

Toward a Unifying Hypothesis for Schizophrenia and Autism Visual Fragmentation

To the Editor: Neurobiological research shows that consciousness has some neural correlates in the brain.¹ Understanding these neural correlates may be helpful in revealing the mechanisms of consciousness defects in disorders like autism and schizophrenia. Neurobehavioral studies suggest that autistic patients do not integrate visuo-perceptual information efficiently, which leads to the fragmented visual world of autistic patients. Surprisingly, “perceptual fragmentation” and “inefficient neuro-integrative perceptual processing” have also been described in schizophrenia.² Why, despite important differences, do these two diseases have such remarkable similarities? Certainly, the answer should be investigated at the cellular-molecular level. However, since the brain is a complex system with multiple interacting elements, it will be difficult to study the details at this level without having any initial insight. It seems that a systemic approach helps to solve the puzzle by bridging the distance between the cellular-molecular level and gross behavioral aspects. Some theories of consciousness provide such a systematic view, two of which are the “dynamic core hypothesis” (DCH) and “infor-

mation-integration theory” (IIT). In this notion, consciousness arises from a group of neurons (the dynamic core) which, in their dynamics, through reentrant interactions in the thalamocortical system, are simultaneously differentiated and integrated. Based on these theories, in conscious states, the brain can integrate the many different parts (modalities) of a scene to form a unitary view, which would be perceived as an integrated experience.^{3,4} The authors hypothesize that the fragmented visual world of autistic and schizophrenic patients is interpretable based on DCH and IIT viewpoints: the integration of the dynamic core, which contains the neuronal cluster that produces the visuo-perceptual conscious state, is somehow diminished in both diseases, which causes the perceiver to have fragmented and nonunitary visual experience. For determining the main neurobiological cause, some scientific clues may help us: 1) the effect of dopamine overstimulation in schizophrenia is strongly suggested in many research findings; 2) dopamine involvement in autism is implicated in some studies; 3) it has been claimed that striatum is not a simple relay, and records in animal research show that striatal neurons could integrate spatial information (the effective contribution of dopaminergic signaling to the modulation of neuronal activity in striatum has also been reported); and 4) the concept of involvement of dopaminergic neurons in the regulation of

the different cognitive aspects of behavior is illustrated in various experiments.⁵ Considering the abovementioned points, we believe that dopamine is involved in integration of information in dynamic core during visuo-perceptual tasks, which are disturbed both in schizophrenia and in autism. Since DCH and IIT present quantitative measures of neural system integration, using these measures may help us in understanding the role of dopamine in the mechanisms leading to visuo-perceptual fragmentation. Surely, experimental research is needed to validate our hypothesis.

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