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Title: Connectivity and cognition in autism spectrum disorders: where are the links?

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The idea that autism spectrum disorders (ASDs) are a consequence of reduced or atypical neural connectivity has considerable intuitive appeal and a growing evidence base, but struggles to address both the extreme clinical heterogeneity amongst individuals with an ASD diagnosis and the fact that atypical brain connectivity is clearly non-specific to ASDs (1). Rather than treating atypical connectivity as an explanation for ASDs per se, an alternative approach may be to consider the relationship between specific connections within the brain and specific symptoms or cognitive differences associated with the diagnosis. However, given the huge number of neural pathways, combined with the many different behavioural symptoms and cognitive differences associated with ASDs, a major challenge will be to distinguish meaningful cognition-connectivity links from chance correlations.

These issues are highlighted by the recent PNAS paper by Abrams and colleagues (2), who proposed that weakened connections between the posterior superior temporal sulcus (pSTS) and frontostriatal regions mean that people with ASDs find speech stimuli less intrinsically rewarding, leading to the communication impairments associated with the disorder. Consistent with this proposal, the authors reported that, relative to typically developing controls, children with an ASD diagnosis showed a reduction in coactivation between the pSTS and fronto-striatal regions; and that, within the ASD group, this putative index of functional connectivity was correlated with the children’s reciprocal communication skills.

There are, however, a number of missing links in the authors’ argument. Abrams et al. employed a resting state paradigm, thereby avoiding the concern that differences in brain activity may be a consequence rather than a cause of behavioural
differences; but they provided no evidence that listening to speech activates the “reward centres” of the brain in typically developing individuals, or that this effect (if it exists) is reduced in ASDs. They also failed to provide any behavioural evidence that their participants with ASD found speech stimuli less rewarding than control participants or, critically, that individual differences in the reward value of speech correlated with patterns of connectivity. Lastly, it is unclear whether the association between communication skills and pSTS-frontostriatal connectivity was specific either to the clinical or the neural measure.

These observations indicate the need for clearly defined minimum criteria for establishing specific connectivity-cognition links in future studies. First, investigation should be restricted to pairs of brain regions where there is evidence for a direct physical connection, and where both regions are activated during the cognitive process in question. Second, the cognitive process has to be measured directly and reliably. Third, it must be demonstrated that the cognitive and connectivity measures are more strongly associated with each other than they are with other cognitive or connectivity measures. Finally, given the considerable scope for false positives, we should all retain a healthy degree of caution in our interpretation of such findings until they are replicated.
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References
