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Mercury, Lead, and Zinc in Baby Teeth of Children with Autism Versus Controls

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This study determined the level of mercury, lead, and zinc in baby teeth of children with autism spectrum disorder (n = 15, age 6.1 ± 2.2 yr) and typically developing children (n = 11, age = 7 ± 1.7 yr). Children with autism had significantly (2.1-fold) higher levels of mercury but similar levels of lead and similar levels of zinc. Children with autism also had significantly higher usage of oral antibiotics during their first 12 mo of life, and possibly higher usage of oral antibiotics during their first 36 mo of life. Baby teeth are a good measure of cumulative exposure to toxic metals during fetal development and early infancy, so this study suggests that children with autism had a higher body burden of mercury during fetal/infant development. Antibiotic use is known to almost completely inhibit excretion of mercury in rats due to alteration of gut flora. Thus, higher use of oral antiobiotics in the children with autism may have reduced their ability to excrete mercury, and hence may partially explain the higher level in baby teeth. Higher usage of oral antibiotics in infancy may also partially explain the high incidence of chronic gastrointestinal problems in individuals with autism.

Autism is a severe developmental disorder that involves social withdrawal, communication deficits, and stereotypic/repetitive behaviors. The causes of autism are unknown, but both genetic and environmental factors have been implicated. The purpose of this study was to investigate the environmental factor of heavy metals (mercury, lead) toxicity.

A thorough review by Bernard et al. (2001) reported that all of the major symptoms reported in the literature for autism were also reported for cases of infantile mercury poisoning, including especially language/communication problems and social withdrawal. Therefore, they suggested that autism was primarily due to infantile exposure to mercury. Their hypothesis is plausible because mercury exposure at hazardous levels is common in the United States and other countries; the Food and Drug Administration (FDA) estimates that 1 in 6 women in the United States have mercury levels that increase the risk of neurological damage to their children. (Mahaffey et al., 2004) The major sources of mercury exposure for infants are (1) maternal seafood consumption, (2) maternal mercury amalgam dental fillings, and (3) thimerosal (an ethylmercury compound) in childhood vaccines and in anti-RhoD immune globulins given to Rh-negative mothers during pregnancy. Thimerosal was largely but not totally removed from childhood vaccines by 2004.

Mercury toxicity might occur either due to high exposure, or due to a decreased ability to excrete mercury, with the latter case seeming to be the primary issue in autism. The primary mechanism for excreting mercury involves its binding to glutathione and then being excreted in the bile (Ballatori & Clarkson, 1985). Infants are poor excretors because they produce less glutathione (Ballatori & Clarkson, 1984) and because they are usually on all-milk diets (which decreased mercury excretion by a factor of 3 in a study of rats); thus, they are especially vulnerable to mercury poisoning (Rowland et al., 1984).

Infants with autism were even more vulnerable to mercury toxicity, because their glutathione is much lower than in typical children (James et al., 2004; Audhya, 2004) and a higher fraction of their glutathione is oxidized (James et al., 2004). Further, two studies (Konstantareas & Homatidis, 1987; Adams et al., 2003) found that children with autism had much higher usage of oral antibiotics, which (in rats) resulted in a near-total loss of the ability to excrete mercury (Rowland et al., 1980, 1984). The reason appears to be that normal gut anaerobes are able to convert methylmercury (which is rapidly absorbed) into inorganic mercury (which is poorly absorbed and hence mostly excreted). In contrast, most strains of yeast

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