

DR. ALLAN D. BASS (1910-2005)

Allan D. Bass was Professor and Chairman of the Department of Pharmacology at Vanderbilt University School of Medicine from 1953 until 1973. He served as Associate Dean for Biomedical Sciences from 1973 until his retirement in 1975 and as Acting Dean of the School of Medicine from 1973 to 1974. Additionally, he was Associate Chief of Staff for Education at the Veterans Administration Hospital from 1974 to 1981.

Dr. Bass was nationally recognized as an outstanding administrator, scientist, and educator. He participated actively in many national societies, including The American College of Physicians, American Society for Pharmacology and Experimental Therapeutics (President, 1967-1969) and the American Medical Association Council on Drugs (1962-1972). Dr. Bass was active in scientific research for more than four decades, and wrote many professional papers on such topics as anthelmintics, skin sterilizing agents, sulfonamides, adrenal corticosteroids, hormones, and chemical transmitters.

Dr. Bass was responsible for the development of Vanderbilt's Department of Pharmacology from a relatively small entity to a program that has achieved national recognition. As an educator, Dr. Bass always made every effort to assist each student to reach his or her academic potential and to meet professional requirements. Predoctoral and postdoctoral students trained in pharmacology during Dr. Bass' tenure now reside throughout the nation and several foreign countries, and many have held leadership positions in academia, government and the pharmaceutical industry.

The Allan D. Bass Lectureship, established in 1977, celebrates scientific inquiry and communication. The program is made possible through the generosity of many colleagues in the scientific community and by gifts from present and former students and faculty members at Vanderbilt University and the Vanderbilt Department of Pharmacology. Continuing support by alumni and friends is essential to the success and vitality of our training and research programs.

PREVIOUS ALLAN D. BASS LECTURERS INCLUDE:

1977	Avram Goldstein	1995	David E. Clapham
1979	James R. Gillette	1998	Phillip Needleman
1981	James A. Miller	2003	Susan S. Taylor
1981	Elizabeth C. Miller	2005	Morgan Sheng
1983	Norman Weiner	2007	Brian K. Kobilka
1985	Elliot S. Vesell	2009	Xiaodong Wang
1987	Richard W. Tsien	2012	Thomas C. Südhof
1989	John R. Blinks	2014	Peter Doherty
1993	Roger A. Nicoll		



JOSEPH T. COYLE, M.D.

N-METHYL-D- ASPARTATE RECEPTOR DYSFUNCTION
IN SCHIZOPHRENIA: A TARGET FOR MORE
EFFECTIVE TREATMENTS

MARCH 10, 2016

4:00 P.M.

208 LIGHT HALL

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PHARMACOLOGY (ALLAN D. BASS ENDOWED LECTURE SERIES)

VANDERBILT  UNIVERSITY
MEDICAL CENTER

N-METHYL-D- ASPARTATE RECEPTOR DYSFUNCTION IN SCHIZOPHRENIA: A TARGET FOR MORE EFFECTIVE TREATMENTS

Sixty years after the introduction of antipsychotic drug treatment for schizophrenia, the disorder still results in life-long disability because of persistent cognitive impairments and motivational deficits. Post-mortem and imaging studies have revealed a consistent neuropathology with atrophy of pyramidal neuronal dendrites and down-regulation of cortical GABAergic interneurons. Several findings suggest that impaired NMDA receptor glutamatergic neurotransmission accounts for these symptoms. The NMDA receptor is a molecular coincidence detector requiring the simultaneous depolarization of the nerve, binding of the neurotransmitter, glutamate and the binding of the modulator, D-serine in the cortex. The NMDA receptor directs neural plasticity and formation of memories. Silencing the gene for serine racemase (Srr-/-) that synthesizes D-serine recreates the atrophy of cortical pyramidal neurites, down-regulation of cortical GABAergic neurons, reduced NMDA receptor function, cognitive impairments and anhedonia observed in schizophrenia. These deficits could be reversed by treatment with D-serine in adulthood. Treatment with a mGluR5 positive allosteric modulator, which indirectly enhances NMDA receptor function, also remediates the Srr-/- deficits. A recent genome wide association study has revealed several genes encoding proteins interacting with the NMDA receptor or its down-stream mediators are associated with increased risk for schizophrenia, pointing to more effective treatment strategies.



JOSEPH T. COYLE, M.D.

**EBEN S. DRAPER CHAIR OF PSYCHIATRY AND
NEUROSCIENCE, HARVARD MEDICAL SCHOOL
CHIEF SCIENTIFIC OFFICER, MCLEAN HOSPITAL
MEMBER, INSTITUTE OF MEDICINE**

Joseph T. Coyle, M.D. holds the Eben S. Draper Chair of Psychiatry and Neuroscience at Harvard Medical School. From 1991 to 2001, he served as Chairman of the Department of Psychiatry at Harvard Medical School. He received his medical degree (M.D.) from Johns Hopkins School of Medicine. His research interests include developmental neurobiology, mechanisms of neuronal vulnerability and psychopharmacology. He is a member of the National Academy of Medicine (1990), a fellow of the American Academy of Arts and Sciences (1993), and fellow of the American Association for the Advancement of Science (Chair of the Section on Neuroscience, 2013-14). He was elected President of the Society for Neuroscience (SFN) in 1991. He has received numerous awards including A.E. Bennett Award from the Society of Biologic Psychiatry, the John Jacob Abel Award from ASPET, the Daniel Efron Award from ACNP, the McKnight Scholar in Neuroscience Award, the Lieber Prize for Research on Schizophrenia from NARSAD and the Julius Axelrod Award for Neuropharmacologic Research from SFN (2013).
