Cognitive discoordination in schizophrenia and autism

Commentary on Phillips W. A., & Silverstein, S. M. (2003). Convergence of biological and psychological perspectives on cognitive coordination in schizophrenia. *Behavioral and Brain Sciences, 26*, 65-138.

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Abstract

Phillips and Silverstein suggest that schizophrenia and autism may share a common underlying deficit in cognitive coordination. This commentary highlights some of the similarities and differences between the two disorders and argues that comparison of autism and schizophrenia may prove useful in refining the cognitive discoordination account.

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Phillips and Silverstein (P&S) argue that cognitive discoordination is the underlying cause of schizophrenia, but may also be involved in other neurodevelopmental conditions such as autism. This view is consistent with theoretical accounts suggesting that many of the behavioural features of this disorder result from difficulties in processing information in context (Frith, 1989), and with our own hypothesis that autism is associated with a deficit in the integration of ongoing activity in diffuse brain regions (Brock, Brown, Boucher, & Rippon, 2002). There are, of course, clear differences between the two disorders: Autism is commonly diagnosed in the first few years of life, whereas the onset of schizophrenia is typically much later. Moreover, positive symptoms of schizophrenia such as delusions and hallucinations are exclusion criteria for a diagnosis of autism (American Psychiatric Association, 1994). Nevertheless, there is considerable overlap between the disorders. In particular, negative symptoms of schizophrenia such as social withdrawal, stereotyped behaviour, and lack of communication are also typical features of autism (Frith & Frith, 1991; see also Konstantareas & Hewitt, 2001; Sheitman, Kraus, Bodfish, & Carmel, in press).

Frith (1992; see also Frith & Frith, 1991) has previously suggested that autism and schizophrenia may share a common core deficit in metarepresentation - that is in the ability to represent intentions and beliefs (cf. Baron-Cohen, 1995; Frith & Corcoran, 1996). According to this account, similarities between autism and schizophrenia in terms of negative symptoms can be explained by difficulties in representing one's own goals. Moreover, differences between the two disorders in terms of positive symptoms are assumed to reflect the different age of onset of the metarepresentational deficit. In

schizophrenia, such capacities are assumed to develop relatively normally before the onset of the disorder, and positive symptoms arise because the content of propositions (e.g., '*catch the bus'*) become detached from the context indicating whether they are internally or externally generated (e.g., 'I intend to *catch the bus*' or 'Mary wants me to *catch the bus*'). In contrast, Frith argued that most individuals with autism never develop metarepresentations, so the abnormal experience of 'free-floating' propositions cannot occur. However, Frith's account struggles to explain other 'lower-level' deficits that are common to both disorders, such as the impaired ability to integrate context in language processing (e.g., Happe, 1997; Titone, Levy, & Holzman, 2000), and abnormalities in visual perception (e.g., Happe, 1999; Silverstein et al., 1996). Instead, as P&S argue, such findings suggest that there may be a broader deficit in cognitive coordination in both schizophrenia and autism, and that impairments in metarepresentation are one consequence of this (cf. Brock et al., 2002; Leiser & Bonshtein, in press).

Nevertheless, it may be premature to assume that *descriptive* similarities between autism and schizophrenia reflect common underlying cognitive and perceptual mechanisms. Indeed, where similar paradigms have been used with the two groups, results indicate that there are similarities but also subtle differences. For example, both groups show high motion coherence thresholds (e.g., Chen, Nakayame, Levy, Matthysse, & Holzman, 2003; Milne et al., 2002), but show different patterns of responses to local-global hierarchical figures (e.g., Bellgrove, Vance, & Bradshaw, 2003; Plaisted, Swettenham & Rees, 1999) and differential susceptibility to certain visual illusions (Ropar & Mitchell, 1999; Uhlhaas, Silverstein, Phillips, & Lovell, in press).

A further issue concerns the underlying neural correlates of cognitive discoordination. P&S suggest that schizophrenia may be associated with abnormalities in gamma-band cortical activity that is involved in integrative brain function - a view confirmed by recent EEG studies of schizophrenia (see Lee, Williams, Breakspear & Gordon, 2003). Two recent studies have also reported atypical gamma EEG activity in autism. Grice et al. (2001) observed that, whereas unimpaired adults demonstrated increased frontal gamma activity to upright faces and decreased activity to inverted faces, adults with autism showed an increase in induced frontal gamma activity to both upright and inverted faces. Similarly, Brown, Gruber, Boucher, Rippon, and Brock (in press) found that, relative to learning-disabled controls, adolescents with autism showed a significant increase in induced parietal gamma activity when presented with an illusory Kanizsa shape in an array of 'pacman' shapes (there were no group differences when there was no illusory shape).

These studies indicate that integrative brain activity is atypical in both autism and schizophrenia. However, given the differences in age of onset, it seems unlikely that such activity is atypical in the same way in the two disorders. Cyclical changes in levels of EEG coherence between distant cortical sites during early childhood indicate that brain development at this stage features periods of increasing connectivity that are followed by periods during which these connections are pruned (Thatcher, 1992). Brock et al. (2002) therefore hypothesised that cognitive discoordination in autism is caused by either limited growth or excessive pruning of these *long-range* connections early in development. In contrast, schizophrenia has been linked to excessive pruning of *short-range* connections

within frontal cortex that occurs during adolescence (e.g., Hoffman & McGlashan, 1997; Lewis & Gonzalez-Burgos, 2000).

In summary, there are interesting parallels between autism and schizophrenia that may, as P&S suggest, be explicable in terms of a common underlying deficit in cognitive coordination. Nevertheless, there are clear differences between the two disorders, although the later age of onset of schizophrenia would appear to be crucial in explaining many of these differences. With this in mind, future research investigating the similarities and differences between the two disorders in terms of underlying neural, perceptual and cognitive processes may provide a useful means of refining cognitive discoordination accounts of both autism and schizophrenia.

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