Edwin Cook, Jr., M.D.
MIND Institute Distinguished Lecturer Series – January 10, 2018

Biographical Information

Ed Cook attended Southern Methodist University and the University of Texas Medical Branch at Galveston. He trained in Adult and Child and Adolescent Psychiatry at the University of Chicago. After completion of training, he was promoted to the role of Professor of Psychiatry, Human Genetics and Pediatrics at the University of Chicago until his move to the University of Illinois at Chicago in 2005.

Ed is now the Director of Child and Adolescent Psychiatry and the Earl M. Bane Distinguished Professor of Psychiatry at the University of Illinois College of Medicine His program of research focuses on collaborative molecular genetic studies of autism spectrum disorder, with an emphasis of studies of relationships between genotype and phenotype. He also focuses on study of the biomarker, hyperserotonemia, in autism. The goal of Dr. Cook's research is the development of improved pharmacological treatments of autism. He has assessed and treated children, adolescents and adults with autism for over 30 years, including following many patients for over 25 years.

Presentation Abstract (4:30pm presentation)
Translating the complex genetics of autism

Substantial progress has been made in identifying specific genetic variants contributing to autism, with the pace of these findings accelerating over the past decade. This talk will review evidence that supports the role of genetics in the complex causes of autism. Although more are predicted, at least 72 genetic variants have been identified, including 66 affecting a single gene (e.g. *CHD8*, *SCN2A*) and 6 leading to fewer or more copies of a region on a chromosome (e.g. maternal chromosome 15q11-q13 duplication). Since 3-5% of individuals with autism may currently be diagnosed as having a specific genetic condition associated with their autism, this knowledge may provide important clinical information such as risk for additional medical problems such as ADHD or epilepsy. In addition, study of these *de novo* genetic variants is providing information about the complexity of brain development that contributes to autism and is guiding the development of new therapeutics for autism and related comorbidities. Many misconceptions related to the complexity of autism will be addressed. As one example, the most clinically relevant genetic findings are not present in parents and not inherited. Conversely, most cases of autism are due to many, many genetic variations added together to increase risk for autism.