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# The effects of static magnetic field on action potential propagation and excitation recovery in nerve ☆

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**Abstract**

Calculations using the Hodgkin–Huxley and one-dimensional cable equations have been performed to determine the expected sensitivity of conduction and refractoriness to changes in the time constant of sodium channel deactivation at negative potentials, as reported experimentally by Rosen (Bioelectromagnetics 24 (2003) 517) when voltage-gated sodium channels are exposed to a 125 mT static magnetic field. The predicted changes in speed of conduction and refractory period are very small.


*Keywords:* Action potential; Nerve conduction; Magnetic field; Refractory period; Sodium channel kinetics

## 1. Introduction

The preceding article (Saunders, 2004) describes a paradox in relation to nerve conduction and excitation. This is that Rosen (2003) has reported an effect of static magnetic fields on voltage-gated sodium channel kinetics, whereas work by Gaffey and Tenforde (1983) and others (see Saunders, 2004, for further references) show no effect on either conduction velocity or on

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refractory period. In principle, changes in ion channel kinetics should be reflected in changes in excitation, conduction and recovery.

A possible explanation for the paradox is that since the largest effect reported by Rosen is an increase in the sodium channel activation ($m$) time constant, $\tau_m$, at negative potentials only, the results might be explained by a reduction of the deactivation rate function $\beta_m$, with little or no effect on the other rate functions of sodium channel activation and inactivation, i.e., $\alpha_m$, $\alpha_h$ and $\beta_h$.

Conduction velocity must be strongly dependent on $\alpha_m$ but may be only weakly dependent on $\beta_m$. Most of the early depolarization, at potentials where $\beta_m$ would be significant, is a passive spread of current from already excited regions (Jack et al., 1975, Figure 10.2), and the later stages of depolarization take place at potentials at which $\beta_m$ is negligible. Moreover, even during the early stage, the back reaction, determined by $\beta_m$, will be small because $m$ is small.

In this article we describe computations and analyses to test this explanation quantitatively. We encountered a technical difficulty, which is that in the Hodgkin–Huxley formulation of channel kinetics, any change in one or both of the rate functions automatically changes the voltage-dependence of the steady-state activation curve. Thus, decreasing $\beta_m$ to achieve an increase in $\tau_m = 1/(\alpha_m + \beta_m)$ automatically increases the steady-state value of $m$, namely $m_\infty(V) = \alpha_m/(\alpha_m + \beta_m)$, at negative potentials. Since the sodium channel current is very large compared to the ionic currents at rest, even a relatively small increase at the foot of the activation curve (which may not be detectable experimentally) will disturb the resting potential. We have used two methods to avoid this. The first (in Section 3) involves keeping the activation curve constant and using curves fitted to the voltage dependence of $\tau_m$ to compute the rate at which $m$ approaches its steady-state value. The second (in Sections 4 and 5) involves adjusting the leakage current, within experimental limits, to keep the resting potential constant.

## 2. Summary of changes due to magnetic field

In this section we review the experimental changes in the sodium channel kinetics due to the application of a magnetic field (Rosen, 2003). The (TTX-sensitive) sodium channel current was recorded in GH3 cells using the whole cell patch clamp method. The cells were exposed to a 125 mT magnetic field for 150 s and the sodium currents were recorded before, during and after the field was applied. Experiments were carried out at a range of temperatures from 25 to 37 °C, although significant (and large) changes were only observed at 35 and 37 °C. This strong temperature dependence of the effect of the magnetic field is similar to the highly non-linear dependence of $\tau_m$ on temperature (Rosen, 2001). The following results are from recordings in six cells at 35 and 37 °C.

1. When the field was applied, there was a 'slight shift' in the current–voltage relationship and less than a 5% reduction in the peak current. These changes were transient and reversed within 100 s of the field being turned off. Since these changes were small, we shall exclude them from our model.
2. The activation time constant $\tau_m$ changed by a significant amount at low potentials when the field was applied, and these changes persisted for at least l00 s after the field was removed. Rosen's results are re-plotted in Fig. 1.
3. There was no significant change in the inactivation time constant $\tau_h$ when the field was applied.