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Cover Image: The image on the cover shows a hypothetical model whereby glutamate is released from an intracortical pyramidal neuron and binds to an NMDA receptor on a GABA-ergic interneuron. GABA is then released and binds to receptors on the axon of another glutamate pyramidal neuron. This inhibits the neuron, thus reducing the release of cortical glutamate. The GABA interneuron and its NMDA synapse from the first neuron to the second is the hypothetical site of glutamate dysfunction in schizophrenia.

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CNS Spectrums aims to be the premiere journal covering all aspects of clinical neurosciences, neurotherapeutics and neuropsychopharmacology. From 2012 the journal will primarily focus on the publication of authoritative, cross-disciplinary review and opinion material publishing advances and controversial issues with pertinence to the clinician. In particular we aim to publish reviews and articles in translational neuroscience, biological psychiatry and neuropsychopharmacology that explain clinically relevant neuroscience discoveries in a way that makes these findings accessible and understandable to clinicians and clinical investigators. We will emphasize new therapeutics of all types in clinical neurosciences, mental health, psychiatry, and neurology, especially first in man studies and proof of concept studies. Our focus will be not just drugs, but novel psychotherapies and neurostimulation therapeutics as well. CNS Spectrums will in addition, continue to publish original research and commentaries that focus on emergent areas of research. Subject coverage shall span the full spectrum of neuropsychiatry focusing on translational issues and those crossing traditional boundaries between neurology and psychiatry.

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