Neuropsychol Rev (2009) 19:275–276
DOI 10.1007/s11065-009-9100-7

LETTER TO THE EDITOR

Autism is a Portmanteau Syndrome

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Received: 30 March 2009 / Accepted: 6 May 2009 / Published online: 26 May 2009
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Editor:

I whole-heartedly agree with Professor Belmonte and his colleagues that case studies are important, and that more autism theory syntheses could be constructed.

The key difference between my view of autism and that of Belmonte et al. (2009) is that they believe there is one shared neurofunctional deficit in autism. My research conducted with others, and my review of the research and theories of others has led me to conclude that brain deficit variation in autism as currently diagnosed is too great to assume that one shared neurofunctional deficit will "be found to provide a unifying causal explanation for autism" (2008, p. 284). I am not alone in this view: Reiss (2009) asserted "it is unlikely that a single cause or pathophysiological mechanism will be described that applies to most individuals currently diagnosed with autism" (p. 91).

Belmonte and colleagues, however, labeled my argument a "defeatist fallacy". But there is no defeat and no fallacy in viewing autism as an aggregation of many micro-subgroups of neural deficits. Only if you believe, as Belmonte and colleagues do, that there is one pan-autism neural information processing deficit, can you interpret the un-reconciled existing variation in autism as defeating.

Belmonte and colleagues deny that their generalizations in Bonneh et al. (2008) constitute an ad hoc theory. However, A. M. is one individual with autism in whom a stronger sense representation extinguishes a weaker one. Bonneh et al. (2008) proposed that A. M.'s abnormal processing: "could be common in autism" (p. 641); "may be a general property of autism" (p. 648); is "a valuable window onto the very large population of 'low-functioning' individuals with autism" (p. 636); and, "may be common to many people with low-functioning autism" (p. 650).

Lakatos (1978) argued that generalization based on new data constitutes an "ad hoc theory" if the generalization fails to work for a synthesis with existing theories. In fact, Bonneh et al. (2008) argued *against* synthesis. They asserted that the hyper- and hyposensitivity theories were "unlikely to account for" (p. 647) A.M.'s abnormal processing, and that the monotropism theory posited monochannel response under perceptual load, whereas A. M.'s monochannel perception occurred without "any apparent perceptual load" (p. 648). Because Bonneh et al. proposed A.M.'s processing as a "general property of autism", but claimed A.M.'s processing was not consonant with three existing theories, consequently their claims constitute an ad hoc theory.

Belmonte and colleagues claim that I argued that autism is a set of single-variable relationships linearly aligned from genes and environment to behavior. However, nowhere in my paper does this view appear. What I do believe is that autism is a portmanteau syndrome, e.g., is a large carryall syndrome in which can be found very many different gene mutations and chromosomal copy number variants, very many different neural deficits, and very many different patterns of behavioral expression (Waterhouse 2008). As even autism endophenotypes have multiple genetic sources (Waterhouse, in press), a reasonable assumption is that

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causal relationships in autism will include convergence and divergence at every level.

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