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Justin S Blackwell* (jblackwell@uta.edu), Department of Mathematics, University of Texas at Arlington, P.O. Box 19408, Arlington, TX 76019. *A Modeling Study of Synaptic Neurotransmission and Independent Signaling of NMDA Receptors*. Preliminary report.

In a synapse, spontaneous and action-potential-driven neurotransmitter releases were generally assumed to activate the same set of postsynaptic receptors. New experimental and numerical results now support the premise that these two different release events activate distinct sets of NMDA receptors and independently signal postsynaptic receptors in postsynaptic terminals larger than $\sim 0.2 \mu\text{m}^2$. A computational model is constructed to simulate the diffusion of the neurotransmitter, glutamate, within the synaptic cleft as well as the receptor kinetics to determine opening probabilities that give rise to postsynaptic currents. We consider various factors within the synapse that can accommodate independent signaling in smaller synapses, such as variations in the spatial domain, release rate, and diffusion coefficient of the neurotransmitter. The modeling study collaborates with experimental results and gives a plausible explanation for synaptic noise from spontaneous release to be independent from action-potential-driven synaptic signals. (Received September 22, 2010)