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

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

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Neurophysiology of sensory memory: A potential tool for early diagnosis of schizophrenia



Dean F. Salisbury, Ph.D.

Director, Cognitive Neuroscience Laboratory, McLean Hospital
Associate Professor, Department of Psychiatry, Harvard Medical School

**Kennedy Center (Room 901):
12:00pm, Thursday, May 17th, 2012**

Biographical note: Dean Salisbury, Ph.D. received his B.A. in the Whittier Scholars Program with an interdisciplinary major of Psychology, Sociology, and Education from Whittier College, California in 1985, followed by an M.A. (1989) and Ph.D. (1990) in Biological Psychology from the State University of New York at Stony Brook. He is Director of the Cognitive Neuroscience Laboratory at McLean Hospital since 2004. His research interests cover Cognitive Neuroscience and Clinical Cognitive Neuroscience, particularly cognitive abnormalities in schizophrenia. Prof. Salisbury's research focuses on the interplay between semantic memory neural networks subserving concept storage and verbal working memory systems that allow adaptive and flexible human behavior in the face of unique current situations. Ongoing research also examines basic auditory sensory processing and memory functioning (e.g. N1, MMN, P3) to determine where abnormalities occur in patients relative to controls, and MR measures determine whether the neural substrates of these brain processes are also affected in the diseases, utilizing longitudinal assessments of first episode patients.

Abstract: Schizophrenia is associated with well-known deficits in higher-order cognitive processes, reflected, for example, in thought disorder, delusions, and impaired working memory. Recent work has indicated deficits in low-level sensory processing as well. One particular event-related potential called mismatch negativity (MMN), a brainwave extracted from the EEG, is sensitive to pre-attentive sensory cortex detection of deviant events in the environment in service of orienting responses. MMN appears to be sensitive to N-methyl-D-aspartate (NMDA) receptor activity, and thus is highly relevant as a functional cortical index for the glutamate model of schizophrenia. Controversy has arisen whether MMN can be used to identify schizophrenic subjects prior to their first overt breakdown. The talk will review the existing data on MMN in schizophrenia with reference to the underlying neurochemical and neocortical circuit basis of the disorder, and introduce a new set of experiments aimed at developing new MMN tasks sensitive to the subtle biochemical perturbations that may be present during the disease prodrome.