

Effect Of A 1000 μ T, 60 Hz Magnetic Field On Spike Timing In Cortical Neurons: A Modeling Study

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INTRODUCTION

There is experimental evidence that extremely low-frequency (ELF) electromagnetic fields (EMF) affect human neurophysiological measurements such as electroencephalography (EEG) recordings (see [1] for a review). In computational neurosciences, mathematical models of brain structures have been developed and validated, offering original tools to study how an ELF EMF signal may interact with the human brain. As a first step, we present a computational model of a cortical neuron exposed to an external magnetic field (MF) to obtain a quantitative estimate of MF exposure on single neuron dynamics. Hence, we study a spiking neuron model subject to a 1000 μ T, 60 Hz magnetic field exposure, resulting in a maximal membrane depolarization of 233 μ V.

MATERIALS AND METHODS

MF exposure induces an electric field $E(t)$ in brain tissue, resulting in membrane depolarization [2] depending on polarization length λ_p [3] and cell orientation θ with respect to $E(t)$ [2]. The membrane depolarization during exposure to an AC field of pulsation ω is $\delta V(t) = \pi R B_0 / \lambda_p \cos \theta [\cos(\omega t) + \omega \tau \sin(\omega t)] / (1 + \omega^2 \tau^2)$ where τ is the Maxwell-Wagner time constant and R the exposure radius. This term is included into the Izhikevich model (detailed in [4]) such as $V(t) = v(t) + \delta V(t)$. We simulated 10 s of exposure of a single cell driven by a current $I = 5$ pA to a 60 Hz MF of amplitude $B_0 = 1000$ μ T (giving $E(t) = \pi R / B_0 \cos(\omega t)$). Model equations were solved using a 4th-order Runge-Kutta method (time step $\Delta t = 10$ μ s, a time step of $\Delta t = 1$ μ s was also used to check that results were unchanged).

RESULTS

We computed the membrane potential of a spiking neuron in regular spiking (RS) and bursting (CH) modes [4] with and without 60 Hz MF exposure during 10 s. Spike times are impacted as shown in Figure 1, depending on the relative phase between stimulus and neuronal oscillation. Indeed, membrane responsiveness depends on the oscillation phase, since membrane dynamics is described by nonlinear equations [4]. Furthermore, our results indicate that CH neurons spike times are more impacted than RS neurons (roughly by a factor 3), suggesting that bursting neurons are more sensitive to MF exposure.

DISCUSSION

We show that periodic, sub-millivolt membrane depolarization may delay or advance spike-timing, consistent with previous experimental results [5]. This is of potential importance, since spike times shape synaptic weights modulation via spike-timing dependent plasticity [6], and thus network activity. Consequently, it is plausible that long-term MF

exposure induces detectable changes in synaptic weights. Therefore, it would be useful to investigate how these spike-timing modulations translate into network activity modulation via synaptic weights reshaping.

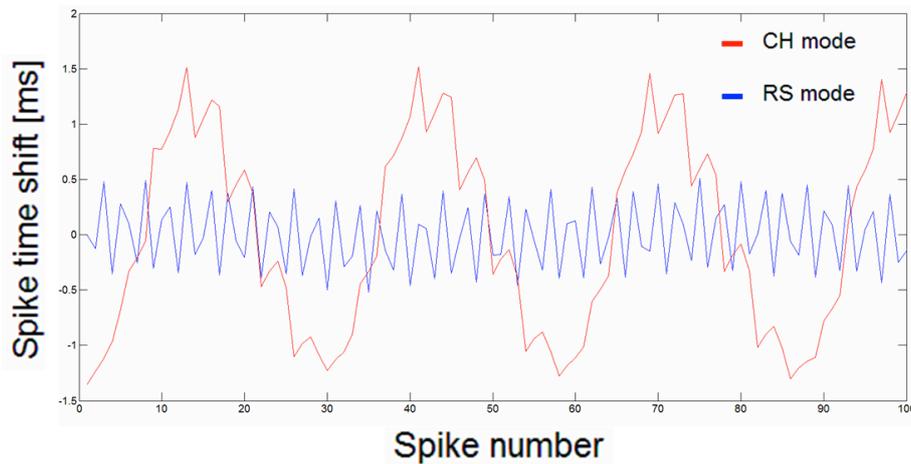


Figure 1: Field-induced shift of spike times for the first 100 spikes of a cortical neuron model in two spiking patterns: RS (blue curve) and CH (red curve), during MF exposure ($\theta=1$, $B_0=1000 \mu\text{T}$, $f=60 \text{ Hz}$, $R=5 \text{ cm}$, $\tau=20 \text{ ms}$, $\lambda_p=500 \mu\text{m}$ [3]).

CONCLUSIONS

In the limit of the biophysical mechanisms included in the model, we suggest that a $1000 \mu\text{T}$ MF at 60 Hz does not induce, but modulate neural activity by shifting spike timing, depending on the relative phase between the MF and cell oscillation, and the spiking pattern (regular or bursting). Such brain oscillations modulation (by transiently increasing/decreasing the period of single cell oscillations) may explain partly EEG changes during ELF EMF exposure [1]. Further work at the neural network level is needed to verify this possibility.

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