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# Critique or Misrepresentation? A Reply to Timimi et al.

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In rebuttal to Timimi et al., we show that their critique is not a form of reasonable scientific debate with informed, constructive criticism, but merely a misrepresentation of the existing scientific literature on ADHD apparently designed to convince the scientifically uninformed of its nonexistence and of the misuse of medications for its management. We show their argument to be based on faulty logic, selective citation, misreprensentation of individual studies, ignorance of the vast literature on ADHD, and innuendo that maligns the integrity of scientists studying the disorder. Our original International Consensus Statement on ADHD remains untarnished by this faux critique – indeed it was intended to refute just such unsupported and unsupportable criticism that often appears in the popular media.

**KEY WORDS:** ADHD; attention deficit hyperactivity disorder; etiology; medication; psychosocial management.

We thank the Editors for this opportunity to reply to the critique by Timimi et al. (2003) of the International Consensus Statement Barkley et al., 2002. Space is limited, and so we must be direct. Like much criticism of ADHD and its investigators that has appeared in lay publications, this critique misconstrues our motives, misrepresents the scientific literature on ADHD, engages in faulty logic concerning the basis for viewing conditions as disorders, selectively cites a few reports in support of their assertions while ignoring a much larger body of research opposing them, and uses innuendo to malign the integrity of scientists whose research supports the existence of this disorder.

Contrary to Timimi et al., we can find no compelling evidence in scientific journals or scientific meetings that the validity of the disorder has been "shaken by criticism." Indeed, with as many as 20 or more papers related to ADHD per month published in scientific journals internationally, the genuineness of ADHD as a disorder appears to be alive, well, and on solid-scientific ground, continuing to usefully drive programmatic research. Any "debate" over the legitimacy of ADHD as a valid disorder exists only in some segments of the popular media, not in the scientific community. That is why our sizeable group produced the Consensus Statement. Drawn largely from the membership of the International Society for Research in Child and Adolescent Psychopathology, which has no commercial or vested interests, the signers clearly represent the largest single group of mainstream scientists studying ADHD ever assembled for a common cause. Our aim, clearly stated in our introduction, was to confront misrepresentation of the disorder in the popular media using the status of the science on ADHD to refute it. It was not, as these critics disingenuously claim, "to forestall debate," to act "completely counter to the spirit and practice of science," or "to close down debate prematurely and in a way not becoming of academics," or other such assertions. All such misrepresentations

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are straw men bearing no resemblance to our stated intentions.

Given the ease with which Timimi et al. dismiss ADHD as a legitimate disorder, we can rightly ask "How would they know?" What specific standards or criteria have they applied to the wealth of scientific literature on ADHD that permit such a conclusion? Specifically, just how do they distinguish valid disorders from mere problems in living, the consequences of "cultural anxiety," or parents "loaded with anxiety" in their child-rearing? Apparently, they have no such standards for none are set forth against which to judge the merits of their argument. We made our dual standards obvious: (1) valid disorders are failures or severe deficiencies in psychological adaptations (functional mental mechanisms) that are universal to humans and (2) the failures or deficiencies result in harm (increased morbidity, mortality, or impairment in major life activities) (Wakefield, 1997, 1999). And we provided more than 400 references to scientific studies that attest to ADHD meeting these standards—we could have cited five times as much. Our critics can site but three reviews (not experimental research) and two studies by Ford et al. that they claim raise issues about the validity or etiologies of ADHD.

Tellingly, even the studies by Ford et al. are misrepresented as to their findings and conclusions. Those studies do not show that ADHD arises from child maltreatment and trauma exposure but that histories of such circumstances may be present to a greater than expected degree in ADHD, especially where ODD may also be present. Here is what Ford et al. (2000) actually concluded: "The finding that ADHD was associated with lesser likelihood of having experienced maltreatment than ODD and the absence of an increased risk for accident/illness trauma in ADHD (compared to adjustment disorder), is consistent with studies indicating that *biological and nontraumatic parent/family factors are critical in the etiology and treatment of ADHD* (emphasis added)." (p. 213) And further on, they state: " Another limitation is the study's cross-sectional design, which does not permit the detection and clarification of crucial causal and temporal relationships linking trauma, PTSD symptoms, and the disruptive behavior disorders." Yet that is what Timimi et al. tried to assert. Thus, where we stand accused of implying cause from correlational evidence, it is our critics who have been caught doing so.

Timimi et al. challenge the view of ADHD as a legitimate disorder on several grounds, none of which are logical or scientifically defensible. While professing no explicit standards against which real disorders are to be judged, the critique does imply what their grounds for dismissal might be. To them genuine disorders: (1) cannot exist without some "medical test" being available for their diagnosis; (2) cannot change in having their defining features revised or improved upon across their history; (3) cannot vary in prevalence across segments of society, countries, or geographic regions; (4) cannot have other disorders coexist with them (comorbidity); (5) must have a distinct and specific neurobiological lesion identifiable as their etiology; and (6) cannot share heritability or other contributing factors that may overlap with other disorders. Applying all these criteria as the standard for defining disorders would rule out all currently known psychiatric disorders as being valid not to mention numerous medical ones as well, including Alzheimer's and Parkinson's disease, multiple sclerosis, cancer, HIV/AIDS, seizure disorders, sickle cell anemia, etc. None of these disorders could withstand comparison to such a set of criteria for concluding a disorder to be valid. Such unscientific challenges to the validity of ADHD are not just misguided but harmful because they can serve to misinform policy-makers and the public and thereby restrict, reduce, or eliminate access to services for them. We address each briefly below:

- Certainly the fields of medicine, psychiatry, pediatrics, and clinical psychology, among others, strive to develop accurate, reliable, objective tests for disorders where they may be possible. And surely some clinicians need to do a better job of evaluating and treating those who seek services from them. Yet the absence of such tests or the occasional clinical misdiagnosis or mismanagement of disorders is not evidence against the existence of a disorder. If this inappropriate standard were true, then all major mental and developmental disorders, including schizophrenia, bipolar disorder, Tourette's syndrome, mental retardation, autism, to name just a few, and many medical disorders (see above) could not be considered as valid disorders.
- Just because definitions of ADHD have been refined and improved across the history of the disorder provides no logical justification against it being a disorder. How could this be so if science is an enterprise of test, refine, then test again? If disorders had to be

defined precisely and accurately when first they appear in history and could not undergo change with subsequent revision and refinement then there would be no disorders. For no disorders of which we are aware in either medicine or mental health have been defined precisely and unchangingly when first recognized. Such a standard clearly ignores the Darwinian self-corrective process that is the scientific enterprise—proposing, testing, and refining based on the evidence so obtained. We can and should expect the *DSM-V* diagnostic criteria for ADHD to be somewhat different from those in *DSM-IV* (e.g., greater developmental sensitivity to different ages). This will arise purely from science being a self-correcting enterprise. It would not provide evidence against the disorder.

- This is a nonstarter that overlooks the reality that different definitions applied at different stages in the history of a disorder may yield differing prevalence figures, as would be expected. It also ignores the real likelihood that disorders vary as a function of demographic parameters such as geographic location, population density, sex, nutrition, prenatal care, access to medical care, exposure to biological hazards, etc. If this assertion of Timimi et al. were true, then the variations frequently noted in the geographic and demographic distributions of cancers, sickle cell disease, or multiple sclerosis, or mental disorders such as psychosis or Tourette's Syndrome, to chose but a few, are prima facie evidence against their being valid disorders when judged against this implied standard. That different countries and cultures may manifest different levels of prevalence or severity of a disorder raises good reasons to study those differences for what they may yield about refining its nature and causes yet they would hardly invalidate the existence of a disorder. Here Timimi et al., true to pattern, once more misrepresent their references. The paper by Rappley et al. (1995) is cited as evidence for intrastate variability of disorder (Michigan) when it was in fact about intrastate variations in prescribing patterns as its title clearly shows. Prescribing patterns may vary for many legitimate reasons, including the very real contemporary problem of grossly uneven access to care across segments of society, and do not directly indicate a disorder's actual prevalence within the population.
- Because ADHD coexists with other disorders, it is said, "the concept of ADHD is inadequate to explain clinical reality," whatever that may mean. As a standard for judging the status of a disorder, this makes no sense. Nearly all of the mental disorders and many medical ones can co-occur with others yet remain separable and genuine disorders. Again, what science strives to do is study such patterns of coexistence for what they may have to say about how disorders relate, develop, and even contribute to each other and why. That does not invalidate the disorder.
- Neuroimaging research on ADHD can tell us nothing about its biological bases because the crucial comparison of unmedicated ADHD children to age matched controls has not been done. The Neuroimaging findings could be the result of medication treatment. Unfortunate for this position is that such a study has been done (Castellanos et al., 2002) and its results were very much in keeping with other previous neuroimaging studies of ADHD in which group differences were obtained. More importantly it was able to demonstrate that such differences are persistent over time as shown in serial scans. That this study used both never-medicated and medicated ADHD children and found comparable differences from normal for each ADHD group refutes this point by Timimi et al. So does an earlier study on deficient neuronal inhibition assessed via transcranial stimulation of the motor region using stimulant naïve children (Moll, Henreich, Trott, Wirth, & Rothenberger, 2000).
- Critics of ADHD may wish to view the evidence for its striking pattern of genetic heritability as being "open to interpretation" but that does not make it so. No one cognizant of the current scientific literature doubts the consistency of findings in this area of research. It has repeatedly shown ADHD and its symptoms to be among the most genetically influenced psychiatric conditions across multiple studies in multiple countries (see Levy & Hay, 2001; and our original references). These studies involved hundreds or even thousands of identical and fraternal twin pairs. Others involved comparisons of siblings reared together to those reared apart

> or comparisons of biological versus adoptive families of ADHD children. All these different methods supported the strong level of heritability of this disorder and its defining symptoms. Indeed, there exist no studies that would refute this clear pattern of results. The fact that other disorders may share some genetic susceptibility with ADHD is hardly an indictment against it as a valid disorder—such shared genetic vulnerabilities are found in other mental and medical disorders as well.

Though it was a very minor point in our statement, Timimi et al. aim much of their criticism at the rise in medication use for the management of ADHD as if such an increase alone were de facto evidence of something scandalous or reprehensible taking place in our professions. Interestingly, they ignore the rise in empirically based behavioral, psychosocial, and special educational treatments that have occurred simultaneously, though these can, often in combination with medication, effectively assist with the reduction in symptoms and impairments for many children. Their logic would extend to these psychosocial forms of treatment as well. If the identification and care of psychiatrically ill children improves in any way from some earlier historical bench mark, our critics imply, then this is explicit evidence that something is dramatically wrong with the mental health profession. Obviously this ignores the fact that this is precisely what takes place from efforts to address public mental health problems. True to form, our critics cite the only outlier study of prevalent stimulant use; that being the one done by Lefever et al. (1999) in the Norfolk, VA area that claimed to have found an exceptionally high level of stimulant prescribing relative to other regions and large databases. Other more numerous studies showing considerably lower prescribing rates go unmentioned though they are clearly from larger databases and are likely more representative of U.S. patterns. What the critics do not know is that a subsequent study just completed has been unable to replicate the earlier Lefever et al. results for that same region and finds a prescribing prevalence closer to 3%, in keeping with other studies of other regions (Hathaway, personal communication, September, 2003). The reasons for such a gross disparity of results deserve investigation. The more obvious and sensible interpretation for the rise in medication use is that it has occurred because of the increasing evidence for the safety and effectiveness of some medications for the symptomatic management of the disorder, the increased recognition that girls, teens, and adults can also have this disorder, changes in special education regulations, and the rise in public awareness about the disorder, among other legitimate factors.

Finally, there is the scientifically flimsy "cultural perspective" offered up by these critics as a competing theory for the origins of ADHD in contrast to the neuropsychological, neurological, and genetic ones that have substantial support in the science on this disorder. Thus, ADHD is said to originate in the "collapse of the moral authority of adults" (unexplained), anxieties in contemporary child rearing (also unexplained), and drug company strategies to expand markets to children using unwitting ADHD scientists in their campaign (innuendo). No evidence is offered to support any of these assertions though they are presented as undeniable facts based solely on citations of politically motivated editorials and highly biased trade books. Fortunately, we have reached a state in the mental health sciences where sufficient data are available on disorders like ADHD such that all ideas about it no longer get prizes. Theories and hypotheses about the origins of ADHD must have consequences to be useful; that is, they must be testable against the sizeable and increasing body of scientific findings available on it for consistency with that database. When this is done, the vague pontifications of these critics do not square with the available data.

There is no evidence to show that ADHD arises from any such unsupportable cultural perspectives as claimed by these critics. Indeed, studies of twins are an excellent means of testing such environmental hypotheses about disorders. Modern statistics can be applied to such data sets that can discern the extent to which variation in the population in certain traits or disorders can be attributed to common, shared, or rearing environment, to unique events that occur only to the affected family member, or to genetics. The hypotheses of our critics clearly fall within the common or shared environmental variation tested in such twin studies. To date, all twin studies have found no significant contribution of shared environment to the symptom expression of ADHD. They do find a small but significant contribution of unique environmental events (some or all of which can be due to biohazards experienced by the child as well as unique social influences from outside the home). But they consistently find a substantial genetic contribution to ADHD within the population. These numerous

studies, and many other lines of evidence, directly refute the nonexpert folk wisdom offered by these critics as to the origins of ADHD. In fact, hundreds of studies finding differences between ADHD and community control children not to mention the numerous cross-cultural studies on the prevalence of ADHD would be evidence against their vaguely framed hypothesis. For their hypothesis asserts that there is nothing unusual about these children in any way—it is all to be found in parental child-rearing and some amorphous concept called "cultural anxiety" prevalent in the United States or western world. Such views are at worst a continuation of the past 50 years of parent bashing stemming from psychoanalytic, radical behavioral, and poppsychology perspectives on children's mental disorders (see Harris, 1997; Pinker, 2002 for discussions). These are historical dead ends in understanding child psychopathology. At best this hypothesis is a form of scientific buck-passing for it can generate no useful understanding of disorders, predictions that can drive informative programmatic research, or insights into effective treatments. All one need do, apparently, is "engage with the interpersonal realities of human life" using the untested methods of Peter Breggin. Such unscientific views and treatments are unpersuasive when judged against the abundant scientific evidence. As we urged in our initial statement, such views deserve to be ignored in the popular media.

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