Stimulus-specific adaptation (SSA) occurs when neurons decrease their responses to frequently-presented (standard) stimuli but not, or not as much, to other, rare (deviant) stimuli. SSA is present in all mammalian species in which it has been tested as well as in birds. SSA confers short-term memory to neuronal responses, and may lie upstream of the generation of mismatch negativity (MMN), an important human event-related potential. Previously published models of SSA mostly rely on synaptic depression of the feedforward, thalamocortical input. Here we study SSA in a recurrent neural network model of primary auditory cortex. When the recurrent, intracortical synapses display synaptic depression, the network generates population spikes (PSs). SSA occurs in this network when deviants elicit a PS but standards do not, and we demarcate the regions in parameter space that allow SSA. While SSA based on PSs does not require feedforward depression, we identify feedforward depression as a mechanism for expanding the range of parameters that support SSA. We provide predictions for experiments that could help differentiate between SSA due to synaptic depression of feedforward connections and SSA due to synaptic depression of recurrent connections. Similar to experimental data, the magnitude of SSA in the model depends on the frequency difference between deviant and standard, probability of the deviant, inter-stimulus interval and input amplitude. In contrast to models based on feedforward depression, our model shows true deviance sensitivity as found in experiments.

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