SYNAPTIC DYNAMICS WITH SHORT-TERM FACILITATION AND DEPRESSION: A STOCHASTIC MODEL. V. Matveev* and X.-J. Wang. Dept. of Physics and Volen Center for Complex Systems, Brandeis Univ.; Waltham, MA 02254.

We explore the impact of short-term plasticity on synaptic transmission by studying the response of a stochastic model synapse to input spike trains of different temporal structure. Analyzing the case of a constant-frequency stimulus we find that: (i) steady-state probability of release (PR) is inversely proportional to stimulation rate as in other existing models; (ii) coefficient of variance of the response diverges with increasing input rate as $\sqrt{rate}$; (iii) Correlation between two successive release events is negative when PR is proportional to the number of available synaptic vesicles (SV) and can be positive when PR depends non-linearly on SV pool size, as is the case when at most one release per action potential is assumed. Further, we test the hypothesis that short-term facilitation can enable synapses to filter out single spikes and favor bursts of action potentials; driving the synaptic model with burst-containing spike trains we find that, given a fixed firing rate, transmission is most efficient when the input is organized into clusters of spikes. An opposite result holds for a strongly depressing synapse. We also show that the temporal correlations present in the input signal are significantly reduced by the synapse. Experimental recordings exist for cortical synapses revealing more than two-fold depression in response to a paired-pulse stimulus; we conclude that this effect cannot be accounted for by vesicle depletion, and propose mechanisms that would explain this fast initial phase of depression, including inactivation of release machinery, and introduction of an intermediate "primed" vesicle state. Lastly, we explore the possible role of delayed inhibitory feedback to vesicle release by incorporating model $GABA_B$ autoreceptors into our synaptic scheme, and find the dynamic range of the synapse to be significantly increased due to reduction of saturation of synaptic response at high stimulation rates. (Supported by the Alfred P. Sloan Foundation and ONR grant N00014-95-1-0319).