Modeling Habituation of Auditory Evoked Field using Neural Mass Model

Peng Wang & Thomas R. Knösche

Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany

pengwang@cbs.mpg.de

Introduction

In the auditory modality, repetitions of stimuli with short time interval usually lead to a decrease of the auditory evoked potentials/fields, in particularly its N100 component [1]. One generally accepted explanation of such stimulus specific short-term habituation is the decrease of the synaptic connectivity [2]. To investigate and mimic the underlying mechanism of this phenomenon: (1) we extended the biologically plausible neural mass model (NMM) of Jansen and Rit [3] with more realistic inter-laminar connections according to animal studies [4] to account for the basic dynamics of the auditory cortical response in source space; (2) we implemented a learning rule, which is based on the synaptic vesicle recycling process [5], to dynamically modify the excitatory connections among the subpopulations in order to explain the decrease and recovery of the auditory response as a function of the stimulus repetition; (3) we investigated three possible inter-laminar signal pathway using a Bayesian inference scheme [6].

Methods

Model

Model of a single cortical column/ neural source

Under the notion that a single dipole within the primary auditory cortex is able to account for the major component of the N100m peak, we use our cortical column model to mimic the dynamics of the auditory evoked fields. We pro-pose an extension to the Jansen & Rit model, comprising 5 neural masses and 11 synaptic connections (Fig. 1). The Jansen & Rit system comprises a differential operator for the temporal synaptic-dendritic dynamics receiving input from connected neural masses as well as externt sources through a sigmoid function.

$$\ddot{u} = \frac{H}{\tau}\left(\sum_i W_i C_i sigm(u_i) + Input\right) - \frac{2}{\tau}\dot{u} - \frac{1}{\tau^2}u$$

where u is the averaged membrane potential, H is the synaptic gain, τ is the synaptic time const. W as well as C control the connection strength between two neural populations (For more details about the parameter please see [3].)

Model of habituation

We hypothesize that the decrease of the N100 amplitude can be be explained by a reduction of the excitatory intra-connectivity to between the neuronal populations (pyramidal cells and excitatory interneurons to any other type), which is implemented in the following differential equation:

$$\begin{cases} \dot{W} = -n_1 W \dfrac{Q}{Q_{\max}} + n_2(1-W), & (Q \geq 0) \\ \dot{W} = n_2(1-W), & (Q < 0) \end{cases}$$

where $W(0 \leq W \leq 1)$ is the weight that scales the synaptic connection efficiency between two neuronal populations. Q is the pre-synaptic firing rate. $Q_{\max}$ denotes the maximally possible firing rating. n_1 and n_2 govern the habituation and the recovery rates. The first term of the differential equation describes the releasing of the synaptic vesicles from the vesicle pool as a function of the current activity of the neural subpopulations and causes the decrease of the connectivity due to the reduction of the vesicle release probability. The second term describes the recycling of the vesicles back to the pool and causes spontaneous recovery of the connectivity.

Model estimation

We propose three different hypotheses concerning the information pathways following the arrival of the bottom-up input at EINs in layer IV: (1) Model A: information follows a serial pathway, where the information first ascends from layer IV to the sPCs in layer II/III and then goes down to the dPCs in layer V; (2) Model B: information follows parallel pathways, it flows simultaneously from layer IV to both layer II/III and layer V; (3) Model C: both serial and parallel path-ways are present. The Data favored model is the model with the highest model evidence [7].

Data

Stimulation Six healthy human volunteers were stimulated by earphones with 160 trains of ten identical tones (900Hz, 15ms) each. The tones were separated by 500ms and the trains were separated by 10s. The participant was watching a silent movie during recording.

Data preparation Magnetoencephalographic (MEG) data were recorded at a sampling rate of 1000Hz using 306 MEG channels. 153 channels (51 magnetormeters, 102 planar gradiometers), which covered the right hemisphere, were used for fitting the single equivalent current dipole (ECD) model around the right auditory cortex. The data were averaged over trains offline (time window: -100ms to 2500ms, including five stimuli, first stimulus was pre-sented at the time point 0). A band pass filter (1 Hz to 20 Hz) was applied to reduce the noise. The ECD was esti-mated from the time window from 60ms to 130ms.

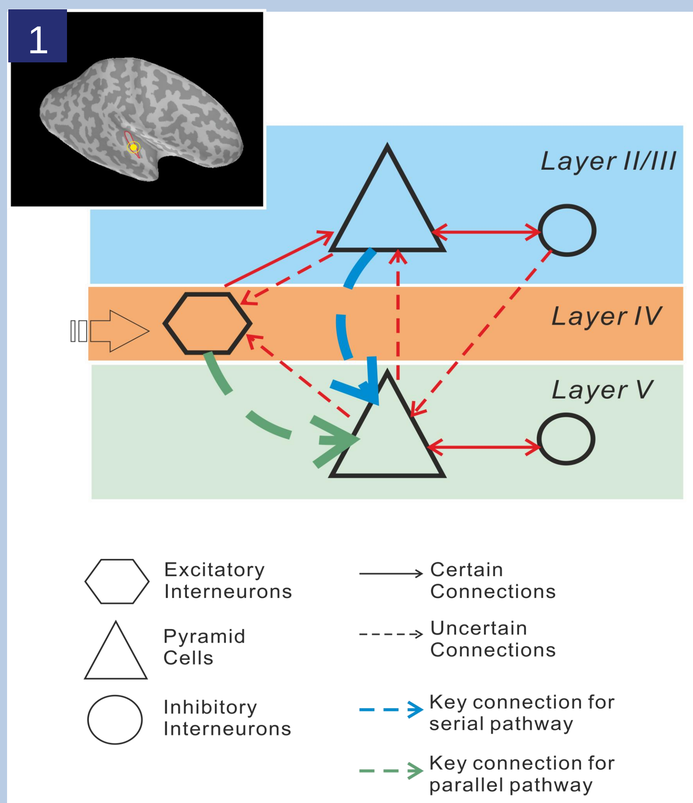

Figure 1. The extended neural mass model was constructed with 5 neural populations and 11 synaptic connections. The neural populations account for the excitatory interneurons, mainly spiny stellates in layer IV, superficial pyramidal cells in supragranular layers II/III, deep pyramidal cells in infragranular layer V, as well as two for the supragranular and infragranular inhibitory inter-neurons . The synaptic connections are motivated by the in vivo animal study [4] and divided into two subgroups. The certain connections are as-signed with non-zero priors and the uncertain connections are assigned with zero priors in the Bayesian inference scheme [6]. The model output, which was the superposition of the average depolarization of the two pyramid cells populations, was assumed to proportional to the dipole dynamics.

Results

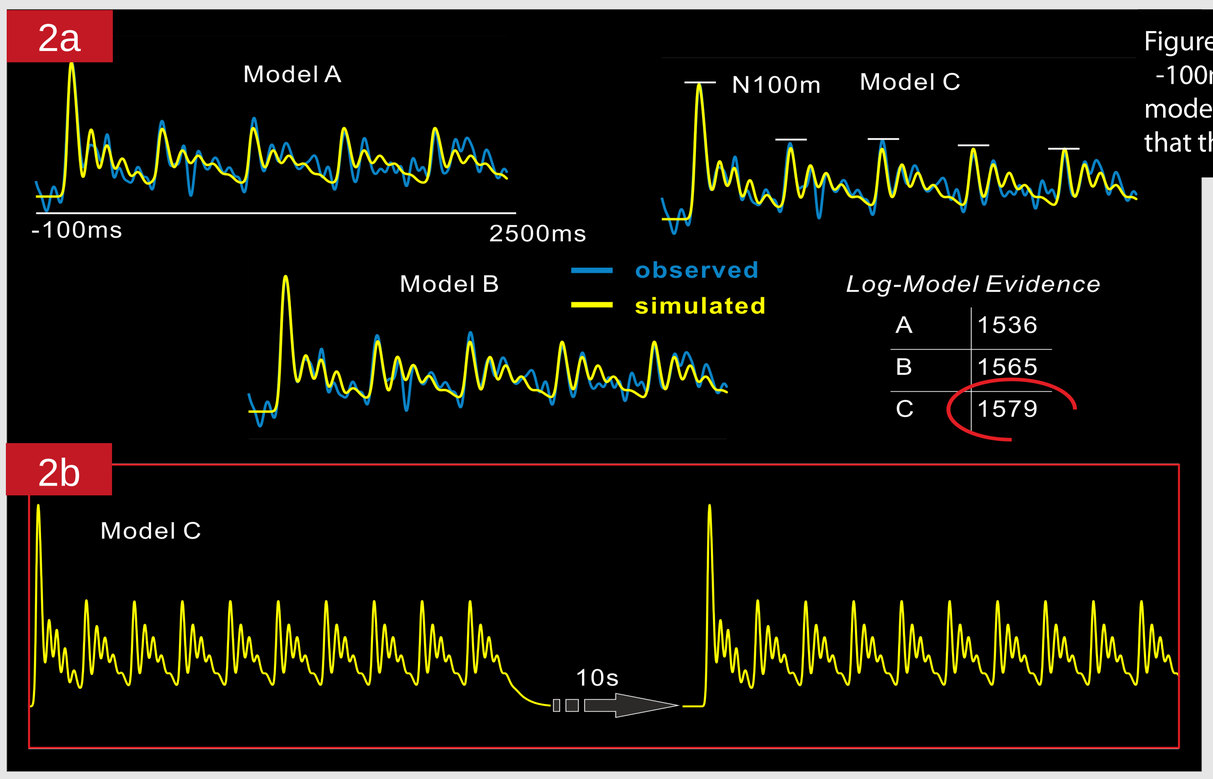

Figure 2. Observed and simulated auditory evoked fields in source space (subj. 2). (a) Data epoch from -100ms to 2500ms of three different models: A (serial pathway), B (parallel pathway) and C (both). The model evidence indicate that the data favour Model C. (b) Simulated data using model C to demonstrate that the N100m peak recovers during the 10s stimulus free time and the habituation cycle anew.

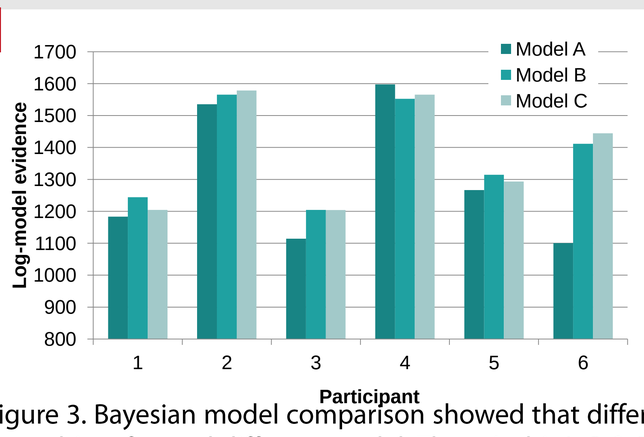

Figure 3. Bayesian model comparison showed that different subject favored different model. Three (subj. 1, 3 & 5) suggested Model B (parallel pathway), two (subj. 2 & 6) supported Model C (both serial and parallel pathway) and one(subj. 4) suggested Model A.

Our models yielded a reasonable fit of the data (suppression of the N100m peak) (Fig. 2a) and also showed the features of the recovery at the absence of the stimuli (Fig. 2b). We analyzed the possible inter-laminar signal pathways based on model comparison at the within-subject level (Fig. 3). A difference in log-model evidence of three is considered strong evidence in favour of the more likely model. The best models of each subject, given the data, seemed to be not uniform, however, except for one subject, the parallel pathway was necessary.

Discussion & Conclution

We present a mathematically simple but biologically plausible model to mimic the synaptic plasticity in terms of the stimulus specific adaptation and recovery in a neural mass aspect. Furthermore, we studied the intra-laminar signal pathway with the non-invasive measurement method. Note that our model-based analysis is strongly dependent on (i) the a priori knowledge of the local wiring in a cortical column, which is obtained by in vivo animal studies as well as (ii) the given experimental data. In other words, it only suggests the most "optimized" result among all given hypotheses.

This study demonstrates that NMMs can be specifically modified to reproduce important features of MEG signals, which represent cognitive processes. The advantage of using the modeling technique in MEG analysis is its ability to pinpoint a specific neural network structure or a neuronal mechanism underlying normal or pathological cognitive processes in the human brain.

Reference

1. Rosburg T, Trautner P, Boutros NN, Korzyukov OA, Schaller C, Elger CE, Kurthen M (2006), '*Habituation of auditory evoked potentials in intracranial and extracranial recordings*', Psychophysiology, 432:137-144.
2. Bear MF, Connors BW, Paradiso MA (2007), '*Neuroscience: Exploring the brain*', Lippincott Williams & Wilkins.
3. Jansen, B. H., G. Zouridakis, et al. (1993). '*A neurophysiologically-based mathematical model of flash visual evoked potentials*', Biological Cybernetics, 68: 275-283.
4. Thomson, A. M., D. C. West, et al. (2002). '*Synaptic connections and small circuits involving excitatory and inhibitory neurons in layers 2–5 of adult rat and cat neocortex: triple intracellular recordings and biocytin labelling in vitro*' , Cerebral cortex, 12: 936.
5. Sara Y., Mozhayeva M.G., Liu X., Kavalali E.T. (2002), '*Fast vesicle recycling supports neurotransmission during sustained stimulation at hippocampal synapses*', The Journal of neuroscience, 22: 1608-1617.
6. Friston K, Glaser D, Henson R, Kiebel S, Phillips C, Ashburner J (2002), '*Classical and Bayesian inference in neuroimaging: applications*', Neuroimage , 16:484-512.
7. Penny, W., K. Stephan, et al. (2004). '*Comparing dynamic causal models*', NeuroImage,22: 1157-1172.