Biographical Information
S. Jill James, Ph.D., is a research professor in the Department of Pediatrics at the University of Arkansas for Medical Sciences and director of the Autism Metabolic Genomics Laboratory at the Arkansas Children’s Hospital Research Institute. Dr. James has over 25 years of research experience and is nationally and internationally recognized as an expert on one-carbon metabolism and pathways of transmethylation and transsulfuration. She was funded for 12 years by the American Cancer Society to study epigenetic mechanisms of carcinogenesis at the FDA-National Center for Toxicological Research. In 2003, she joined the University of Arkansas for Medical Sciences where she has studied mechanisms of gene-environment interaction in the etiology of Down Syndrome, congenital heart defects, and most recently autism. Her autism research is focused on metabolic, genetic, and epigenetic abnormalities in children with autism and also in their mothers, and is supported by funding from NIH for “Metabolic biomarkers of autism: predictive potential and genetic susceptibility,” and from the Department of Defense for “Redox imbalance as a vulnerability phenotype in autism.” She is the Arkansas site PI for the national Autism Treatment Network consortium. Dr. James has published over 130 peer-reviewed papers and recently received the American Society for Nutritional Sciences award for innovative research contributing to the understanding of human nutrition. Dr. James completed her undergraduate degree in biology at Mills College in Oakland, CA, and her PhD degree in nutritional biochemistry at UCLA.

Presentation Abstract (4:30 pm)
Oxidative Stress and the Metabolic Pathology of Autism
The metabolic pathology of autism has been relatively unexplored, despite the fact that metabolic abnormalities have been implicated in the pathogenesis of many other complex neurobehavioral disorders. Dr. James uses a targeted approach to autism “metabolomics” that focuses on the dynamics of an integrated metabolic pathway that is essential for the regulation of oxidative stress and epigenetic gene expression. A candidate pathway approach to autism pathology provides an integrated reflection of the combined influence of genes and environment on metabolic phenotype. As such, an abnormal accumulation or deficit of specific metabolites in a defined pathway can provide clues about relevant candidate genes and/or environmental exposures. Unlike changes in DNA gene sequence, both redox and epigenetic alterations are dynamic processes that are inherently reversible. Thus, a deeper understanding of these alterations could provide new insights into the basic neurobiology of autism and, subsequently, lead to novel targeted therapeutic strategies to treat and possibly prenatally prevent the development of autism. In her presentation, Dr. James will review her research into folate-dependent methionine transmethylation and transsulfuration metabolism and the functional impact of systemic metabolic imbalance on genome-wide DNA methylation and oxidative damage in children with autism and also in their mothers. Evidence for cumulative oxidative damage/inflammation and epigenetic dysregulation would provide a plausible mechanism by which the environment could modulate predisposition to autism.