

Modeling the primary auditory cortex using dynamic synapses: Can synaptic plasticity explain the temporal tuning?

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Received 11 January 2006; received in revised form 20 March 2007; accepted 20 March 2007

Available online 24 March 2007

Abstract

The molecular mechanisms underlying the temporal plasticity (temporal tuning) of cortical cells remain controversial. Experimental observations indicate that the neuronal responses at the primary auditory cortex are affected by behavioral learning. In this paper, we present a minimal feed-forward model of the primary auditory cortex, based on the dynamic synapse and the leaky integrate-and-fire neuron models, in order to search for the origin of the observed plasticity. We demonstrate that the frequency response of the model is markedly modified by regulating the contribution of synaptic facilitation to the short-term dynamics of synapses (U_1). Consequently, we propose that the variation of this parameter may be responsible for primary auditory cortex enhancement achieved by behavioral training. Based on our model, we assume that the contribution of facilitation arises from the amount of Ca^{2+} influx each time an action potential arrives at the nerve terminal. Regardless of what really leads to the long-term variation of Ca^{2+} influx, we suggest that this process is responsible for the temporal tuning of responses observed in experimental studies. We believe that measurement of the long-term variation of Ca^{2+} influx at the pre-synaptic area of the cortical cells in auditory learning trials would be the first step to validate our hypothesis.

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Keywords: Primary auditory cortex; Synaptic plasticity; Modeling; Cortical temporal plasticity

1. Introduction

The primary auditory cortex (A1) has always been the focus of neurophysiologists' attention for its fundamental role in auditory signal processing. Recent findings suggest that it might serve as a general-purpose hub of the auditory pathway, forming a basis for the representation of the features of auditory signals (Griffiths et al., 2004). One of the most interesting functions observed in A1 is the temporal tuning (plasticity) of neural responses, which means that the characteristics of responses change with learning. For instance, Kilgard et al. (2001) have observed that auditory learning in adult rats leads to changes in the firing rate of cortical neurons in response to different frequencies of sound. As another example, an experiment

on the primary auditory cortex of rats being trained in a "sound maze" indicates that behavioral learning can affect the response magnitude of cortical neurons, without remarkably changing the latency (Bao et al., 2004). A more rapid plasticity in A1 cell responses has also been reported for domestic ferrets under behavioral training (Fritz et al., 2003).

Several phenomena are assumed to underlie the plasticity of cortical cell responses in A1, but there is no global agreement on the issue (Kilgard et al., 2001, 2002; Wehr and Zador, 2005). Kilgard and Merzenich (1998) have proposed that the alteration of synaptic time constants, intrinsic temporal characteristics or network structural transformation may account for the observed plasticity. Krukowski and Miller (2001) have suggested that the plasticity may arise from the variation of NMDA receptor conductances in cortical inhibitory–excitatory circuitry. In their model, the variation of conductances modifies the

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ration of excitation and inhibition in the entire circuitry, and provides a way to tune the frequency response of cells. Finally, Kilgard et al. (2002) have definitely enumerated three possible causes, including lowering or raising of spike thresholds, increased or decreased synaptic strength, and added or reduced number of neural connections; however, they have not evaluated their hypotheses experimentally.

Inspired by the efforts of Denham (2001) and Loebel and Tsodyks (2002) in using depressing synapses to model some aspects of the auditory cortical cells, we propose a minimal feed-forward model of the primary auditory cortex, comprised of excitatory dynamic synapses (Markram et al., 1998) and leaky integrate-and-fire (LIF) neurons (Izhikevich, 2005), in search for the origin of temporal plasticity in auditory cortical cells. More specifically, we exploit three LIF neurons at three layers: one to represent a pre-thalamic sensory neuron, the second to represent a thalamic cell, and the third to stand for a cortical neuron at the input layer (layer 4) of A1, which gets direct input from the thalamus. Thus, two excitatory dynamic synapses are utilized: one to represent a pre-thalamic sensory afferent, and the other to stand for a thalamocortical connection. The assumption of feed-forward (Suder et al., 2002; Reyes, 2003) and excitatory (Hu et al., 1994) structure for the modeled region of the auditory pathway seems biologically plausible.

We will show that the presented model is able to imitate the response properties of the A1 cells, obtained by Bao et al. (2004) in an experimental study. To this end, we will initially use the genetic algorithm in order to optimize some of the model parameters. We are obligated to apply this method since the real biological values of some parameters are not accessible. Afterwards, we will study if the plasticity of synaptic parameters (synaptic plasticity) could be responsible for the observed (Bao et al., 2004) temporal tuning in the model. This will be investigated over various aspects of the responses, including repetition rate transfer function (RRTF), response latency and single burst response magnitude. Based on the proposed model and the results, we will go for the possible biological origin(s) of temporal plasticity in the auditory cortex.

2. Methods

2.1. The stimulus

The stimulus signals consist of repetitive noise bursts, in accordance with Bao et al. (2004). Each noise burst is comprised of an ensemble of white Gaussian noise modulated by a pulse signal with 25 ms duration and 5 ms rise (fall) time. The power of the white noise is set to one, and the repetition rate of the bursts ranges from 2 to 20 pulses per second (pps).

We apply the generated stimulus signals to the first layer neuron of the model, as an excitatory post-synaptic current.

2.2. The model

The proposed model is comprised of a three-layer feed-forward network of neurons. Each layer contains one neuron in our attempt to simplify the model as much as possible. The neurons are connected to each other via dynamic synapses, as depicted in Fig. 1. The biological interpretation of the model structure is as follows: the first neuron represents a pre-thalamic sensory cell, the second one represents a thalamic neuron, and the third one stands for a cortical layer 4 cell. The “layer 4” neuron serves as the output of our model since the data captured by Bao et al. (2004) are mostly from this layer.

The input of the model is the post-synaptic current stimulating the first neuron, while the output is the sequence of action potentials generated by the third neuron.

2.2.1. The neuron model

The LIF model is utilized for simulating a neuron having ohmic leakage current, in which the membrane potential is obtained as the leaky integral of the post-synaptic currents due to the release of neurotransmitter vesicles. The statement can be formulated as (Izhikevich, 2005)

$$C \frac{dV(t)}{dt} = I(t) - g_{leak}(V(t) - E_{leak}), \quad (1)$$

where V is the membrane potential, I is the ionic current caused by neurotransmitter release, C is the mean membrane capacitance, g_{leak} is the mean conductance due to leakage, and E_{leak} is the minimum mean membrane potential at which the outward leakage current starts. When the membrane potential V reaches a threshold value, the neuron fires an action potential (spike) and V is reset to potassium equilibrium potential, E_K , which is typically less than E_{leak} . Right after the action potential, the neuron is in its refractory period, during which it is less excitable (Izhikevich, 2005). We have modeled this behavior by including a constant refractory period value for all neurons, during which they do not fire any action potentials.

2.2.2. The dynamic synapse model

The deterministic model of dynamic synapse, which encompasses short-term synaptic mechanisms, has been introduced and comprehensively discussed by Tsodyks et al. (1998). The model encompasses a variety of short-term mechanisms that regulate the synaptic strength. However, some simpler models have been proposed based on this model (Markram et al., 1998; Fuhrmann et al., 2002) in which there are two distinct short-term synaptic mechanisms, depression and facilitation, each of which is modeled by a first-order differential equation. In this study, we utilize the model introduced by Markram et al. (1998).

The depression phenomenon takes place since the readily releasable neurotransmitter pool declines following each

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