continued)

Test system, end-point	Exposure conditions	Genotoxic agent	Results and comments	Reference
Mouse m5S cells; chromosomal aberrations	2450 MHz CW or PW; SAR, 5, 10, 20, 50, 100 W/kg; 2 h	-	No effect	Komatsubara et al. (2005)
Chinese hamster V79 cells; chromosomal aberrations	7700 MHz, 300 mW/cm ² ; 15, 30, 60 min	-	Induction of chromosomal aberrations	Garaj-Vrhovac et al. (1990b)
Chinese hamster ovary cells; chromosomal aberrations	2450 MHz pulsed-wave; 490 mW/cm ² , SAR, 33.8 W/kg, 2 h	Simultaneous exposure to adriamycin or MMC	No effect of RF radiation alone No effect by RF radiation on aberrations induced by adriamycin or MMC	Kerbacher et al. (1990)
Chinese hamster V79 cells; chromosomal aberrations (structural)	7700 MHz, 30 mW/cm ² , 15, 30, 60 min	-	Increased frequency of chromosomal aberrations, including dicentric and ring chromosomes	Garaj-Vrhovac et al. (1991)
Chinese hamster ovary cells; cell-cycle progression	2450 MHz pulsed-wave; 490 mW/cm ² , SAR, 33.8 W/kg, 2 h	Simultaneous exposure to adriamycin	No effect of RF radiation alone No influence on cell-cycle progression caused by adriamycin	Ciaravino et al. (1991)
Chinese hamster V79 cells; cell growth (cell count, microtubule structure)	935 MHz; SAR: 0.12 W/kg; 1, 2, 3 h	-	Alteration of microtubule structure after a 3-h exposure; significantly decreased growth was noted in cells exposed for 3 h, at 3 d after exposure	Pavacic & Trosic. (2008)
Chinese hamster V79 cells; cell-proliferation kinetics, analysis of microtubule structure, mitotic index	935 MHz CW; SAR, 0.12 W/kg; 1, 2, 3 h	-	Alteration of microtubule structure; no effect on mitotic index. Cell proliferation was reduced at 72 h after exposure in cells exposed for 3 h. Slower cell-division kinetics	Trosic & Pavacic (2009)
Chinese hamster V79 cells; survival	7700 MHz; 0.5, 10, 30 mW/cm ² ; 10, 20, 30, 60 min	-	After 8 days of post-incubation: reduced cell survival related to power density and exposure time	Garaj-Vrhovac et al. (1991)

4-NQO, 4-nitroquinoline 1-oxide; CA, chromosomal aberrations; CDMA, code-division multiple access; CHP, cumene hydroperoxide; CPA, cyclophosphamide; CW, continuous wave; DSB, DNA double-strand breaks; FDMA, frequency-division multiple access; h, hour; *Hprt*, hypoxanthine-guanine phosphoribosyl transferase gene; min, minute; MMC, mitomycin C; MMS, methylmethane sulfonate; MN, micronuclei; SA, sodium azide; SCE, sister-chromatid exchange; SSB, DNA single-strand breaks

The alkaline comet assay and a test for chromosome aberrations *in vitro* were used to investigate the effects of 835 MHz RF radiation (4 W/kg), alone and in combination with the clastogens cyclophosphamide (CP), 4NQO and ethylmethane sulfonate (EMS), in L5178Y *Tk*^{+/−} mouse-lymphoma cells (to assess DNA breakage) and in Chinese hamster lung fibroblasts (to measure chromosome aberrations). In the latter cells, no effect was observed from RF radiation, alone or in combination with CP or EMS, but in the mouse-lymphoma cells a potentiating effect was noted on DNA strand-break induction after exposure to RF radiation following treatment with CP or 4NQO (Kim *et al.*, 2008a).

V79 Chinese hamster cells were exposed for 1, 2, or 3 hours to RF radiation at 935 MHz, generating an electric field-strength of 8.2 ± 0.3 V/cm and an average SAR of 0.12 W/kg. The microtubule structure in these cells was analysed by use of an immunocytochemical method. After 3 hours of exposure, microtubules in exposed cells were found to be altered compared with those in unexposed control cells. Three days after exposure, cell proliferation was significantly decreased in samples that had been exposed for 3 hours. Exposure to RF radiation at 935 MHz affects the structure of microtubule proteins, which consequently may obstruct cell growth (Pavicic & Trosic, 2008; Trosic & Pavicic, 2009).

(ii) 2450 MHz

The assay for forward mutation at the thymidine kinase locus in L5178Y mouse lymphoma cells was used to investigate the effects of a 4-hour exposure to RF radiation at 2450 MHz (power density, 48.8 mW/cm²; SAR, 30 W/kg), alone and in the presence of the chemical mutagen MMC (0.1, 0.2, 0.3 µg/ml). Exposure to RF radiation alone was not mutagenic, and it did not alter the effects of MMC with regards to cell proliferation or mutation induction (Meltz *et al.*, 1989). A similar experiment involving exposure to RF radiation combined with proflavin – a

DNA-intercalating drug – gave similar results (Meltz *et al.*, 1990).

In a cytogenetic study, CHO cells were exposed to pulsed-wave RF radiation at 2450 MHz (SAR, 33.8 W/kg), for 2 hours in the absence or presence of MMC (0.075 or 0.1 µg/ml) or adriamycin (0.175 µg/ml). The experimental conditions resulted in a maximum temperature increase of 3.2 °C. With respect to the induction of chromosomal aberrations, no effect was found that could be ascribed to the exposure to RF radiation (Kerbacher *et al.*, 1990).

CHO cells were exposed simultaneously to adriamycin (10^{−6} M) and pulsed-wave RF radiation at 2450 MHz (SAR, 33.8 W/kg) for 2 hours, or to adriamycin only. There was no effect of exposure to RF radiation on adriamycin-induced changes in cell progression or SCE frequency (Ciaravino *et al.*, 1991).

Micronucleus formation in Chinese hamster ovary (CHO) K1 cells was measured after exposure of the cells to RF radiation at 2450 MHz in four different scenarios: (1) exposure for 18 hours at average SARs of 13, 39 or 50 W/kg (input power, 7.8 W), which had no effect on micronucleus formation; (2) exposures corresponding to SARs of 78 or 100 W/kg (input power, 13 W), which produced a significant increase ($P < 0.01$) in micronucleus frequency; (3) treatment with the clastogenic compound bleomycin alone, or with bleomycin followed by irradiation for 18 hours at SARs of 25, 78 or 100 W/kg, which resulted in enhancement by RF radiation (at SAR values of 78 and 100 W/kg) of the effect of bleomycin alone; and (4) incubation at 39 °C for 18 hours as a high-temperature control; this last experiment also showed an increase in micronucleus frequency, albeit less strong than that after exposure to RF radiation. In a subsequent study, the authors reported a significant increase in micronucleus formation in cells exposed to RF radiation at 2450 MHz at SARs of 100 or 200 W/kg for 2 hours, but no effect of the combined exposure to RF radiation and bleomycin. Sham-exposures

at higher temperatures (38–42 °C) also increased the frequency of micronuclei, which indicates that the effects at the high SAR levels may have been thermal in nature ([Koyama et al., 2003, 2004](#)).

C3H 10T½ mouse fibroblasts were exposed to continuous-wave RF radiation at 2450 MHz (SAR, 1.9 W/kg) for 2 hours and processed for measurement of alkali-labile DNA damage and/or DNA–protein or DNA–DNA crosslinks. No effect was noted for any of these end-points ([Lagroye et al., 2004b](#)).

The induction of chromosomal aberrations was investigated in murine m5S cells exposed to continuous- or pulsed-wave RF radiation at 2450 MHz (average SARs of 5, 10, 20, 50 or 100 W/kg) for 2 hours. No significant differences were observed following exposure at any SAR compared with sham-exposed controls. There was also no difference between exposure to continuous-wave and pulsed-wave RF radiation ([Komatsubara et al., 2005](#)).

CHO-K1 cells were exposed to RF radiation at 2450 MHz (SAR, 5–200 W/kg) for 2 hours, after which *Hprt* gene mutations were scored. There was no mutation induction by exposure to RF radiation alone. An increase in the mutation frequency was found in cells exposed to RF radiation (SAR, 100 or 200 W/kg) in combination with bleomycin, but this may have been a thermal effect ([Koyama et al., 2007](#)).

(iii) 7000–9000 MHz

Cultured V79 Chinese hamster cells were exposed to continuous-wave RF radiation at 7700 MHz (power density, 30 mW/cm²) for 15, 30, or 60 minutes. In comparison with the controls, there was a higher frequency of specific chromosome lesions and a reduction in the incorporation of [³H]thymidine, showing inhibition of entry into S-phase ([Garaj-Vrhovac et al., 1990b](#)).

In a further study, the same authors reported a decrease in the number of V79 cell colonies, which was related to the power density and

the duration of exposure. Significantly higher frequencies of specific chromosomal aberrations – dicentrics, ring chromosomes – and micronuclei were observed in the exposed cells ([Garaj-Vrhovac et al., 1991](#)).

Cultures of bovine (*Bos taurus* L.) peripheral blood lymphocytes were exposed to RF radiation at 9000 MHz (SAR, 70 W/kg) for 10 minutes. To evaluate possible cooperative effects with a chemical mutagen, some exposed cultures were also treated with MMC. Exposure to RF radiation induced a statistically significant increase in micronucleus formation, both in the presence ($P < 0.01$) and absence ($P < 0.001$) of MMC ([Scarfi et al., 1996](#)).

(e) Non-mammalian cells

See [Table 4.7](#)

Mutagenic or recombinogenic effects of RF radiation at 900 MHz (GSM; SAR, 0.13 and 1.3 W/kg) were investigated in the yeast *Saccharomyces cerevisiae*. Mutation rates were monitored with a widely used gene-specific assay for forward mutation in the *CAN1* gene, which encodes arginine permease (gene-inactivating mutations lead to canavanine resistance) and with an assay measuring induction of respiration-deficient “petite” clones (small colonies) that have lost mitochondrial function. The recombinogenic effect of RF radiation was investigated with an assay for intrachromosomal deletion and an assay for intragenic recombination at the *ADE2* gene, which encodes an enzyme involved in purine (adenine) biosynthesis. Exposure of *S. cerevisiae* to RF radiation under these conditions did not result in recombinogenic or mutagenic effects ([Gos et al., 2000](#)).

The effects of a 40-minute exposure to pulsed-wave RF radiation at 8800 MHz (SAR, 1.6 W/kg; pulse width, 180 ns; peak power, 65 kW; repetition rate, 50 Hz) were investigated in erythrocytes of the frog *Xenopus laevis* by means of the alkaline comet assay. The temperature rise in the blood samples at steady-state was 3.5 ± 0.1 °C.