2009
YOUNG SCIENTIST RESEARCH FUND
Awards

CHADD is pleased to announce the recipients of the **2009 Young Scientist Research Fund Awards.**

Selected from a pool of qualified applicants by renowned experts in the field, these young researchers are making outstanding contributions to our understanding of AD/HD. The chair of CHADD's professional advisory board, Ann Abramowitz, PhD, will present the awards during CHADD's annual international conference, to be held this year in Cleveland, Ohio. The award program is administered by Myra Cushman, senior health information specialist at CHADD's National Resource Center on AD/HD. The awards are currently supported through generous funding from the McNeil Pediatrics Division of Ortho-McNeil-Janssen Pharmaceuticals, Inc. of Titusville, New Jersey, and by a number of individual donations. *Attention* asked each recipient to summarize her research.

L. Cinnamon Bidwell, MA, a doctoral candidate in clinical psychology and behavioral genetics at the University of Colorado at Boulder, submitted her research entitled "Association of DRD4, DAT1, and 5HTT with Putative Neuropsychological Endophenotypes in Attention-Deficit Hyperactivity Disorder." Bidwell graduated *magna cum laude* from Tufts University with a bachelor's degree in psychology and philosophy. At the University of Colorado, she conducted her graduate training under Erik G. Willcutt, PhD, and collaborated with Bruce F. Pennington, PhD, at the University of Denver.

The usual causal models of AD/HD and other psychopathologies have traditionally assumed that every clinical disorder resulted from a single genetic or environmental risk factor that led to a single cognitive deficit and that this single causal factor was necessary and sufficient to produce the disorder. Data collected more recently, however, suggest that these simple single-deficit explanations, while parsimonious, are inadequate for most disorders. In response, the field has shifted toward new multiple-deficit models. Newer models now explicitly hypothesize that AD/HD and most other complex disorders are due to the combined effects of *multiple* cognitive impairments that arise from *multiple* genetic or environmental risk factors. Bidwell investigates the important links between genetic vulnerabilities, neuropsychological weaknesses, and AD/HD symptoms, with the goal of contributing to the development of a comprehensive causal model for AD/HD.

Bidwell's research into the etiological underpinnings of AD/HD has cut across the fields of clinical psychology and psychopathology, neuroscience, and behavioral and molecular genetics. Her

This year's awards recognize accomplishments in behavioral and molecular genetics

master's thesis demonstrated that the same neuropsychological impairments that often occur in children with AD/HD are also evident in some family members without AD/HD symptoms. Bidwell was awarded a National Research Service Award (NRSA) from the National Institute of Mental Health in order to study the neuropsychology of adult AD/HD using behavioral, structural neuroimaging, and genetic approaches. Her dissertation used a family-based genetic design to examine the relationships among AD/HD, associated neuropsychological impairments, and specific genetic influences, and in turn how these factors confer susceptibility for the disorder through neurocognitive pathways.

Bidwell's training continues with a clinical psychology internship at Duke University Medical Center under the mentorship of Scott H. Kollins, PhD. She is currently completing her internship in general child psychology and pursuing specialized training in the evaluation and treatment of children and adults with AD/HD at the Duke AD/HD program. She is also engaged in research that examines pharmacological and genetic factors in AD/HD and comorbid nicotine dependence. To date Bidwell has authored or coauthored six papers in peer-reviewed journals and twenty-four presentations at national and international meetings. She is also a member of APA, APS, and ABCT. During her internship and upcoming postdoctoral fellowship, Bidwell hopes to develop into an independent researcher and clinician with the knowledge to become a leading expert in the neurobiological and clinical phenomenology of AD/HD and its common comorbidities. ◑

Molly Nikolas, MA, is a doctoral candidate in clinical psychology at Michigan State University. She graduated from the University of Notre Dame in 2002, earning a bachelor's degree in psychology. At MSU, she has studied under Joel Nigg, PhD, and S. Alexandra Burt, PhD, on projects involving the genetics of AD/HD, neuropsychological performance, and disruptive behavior disorders. Nikolas' dissertation, "Youth Appraisals of Marital Conflict and Genetic Risk for Attention-Deficit Hyperactivity Disorder: Examination of Gene x Environment Interactions Using Behavioral and Molecular Genetic Methodologies," makes use of both behavioral and molecular genetic approaches to examine interactions between genetic and environmental factors that contribute to the development of AD/HD. Nikolas plans to pursue a clinical research career investigating gene-environment interplay for AD/HD and associated phenotypes (neuropsychological performance, disruptive behavior disorders).

Understanding the interplay between genetic and environmental contributors (gene x environment interactions, or GxE) to AD/HD is likely a key component to unlocking the specific processes that cause the disorder. While many environmental factors have been linked to AD/HD, marital conflict has been shown to be a strong predictor of child adjustment. Further, children's perceptions of conflict, specifically the degree to which they blame themselves, likely play a determining role as to the impact of conflict on child behavior.

Nikolas' dissertation set out to examine interactions between genetic influences on AD/HD and children's feelings of self-blame in relation to marital conflict using both behavioral and molecular genetic techniques. First, to examine changes at the latent level, Study 1 involved tested GxE interactions among twins. Twin data is useful for examining genetic and environmental contributions to AD/HD because the analyses make use of differing degrees of genetic relatedness (i.e., identical twins share 100 percent of their genes while fraternal twins share only 50 percent). The sample included 246 twin pairs ages 5-16 years from the Michigan State University Twin Registry. The twin data were analyzed to determine estimates of genetic and environmental influences on AD/HD as well as to examine shifts with changes of self-blame. Shifts in genetic and environmental estimates were observed. When self-blame was low, genetic influences on AD/HD were high. Yet, as reports of self-blame increased, genetic effects decreased and environmental effects dramatically increased.

Study 2 next examined GxE interactions with a specific DNA marker—the promoter polymorphism of the serotonin transporter gene. Three hundred and four youth ages 6-18 years from the MSU Attention Study underwent a multi-informant diagnostic procedure. Children were assigned to one of three different "transcription efficiency" groups based on their genotype: a high serotonin group, an intermediate serotonin group, and a low serotonin group. Significant differences in the relationship between self-blame and AD/HD across the three genotype groups emerged. For the low and high serotonin groups, there was a strong relationship between self-blame and AD/HD symptoms, such that increases in self-blame corresponded to increases in AD/HD symptoms. Yet, there was no relationship between self-blame and AD/HD for the intermediate serotonin group.

Overall, Nikolas' dissertation indicates a potential etiological role of self-blame in regard to marital conflict in AD/HD. While these results need to be replicated, they provide a strong test of GxE effects, as significant findings emerged using both behavioral and molecular genetic methodologies. ◑