

## **A Critique of the ADHD Status**

*Adam A. Hill, BA. November 30, 2008.*

*adamahill@gmail.com*

A new article in the online journal *ScienceDaily* promotes a recent study that “for the first time reveals shape differences in the brains of children with ADHD, which could help pinpoint the specific neural circuits involved in the disorder” (Kennedy Krieger Institute, 2008). Sounds promising—perhaps a new way to understand the neurology of ADHD, perhaps a new way to treat ADHD, and perhaps even implications for other learning disabilities. But on closer inspection, the research as reported leaves significant questions unanswered.<sup>1</sup>

The biggest problem is the presentation of learning disabilities, such as ADHD, as exclusively based in neurology, biology, or physiology. While research suggests that a correlation between neurological processes and learning disabilities exists (Leonard, 2001: 159; Wolf, 2007: 185), a wealth of research in the social sciences has also found that disabilities—including learning disabilities—are more socially constructed than purely biological (Carrier, 1983; Kavale, 1980). As Joseph W. Schneider put it, “illness, disease, and disability are not ‘givens’ in nature . . . but rather *socially constructed* categories that emerge from the interpretive activities of people acting together in social situations” (Schneider, 1988: 65). A useful synthesis of the two views can be found in Michelle Fine and Adrienne Asch’s article, “Disability beyond Stigma” (1988).

In addition to the debate between constructionism and essentialism, the study (at least as reported in the *ScienceDaily* article) interprets its results in a troubling way. The research found that “boys with ADHD had significant shape differences and decreases in overall volume of the basal ganglia compared to their typically developing peers. Girls with ADHD did not have volume or shape differences, suggesting sex strongly influences the disorder's expression” (Kennedy Krieger Institute, 2008). In other words, the research finds a relationship between brain composition and ADHD symptoms, at least in boys, but it is unclear to what extent brain composition *causes* ADHD symptoms. Another way of interpreting the results, however—one that seems more reasonable—is that ADHD is

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<sup>1</sup> To be fair, it is worth noting that the official findings from the study have not been publicly released yet. Perhaps some of the concerns raised in this essay will be answered in the formal publication, which is scheduled for December 2008.

*not* primarily neurological, but rather primarily a socially defined characteristic that encompasses certain behavioral habits and learning styles.

If ADHD were nothing more than a set of observable symptoms caused by neurological processes and brain composition, then the symptoms would be positively correlated (in the case of varied shape) or negatively correlated (in the case of size) in *all* afflicted children, not just boys. It is known that boys and girls learn in different ways (Kindlon and Thompson, 1999; Gurian and Stevens, 2005), and this ADHD study could just as easily conclude that neurological differences affect learning style rather than the authors' conclusion that neurological impairments cause a learning disability. Furthermore, studies have shown the amount and quality of learning in which a person engages contributes to the size of the basal ganglia (Leonard, 2001: 159); thus, social factors that lead to decreased literacy, inadequate education, and academic indifference may also be contributors to the neurological differences that the study attributes to ADHD.

This possibility is not even addressed in the *ScienceDaily* summary of the research. The inattention afforded this question is particularly disturbing because previous research found that the basal ganglia—the portion of the brain that is apparently smaller in boys with ADHD—is also “critical to habits, addiction and procedural learning” (Kanellos, 2005). According to a Massachusetts Institute of Technology study,

Habitual activity . . . changes neural activity patterns in [the basal ganglia]. These neural patterns created by habit can be changed or altered. But when a stimulus from the old days returns, the dormant pattern can reassert itself, . . . putting an individual in a neural state akin to being on autopilot (Kanellos, 2005).

The implication here is that even if having a smaller or unusually shaped basal ganglia results in behaviors associated with ADHD, the shape, size, and activity of the basal ganglia are influenced by habits, which are in turn influenced by social circumstances. An appropriate interpretation, then, might suggest treating ADHD not through medication but through improvement of an affected child's environment. If ADHD is a disability, it is important to remember that the notion of disability does not rise from a “faulty” human body, but from a faulty environment that curbs a person's intellectual and physical growth. As Jenny Morris (1991: 41) notes, “It is not the physical disability itself but the social and economic circumstances of the experience which can lead to a diminished quality of life.”

The research does not seem to consider whether it is the educational environment that is failing, rather than the child's brain. For example, Maryanne Wolf shows that the process of learning to read is fundamentally *different* between children who develop normally and those who develop with dyslexia (Wolf, 2007: 185). Perhaps children diagnosed with learning disabilities would have better outcomes if the parents, teachers, and social workers in their lives treated their neurological processes as *different* rather than *substandard* and *diseased*. In other words, rather than merely examining the brains of different children—seeking neurological problems and pharmaceutical solutions while ignoring problems based more in social roles than in perpetual biological disorder—we could additionally accommodate all learning styles. The current trend in the United States seems to reward a normative learning style and penalize all others—a strategy that inhibits society's ability to develop every child into an adult who is fully participatory and a successful contributor to society in her or his own way.

My point is not to suggest that neurological functions and genetics play no role in how children learn or behave. To the contrary, I tend to view most situations as a soft essentialist (i.e., I think genetics and biology play a large role in our individual patterns but that the social meaning and roles ascribed to those patterns are more relevant to our development as humans). I am merely asking whether the findings of this study were interpreted properly. By ignoring the social elements of learning disability, conflating trends in diagnosed boys so that they apply to all diagnosed children, and by overlooking the other key functions of the basal ganglia, it seems clear to me that the study—at least as summarized in the *ScienceDaily* article (which was written by the study's authors themselves)—does not provide an appropriate interpretation of its findings. I, for one, eagerly await the publication of the full study to determine whether this is an example of poor research or merely a poor press release.

*ADAM A. HILL has a Bachelor of Arts degree in social science from Metropolitan State University. His essay, "Finding Historical Place: How Education Develops Students into Citizens," won first place in Sociologists of Minnesota's 2008 undergraduate student paper competition.*

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