

TALK OVERVIEW  
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Research into the pathophysiology and genetics of autism may inform the identification of environmental susceptibility factors that promote adverse outcomes in brain development. Conversely, understanding how low level chemical exposure influences molecular, cellular and behavioral outcomes relevant to the development of autism will enlighten geneticists, neuroscientists and immunologists about autism's complex etiologies, and possibly yield novel intervention strategies. Dr. Pessah will present recent results from M.I.N.D. Institute investigators participating in the UC Davis Center for Children's Environmental Health and Disease Prevention. First he will review published findings about mercury levels in Northern Californian children participating in the CHARGE (Childhood Autism Risk from Genes and the Environment) case control epidemiological study. How mercury levels correlate with gene expression in blood differ significantly in individuals with autism when compared to age-matched individuals with neurotypical development, suggesting divergent responses. Second, Dr. Pessah will present two examples of gene x environment interactions that may contribute to autism risk: (1) pesticides that interfere with  $\gamma$ -aminobutyric acid (GABA) neurotransmission; and (2) persistent organic pollutants that alter calcium regulated pathways. Inherent imbalances in neuronal connectivity in children at risk for autism are likely to provide the biological substrate for enhanced susceptibility to environmental chemicals that target these signaling systems.