models) and/or an increase in other nodes; and sensory gating or filtering (Miyake et al. 2000). Through these processes, inhibition provides interference control and assists in processing relevant information. Although inhibition and working memory are associated constructs, they are not entirely overlapping processes. While P&S discuss inhibition as one aspect of cognitive coordination, they discuss inhibition primarily in terms of its service in the maintenance of context. However, the disinhibition seen in patients with schizophrenia encompasses a broader array of impaired processes. In addition to disinhibition of prepotent responses such as those seen during Stroop and anti-saccade tasks, schizophrenia patients also manifest sensorimotor gating deficits as measured through prepulse inhibition paradigms (cf. Braff et al. l999) and perseveration in behavioral responses as well as in language production. We would therefore assert that disinhibition, in addition to failure to maintain contextual representations, is part of the cognitive signature of schizophrenia.

The P&S model departs from that of Meehl (l962; l989) by allowing for etiological as well as phenomenological heterogeneity. Indeed, the authors assert that their model is particularly relevant for the disorganized subtype of schizophrenia. In the final section of their paper, P&S present several avenues for further theorizing and investigation. One further avenue for refining the P&S model might be to relate other aspects of neurochemical dysregulation, for example, the effects of serotonergic hallucinogens and/or dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis (cf. Walker & DiForio l996) to the coordinating interactions that are implemented via NMDA-receptors. Such theorizing might account for the exacerbation of at-risk individuals' cognitive impairments under conditions of stress.

In summary, the P&S model fails to link the NMDA-mediated cognitive dysmetria specifically to the development of schizophrenia and spectrum disorders. Nonetheless, we believe that it provides a valuable heuristic for our understanding of schizophrenia and are encouraged by its potential for further research inquiry.

Linking brain to mind in normal behavior and schizophrenia

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Abstract: To understand schizophrenia, a linking hypothesis is needed that shows how brain mechanisms lead to behavioral functions in normals, and also how breakdowns in these mechanisms lead to behavioral symptoms of schizophrenia. Such a linking hypothesis is now available that complements the discussion offered by Phillips & Silverstein (P&S).

This interesting target article emphasizes the important role of "cognitive coordination" – notably contextual disambiguation and dynamic grouping – in schizophrenia, and proposes that a failure of such coordination may occur during schizophrenia as a result of reduced ion-flow through NMDA glutamate receptors. The article summarizes a variety of useful data that support this hypothesis to varying degrees. There is, however, a large gap in the authors' argument that would need to be filled for the hypothesis to be more compelling: Namely, no linking hypothesis is supplied to explain how brain mechanisms of cognitive coordination work, or how they generate contextual properties at the behavioral level in normal individuals; or how the proposed breakdown of these mechanisms leads to schizophrenic symptoms. As a result, the authors' arguments are basically arguments of consistency with some known data, rather than arguments from which a compelling deduction of cause and effect can be derived.

Because of this absence of computational rigor, some of their statements can be confusing or even misleading. For example, the authors write that "subsets of neuronal responses are grouped by synchronizing the spiking activity of which they are composed" (target article, sect. 1, para. 2). This seems to say that synchrony causes grouping, whereas it is detailed neural models of vision which show how grouping can sometimes cause synchrony (Grossberg 1976; Grossberg & Grunewald 1997; Grossberg & Somers 1991). A second possible cause of confusion is that mechanistically different types of cognitive coordination tend to be lumped together, rather than distinguished by their unique characteristics. For example, the authors mention working memory in the prefrontal cortex (PFC) as an example of cognitive coordination (sect. 1) but do not indicate that the circuits that control working memory in the PFC (e.g., Bradski et al. 1994; Grossberg 1978) can have different organization and properties than, say, those which govern grouping in V1 and V2 (e.g., Grossberg 1999; Grossberg & Raizada 2000).

The authors' working hypothesis is "that cortical activity is coordinated by widely distributed local interactions within and between regions, as well as by top-down strategic commands" (sect. 1, para. 12), a view that is presently shared by many neuroscientists. Stated so broadly, this hypothesis is hard to use effectively. In fact, detailed models of the laminar architecture of the neocortex now clarify how such bottom-up, top-down, and horizontal interactions are organized for purposes of perceptual grouping, attention, development, and learning (e.g. Grossberg 1999; Raizada & Grossberg 2003), and would be a good foundation for testing the authors' general hypothesis. These LAMINART models have qualitatively explained and quantitatively simulated key grouping and attentional effects about visual *What* stream processing that the authors review; for example, in section 2.1. The authors then immediately discuss processing in the *Where* cortical stream of coherent object motion. Although these *What* and *Where* processes share some mechanisms, they also have computational complementary properties (Grossberg 2000b), as a result of the differences between generating orientationally sensitive groupings of object form and generating directionally sensitive groupings of object motion. Key data about coherent object motion have also been quantitatively simulated (Chey et al. 1997; Grossberg et al. 2001), and one can now study precisely how form and motion processes differ, and how they interact to overcome their complementary deficiencies.

Models have elsewhere been proposed that attempt to show how cognitive-emotional neural processes can explain normal behavioral and brain data about learning in animals and humans, and prescribed breakdowns in these models give rise to schizophrenic negative symptoms. These models include feedback interactions between brain regions such as the sensory cortex, amygdala, and prefrontal cortex, and propose how an overaroused or underaroused opponent process in the amgydala or prefrontal cortex can lead to negative symptoms (Grossberg 1984; 2000c). These models do include a possible role for DA (dopamine) dysfunction in giving rise to overaroused or underaroused condition. Given the derivation of these models from properties of cognitive-emotional learning, they may also accommodate problems with NMDA-receptors. Related models of sensory and cognitive learning suggest how tonically hyperactive volitional signals, say from the basal ganglia, could lead to positive symptoms like hallucinations (Grossberg 2000a), as a manifestation of the learned top-down expectations that normally help to stabilize learning and, along the way, focus attention on, and prime, the brain to selectively process objects of interest.

https://doi.org/10.1017/S0140525X03300020 Published online by Cambridge University Press