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Research Interest: Molecular and Developmental Basis of Neuropsychiatric Disorders.

Dr. Levitt studies the molecular and cellular mechanisms that control the development of the forebrain, and the causes for developmental and neuropsychiatric disorders such as autism, anxiety and schizophrenia. The brain begins to establish functional circuits before birth, but has its greatest growth period from birth until around three years of age. The brain then undergoes a substantial period of remodeling during childhood and adolescence. The laboratory is comprised of a group of highly collaborative researchers who investigate the genetic basis for the establishment of forebrain circuits that regulate mood, emotion, stress and complex higher functions. Human genetic studies of candidate genes and molecular pathways leverage the laboratory’s studies of model mouse systems. The goal of combining animal model and human genetic research is to identify mutations and polymorphisms that may alter function of genes sufficiently to increase the risk for expressing a particular disorder, and in predicting the response to treatments.

The laboratory applies a variety of tools in the research projects. For example, microarrays, which contain information on the entire genome, are used to develop gene expression profiles that may be unique to specific neuropsychiatric disorders and in genetically altered mice. The laboratory uses modern technology to create conditional genetically engineered mice to express mutations of specific genes that may be involved in psychiatric disorders. By using sophisticated in utero surgical methods, genes are introduced into specific developing brain regions to disrupt normal patterns of gene expression and brain development. Cell-based systems are employed to evaluate functional implications of human polymorphisms and mutations identified in human genetic studies. Humanized mice are being engineered to test specific hypotheses based on the cellular approaches. These integrated approaches move beyond correlations to direct implication of gene pathways and cellular functions that lead to human brain disorders.

Selected Publications:

