Paul H. Patterson, Ph.D.

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Biographical Information

Paul H. Patterson, Ph.D., is the Anne P. and Benjamin F. Biaggini Professor of Biological Sciences at the California Institute of Technology. He is a Midwesterner who grew up in Chicago and attended Grinnell College in Iowa. He obtained a PhD at Johns Hopkins University in biochemistry, and began his work in developmental neurobiology with studies of dissociated neuronal cultures while on the faculty at Harvard Medical School. While at Caltech, his group developed a mouse model of mental illness based on the risk factor of maternal infection. This work has also involved several collaborations with researchers at UC Davis. Patterson's group is also testing various types of gene therapy for treatment of mouse models of multiple sclerosis and Huntington's disease. Patterson is currently serving on the scientific advisory boards of the International Rett Syndrome, the John Douglas French Alzheimer's, the Autism Speaks, and the Hereditary Disease foundations.

Presentation Abstract (4:30 pm)

Gut-Brain-Immune Connections: Modeling an Environmental Risk Factor for Autism

While autism is a neurodevelopmental disorder characterized by language and social deficits, recent studies have highlighted striking dysregulation in the neural, peripheral, and enteric immune systems of autistic individuals. There are also reports that subsets of children with autism spectrum disorder (ASD) display gastrointestinal (GI) abnormalities, including increased intestinal permeability and altered composition of GI microbiota. To explore potential connections between GI problems and the brain and behavior, we use a mouse model of an ASD risk factor, maternal infection or maternal immune activation (MIA). Immune activation for one day during gestation results in adult offspring that display the cardinal ASD behaviors and neuropathology. MIA offspring also display decreased intestinal barrier integrity (leaky gut) and corresponding changes in levels of tight junction proteins. These symptoms are associated with altered expression of colon cytokines and changes in serum metabolite levels. Postnatal treatment with the human commensal bacterium *B. fragilis* for one week permanently ameliorates these GI abnormalities, and normalizes certain serum metabolites as well as many of the ASD-related behaviors. These results reinforce the potential relevance of the gut-brain axis for ASD, where manipulation of the intestinal microbiome can influence GI physiology and behavioral performance. The findings also raise the possibility of testing a probiotic therapy in individuals with ASD and GI symptoms. Moreover, the altered serum metabolite profiles in the MIA mouse model raise the possibility of testing particular metabolites as candidate biomarkers for subsets of human ASD. A collaboration with researchers at the MIND Institute has recently validated the rodent MIA model using non-human primates.