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1983	B.A. in psychology (with honors), Haverford College
1989	Ph.D., Johns Hopkins University School of Medicine, Department of Pharmacology and Molecular Sciences
1989-1995	Post-doctoral fellow, Stanford University Medical Center, Department of Molecular and Cellular Physiology
1995-present	Assistant Professor, Kennedy Krieger Institute, Department of Neurology and Johns Hopkins University School of Medicine, Department of Neuroscience

Honors

1989	The Sandoz Award, Johns Hopkins School of Medicine, for outstanding research
1989-1992	Helen Hay Whitney Foundation, post-doctoral fellowship
1991	Cosmetic Chemists Award, The Society of Cosmetic Chemists
1997-1999	Basil O'Connor Starter Scholar Research Award, March of Dimes

Analysis of gene expression in human brain diseases using high density microarrays

For many disorders of the human brain such as autism, schizophrenia and mental retardation, the primary genetic defects are not known. Additionally, a variety of secondary changes in gene expression may occur in these diseases as the brain compensates for the disruption of some pathway perturbed by the primary gene defect(s). For human disorders that affect cognition or other higher mental functions such as language skills, animal models may be inadequate because they cannot accurately represent the pathological changes. In addition, human brain biopsy material is not available except in extreme cases involving invasive surgery. Thus, an important approach to studying human brain disorders is to analyze postmortem brain samples. We have measured gene expression in brain samples from patients with autistic disorder (n=12) and a related pervasive developmental disorder, Rett Syndrome (n=9) as well as 25 age-, gender-, and regionally-matched controls. We measured the expression levels of up to 20,000 genes using the Atlas (CLONTECH Laboratories), GeneFilters (Research Genetics) and Micromax (NEN Life Sciences) cDNA microarrays. We performed a series of control experiments in normal human brain samples to measure differences in expression profiles based upon factors such as age at death, gender, brain region, postmortem interval, and comparison of gene expression in brain to astrocytes and to fibroblasts. In Rett Syndrome, we found a consistent up-regulation of a group of glial genes using several array techniques. These changes in gene expression were confirmed by PCR, Western blotting, and by protein microsequencing of postmortem Rett Syndrome brains.