

Proximity to point sources of environmental mercury release as a predictor of autism prevalence

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Received 2 October 2006; received in revised form 16 January 2008; accepted 4 February 2008

Abstract

The objective of this study was to determine if proximity to sources of mercury pollution in 1998 were related to autism prevalence in 2002. Autism count data from the Texas Educational Agency and environmental mercury release data from the Environmental Protection Agency were used. We found that for every 1000 pounds of industrial release, there was a corresponding 2.6% increase in autism rates ($p < .05$) and a 3.7% increase associated with power plant emissions ($P < .05$). Distances to these sources were independent predictors after adjustment for relevant covariates. For every 10 miles from industrial or power plant sources, there was an associated decreased autism Incident Risk of 2.0% and 1.4%, respectively ($p < .05$). While design limitations preclude interpretation of individual risk, further investigations of environmental risks to child development issues are warranted.

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Keywords: Mercury; Autism; Environment; Distance; Industry

Introduction

Mercury is a heavy metal found naturally in trace amounts in the earth's atmosphere in differing forms—as elemental vapor, reactive gaseous compounds, or particulate matter. Studies show that background levels of environmental mercury deposition have steadily increased several fold since the pre-industrial era (Schuster et al., 2002), with the largest source of potentially adverse exposures coming primarily from coal-fired utility plants (33%), municipal/medical waste incinerators (29%) and commercial/industrial boilers (18%)—estimated to be responsible for 158 tons of environmental mercury released per year in the US (Environmental Protection Agency, Report to Congress, 1997). Other sources include hazardous waste sites, cement factories, and chlorine production plants. According to the Agency for Toxic Substances and Disease Registry (ATSDR), next to arsenic and lead,

mercury is the third most frequently found toxic substance in waste facilities in the United States (ATSDR, 2001).

Mercury is now widespread in the environment (EPA, 1997; ATSDR, 2001). The long-range atmospheric transport of mercury (Ebinghaus et al., 2001), and its conversion to organic forms through bio-accumulation in the aquatic food chain has been known for some time (MacGregor, 1975; Mahaffey, 1999). Notwithstanding, there are emerging concerns over the potential adverse effects of ambient levels of environmental mercury during early childhood development. There is sufficient evidence that children and other developing organisms are particularly susceptible to the adverse neurological effects of mercury (Landrigan and Garg, 2002; Grandjean et al., 1995; Ramirez et al., 2003; Rice and Barone, 2000).

Evidence from animal studies suggests that neonates lack the ability to efficiently excrete both methylmercury (Rowland et al., 1983) and inorganic mercury (Thomas and Smith, 1979), and that there is a higher lactational transfer of inorganic mercury than methylmercury (Sundberg et al., 1991a,b). Correspondingly, it has been shown that infants exposed via milk from mothers who were accidentally

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