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Message from the Editor: In this issue of the Reviews & Reports, Dr. Kouris reports on a neurodevelopmental disorder known as "Toxic Brain Injury." Dr Kouris discusses several toxins which when exposed to in childhood can lead to childhood-onset brain dysfunction. Examples include lead, mercury, and polychlorinated biphenyls (PCB's). Other important examples include alcohol, cocaine, and tobacco smoke. Regarding the latter, it is not generally appreciated that there is mounting evidence that fetal exposure to maternal tobacco smoke may contribute to mental retardation, cerebral palsy, epilepsy and autism. Health care providers and the general public need to be aware of this possibility. *—Philip May, MD*

Update on the Role of Environmental Toxins in Neurodevelopmental Disabilities.

By Steven Kouris, DO, MPH, MS

Toxic exposures during pregnancy and early childhood continue to play an important role as a preventable cause of neurodevelopmental disabilities in the U.S. and around the world. Identifying and eliminating these toxins should be a priority but the task is made exceedingly difficult due to the severe limits of scientific knowledge in this area as well as the competing interests of industry and commerce.

I recently had the pleasure of attending an outstanding environmental health conference at the University of Wisconsin in Madison. Jointly sponsored by the UW Medical School Office of Continuing Education and Department of Family Medicine as well as Madison Physicians for Social Responsibility, Sierra Club Great Lakes Program and the Wisconsin Conservation Power Project, the conference was entitled, "Making the Connection: Human Health and Environmental Exposures." I will briefly highlight the portions that pertained to childhood neurodevelopment.

From conception through at least the first decade of life, children are recognized to have a heightened susceptibility to environmental insult. Additionally, critical windows of vulnerability exist relative to brain development and proper maturation of the central nervous system. Children's overall exposure to environmental contaminants is also increased compared to adults. Many substances a mother comes in contact with may cross the placental barrier and affect her fetus. After birth, a child still has an incomplete blood-brain barrier, immature metabolic pathways, and disproportionately greater intestinal absorption of nutrients and contaminants. Pound for pound, children take in more air, water and food than adults. Dwelling nearer to the ground with an enlarged surface area, children are exposed to more dust and soil, heavy vapors and any contaminants present on floors and in carpets. And as parents of young children know, everything goes into a child's mouths as well.

Manifestations of abnormal development caused by toxic exposure can range from fetal death and structural birth defects to retarded growth and developmental or behavioral disability. An estimated 12 million children in the U.S. now suffer from one or more developmental disabilities. In some parts of the country, rates of reported autism and other developmental disorders are markedly increased. As mounting concerns for possible environmental factors are publicly expressed, little information has been forthcoming. Surprisingly, there exists no human safety data for the vast majority of chemicals we are routinely exposed to in our environment, and currently, there is no nationwide system for collecting toxic exposure data.

What is known is restricted to a relatively small number of substances. Extensive data exist on the effects of lead, mercury, alcohol, nicotine and PCB's (polychlorinated biphenyls). Less extensive but substantial data exist for some neurotoxic pesticides and solvents, other than alcohol. There are still fewer data on other compounds such as man-

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ganese, fluoride and brominated flame-retardants.

Lead is a well-known neurodevelopmental toxin that impairs intellect and learning ability, produces inattention, impulsiveness and hyperactivity, and, more recently, has been causally linked to predatory aggression in animals and juvenile delinquency in humans. As this substance has been removed from paints and fuels, exposure levels have steadily and markedly dropped. Unfortunately, the toxic effects continue to be seen even at very low levels (2.5 ug/dl) of exposure. It appears that perhaps no level of exposure is safe for children after all.

Mercury, likewise, is a well-documented toxin. Effects from high dose exposure during pregnancy include impaired intellect, seizures, visual, auditory and sensory disturbances, cerebral palsy, abnormal movements with problems walking, swallowing and speaking. Low doses impair motor skills, attention, memory, language and visual spatial abilities. As a result, strict limits are advised for fish consumption, especially by pregnant mothers. Coal-fired power plant emissions are also being more closely regulated.

PCB's, a class of chemicals used to insulate electrical transformers, are no longer produced. PCB's persist indefinitely in the environment, however, and bioaccumulate in the food chain. Effects in the exposed infant include reduced birth weight, head circumference, gestational age, and impaired performance on the Brazelton Neonatal Behavioral Assessment in the areas of motor immaturity, emotional ability and startle response. In early childhood, effects may include problems with memory, attention, verbal ability, information processing, psychomotor development and mood regulation. Older children may have diminished intellect, memory, attention and reading comprehension.

The most commonly used brominated flame retardants (PBDE's) resemble PCB's in chemical structure. Exposure to these chemicals during critical windows of brain development decreases memory and learning in animals. No human data exist yet, but these chemicals are now documented to be present in human breast milk in U.S. and Canadian populations.

Organophosphate pesticides cause decreased brain weight, decreased cholinergic receptors, hyperactivity, motor and coordination problems in laboratory animals. Human effects are unknown; however, high rates of human exposure are reported. Among urban newborns, 95% to 100% were exposed. One study found a correlation between umbilical blood levels and head circumference. A CDC study found organophosphate pesticide residues in 75% of the general U.S. population and 90% of children, with the highest concentrations found in the children.

Despite the lack of incontrovertible evidence linking all environmental toxins to specific health outcomes, adoption of a precautionary principle was proposed by conference organizers: "When an activity raises the threats of harm to the environment or human health, precautionary measures should be taken, even if some cause and effect relationships are not yet fully established." It remains to be seen whether regulatory authorities will see the wisdom in such an approach.

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INSTRUCTIONS

For CME Credit read the editorial on page 49 and the article that follows and complete the Content Test and CME Evaluation Form on page 52. Please read "Information and Instructions" following the article.

Specific learning objectives for this CME activity (please refer to general objectives).

Upon completion of reading of this article the learner will be able to:

- List common environmental toxins that may cause brain injury in childhood
- Explain why children are more susceptible than adults to toxic brain injury

Upon receipt and acceptance of the completed evaluation form/post-test, the AADMD CME Program will maintain on file a record for 6 years designating your credits earned. If you should need a written verification contact Philip May, MD at (908) 510-3062.