Stimulus-specific adaptation in a recurrent network model of primary auditory cortex

Neurons in the auditory cortex respond strongly to stimuli that are deviant within a regular, repetitive sequence—a phenomenon called stimulus-specific adaptation. This enhancement of responses to deviants is strong and widespread in auditory cortex, but not in the brain area that provides its major input. Therefore, it is thought to be generated in the input to auditory cortex or in the cortex itself. We show that stimuli-specific adaptation can be generated in a simple neural network model where the connections between neurons exhibit synaptic depression, meaning they become weaker with each subsequent activation and only regain their strength slowly. Synaptic depression is known to occur in synapses between neurons in auditory cortex.

In contrast with previous models formulated by our lab and others, which included depression only in the synapses that convey the input to the cortex, in the current model the network structure and the depression of intracortical synapses play a key role: Stimuli evoke population events, in which almost all of the neurons within a cortical column fire at once. These population events propagate across the cortical sheet. However, they also leave in their wake an area where the synapses are depleted, where no population event can occur or invade until these synapses replenish. Stimulus-specific adaptation takes place when the stimuli forming the repetitive sequence cease to generate events that can propagate to other areas, while the deviant stimuli do evoke such events.

Using computer simulations of our model network, we showed that stimulus-specific adaptation in the model depends on various parameters in a way similar to what was found in recordings from auditory cortex (these parameters include the probability of deviant stimulus presentation, the time interval between stimuli, sound intensity etc.). Furthermore, our model could explain certain properties of the deviance detection found in cortex better than models that are based on synaptic depression only in the input connections. Our model shows that the enhancement of responses to unexpected stimuli may be a simple process, requiring only a simple connectivity rule and synaptic depression. On the other hand, it suggests that stimulus-specific adaptation is not easily obtained in a neural network, requiring rather tight control or regulation of physiological parameters. We provide testable predictions that differentiate our model from the said feedforward models. Given its agreement with experimental data and its advantage over feedforward models, our model is a useful starting point for studying the circuit mechanisms that underlie cortical responses to unexpected stimuli.
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