The Lorentz force on ions in membrane channels of neurons as a mechanism for transcranial static magnetic stimulation

Manuel J. Freire, Joaquín Bernal-Méndez, and Alberto T. Pérez

ABSTRACT
Transcranial static magnetic stimulation is a novel noninvasive method of reduction of the cortical excitability in certain neurological diseases that makes use of static magnetic fields generated by permanent magnets. By contrast, ordinary transcranial magnetic stimulation makes use of pulsed magnetic fields generated by strong currents. Whereas the physical principle underlying ordinary transcranial magnetic stimulation is well known, that is, the Faraday’s law, the physical mechanism that explains the interaction between neurons and static magnetic fields in transcranial static magnetic stimulation remains unclear. In the present work, it is discussed the possibility that this mechanism might be the Lorentz force exerted on the ions flowing along the membrane channels of neurons. The overall effect of the static magnetic field would be to introduce an additional friction between the ions and the walls of the membrane channels, thus reducing its conductance. Calculations performed by using a Hodgkin–Huxley model demonstrate that even a slight reduction of the conductance of the membrane channels can lead to the suppression of the action potential, thus inhibiting neuronal activity.

Introduction
Transcranial magnetic stimulation (TMS) is a well-established noninvasive method of brain stimulation for diagnosis and treatment of neurological diseases that it is based on the application of strong and short pulses of magnetic field (typically 1 T of amplitude and 300 μs of duration) generated by current-fed coils (Barker et al. 1985). The physics underlying TMS is well known and it is based on the induction of currents in neurons by virtue of the Faraday’s law. Protocols for TMS therapy are well established, being the theta-burst protocol the most extended to induce long-lasting neural changes (Gutiérrez-Muto et al. 2020). Transcranial static magnetic stimulation (tSMS) is a novel noninvasive form of brain stimulation, that makes use of static magnetic fields (SMFs) created by permanent magnets to reduce cortical excitability in humans (Oliviero et al. 2011)(Silbert et al. 2013)(Arias et al. 2017). Thus, tSMS can produce long-lasting effects that can modulate motor cortex excitability in patients with Parkinson’s disease (Dileone et al. 2017). It must be noted that a new transcranial stimulation technique, referred to as magneto-acoustical stimulation, also makes use of a SMF but in combination with an ultrasonic wave to generate an electric current in an area of interest in the brain (Yuan et al. 2016b) (Yuan et al. 2016a). Experimental evidences show that SMFs of moderate values (tens to hundreds of mT) can interfere with physiological brain functions (Oliviero et al. 2011) (Silbert et al. 2013)(Arias et al. 2017). There is also experimental evidence of effects produced by even greater SMFs in Magnetic Field Resonance (MRI) exams (Nojima et al. 2015)(Roberts et al. 2011). Moreover, the interaction of moderate SMFs with excitable membranes of different biological systems has been extensively reported (Rosen, 2003a)(Rosen, 2003b) (McLean et al. 1995)(Coots et al. 2004)(Ye et al. 2004). Despite these evidences, a physical mechanism accounting for the interaction of moderate SMFs with neurons has not been identified yet. A better understanding of the physical phenomena underlying this interaction would help to fine tune the treatment in tSMS.

At a fundamental level, two kinds of physical mechanisms seem to be feasible candidates to provide an explanation: the magnetic behaviour of the constituent molecules of excitable membranes in the presence of a SMF, and the interaction between a SMF and moving ions in neurons through the Lorentz force. Within the first perspective, it has been suggested that the reorientation of diamagnetic anisotropic molecules in the cell membrane can be responsible for the influence of
moderate SMF on the cell membrane (Rosen, 2003b). The second hypothesis has been used to investigate, from a theoretical point of view, the influence of SMFs on the ion current that flows along the axon and is associated with the propagation of the action potential (AP) in nerves (Wikswo and Barach 1980) (Jamasb 2017). From the analysis carried out in (Wikswow and Barach 1980) (Jamash 2017), it follows that the Lorentz force exerted by moderate SMFs on the ions flowing along nerves cannot appreciably affect the propagation of the AP. Nevertheless, the AP is associated not only with the ion flux along axons but also with the ion flux along membrane channels. Regarding this, it is interesting to note that it has been suggested that ion channels of neurons can be modelled as FET transistors (Bezanilla 2005). Also, it is well known that SMFs can affect the performance of FET transistors in MRI preamplifiers due to the Lorentz force in charge carriers (Possanzini and Boutinele 2008). Thus, in the present work, it is discussed the possibility that the AP can be affected by moderate SMF through the Lorentz force exerted on the ions flowing along the membrane channels in neurons. To support this hypothesis, a dimensional analysis is carried out to estimate the ratio between the Larmor radius of the ions in the presence of a SMF with a value typical of the TSMS (Oliviero et al. 2011), and the dimensions of the cross section of human axons and membrane channels. Based on this analysis, it is suggested that, although moderate SMFs cannot affect the ion flux through axons, it may affect the ion flux along membrane channels. It is also suggested that the effect of the Lorentz force is to introduce an additional friction between the ions and the walls of the membrane channels. Since the conventional friction between the ions and the walls accounts for almost 2/3 of the conductance value of the channels (Chung et al. 1999), we conclude that the ultimate effect of the Lorentz force is to reduce significantly the conductance of the membrane channels in neurons through an additional friction of the ions with the walls of the membrane channels. Results for the AP obtained with a Hodgkin-Huxley (HH) model (Touretzy et al. 2013) reveal that a slight reduction of the conductance of the sodium (Na⁺) channel can lead to the suppression of the AP.

This document is organized as follows. The next section presents an analysis that rules out the effect of Lorentz force associated with moderate SMFs on ions flowing along axons as a cause of neuron inhibition. Also, the comparison between the Larmor radius, and the size of the region of conduction is presented as a suitable benchmark to determine whether Lorentz force can alter the flow of ions. This criterion is employed in the subsequent Results and Discussion section to show that membrane channels might see its conductance decreased by a Lorentz force such as that created by a moderate SMF, and that the expected decrease can actually suppress the AP. Finally, conclusions are presented in the last section.

Analysis

As it is well known, the Lorentz force is the force exerted on a charged particle moving in the presence of a SMF. Because this force is perpendicular to both the velocity of the particle and the direction of the SMF, it makes the particle to describe a circular trajectory in a plane perpendicular to the SMF. The radius of this trajectory is referred to as the cyclotron radius or Larmor radius, \( R_L \), and it is given by \( R_L = \frac{mv}{qB} \), where \( m \), \( v \) and \( q \) are the mass, velocity and charge of the particle, respectively, and \( B \) is the amplitude of the SMF.

The AP propagating through the axon of neurons is associated with a longitudinal ion current flowing along the axon. In the presence of a SMF, due to the Lorentz force, the ions flowing along the axon undergo a deflection of their trajectory which produces a transverse current. In (Wikswow and Barach 1980) it is theoretically analyzed for the first time the order of magnitude of the SMF necessary to produce an appreciable deflection in the longitudinal current associated with the propagation of the AP in the axons of human neurons. The calculations in (Wikswow and Barach 1980) show that a magnetic field on the order of 25 T is necessary to produce a deflection or reduction of 10% in the ion current along the axon. Such a field is several orders of magnitude greater than a moderate SMF such as that employed in TSMS, and it is even an order of magnitude greater than typical SMF in MRI systems. Moreover, in (Jamasb 2017) a deeper analysis estimates the effect of this deflection in the AP by means of a HH model where a term that accounts for the transverse current that appears as a consequence of the deflection is added in the differential equations, this term being proportional to the value of the SMF. In (Jamash 2017) it is defined a ratio \( \alpha \) between the transverse current and the longitudinal current, and it is expressed as a relation between the value of the SMF, \( B \), and the transverse mobility of the ions, \( \mu \), as \( B = \alpha/\mu \). The calculations in (Jamash 2017) show that, in particular, a moderate value for the SMF of \( B = 11 \) mT will produce a reduction of 5% (corresponding to \( \alpha = 0.05 \)) in (Jamash 2017) in the longitudinal current in the axon. In (Jamash 2017) it is shown that, taking this into account in the HH model, this will cause a suppression of the AP. This result obtained for a moderate value of a SMF (11 mT) entirely disagrees
with the conclusion in (Wikswo and Barach 1980), which shows that a very high value for the SMF (25 T) is required. This apparent paradox can be solved by noting that the analysis carried out in (Jamasb 2017) assumes an ion mobility of 5 m²/Vs, which is three orders of magnitude larger than values experimentally reported (Wikswo and Barach 1980). For example, in (Wikswo and Barach 1980) the peak axial electric field during the passage of the AP is reported to be $E = 8$ V/m and the ion velocity $v_d = 3.3 \times 10^{-2}$ m/s. Therefore, the ion mobility is $\mu = v_d/E = 0.004125$ m²/Vs. Assuming this much more realistic value for $\mu$, the required SMF for a reduction of 5% in the longitudinal current in (Jamasb 2017) will be 14.7 T, which is closer to the order of magnitude of the 25 T value estimated in (Wikswo and Barach 1980).

From the above discussion, it can be concluded that moderate SMFs cannot affect the propagation of the AP in human axons. This same conclusion can also be drawn from a simpler alternative analysis based on the comparison of the Larmor radius with the diameter of the axons and the Larmor frequency with the collision frequency. Consider, for example, a Na⁺ ion, whose mass and charge are: $m = 3.8 \times 10^{-26}$ kg and $q = 1.67 \times 10^{-19}$ C. To estimate the Larmor radius we can assume an ion velocity in the axon of $v_d = 3.3 \times 10^{-2}$ m/s (i.e., the same value as in (Wikswo and Barach 1980)) and a SMF of value $B = 164$ mT. This is the field value measured by the authors for the same magnet used in tSMS in Oliviero et al. 2011, at a distance of 2 cm from the surface of the magnet, which is the distance between the scalp and the motor cortex. With those assumptions, the Larmor radius is $R_L = mv/qB = 48$ nm. This is two orders of magnitude smaller than the typical diameter of the myelinated human axon which is 1 μm in average (Debanne et al. 2011). Moreover, the frequency of collision, defined as $v_c = qE/mv_d$ (Reif 2008), is $v_c = 10^9$ Hz for the values of $E$ and $v_d$ given above, and the Larmor frequency is $\omega_L = eB/m = 7 \cdot 10^5$ Hz. Since Larmor frequency is much lower than the frequency of collision, it can be concluded that the ion movement is ruled by collisions of the ions with other species and that due to the different orders of magnitude of the Larmor radius and the cross section of the axon, the interaction of the ions with the walls of the axon can be neglected. Similar calculations for the potassium (K⁺) ion, with a mass $m = 6.49 \times 10^{-26}$ kg, yield the following results: $R_L = mv/qB = 82$ nm, $\omega_L = eB/m = 4 \cdot 10^5$, $v_c = qE/mv_d = 0.6 \cdot 10^9$ Hz. From these results, it follows the same conclusion as for the Na⁺ ion. Therefore, moderate SMFs cannot affect the transport of ions through the axon, in accordance with (Wikswo and Barach 1980). In the next section, it will be shown that collisions of the ions with the walls of the ion channel cannot be neglected and that actually play a key role.

The discussion presented above suggests that the comparison between the Larmor radius and the dimensions of the axon and between the Larmor frequency and the frequency of collision can be considered as a benchmark to ascertain whether the Lorentz force associated with a given value of SMF affects the ion nerve conduction. In fact, we have just shown that this criterion allows to rule out Lorentz force due to a moderate SMF as the cause of the AP suppression in axons. In view of this, in this work, we propose the effect of Lorentz force on the conductance of membrane channels as an alternative explanation for the effect on the AP of a moderate SMF. To underpin this hypothesis, we will use the benchmark index described above to determine whether a moderate SMF can affect the ion flux along membrane channels. In this regard, a key point to be taken into account is that the size of the cross section of ion channels of excitable membranes is several orders of magnitude smaller than the diameter of the axon.

Results and discussion

In this section, a dimensional analysis is carried out to compare the size of the K⁺ and the Na⁺ channels with the Larmor radius of these ions for moderate SMFs. As a first step, this requires to perform an estimation of the drift velocity of the ions through the channel. Regarding this point, it is important first to determine whether the flow of the ions through the channel can be considered an Ohmic process (or ions should be considered ballistic charges instead). Scientific evidence points out that friction caused by the pore shape and wall tortuosity play an important role in the conductance (Naranjo et al. 2016) (Wallace and Pohorille 2011). Therefore, it is reasonable to consider the flow of ions through the channel as an Ohmic process dominated by the collisions of the ions with the walls, which acts as a friction force that prevents ions from being uniformly accelerated by the transmembrane electric field. Under this assumption, the amplitude of the current can be written as $I = J \cdot S$, where $S$ is the average cross section of the channel and $J$ is the current density, which can be written as $J = qn \nu_d$. In this expression $n$ is the number of ions per unit volume and $\nu_d$ the drift velocity of ions. The number of ions per unit volume can be written as $n = N/V$, where $N$ is the number of ions that can occupy simultaneously the channel and $V$ is the volume of the channel, that can in turn be expressed as $V = SL$, where $L$ is the length of the channel. Therefore, the drift velocity can be expressed as

\[ \nu_d = \frac{qN}{m} \left( 1 - \frac{B^2}{2} \frac{L}{R_L} \right) \]
The K⁺ channel extends 45Å, with a wide segment of length 23 Å and a narrower selectivity filter of radius 1.5 Å and length 12 Å where the ions would have to shed its hydrating waters to enter (Chung et al. 1999) (Doyle et al. 1998). The selectivity filter contains two K⁺ ions (Naranjo et al. 2016) (Doyle et al. 1998), that is, the number of ions that can occupy simultaneously the selectivity filter is \( N = 2 \). Since the amplitude of the current is of the order of picoamperes (Chung et al. 1999), assuming \( I = 1 \text{pA} \) and \( L = 12 \text{Å} \), \( v_d \) can be estimated from (1) as \( v_d = 3.75 \times 10^{-3} \text{ m/s} \). From this estimation of the drift velocity, and taking into account that the mass of K⁺ ion \( m = 6.49 \times 10^{-26} \text{ kg} \), the corresponding Larmor radius for a SMF of value \( B = 164 \text{ mT} \) can be calculated as \( R_L = \frac{mv_d}{eB} = 93 \text{Å} \). Note that this Larmor radius is of the same order of magnitude as the length of the channel, and what it is more important, it is not negligible in comparison with the width of the channel. This conclusion, obtained for the K⁺ channel can be generalized to the rest of channels. To show this, we have performed similar calculations for the sodium channel, using the structural parameters of the channel provided in (Payandeh et al. 2011) and (Kühlbrandt 2016). Thus, taking into account the length of the selectivity filter of 13 Å given in (Payandeh et al. 2011), and that three Na⁺ ions accommodate in the selectivity filter (Kühlbrandt 2016), it is obtained that \( R_L = 39 \text{Å} \) for a Na⁺ channel, i.e. the same order of magnitude as the length of the channel.

From the above discussion it can be concluded that, since the ions do not collide with other species inside the channel but with the walls, and the Larmor radius and the size of the channel are of the same order of magnitude, both the Lorentz force and the collisions with the walls can significantly affect the movement of the ions. In particular, the component of the SMF perpendicular to the axis of the channel will give rise to a Lorentz force acting on the ions directed toward the walls of the channel. As it is well known, the Lorentz force is actually proportional to the cross product of the velocity of the charge times the magnetic field vector. Therefore, the force exerted by a given magnetic field could be different for channels with different orientations. However, since channels are randomly oriented, the order of magnitude of the average effect for any direction of the magnetic field can be estimated by assuming that there exists a component of the magnetic field with the same order of magnitude as the magnetic field itself perpendicular to the axis of the channel.

The effect of Lorentz force on ions for a SMF perpendicular to the axis of the channel is sketched in

\[
v_d = \frac{IL}{Nq}.
\]

The Lorentz force acting on ions inside the narrow channels tends to curve the ion trajectory, whereas at the same time the ions are forced to follow a narrow and straight path. This causes a friction originated by the electrostatic interaction between the ions and the molecules that make up the channel. Summing up, Lorentz Force causes an increased friction acting on ions flowing inside the channel. As mentioned above, the friction accounts for a reduction on the conductance of the channel when compared with ions flowing in bulk media. The additional friction caused by Lorentz force must therefore cause a further reduction of the conductance of the channel.

To estimate whether the decreasing of the conductivity of the channel caused by Lorentz force can be significant, it is interesting to revise the relationship between friction, diffusion and conductance. In the Brownian movement, the Einstein relation relates the friction force with the diffusion coefficient \( D \) as \( D = kT/\eta \gamma \), being \( \eta \gamma \) the friction force in the Langevin’s equation (Reif 2008). In (Chung et al. 1999) the diffusion coefficient of K⁺ in the selectivity filter of the membrane channels is calculated and it is on average 1/3 of the bulk value, whereas in the wider segment of the channel is nearly the same as the bulk value. In the same sense, in (Wallace and Pohorille 2011) it is also reported that the friction is responsible for the diffusion coefficient of K⁺ to be 3 or 5 times lower than in bulk water (\( D = 0.46 \times 10^{-9} \text{ m}^2/\text{s} \) in the channel and \( 2.2 \times 10^{-9} \text{ m}^2/\text{s} \) in bulk water region). Moreover, in (Naranjo et al. 2016) it is pointed out that the different conductance of K⁺ channels might have different causes, the friction among them. Thus, in (Chung et al. 1999) it is shown that the reduction of the diffusion coefficient in the selectivity filter (the narrower part of the channel) influences the overall channel conductance. Those evidences suggest that the additional friction
introduced by the Lorentz Force in the dynamics of ions through membrane channels can result in the reduction of the conductance of the channels. Although the expected reduction of conductance caused by friction due to Lorentz force would be only by a factor of 2 or 3, and therefore similar to the abovementioned contribution of friction with the walls of the channel to reduce the conductance of ions in channels, this additional reduction might be enough to completely suppress the AP. This is due to the fact that the AP generation is quite sensitive to small variations of the conductance values. To illustrate this, Figure 2 shows changes undergone by the transmembrane potential of a neuronal cell segment in response to three consecutive equal stimuli for three different values of the conductance of the fast Na+ channel, which is greatly involved in the onset of the AP. These results have been calculated by solving the differential equations of the HH model of AP generation by means of the HHSim software (Touretzky et al. 2013), a free graphical simulator that provides access to the parameters of the HH model. Figure 2 shows three spikes generated under stimuli for three different values of the conductance of the fast Na+ channel. The first spike corresponds to a conductance of 120 μS, the second spike corresponds to 80 μS and the last spike is for a conductance of 60 μS. For this last value, it can be observed that, even though the change in conductance is only a 25% with respect to the previous value, the AP is almost entirely suppressed.

Regarding these results, note that the magnetic field is not actually an input parameter of the HH model. However, this simulation clearly shows that changes of conductance such as those expected by the effect of the additional friction caused by the SMF used in tSMS have a significant impact on the transmembrane potential, in particular, the suppression of the AP. This is in agreement with the reduction of cortex excitability associated with tSMS (Oliviero et al. 2011)(Dileone et al. 2017)(Silbert et al. 2013)(Arias et al. 2017).

**Conclusion**

The ultimate explanation of the effect of tSMS on neurons remains unknown today. This work offers an explanatory hypothesis and demonstrates its plausibility by means of calculations and numerical simulations. In this work, it is demonstrated that whereas Lorentz Force caused by a moderate SMF is not expected to produce appreciable effects on the ions flowing along the axon of neurons, it might well affect the flux of the ions along the membrane channels in neurons. This is due to the different ratios of the cross sections of axons and membrane channels with respect to the corresponding Larmor radius. It has been shown that in the membrane channels the Lorentz force can effectively produce a friction of the ions with the walls of the channel and that this additional friction might reduce the conductance of the channels. Calculations of transmembrane potential in neurons by using a HH model have illustrated that reductions of conductance of the same order as those expected from the effect of the friction caused by the Lorentz force on ions flowing through membrane channels can effectively suppress the AP in neurons. The provided analysis opens the discussion to make of the Lorentz force acting on ions flowing through membrane channels in neurons a feasible candidate to be the main physical mechanism accounting for the reduction of the excitability of the motor cortex achieved by the tSMS technique.

![Figure 2](image_url)

**Figure 2.** Response of the transmembrane potential (continuous line) of a neuronal cell segment to three consecutive equal stimuli (dashed line). Parameters in the HHSim software: conductances for Na, K, and Cl are set, respectively, to 0.0265μS, 0.07μS and 0.1μS. The conductance of the fast Na+ channel is 120 μS for the first stimulus, 80 μS for the second stimulus and 60 μS for the last stimulus. Note that the AP is almost suppressed in the latter case.
References

Arias, P., L. Adán-Arcay, B. Puerta-Catoira, A. Madrid, J. Cudeiro. 2017. Transcranial static magnetic field stimulation of M1 reduces corticospinal excitability without distorting sensorimotor integration in humans. *Brain Stimul.* 10:340–42. doi:10.1016/j.brs.2017.01.002.

Barker, A. T., R. F. I. Jalinos, and I. L. Freeston. 1985. Non-invasive magnetic stimulation of human motor cortex. *Lancet* 11:1106–07. doi:10.1016/s0140-6736(85)92413-4.

Bezanilla, F. 2005. Voltage-gated ion channels. *IEEE Trans. Nanobiosci.* 4:34–48. doi:10.1109/TNANO.2004.842463.

Chung, S., Toby W Allen, Matthew Hoyles, and Serdar Kuyucak. 1999. Permeation of ions across the potassium channel : Brownian dynamics studies. *Biophysical Journal* 77:2517–33.

Coots, A., R. Shi, and A. D. Rosen. 2004. Effect of a 0.5-T static magnetic field on conduction in guinea pig spinal cord. *J. Neurol. Sci.* 222:55–57. doi:10.1016/j.jns.2004.04.010.

Debanne, D., E. Campanac, A. Bilalows, E. Carlier, G. Alcaraz. 2011. Axon physiology. *Physiol. Rev.* 91:555–602. doi:10.1152/physrev.00048.2009.

Dileone, M., L. M. Mateos, M. C. Carrasco-Lopez, J. Segundo-Rodriguez, N. López-Aristegui, F. Alonso-Frech, M. J. Catalan-Alonso, J. Obeso, A. Oliviero, G. Foffani, et al. 2017. P037 transcranial static magnetic field stimulation-induced modulation of motor cortex excitability in Parkinson’s disease. *Clin. Neurophysiol.* 128:e26. doi:10.1016/j.clinph.2016.10.165.

Doyle, D. A., J. M. Cabral, R. A. Pfuetznner, J. M. Anling Kuo, S. L. Gulbis, B. T. C. Cohen, and R. Mackinnon. 1998. The structure of the potassium channel: Molecular basis of K+ conduction and selectivity. *Science* 280:69–77. doi:10.1126/science.280.5360.69.

Gutiérrez-Muto, A. M., J. Castilla, M. Freire, A. Oliviero, J. Tornero. 2020. Theta burst stimulation: An overview about TMS devices title. *Brain Stimul.* 13:562–64. Press (https://www.brainstimjrnl.com/article/S1935-861X(20)30002-4/fulltext). doi:10.1016/j.brss.2020.01.002.

Jamash, S. 2017. Extension of the neuronal membrane model to account for suppression of the action potential by a constant magnetic field. *Biophysics (Russian Federation)* 62:428–33. doi:10.1134/S0006359107030022.

Kühlbbrandt, W. 2016. Three in a row—how sodium ions cross the channel. *Embo J.* 35:793–95. doi:10.15252/embj.201649049.

McLean, M. J., R. R. Holcomb, A. W. Wamiil, J. D. Pickett, A. V. Cavopol. 1995. Blockade of sensory neuron action potentials by a static magnetic field in the 10 mT range. *Bioelectromagnetics* 16:20–32. doi:10.1002/bem.2250160108.

Naranjo, D., H. Moldenhauer, M. Pincunturero, I. Díaz-Franulic. 2016. Pore size matters for potassium channel conductance. *J. Gen. Physiol.* 148:277–91. doi:10.1085/jgp.201611625.

Nojima, I., S. Kogonemaru, H. Fukuyama, T. Mima. 2015. Static magnetic field can transiently alter the human intracortical inhibitory system. *Clin. Neurophysiol.* 126:2314–19. doi:10.1016/j.clinph.2015.01.030.

Oliviero, A., L. Mordillo-Mateos, P. Arias, I. Panyavin, G. Foffani, J. Aguilar. 2011. Transcranial static magnetic field stimulation of the human motor cortex. *The Journal of Physiology* 589:4949–58. doi:10.1113/jphysiol.2011.211953.

Payandeh, J., T. Scheuer, N. Zheng, W. A. Catterall. 2011. The crystal structure of a voltage-gated sodium channel. *Nature* 475:353–59. Nature Publishing Group. doi:10.1038/nature10238.

Possanzini, C., and M. Boutelje. 2008. Influence of magnetic field on preamplifiers using GaAs FET technology. Proceedings 16th Scientific Meeting, International Society for Magnetic Resonance in Medicine, Toronto, p. 1123. /MyPathway2008/1123.

Reif, F. 2008. *Fundamentals of statistical and thermal physics*. Long Grove, IL: Waveland Pr. Inc.

Roberts, D. C., V. Marcelli, J. Gillen, J. Carey, C. Della Santina, D. Zee. 2011. MRI magnetic field stimulates rotational sensors of the brain. *Curr. Biol.* 21:1635–40. Elsevier Ltd. doi:10.1016/j.cub.2011.08.029.

Rosen, A. D. 2003a. Effect of a 125 mT static magnetic field on the kinetics of voltage activated Na+ channels in GH3 cells. *Bioelectromagnetics* 24:517–23. doi:10.1002/bem.10124.

Rosen, A. D. 2003b. Mechanism of action of moderate-intensity static magnetic fields on biological systems. *Cell Biochem. Biophys.* 39:163–73. doi:10.1385/CBB:39:2:163.

Silbert, B. I., D. D. Pevcic, H. I. Patterson, K. A. Windnagel, G. W. Thickbroom. 2013. Inverse correlation between resting motor threshold and corticmotor excitability after static magnetic stimulation of human motor cortex. *Brain Stimul.* 6:817–20. doi:10.1016/j.brs.2013.03.007.

Touretzky, D. S., M. V. Albert, D. Nathaniel, and A. L. Daw (2013) HHsim: graphical Hodgkin-Huxley simulator. Accessed on 21 February, 2020: http://www.cs.cmu.edu/~dst/HHsim/.

Wilson, Michael A., Chenyu Wei, Pär Bjelkmar, B.A. Wallace, and Andrew Pohorille. 2011. Molecular Dynamics Simulation of the Antiamoebin Ion Channel: Linking Structure and Conductance. *Biophysical Journal* 100 (10):2394–2402. https://doi.org/10.1016/j.bpj.2011.03.054.

Wikswo, J. P., and J. P. Barach. 1980. An estimate of the steady magnetic field strength required to influence nerve conduction. *IEEE Trans. Biomed. Eng.* BME-27:722–23. doi:10.1109/TBME.1980.326598.

Ye, S. R., J. W. Yang, and C. M. Chen. 2004. Effect of static magnetic fields on the amplitude of action potential in the lateral giant neuron of crayfish. *Int. J. Radiat. Biol.* 80:699–708. doi:10.1080/09553000400017424.

Yuan, Y., Y. Chen, and X. Li. 2016a. Theoretical analysis of transcranial magneto-acoustical stimulation with Hodgkin-Huxley neuron model. *Front. Comput. Neurosci.* 10:1–10. doi:10.3389/fncom.2016.00035.

Yuan, Y., Y.-D. Chen, and X.-L. Li. 2016b. A new brain stimulation method: Noninvasive transcranial magneto-acoustical stimulation. *Chin. Phys. B* 25:084301. doi:10.1088/1674-1056/25/8/084301.