
Rose F. Kennedy Intellectual and Developmental Disabilities Research Center (IDDRC)
Seminar Series

Director: Steven U. Walkley, DVM, Ph.D.

Associate Director: John J. Foxe, Ph.D.

Perceptual Organization Deficits in Schizophrenia: Why Do They Happen and Why Should We Care?

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Kennedy Center (Room 901): 12:45pm, Tuesday, March 27, 2012



Biographical Note: Dr. Brian P. Keane graduated summa cum laude at the University of Pittsburgh with majors in Physics & Astronomy, Spanish, and Philosophy. He obtained a Ph.D. in Philosophy at Rutgers University in 2006 and a Ph.D. in Psychology at the University of California, Los Angeles in 2009. Dr. Keane's research is broadly concerned with how the visual system represents a stable, coherent world from a spatiotemporally fragmented retinal image and how such integrative processes become disrupted during mental illness. He is a recipient of the American Psychological Association Dissertation Research Award, the UCLA Dissertation Year Fellowship, and the NIMH National Research Service Award (F32). As part of his NRSA, Dr. Keane utilizes behavioral measures to explore the neurobiological and clinical implications of perceptual organization deficits in schizophrenia.

Abstract: Seeing the world normally requires integrating information over space and time to determine the number and shape of objects that we visually confront. The mechanisms that contribute to this perceptual organization include circuits in V2, lateral occipital complex, and later areas such as the frontal lobe. Although the evidence is increasingly strong that perceptual organization is compromised in schizophrenia, the neurobiological locus of the problem is still disputed. In the first part of the talk, I present behavioral data that indicates that perceptually completing contours is automatic and dissociable from a later shape integration stage. In the second part of the talk, I present psychophysical evidence that the earlier contour completion stage is intact in schizophrenia whereas the shape integration stage is not. Upon pointing out the clinical implications of these results, I suggest that impaired magnocellular processing may explain at least a portion of the visual integrative deficits found in schizophrenia.