Psychiatric illnesses are caused by both genetic and environmental factors. However, almost nothing is known about how these factors interact to give rise to the biological abnormalities that underlie the symptoms of major mental illnesses like schizophrenia. The goal of my research is to elucidate the mechanism by which genes and environment interact to cause psychiatric disorders.

To investigate this question I have focused on genes which act at the nexus between changes in the environment and changes in gene expression in the brain. These genes, called immediate early genes (IEGs), are activated at high levels in the brain in response to stimuli such as stress, social interactions, hormonal fluctuations, and interventions such as administration of drugs and electroconvulsive therapy. To identify the potential role of these genes in cognition and psychiatric illness I have taken a dual approach of studying mice that lack function of these genes, as well as evaluating mutations in these genes in humans.

The results of my studies suggest that mice lacking the IEG Early Growth Response gene 3 (Egr3) display behavioral and physiologic abnormalities consistent with the human illness schizophrenia. These behavioral abnormalities can be reversed by treating the mice with antipsychotic medications that we use to treat human psychiatric illnesses. In addition, the mice display a unique response to the premier antipsychotic medication clozapine, which is characteristic of that seen in patients with schizophrenia. Ongoing studies in my lab are pursuing the brain abnormalities of these mice which may both identify the mechanism of action of clozapine and provide insight into the neurobiological abnormalities that give rise to the symptoms of schizophrenia.

If interested, please email your resume and a 1 page statement describing your interest in doing research and why you are interested in this study to Amanda Maple, PhD at amaple@email.arizona.edu.

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