

Analytical solutions of a non-linear model of information processing in auditory cortex

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Introduction

- The auditory cortex (AC) creates representations of sound sequences by adjusting its dynamics for processing incoming stimuli according to stimulus history. This process is termed temporal binding.
- Previous studies have suggested that the underlying mechanism of temporal binding is short-term synaptic depression (STSD) which can be summarized in one central parameter τ_{rec} , the time constant of recovery from synaptic depression.
- We want to investigate how τ_{rec} might be reflected in classical N1m adaptation phenomena and temporal binding, with a view of exploring whether adaptation could predict temporal binding from the perspective of dynamical neural networks.

Computational model

Model of information processing in AC based on STSD (May et al. 2010, 2013, 2015)

Structure

• Hierarchical organization of monkey AC: 3 core, 8 belt, 2 parabelt fields (Hackett et al., 1998; Kaas & Hackett, 2000). **Serial** feedforward activation from core to belt and to parabelt fields along many parallel routes.



• Fitting the model to experimental data was done by trial and error, which is very slow and sparse. Thus, mostly qualitative simulations were performed so far.

- We aim for an approximated version of the original non-linear model (linearized/ simplified) which retains relevant features (network structure, presynaptic adaptation).
- By means of analytical solutions of the model, we aim to vastly improve fitting the model to experimental data (MEG on single-cell humans, observations on monkeys), and, thus, obtain e.g. more detailed and subject-specific information on auditory processing.

Ultimate Aim

Linearization of firing rate	Approximation of adaptation
Fit to real data	Analytical solution
Model parameters	Implementation in the nonlinear model

• Each field consists of n columns.

Computational Unit: The Column

- A column is a complex, local collection of neurons with similar response properties.
- We model the column in the simplest possible way: Each column consists of an excitatory (e) and an inhibitory (i) cell population each described by one state variable.
- Connections within and between columns are expressed by W_{ee} , W_{ei} , and W_{ie} .



Oberlaender et al. (2012) Cereb. Cortex



Dynamics

Coupled differential equations describing neural interactions: leaky integrator neuron plus presynaptic depression

Our approach

We first linearize the spiking rate and assume that excitatory to excitatory connections (W_{ee}) are symmetric, and all other connection matrices (W_{ei}, W_{ie}, W_{ii}) are diagonal. We can find analytical solutions as a mixture of damped harmonic oscillators (normal modes).

$$\tau_m \Upsilon^{-1} \mathbf{u}(t) = -\Upsilon^{-1} \mathbf{u}(t) + W_{eed} \Upsilon^{-1} \mathbf{u}(t) - W_{eid} \Upsilon^{-1} \mathbf{v}(t) + \Upsilon^{-1} \mathbf{I}_{aff}$$
$$\tau_m \Upsilon^{-1} \dot{\mathbf{v}}(t) = W_{ied} \Upsilon^{-1} \mathbf{u}(t) - \Upsilon^{-1} \mathbf{v}(t)$$
$$u_{d}(t) = \exp(-\gamma_{d} t) (a_{u_{d}} \sin(\delta_{d} t) + b_{u_{d}} \cos(\delta_{d} t)) + f_{u_{d}}$$
$$v_{d}(t) = \exp(-\gamma_{d} t) (a_{v_{d}} \sin(\delta_{d} t) + b_{v_{d}} \cos(\delta_{d} t)) + f_{v_{d}}$$

Presynaptic adaptation A: release of neurotransmitters is dependent only on the excitatory activity of preceding column, i.e. A is a matrix with identical rows.





Thus, excitatory connection strengths become asymmetric, but we still can solve the state equations analytically, because for the eigenvalues the following equality holds: $1 \neq (\Pi I I I I = (1)) = 1 \neq (1 \neq (1))$ 1.

$$\det \left(W_{ee} \operatorname{diag}(\mathbf{a}) - \lambda I \right) = \det \left(\sqrt{\operatorname{diag}(\mathbf{a})} W_{ee} \sqrt{\operatorname{diag}(\mathbf{a})} - \lambda I \right)$$

Connections



Adaptation has a fast onset and a slow recovery phase. We approximate it with an infinitely fast decline of the connection strength and a slow exponential recovery.

$$\frac{da_j(t)}{dt} = -\frac{1}{\tau_o}a_j(t)g[u_j(t)] + \frac{1 - a_j(t)}{\tau_a} \longrightarrow \frac{da_j(t)}{dt} = -\frac{d_j}{\tau_o}\delta(t - t_{stim}) + \frac{1 - a_j(t)}{\tau_a}$$



$$\dot{\mathbf{A}}_{ij}(t) = -\frac{\mathbf{A}_{ij}(t)g[\mathbf{u}_j(t)]}{\tau_{\text{on}}} + \frac{1 - \mathbf{A}_{ij}(t)}{\tau_{\text{rec}}}$$
$$g(u) = tanh(\frac{2}{3}(u - \theta))$$

- state variable of excitatory cell population state variable of inhibitory cell population membrane time constant spiking rate afferent input from auditory pathway $\widetilde{W}_{ee} W_{ie} W_{ei}$ connection matrices presynaptic depression onset and recovery time constant $au_{
 m on}$, $au_{
 m rec}$ threshold
- Experimental paradigm

Standard paradigm to study adaptation: Regular-SOI experiment

• Stimuli are presented at regular stimulus-onset intervals (SOIs) in different blocks and SOI is varied across blocks.

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- Averaging single-trial responses per block produces mean response per SOI.
- Dependence of mean peak amplitude of the AC response on SOI can be described with an exponentially saturating function with adaptation time constant τ_{SOL}





We assume that the signal decline d_i spreads infinitely fast through the network. We approximate this spread by using the non-symmetric equivalent resistance (conductance) matrix approach (resistances with voltage controlled current sources). Conductances⁻¹





- We approximate the original non-linear model while keeping the basic concepts: network structure, oscillatory behavior, presynaptic adaptation.
- The resulting analytical solutions show a remarkable similarity in excitation and adaptation as observed in the non-linear model.
- Are there other possibilities to improve this approximation further?

References



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