

<COMP: BRITISH SPELLING AND USAGE OK>

<CT> **The “mechanism” of human cognitive variation**

<CA>Matthew K. Belmonte

<CAA>*Department of Human Development, Cornell University, G77 Martha Van Rensselaer Hall, Ithaca, NY 14853–4401*

belmonte@mit.edu

<http://www.mit.edu/~belmonte/>

<C-AB>**Abstract:** The theory of psychosis and autism as diametrical disorders offers a tractable and testable view of normal and abnormal human cognitive variation as a function of opposing traits grouped by their selection for maternal and paternal reproductive fitness. The theory could be usefully rooted and developed with reference to the lower-level perceptual and attentional phenomena from which social cognitive modules are developmentally refined.

<C-text>There is a certain mechanistic appeal in theoretical science’s types and dimensions of personality. If we can believe in them, such paradigms hold out to us the hope of rendering a welter of human differences comprehensible in terms of cause and effect, and quantifiable in terms of position along some fundamental axis. To the extent that such models are valid, they garner a great deal in terms of predictive, explanatory, or at least descriptive power. To the extent that they are invalid, they lose a great deal in the

projection of multifaceted reality onto flat theory. So theoretical abstractions are useful only insofar as they remain grounded in the phenomena that they purport to describe. Crespi and Badcock are keenly aware of this tension between theory and phenomenon, symbol and referent, and this is why they conclude with an apology for “overly simplistic terms” and “broad, crude strokes.” Despite this acknowledged coarseness of their model, Crespi and Badcock have little for which to apologise – for it’s the very breadth and force of such strokes that carry science across ideological limits and into new territories.

Like Baron-Cohen’s (2002) empathising–systemising duality, which it extends and supplants, this mentalism–mechanism duality has something of an Aristotelian feel. Indeed, it recalls medicine’s first explanations of disease in terms of imbalance of the humours. In terms comprehensible to the ancient Greeks we might define the psychotic spectrum as an excess of the mentalistic humour and the autism spectrum as an excess of the mechanistic humour, and observe that these imbalances can be associated with unusual cognitive skills – just as Aristotle characterised what we now know as mood disorders as an excess of black bile and observed among his fellow philosophers a certain tendency towards melancholic constitutions (Pies 2007). The difference, of course, is that we don’t imagine our humoral abstractions as unitary, physical substances. Rather, we’re aware that at genetic and biochemical levels of analysis these mentalistic and mechanistic humours are implemented by dauntingly complex genetic networks that interact with the environment and with each other as development unfolds. This underlying complexity, though, is precisely the condition that makes these insubstantial humours so useful: In parallel with efforts to describe autism and schizophrenia in terms

of network interactions among specific genes, we can now describe these conditions in terms of classes of genes united by their selection for maternal and paternal reproductive fitness. Thus the work of Crespi and Badcock enables a trait-level analysis of autism and psychosis that complements gene-level analysis.

As Crespi and Badcock point out, this trait-level view in terms of mentalism and mechanism can in some regards muster greater descriptive and explanatory power than the more immediate view in terms of paternally and maternally imprinted genes themselves, because abnormal biases constructed by such genes can interact with normal programmes and gradients of brain development, spurring knock-on effects as the process of interactive specialisation of neural and cognitive systems unfolds (Johnson et al. 2002; Karmiloff-Smith 2007). Support for the notion of such multilevel cascades of perturbed development comes from studies of schizophrenia demonstrating deficits in early sensory processing (Butler & Javitt 2005; Butler et al. 2007; Uhlhaas & Silverstein 2005) and relating auditory frequency discrimination to deficits in affect recognition (Leitman et al. 2007) and visual size discrimination to deficits in theory of mind (Uhlhaas et al. 2006), from studies of autism demonstrating enhanced perception of first-order stimuli (Mottron et al. 2006) and associating theory-of-mind performance with joint attention (Charman 1997) and perceptual disembedding (Jarrold et al. 2000), and from models relating Crespi and Badcock's cited variations in brain size to systems-level variation in the balance between short-distance and long-distance connectivities (Lewis & Elman 2008). In each of these instances, what seems on face an alteration of higher-order cognition is seen to be related to, and in some cases a consequence of, perturbations at lower levels of

processing – and in this regard the cited conditions are not disorders of the “social brain” exclusively. Though such relationships may be most obvious in cases of clinical disorders, they seem unlikely to be limited to disordered cognition and instead may extend to normal variation – that is, to the entire continuum of cognitive variation between the mentalistic and mechanistic extremes.

Further refinement will be necessary to explore aspects in which the autism spectrum and the less straightforwardly defined “psychotic spectrum” may have been miscast as opposites. As Crespi and Badcock note, some empirical aspects are not comparable between these two populations with existing data. Schizophrenia is diagnosed much later than autism, and therefore there remains a dearth of early developmental observations. For example, although the autistic amygdala is indeed abnormally large during childhood and therefore seems a counterpoint to the abnormally small amygdala in schizophrenia, by adulthood the autistic amygdala likewise has become abnormally small (Aylward et al. 1999; Rojas et al. 2004). Likewise, observations of frontal hypoactivation in autism may depend strongly on the behavioural task and on the latency at which activation is measured – in some contexts autistic frontal systems are hyperactivated (Takarae et al. 2007). Regarding sensory filtering, autism shares with schizophrenia a decrease in prepulse inhibition (McAlonan et al. 2002; Perry et al. 2007), and although people with autism do often focus attention very strongly, in some cases maintaining attention is an issue, and many people with autism fulfill criteria for attention-deficit/hyperactivity disorder (Corbett & Constantine 2006). Familial association of autism and mood disorders (DeLong 2004) calls into question the inclusion of mood disorders within the

“psychotic spectrum” in this context. The connectivity issues too remain to be sorted out; both autism and schizophrenia have been described as deficits in brain connectivity (Frith 2005), but these may be secondary to developmental interactions whose antecedents differ between the two conditions. Despite such areas for refinement, the mentalism–mechanism duality has strong potential to change the way we think about the relation of clinical disorders to normal human cognitive variation, as well as the evolutionary forces that have produced both.

<RFT>References [Matthew K. Belmonte] [MKB]

<refs>

Aylward, E. H., Minshew, N. J., Goldstein, G., Honeycutt, N. A., Augustine, A. M., Yates, K. O., Barta, P. E. & Pearlson, G. D. (1999) MRI volumes of amygdala and hippocampus in non-mentally retarded autistic adolescents and adults. *Neurology* 53:2145–50. [MKB]

Baron-Cohen, S. (2002) The extreme male brain theory of autism. *Trends in Cognitive Sciences* 6:248–54. [MKB]

Butler, P. D. & Javitt, D. C. (2005) Early-stage visual processing deficits in schizophrenia. *Current Opinion in Psychiatry* 18:151–57. [MKB]

Butler, P. D., Martinez, A., Foxe, J. J., Kim, D., Zemon, V., Silipo, G., Mahoney, J., Shpaner, M., Jalbrzikowski, M. & Javitt, D. C. (2007) Subcortical visual dysfunction in schizophrenia drives secondary cortical impairments. *Brain* 130:417–30. [MKB]

Charman, T. (1997) The relationship between joint attention and pretend play in autism. *Development and Psychopathology* 9:1–16. [MKB]

Corbett, B. A. & Constantine, L. J. (2006) Autism and attention deficit hyperactivity disorder: Assessing attention and response control with the integrated visual and auditory continuous performance test. *Child Neuropsychology* 12:335–48. [MKB]

DeLong, R. (2004) Autism and familial major mood disorder: Are they related? *Journal of Neuropsychiatry and Clinical Neuroscience* 16:199–213. [MKB]

Frith, C. (2005) The neural basis of hallucinations and delusions. *Comptes Rendus Biologies* 328:169–75. [MKB]

Jarrold, C., Butler, D. W., Cottington, E. M. & Jimenez, F. (2000) Linking theory of mind and central coherence bias in autism and in the general population. *Developmental Psychology* 36:126–38. [MKB]

Johnson, M. H., Halit, H., Grice, S. J. & Karmiloff-Smith, A. (2002) Neuroimaging of typical and atypical development: A perspective from multiple levels of analysis.

Development and Psychopathology 14:521–36. [MKB]

Karmiloff-Smith, A. (2007) Atypical epigenesis. *Developmental Science* 10:84–88.

[MKB]

Leitman, D. I., Hoptman, M. J., Foxe, J. J., Saccente, E., Wylie, G. R., Nierenberg, J., Jalbrzikowski, M., Lim, K. O. & Javitt, D. C. (2007) The neural substrates of impaired prosodic detection in schizophrenia and its sensorial antecedents. *American Journal of Psychiatry* 164:474–82. [MKB]

Lewis, J. D. & Elman, J. L. (2008) Growth-related neural reorganization and the autism phenotype: A test of the hypothesis that altered brain growth leads to altered connectivity.

Developmental Science 11:135–55. [MKB]

McAlonan, G. M., Daly, E., Kumari, V., Critchley, H. D., van Amelsvoort, T., Suckling, J., Simmons, A., Sigmundsson, T., Greenwood, K., Russell, A., Schmitz, N., Happé, F., Howlin, P. & Murphy, D. G. (2002) Brain anatomy and sensorimotor gating in

Asperger's syndrome. *Brain* 125:1594–1606. [MKB]

Mottron, L., Dawson, M., Soulieres, I., Hubert, B., Burack, J. A. (2006) Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders* 36:27–43. [MKB]

Perry, W., Minassian, A., Lopez, B., Maron, L. & Lincoln A. (2007) Sensorimotor gating deficits in adults with autism. *Biological Psychiatry* 61:482–86. [MKB]

Pies, R. (2007) The historical roots of the bipolar spectrum: Did Aristotle anticipate Kraepelin's broad concept of manic-depression? *Journal of Affective Disorders* 100:7–11. [MKB]

Rojas, D. C., Smith, J. A., Benkers, T. L., Camou, S. L., Reite, M. L. & Rogers, S. J. (2004) Hippocampus and amygdala volumes in parents of children with autistic disorder. *American Journal of Psychiatry* 161:2038–44. [MKB]

Takarae, Y., Minshew, N. J., Luna, B. & Sweeney, J. A. (2007) Atypical involvement of frontostriatal systems during sensorimotor control in autism. *Psychiatry Research* 156:117–27. [MKB]

Uhlhaas, P. J. & Silverstein, S. M. (2005) Perceptual organization in schizophrenia spectrum disorders: A review of empirical research and associated theories. *Psychological Bulletin* 131:618–32. [MKB]

Uhlhaas, P. J., Phillips, W. A., Schenkel, L. S. & Silverstein, S. M. (2006) Theory of mind and perceptual context-processing in schizophrenia. *Cognitive Neuropsychiatry* 11:416–36. [MKB]