Small-world Network Properties and the Emergence of Social Cognition: Evidence from Functional Studies of Autism

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Abstract

Autism has been proposed to stem from a pervasive abnormality of neural information processing possibly involving decreased signal-to-noise in neural systems [6]. Such a final common pathway of dysfunction at the network level may admit a wide fan-in of causal factors at the level of cells, molecules, and genes, and could be expected to interact with normal neurodevelopmental programmes and gradients to fan out into a wide range of abnormalities of cognition and behaviour [1]. Autism may therefore serve as a useful test case for theories of normal brain and cognitive development at the junction of network modelling with developmental cognitive neuroscience. Behavioural and cognitive findings on autism have been dominated by a combination of facilitated processing of local features and impairment in complex, integrative cognition. These behavioural results are supported by functional imaging and quantitative EEG studies that find abnormally strong, unselective responses within sensory regions and abnormally weak functional correlations between distant regions (*e.g.* [5]). One possible explanation for this pattern is abnormal strengthening of local network connectivity at the expense of global connectivity – a combination that may sabotage the 'small-world' property [7] in which cortical networks combine strong local connectivity with short mean path length. Indiscriminately strong anatomical connectivity within local processing regions may actually decrease computational connectivity, if crosstalk is increased to a degree at which relevant stimuli cannot be discriminated from noise [2]. Network abnormalities of this sort may explain an autistic learning style founded on statistical association rather than on instructive focus on relevant stimuli. Aberrant patterns of cortical connectivity are suggested by reports of decreased size of cortical minicolumns [3], early developmental hyperplasia in short-range white matter but not longer-range white matter compartments [4], associations with neuroligins and other substances involved in synaptogenesis and synaptic modification, and comorbidity with disorders that involve abnormal excitability or alterations in synaptic structure, such as epilepsy and Fragile X syndrome. We describe fMRI findings from two visual selective attention tasks that bear on this issue of neural connectivity in the autistic brain. In Experiment 1, adults with autism spectrum disorders (ASDs) and normal controls covertly attended to one or the other stream of rapid serially presented colour stimuli in left and right hemifields, shifting attention in response to cues within the attended stream. In Experiment 2, 10-to-15-year-old boys with ASDs, their non-autistic brothers, and unrelated normal controls performed a cognitively demanding task combining selective attention to location, colour, and orientation. These studies revealed findings consistent with abnormalities of local and long-range connectivity, including (1) abnormally strong and unselective processing within visual areas of cortex, (2) abnormal deactivation of more anterior, integrative brain regions including inferior frontal gyrus, anterior cingulum, frontal pole, and medial temporal lobe, and (3) abnormally low functional correlations between anterior and posterior regions. In addition, other results may reflect cognitive strategies that attempt to compensate for abnormal excitability of sensory cortices, including (4) abnormally strong activation of parietal cortex during suppression of visual distractors and (5) deactivation of auditory cortex during difficult visual discriminations. Preliminary data suggest that non-autistic sibs share some of the anterior deactivations but none of the posterior hyperactivations; sibs are a useful contrast in disentangling the more subtle, primary effects of genetic susceptibilities from the complicated, secondary dysfunctions associated with the full syndrome of autism. Although this hypothesis of autism as a developmental effect of abnormal network properties remains speculative in the absence of any large-scale microanatomical studies directly examining autistic brains across anatomical regions and developmental periods, it provides a useful entry point for the study of social cognition, a complex capacity whose proper development may depend on a balance between connections that subserve local processing and global integration.

References

[1] M.K. Belmonte, E.H. Cook Jr., G.M. Anderson, J.L.R. Rubenstein, W.T. Greenough, A. Beckel-Mitchener, E. Courchesne, L.M. Boulanger, S.B. Powell, P.R. Levitt, E.K. Perry, Y. Jiang, T.M. DeLorey and E. Tierney, "Autism as a disorder of neural information processing: directions for research and targets for therapy," *Mol. Psychiatry,* vol. 9, no. 7, pp. 646-663, 2004. Unabridged edition at `http://www.cureautismnow.org/media/3915.pdf`

[2] M.K. Belmonte, G. Allen, A. Beckel-Mitchener, L.M. Boulanger, R.A. Carper and S.J. Webb, "Autism and abnormal development of brain connectivity," *J. Neurosci.,* in press 2004.

[3] M.F. Casanova, D.P. Buxhoeveden, A.E. Switala and E. Roy E, "Minicolumnar pathology in autism," *Neurology,* vol. 58, no. 3, pp. 428-432, 2002.

[4] M.R. Herbert, D.A. Ziegler, N. Makris, P.A. Filipek, T.L. Kemper, J.J. Normandin, H.A. Sanders, D.N. Kennedy, V.S. Caviness Jr., "Localization of white matter volume increase in autism and developmental language disorder," *Ann. Neurol.,* vol. 55, no. 4, pp. 530-540, 2004.

[5] M.A. Just, V.L. Cherkassky, T.A. Keller, N.J. Minshew, "Cortical activation and synchronization during sentence comprehension in high-functioning autism: evidence of underconnectivity," *Brain,* in press 2004.

[6] J.L.R. Rubenstein and M.M. Merzenich, "Model of autism: increased ratio of excitation/inhibition in key neural systems," *Genes Brain Behav.,* vol. 2, no. 5, pp. 255-267, 2003.

[7] O. Sporns, G. Tononi and G.M. Edelman, "Theoretical neuroanatomy: relating anatomical and functional connectivity in graphs and cortical connection matrices," *Cereb. Cortex,* vol. 10, no. 2, pp. 127-141, 2000.