Questioning the Treatment for ADHD

MY ROLE IN THE RITALIN (METHYLPHENIDATE) class action suits is mischaracterized by Eliot Marshall in his News Focus article "Planned Ritalin trial for tots heads into uncharted waters" (17 Nov., p. 1280). He says that I "signed up as an expert witness," but my role extended beyond that. Although I am not at present an expert in these cases, my books and scientific publications (*1*) provided the detailed basis for the legal allegations, and I helped formulate the original legal filing for the cases. Furthermore, my reasons for opposing the use of stimulant medication for the treatment of so-called attention deficit hyperactivity disorder (ADHD) go beyond the absence of a biological basis for the putative disorder.

First, stimulant drugs are dangerous. One prospective study indicated that the use of prescription stimulants in childhood predisposes children to abuse cocaine in young adulthood (*2*). Another study found that children diagnosed with ADHD had a 9% risk of developing psychotic symptoms when treated with stimulants (the control group had no such symptoms) (*3*). In my own reviews of controlled clinical trials, I found that stimulants can cause, for example, growth retardation, depression, and obsessive-compulsive disorder (*1*). In addition, animal studies indicate that stimulants in short-term clinical doses permanently change the brain and even destroy neurons (*4*). In terms of developmental neurotoxicity, the potential effects of amphetamines on 4- to 6-year-old children should rule out performing clinical trials involving this age group, such as the Preschool ADHD Treatment Study discussed in Marshall's article.

Second, stimulants exert their "therapeutic" effect by suppressing autonomous and spontaneous behavior. They also enhance obsessive-compulsive behavior (*1*). This compulsive over-focusing is mistaken for improved attention. Meanwhile, there is no evidence that stimulants improve learning or academic performance, but rather that they tend to impair cognitive function.

Third, the collection of behaviors subsumed in the diagnosis ADHD, including squirming in a seat and talking out of turn, are not "symptoms" and do not reflect a syndrome. They are behaviors that disrupt classrooms and can be caused by anything from normal childhood energy to boring classrooms or overstressed parents and teachers. We should not suppress these behaviors with drugs; we should instead identify and meet the needs of our children in the school and the home. Children often improve in their behavior and school performance when parents and teachers find better approaches to disciplining them and to engaging their natural desire to get along with adults and to learn.

PETER R. BREGGIN*

4628 Chestnut Street, Bethesda, MD 20814, USA

*Director of the International Center for the Study of Psychiatry and Psychology

References and Notes

1. P. Breggin, *Talking Back to Ritalin: What Doctors Aren't Telling You About Stimulants for Children* (Common Courage, Monroe, ME, 1998); *Int. J. Risk Safety Med.* **12**, 3 (1999).
2. E. Cherland and M. B. Fitzpatrick, *Can. J. Psychol.* **44**, 811 (1999).
3. N. Lambert and C. Hartsough, *J. Learn. Disability* **31**, 533 (1998).
4. W. P. Melega *et al.*, *Behav. Brain Res.* **84**, 258 (1997); W. P. Melega *et al.*, *Brain Res.* **766**, 113 (1997).

Letters to the Editor

Letters (~300 words) discuss material published in *Science* in the previous 6 months or issues of general interest. They can be submitted by e-mail (science_letters@aaas.org), the Web (www.letter2science.org), or regular mail (1200 New York Ave., NW, Washington, DC 20005, USA). Letters are not acknowledged upon receipt, nor are authors generally consulted before publication. Whether published in full or in part, letters are subject to editing for clarity and space.

CREDIT: ROBERT BUKATY/AP

REGARDING THE PRESCHOOL ADHD TREATMENT Study (PATS) that Marshall describes in his article—there is no disease. No proof exists that ADHD is a disease with a validating abnormality. Yet the public is told it is a "disease" (*1*), that it is "neurobiologic" (*2*) or "neurobehavioral" (*3*). At the National Institutes of Health (NIH) Consensus Conference on ADHD in 1998, W. B. Carey, a professor of pediatrics at the University of Pennsylvania School of Medicine, testified that "ADHD...appears to be a set of normal behavioral variations" (*4*). The Consensus Conference Panel concluded, "we do not have an independent, valid test for ADHD...no data...indicate that ADHD is due to a brain malfunction" (*5*). In that children who would be the research subjects in the PATS study have no demonstrable disease, there is no justification for giving them Schedule II stimulant medications.

FRED A. BAUGHMAN JR*

1303 Hidden Mountain Drive, El Cajon, CA 92019, USA

*Retired neurologist

References and Notes

1. *NIH Publication No. 94-3572* (NIH, Washington, DC, 1994), p. 7.
2. J. C. Heavener Jr., *Attention* **6** (no. 4), 7 (2000).
3. "Clinical practice guideline: Diagnosis and evaluation of the child with attention-deficit/hyperactivity disorder," *Pediatrics* **105**, 1158 (2000).
4. W. B. Carey, *Program and Abstracts of the NIH Consensus Conference on ADHD*, 16 to 18 November 1998, Bethesda, MD (NIH, Washington, DC, 1998), pp. 33–36.
5. Final Statement of the Panel of the NIH, Consensus Conference on ADHD, as distributed to the public and the press, 18 November 1998.

Biochemistry of Neurodegeneration

THE NEURODEGENERATION ASSOCIATED with diseases such as Parkinson's and the Lewy body variant of Alzheimer's is suggested by B. I. Giasson and colleagues in their report (3 Nov., p. 985) to be linked to oxidative damage, specifically the selective nitration of tyrosine residues in α-synuclein, an abundant neuronal protein. Aggregations of this protein form the brain lesions indicative of the above diseases and others, collectively referred to as neurodegenerative synucleinopathies.

The authors, using monoclonal antibodies, detected selectively nitrated α-synuclein (nitrotyrosines in the COOH-terminal region) in lesions in postmortem brains that had different synucleinopathies. The modification of α-synuclein, however, is unlikely to be either selective or causative. Nitric oxide can ameliorate oxidative damage despite the formation of oxidants such as peroxynitrite