Environmental Mercury Release

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Mercury is a well-defined neurotoxin and now widespread in the environment (EPA, 1997). Next to arsenic and lead, mercury is the third most frequently found toxic substance in U.S. waste facilities (ATSDR, 2001).

While the long-range atmospheric transport of mercury (Ebinghaus et al., 2001) and its conversion to toxic forms through bio-accumulation in the aquatic food chain have been known for some time (Stopford and Goldwater, 1975), more recent concern involves mercury pollution as it effects early childhood development. In a 2000 report, the National Academy of Sciences' National Research Council estimated that each year about 60,000 children may be born in the United States with neurological problems because of exposure to methylmercury in utero.

Body burdens of mercury accumulation can be a result of exposure to a wide range of environmental sources, such as industrial emissions, occupational exposures, dental amalgams, fish consumption (EPA, 1997), or through mercury-based preservatives used in some vaccines (Freed et al., 2002). The largest source of potential population exposure comes from coal-fired utility plants, municipal/medical waste incinerators, and commercial/industrial boilers—estimated to be responsible for 158 tons of environmental release per year in the United States (EPA, report to Congress, 1997).

Other sources include hazardous waste sites, cement factories and chlorine plants. While the acute neurotoxicity of mercury is well known (ATSDR, 2001), population risks associated with low-level persistent exposure are poorly understood (NAS, 2000); yet reports implicate mercury in the causation of various developmental and learning disabilities (Ramirez et al., 2003), including autism (Bernard et al., 2001, 2002; Vojdani et. al., 2003).

Environmental Mercury Investigations

We have investigated the hypothesis that environmental mercury may be associated with population autism rates. Our preliminary investigation was initiated by noting that changes in autism over time corresponded with geographic regions where mercury and other toxic environmental releases were at the greatest level.

The top portion of Figure 1 shows the geographic trends in autism over approximately 10 years in Texas. The bottom portion depicts a geographic correspondence of environmental toxic release. The bottom right panel of Figure 1 shows that counties with the greatest rate of change in autism are either counties with the highest levels of toxic releases or those that border counties with the highest levels. There are some notable exceptions. One is Brewster County (the large county bordering Mexico in the west, with the dark border representing a rapid increase in autism but no reported toxic release). Interestingly, the economic history of this county includes being the leading producer of mercury in the United States.

Our recently published study demonstrates an association between environmental mercury release and autism in Texas. We found that for every 100 pounds of environmentally released mercury there was an associated six percent increase in the rate of autism (Palmer et al., 2006). Critics have argued that "exposure" was ill defined, and that distance to exposure sources would have been a better proxy for population exposure. Further, causal inference was limited because the study was cross-sectional and ecological rather than individual in nature.

To address some of these concerns, we conducted a second study in which we demonstrated that distance to industrial sources of mercury was inversely related to changes in the rate of autism over the last 15 years. These findings are consistent with existing research studies conducted with soil and plants (Wang and Shi, 2003; Kalac, 1991), and humans (Kurttio et al., 1998; Horvat, 2003), which demonstrate that proximity to mercury sources is related to greater burdens of mercury.

In a recent analysis, we used data from the U.S. Environmental Protection Agency's National Air Toxics Assessment (US EPA NATA) of 1996, based on a comprehensive analysis of mercury emissions obtained from various state and local air pollution control agencies. Associating this data with statewide-level autism data obtained from the "U.S. Department of Education Office 25th Report to Congress," we show that autism rates among children three-to-five years old in 2000 (e.g., those children conceived or born between 1995 and 1997) were significantly higher among states with greater concentrations of ambient air mercury per square mile. Figure 2 depicts this association.

Ambient air mercury was found to explain 20 percent of state-level autism rates. This association remains significant after adjusting for relevant factors, including baseline levels of autism, percentage of state spending for education and number of pediatricians.

Consistent with our results, Windham et al., (2006) demonstrate that mercury in the air is related to increased risk of autism. Compared to families living in areas of lower air concentrations of mercury, the researchers found that families living in higher-concentration areas were significantly more likely to have autistic children.

Taken together, these studies offer important justification

for further investigations. While our studies demonstrate a positive association between environmental mercury release and autism, they are preliminary because it is potentially erroneous to draw conclusions about individual risk from population-based ecological studies such as ours. These studies serve as the first phase of a larger study initiative involving the connection between environmental neurotoxins and autism.

We currently are pursuing studies that will involve understanding the interaction between genetic susceptibility and amounts of toxic environmental exposures. We suspect that persistent environmental toxic exposures in the presence of a genetic predisposition for poor detoxification of neurotoxins will put individuals at risk for developing autism. Knowing the specific combinations of environmental exposures and genetic predispositions can inform the development of targeted intervention strategies geared toward preventing autism.

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