We tested the same subjects for ToB, adapting the battery designed by Johnson and Carey (1998). This battery relies on two levels of understanding biology, known to discriminate younger from older children. The lower level relies on acquired information about living beings. The higher level marks accession to the understanding of living beings as functional systems, which gives meaning to biological functions. (For example, because all organisms expand energy, they all must eat somehow, even if they have no apparent food ingesting organs.) In their study of Williams Syndrome (WS) patients, Johnson and Carey found that for all their verbal fluency, WS patients remain at the lower, childlike level of understanding.

Schizophrenic patients test normal on ToB, even during acute episodes. One explanation for this difference between ToM and ToB would be to posit a "module" or cerebral specialization for ToM (Povinelli & Preuss 1995). The alternative, "theory-theory" view, maintains that ToM is acquired like any other naïve theory (Gopnik & Wellman 1992). On that view, it would seem difficult to account for the dissociation of ToB and ToM.

We suggest that the challenge of ToM may be different from that encountered in other naïve theories (Leiser 2001). The tasks used to test for ToM require coordination of several pieces of information. Integration of multiple relations is a specific source of cognitive complexity (Astington et al. 2002; Halford et al. 1998; Waltz et al. 1999). In the false beliefs tasks, subjects must hold separate and coordinate the actual state of affairs, the first character's beliefs about them, and the second character's beliefs about the first one's. This coordination is evidently beyond schizophrenics. In Sarfati's paradigm tasks, selecting the right answer implies building a context for the character's actions, and this requires coordination of the successive steps. Failing this complex contextual disambiguation, subjects fall back on either familiar actions to provide meaning, or use a much reduced context, consisting of the last picture only. If this line of reasoning is correct, we would have in ToM a symptom that arises, not from a module, but from the vulnerability of ToM to a deficit in coordination. Preservation of ToB, by contrast, can be explained by the absence of such coordination once the higher level of understanding is achieved.

This conclusion remains tentative as an account of deficits specific to schizophrenia. The authors's description of the effects of "schizomimetic" drugs fit psychotic-like state in general, yet nonschizophrenic psychoses (e.g., affective psychosis) do not damage ToM to the same extent as schizophrenic psychosis (Bonshtein & Leiser, in preparation; Sarfati et al. 1997). Equally, Sarfati et al. (1997) reported that a breakdown of schizophrenia into subtypes is unrelated to the severity of deficit in ToM, except for disorganized schizophrenia, which is associated with severe deficit in ToM. But this condition is characterized by a breakdown of personality and further traits that render cognitive collapse almost self-evident.

Reconciling schizophrenic deficits in top-down and bottom-up processes: Not yet

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Abstract: This commentary challenges the authors to use their computational modeling techniques to support one of their central claims: that schizophrenic deficits in bottom-up (Gestalt-type tasks) and top-down (cognitive control tasks) context processing tasks arise from the same dysfunction. Further clarification about the limits of cognitive coordination would also strengthen the hypothesis.

Phillips & Silverstein (P&S) put forward a hypothesis that addresses an important middle ground between a purely biological level of analysis and one that is entirely behavioral or cognitive. Such a cognitive neuroscience approach to schizophrenia is informative because it takes advantage of constraints from each domain to build a more comprehensive (and more convincing) story about the nature of schizophrenic psychopathology. In addition to the substantive theory put forward in the target article, this is a strong example of how to build hypotheses that incorporate, rather than simply pay lip service to, multiple levels of analysis.

The authors' hypothesis is particularly ambitious in that it attempts to weave together two competing domains of psychopathological research. One tradition of research has identified abnormalities in a variety of bottom-up perceptual processes, including the Gestalt phenomena noted by the authors, as well as backward masking, mismatch negativity, and other physiological and behavioral effects associated with posterior regions of the brain. A second tradition has focused on impairments in top-down processes such as executive functioning, working memory, attentional control, and other higher cognitive processes associated with the prefrontal cortex. There is a growing body of evidence for specific deficits in both traditions of research. The authors hypothesize that the distinction between schizophrenic deficits in bottom-up holistic perceptual processes and top-down control processes is unparsimonious and misleading; both bottom-up and top-down processes are impaired by the same mechanism, NMDA-hypofunction.

Science is often pushed forward by bold claims like this. For example, Cohen and Servan-Schreiber (1992) used computational modeling to demonstrate that schizophrenic impairments in attentional control and some working memory maintenance processes could be accounted for by a single mechanism (reduced gain attributable to tonic dopamine hypoactivity in prefrontal cortex). While the present authors have also developed computational models to support their claims about cognitive coordination, they have not yet taken the important steps of (1) demonstrating that the same model accounts for nontrivial, normal behavioral results in top-down control and bottom-up Gestalt tasks, both of which they refer to as "context processing" tasks, and, especially, that (2) impairments in such context processing associated with NMDA-hypofunction can reproduce impairments similar to those observed in schizophrenia patients *in both domains.* For example, it would be very compelling to find that the same lesion in Phillips and colleagues' models (e.g., Phillips et al. 1995) accounted for the patterns of impairment and spared performance in schizophrenia observed in both Place and Gilmore's (1980) Gestalt numerocity paradigm and in the expectancy manipulation of the AX task (MacDonald et al. 2003; Servan-Schreiber et al. 1996). Until such time, the argument that a single impairment leads to abnormal performance on both bottom-up and top-down tasks that require cognitive coordinating, is not compelling enough to reform this traditional distinction.

The task of breaking down artificial distinctions, if they exist, would be aided by greater clarity around the core construct in the hypothesis of cognitive coordination. The authors define "coordinating interactions" as "those that affect the salience or dynamic grouping of neuronal signals without changing what they mean. Such interactions do affect the interpretation of stimulus inputs, however" (sect. 1, para. 11). As it stands, the concept could be read to cover the waterfront of cognitive tasks (all of which require coordinating a task set with specific incoming stimuli). The authors have provided a number of specific examples of what they feel qualify as tasks of cognitive coordination, but it is not clear what tasks do *not* require cognitive coordination, or whether all tasks require this, but to varying degrees. If all tasks require cognitive coordination, the hypothesis becomes more difficult to test, as performance is confounded by other task demands generally present for cognitive tasks (including motivation, intelligence, and so on).