Neurodevelopmental Effects of Prenatal Exposure to Environmental Toxins: Scope of the Issue and Survey of Recent Literature

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PRESENTATION FORMAT: Panel Presentation

TOPIC/TARGET AUDIENCE: Participants interested in environmental health, maternal and child health; toxicology and epidemiology

ABSTRACT: In The Lancet Neurology in 2014, Drs. Philip Landrigan and Philippe Grandjean referred to rising rates of neurodevelopmental disorders in children as a "pandemic," affecting 10-15% of all births. Medical societies representing obstetricians, gynecologists, pediatricians, and reproductive medicine recognize that ubiquitous parental exposure to neurotoxicants can have profound and lasting effects on brain function. Mercury, lead, and pesticides are well recognized neurotoxicants; emerging science also recognizes developmental neurotoxic effects of air pollution and many industrial chemicals, including those found in consumer products. We will discuss the scope of the issue and evidence of change ("Centers for Disease Control and Prevention"), implications for public health, and present a literature survey of current and emerging science statistically linking traits of attention deficit hyperactivity disorders, autism spectrum disorders, and learning disorders with in-utero and early life exposures to environmental toxicants, using as a guideline the list of ten neurotoxic agents generated in a recent workshop at the Mt. Sinai Children's Environmental Health Center in NYC exploring environmental causes of neurodevelopmental disorders. By sharing current, emerging and suggestive science, we hope to create a common understanding, with the aim to inform policy and practical solutions.

OBJECTIVE(S): Participants will list ten neurotoxicants and describe at least three potential developmental outcomes of prenatal exposure to those substances.

Participants will discuss ways in which vulnerable groups, particularly women of childbearing age and very young children, can minimize exposure to several groups of neurotoxic chemicals. Empowered by the knowledge presented in our talk, participants will formulate public policy that results in decreased neurotoxic chemicals in the environment.

PANEL ABSTRACT 1: Marjorie Kircher, MS OTR

An overview and scope of the issue as defined in the general abstract will first be discussed, including prevalence, toxic environmental agents, developing brain vulnerability, epigenetic mechanisms, and economic implications. The developmental neurotoxicity of lead, mercury, and PCBs (polychlorinated biphenyls) has been well-established for decades and policy has been enacted at the federal and state levels to control our exposures. However, mercury emissions still occur from coal-fired power plants, and aviation fuel used by piston-engine aircraft is leaded. All three of these toxins are still of concern with continued human exposure; they persist in the environment and bioaccumulate, passing up the food chain and into the human body (methylmercury and PCBs from fish; lead from old paint dust and

chips, soil, and lead shot from hunting). The probable mechanism of brain disruption by lead and methymercury is inflammation in neural cells compromising development, and for PCBs, endocrine disruption affecting prenatal brain development. Outcomes in children at later ages after pre-natal exposures included traits of ADHD, lower or impaired cognition, and autism spectrum disorder.

PANEL ABSTRACT 2: Theodora Tsongas, PhD, MS

There is evidence of endocrine disrupting effects in humans associated with exposures to chemicals found in household and personal care products, including Polybrominated diphenyl Ethers (PBDEs) which act as flame retardants, phthalates, BPA, organophosphate (OP) pesticides, and fluorinated chemicals (specifically PFOA/S). This is important to keep in mind as we look at neurodevelopmental effects associated with prenatal exposures and possible differences in neurodevelopmental outcomes based on gender. Biological pathways associated with prenatal exposure to these chemicals can disrupt the equilibrium of the mother's thyroid hormone system, subsequently impairing development of cerebral architecture in the fetus. BPA and phthalates also disrupt sex hormone production. OP pesticides inhibit effects on cholinesterase enzymes affecting fetal and young children's nervous system development. Outcomes in children at later ages after pre-natal exposure included traits of ADHD, lower or impaired cognition, anxiety, depression, increased aggression, and traits of autism.

PANEL ABSTRACT 3: Patrick O'Herron, MD, FACS

Air pollution control has developed along two pathways: one for criteria pollutants (or smog) and one for air toxics. Smog pollutants have steadily declined under mandates by the EPA and Oregon DEQ. However, air toxics have not been sufficiently controlled or reduced. Dr. Frederica Perrera and her colleagues at Columbia University and in several other locations around the world, have developed a novel research method for measuring pregnant women's exposures to Polycyclic Aromatic Hydrocarbons (PAHs) from diesel exhaust throughout pregnancy (they wore air monitors in backpacks); their children were followed after birth at later ages, and significant associations were shown correlating air pollution exposure to neurodevelopmental issues such as reduced IQ, anxiety, depression, and traits of ADHD. Other studies using spatiotemporal models of exposure showed similar findings, including associations of autism with fetal exposure to air pollution from residential proximity to major roadways. Proposed mechanisms of brain disruption by air toxins include inflammation and mitochondrial damage in brain cells. Discussion will follow of proposed policies and practical solutions for reducing exposure to neurotoxicant chemicals.

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