

No New Neurobiology Yet for Autism

John Robert Skoyles, PhD

Minshew and Williams¹ argue for a "new neurobiology of autism" that identifies it as "a large-scale neural systems disorder with alterations in cortical systems connectivity." The basis for this conclusion are studies that compare matched groups of autistic and nonautistic subjects.

Their hypothesis is interesting, but they omit discussion of counter examples relevant to their hypothesis. If autism links to abnormalities in "cortical systems connectivity," then this requires that nonautistic individuals do not show such abnormalities. However, these are widely found.

First, they are present in individuals in the nonautistic group of the studies they cite. The autistic-related traits of these studies derive from group average comparisons that hide—as can be seen in inspection on the nonautistic control group data—individuals who have the same trait as that identified at the group level with autism. None of the reviewed physical or functional traits indeed identified with autism in the cited studies is, in fact, specific to autism. Moreover, such normal "outliers" argue that from a population perspective, the majority of individuals with a trait identified by group analysis with autism, in fact, paradoxically do not have autism. This is because a small percentage of a large number (the population of nonautistic individuals) can easily be larger in absolute terms than a high percentage of a tiny one (individuals with autism make up only a small fraction of the total population).

Second, many individuals exist with neurological conditions that cause severe white matter axon connection abnormalities without this also causing in them concomitant autism. For example, hydrocephalus does not associate with autism even though it can produce profound diminution of intrahemispheric white matter connectivity: consider for instance, the recent case (and the accompanying magnetic resonance imaging scan) in *Lancet*.²

Acknowledging the existence of such counter examples matters because they allow potentially better focused hypotheses. For example, autism might relate to a specific type of large-scale neural systems disorder. But without researching the varieties of large-scale neural systems disturbances that occur in nonautistic individuals, that specific disturbance and its exact nature will be hard to identify.

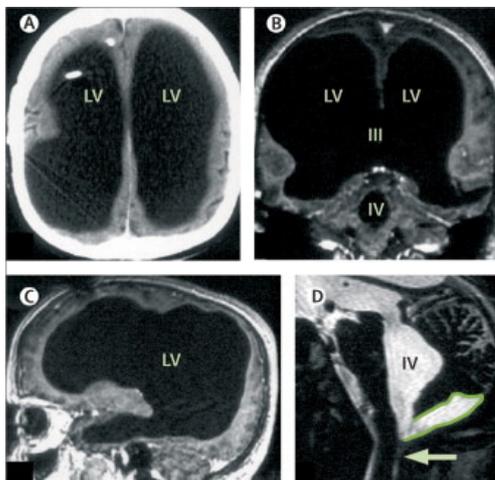
AUTHOR INFORMATION

Correspondence: Dr Skoyles, Centre for Mathematics and Physics in the Life Sciences and Experimental Biology, University College London, 4 Stephenson Way, London NW1 2HE (j.skoyles@ucl.ac.uk).

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1. Minshew NJ, Williams DL. The new neurobiology of autism: cortex, connectivity, and neuronal organization. *Arch Neurol*. 2007;64(7):945-950.

2. Feuillet L, Dufour H, Pelletier J. Brain of a white-collar worker. *Lancet*. 2007;370(9583):262.



An individual with hydrocephalus and mass disruption of cerebral cortex white matter connections but no autism. Minshew and Williams argue that such disruption underlies autism. They, unfortunately did not do a search for counter-examples to this theory. Figure from Feuillet L, Dufour H, Pelletier J. Brain of a white-collar worker. *Lancet*. 2007;370(9583):262.