AUTISM OVERFLOWS WITH SYNTHESES

Editor:

We were surprised to find our case study "Cross-modal extinction in a boy with severely autistic behaviour and high verbal intelligence" (Bonneh et al. 2008) impugned by Lynn Waterhouse (2008) as an example of "unsynthesized and hoc theories" cluttering autism research. Our article never purports to constitute a theory in itself, and from its very beginning relates our observations of single-channel perception to an established theoretical context described in terms of stimulus overselectivity (Lovaas et al. 1979), monotropism (Murray et al. 2005), and impaired attention shifting (Allen & Courchesne 2001). Professor Waterhouse misattributes to us a causal claim that "autism results from monochannel of [sic] winner-takes-all perceptual processing" when in fact all that we claim is that our case results support the existence of such winner-takes-all processing in autism. Our point is not to clutter and to contend with existing theory, but rather to extend such theory to 'low-functioning' cases in a way that might further the very synthesis to which Professor Waterhouse and we all aspire-- hence our unifying theme of perturbed neural interactions (Rubenstein & Merzenich 2003).

Though integrative, cooperative autism research has a long way to go still, Professor Waterhouse's assertion that "the field has not made progress in creating a synthesized, standard predictive causal theory of autism" seems to assume that statements that do not explicitly cite or support each other must necessarily be in conflict and competition. This assumption of irreconcilability is a defeatist fallacy. As Professor Waterhouse herself observes, ideas of weak central coherence (Happé & Frith 2006) and the various takes on abnormal connectivity (Castelli et al. 2002; Just et al. 2004; Courchesne & Pierce 2005a; Takarae et al. 2007; Wilson et al. 2007) offer great potential for synthesis. So do reports of complementary anatomical (Courchesne & Pierce 2005b) and physiological (Kennedy & Courchesne 2008) abnormalities. We ought perhaps to have noted explicitly the consistency of our cross-modal results with this emerging, integrated theory of perturbed neural information processing. The synthetic potential remains, though, even when authors themselves might not take note of it, or worse, when they try to set themselves apart by seizing on rhetorical rather than data-driven distinctions.

We do not accept that autism is as simple as a collection of single-variable relationships that remain segregated through all levels of function from genes and environment to behaviour. The true picture is likely much more tangled, converging from multiple causes and diverging into multiple endophenotypes but traversing a common core of abnormal neural information processing (Belmonte et al. 2004b). This computational abnormality can arise from various, even complementary perturbations of physical connectivity (Belmonte et al. 2004a); the fact that not all individual cases of autism share the same genetic or environmental antecedents or the same epiphenomena (e.g. early white-matter hypertrophy) need not, therefore, imply any similar lack of unity at a core level. Studies of informative single cases can be important hypothesis-generating steps towards including the under-studied and under-served 'low-functioning' population in the basic research that leads to informed and empirically well founded therapies.

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