

Poster presentation

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## Investigating the interaction of transcranial magnetic stimulation with a model cortical neuron

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### Introduction

Transcranial magnetic stimulation (TMS) is a noninvasive technique that induces neuronal discharge in response to a rapidly changing magnetic field (B-field) directed through the scalp. However, the interaction between cortical tissue and the electric field (E-field) induced by the changing B-field remains unclear. A realistic multi-compartment model of a layer V pyramidal neuron receiving a simulated TMS pulse provides a means to characterize the influence of numerous parameters on cell discharge.

### Background

Surface electromyography (sEMG) is widely used to measure the electrophysiological response of muscle to TMS. In the presence of volitional motor activity, a TMS pulse delivered to a targeted brain region evokes an sEMG waveform that sequentially depicts an onset latency, a multiphasic spike, and a refractory period referred to as the silent period (SP). The relationship between the activity of individual cortical neurons and the peripherally recorded SP is currently undefined, but has been explored [1,2].

### Model and methods

We replicated a compartmental layer V neuron model and delivery of simulated TMS as described elsewhere [2,3]. Computer simulation was used to predict both the discharge response of the cortical neuron to different E-field magnitudes and the duration of the subsequent SP. To better understand the role of the TMS induced E-field, we replaced the stimulus described in [2] with a representation of E-field measurements obtained within our lab [4].

### Results

Simulations utilizing the stimulus set forth in [2] verified that the SP duration increased with stimulus strength [5], where the respective durations were most sensitive to alterations in calcium-dependent potassium conductance [3]. Simulations utilizing the pulse reported in [4] produced similar effects, however, SP duration showed decreased dependence on stimulus strength.

### Conclusion

With respect to SP duration, the computational method for modeling TMS first proposed by Kamitani et al is in agreement with sEMG data from pilot studies within our lab and can possibly be attributed to intracellular calcium dynamics [1,2]. Results obtained following the incorporation of [4] suggest that the silent period observed with sEMG may not be a single neuron phenomenon, but possibly a population response as described in [6].

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