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The plausibility of a role for mercury in the etiology of autism: a cellular perspective

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Autism is defined by a behavioral set of stereotypic and repetitious behavioral patterns in combination with social and communication deficits. There is emerging evidence supporting the hypothesis that autism may result from a combination of genetic susceptibility and exposure to environmental toxins at critical moments in development. Mercury (Hg) is recognized as a ubiquitous environmental neurotoxin and there is mounting evidence linking it to neurodevelopmental disorders, including autism. Of course, the evidence is not derived from experimental trials with humans but rather from methods focusing on biomarkers of Hg damage, measurements of Hg exposure, epidemiological data, and animal studies. For ethical reasons, controlled Hg exposure in humans will never be conducted. Therefore, to properly evaluate the Hg-autism etiological hypothesis, it is essential to first establish the biological plausibility of the hypothesis. This review examines the plausibility of Hg as the primary etiological agent driving the cellular mechanisms by which Hg-induced neurotoxicity may result in the physiological attributes of autism. Key areas of focus include: (1) route and cellular mechanisms of Hg exposure in autism; (2) current research and examples of possible genetic variables that are linked to both Hg sensitivity and autism; (3) the role Hg may play as an environmental toxin fueling the oxidative stress found in autism; (4) role of mitochondrial dysfunction; and (5) possible role of Hg in abnormal neuroexcitory and excitotoxity that may play a role in the immune dysregulation found in autism. Future research directions that would assist in addressing the gaps in our knowledge are proposed.

Keywords: autism; mercury; cellular; oxidative stress; mitochondrial; immune dysfunction

Introduction

Rather than critically examining the extensive literature relating to the possible role of Hg in the etiology of autism, this review focused specifically on the biological plausibility of the hypothesis from a cellular perspective. This is an essential prerequisite for furthering our understanding of Hg's possible role in autism because, for ethical reasons, one cannot conduct experimental Hg trials on humans. Instead, one needs to rely on the broad discipline of epidemiology to bring together disparate lines of inquiry and methodologies that inform the area. Independent and *a priori* of this, it is of fundamental importance to evaluate the biological plausibility of the hypothesis. A body of epidemiological evidence

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