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BBS COMMENTARY

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Commentator: Stephen Grossberg

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W.A. Phillips and S.M. Silverstein

**LINKING BRAIN TO MIND IN NORMAL BEHAVIOR
AND SCHIZOPHRENIA**

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Abstract

In order 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 and Silverstein.

COMMENTARY

This interesting 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 due to 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 author’s argument that would need to be filled for it to be more compelling; namely, no linking hypothesis is supplied whereby to explain how brain mechanisms of cognitive coordination work, or how they generate contextual properties on the behavioral level, in normal individuals, or how their proposed breakdown of these mechanisms leads to schizophrenic symptoms. As a result, their 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 (p. 2) that “subsets of neuronal responses are grouped by synchronizing the spiking activity of which they are composed.” This seems to say that synchrony causes grouping, whereas detailed neural models of vision show how grouping can sometimes cause synchrony (Grossberg, 1976; Grossberg and Somers, 1991; Grossberg and Grunewald, 1997). 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 prefrontal (PFC) as an example of cognitive coordination (p. 4), but do not indicate that the circuits which control working memory in PFC (e.g., Bradski et al., 1994; Grossberg, 1978) can have a different organization and properties than, say, those which govern grouping in V1 and V2 (e.g., Grossberg, 1999; Grossberg and 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...” (p. 6), a view that is presently shared by many neuroscientists. Stated so broadly, this hypothesis is hard to effectively use. In fact, detailed models of the laminar architecture of 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 and Grossberg, 2003), and would be a good foundation for testing the authors’ general hypotheses. These LAMINART models have qualitatively explained and quantitatively simulated key grouping and attentional effects about visual What stream processing that the authors review; e.g., p. 7. 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) due to the differences between generating orientationally-sensitive groupings of object form vs. 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 precisely study 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 how prescribed breakdowns in these models give rise to negative schizophrenic symptoms. These models include feedback interactions between brain regions like sensory cortex, amygdala and prefrontal cortex, and propose how an over-aroused or under-aroused opponent process in the amygdala or prefrontal cortex can lead to negative symptoms (Grossberg, 1984, 2000c). These models do include a possible role for DA dysfunction. 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, help to focus attention upon, and prime, objects of interest.

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