Synaptic enhancement of cell morphology by controlled energetic transfer by non-thermal radio-frequency waves to multipolar neurons

Christopher A. Tucker

Abstract

This paper will propose a method and means of applying non-thermal radio-frequency waves to the human body via the skin. The author proposes the reason for this would be to improve cell health and by dendrite growth and synaptic boutons which suggest the multiplicity of connections between an axion and its synapse and other neurons. These improved and multiplied connections serve in recovery of patients with neurodegenerative diseases and damage caused by strokes by helping the neural system reroute connections, due to the presence of a compatible and high-quotient energy allowing replication of cells and growth of new pathways.

1 Introduction

Three significant papers [1, 2, 3] have described the use of magnetic resonance emissive technologies, at a level below 0.5 T, which have demonstrated themselves to have very substantive effects upon human neuronal cultures. One of the most notable of the effects was, after fifteen minutes of exposure to a static 0.2 T magnetic resonance tomograph, dramatic changes of morphology marked by the formation of vortexes of cells and exposed branched neurites featuring synaptic boutons appeared. Even a brief exposure to magnetic fields significantly affects the neuroendocrine features of mammalian neurons, where, such an exposure is more involved with differentiation and senescence rather than with apoptosis or necrosis. Second messenger formations, the authors argued, alters the cell response to environmental stimuli [1].

The authors' point [1] is to demonstrate that, while such magnetic field densities are listed in regulatory specifications as being not harmful to humans, perhaps this is not necessarily the case. Although further studies do not indicate this [2], more so because of the lack of understanding of non-thermal effects of radio-frequency waves [3], they nevertheless support the hypothesis that a properly controlled application of magnetic energy can dramatically affect human neuronal tissues. So, the question is: what if we purposefully try to apply quasi-dynamic, non-thermal electromagnetic waves? What would be its benefit and what are the risks?

This paper will propose a hypothesis for a method and means to consider the possibilities of purposefully constructed devices, which are compact and that can readily transfer currents via low-induction fields to the electrical system of the human body without damage creating the conditions to imbue the body with the ability to increase the density of intra-cell connections. The reason for such a technology would be the ability to treat neurodegenerative disorders, which are manifest by weakly-interacting or electrically-starved cells wherein their connections between have degraded [26, 28, 32], for example, damage to electrical tapestries from strokes, amyotrophic lateral sclerosis, and a wide swath of other neurological disorders.

The postulate described in this paper is a novel approach to treatment of a class of degenerative disorders by the method of providing electrically undernourished cells with energy at a characteristic frequency and power ratio so that they can recover and build a richer set of connections because of the influence upon them by the presence of the currents themselves.

2 Quasi-dynamic non-thermal magnetic fields

Low-frequency wireless power transfer in human proximity has demonstrated itself to be a viable means to transmit cold electrical currents efficiently [4-14]. A loop antenna at a very-specifically designed oscillator emitting a radio-frequency magnetic-resonant wave, in general, does not strongly couple to non-magnetized objects, rather, interacts in a non-thermal manner when its incidence angle is shifted perpendicular to the azimuthal axis.

As described by J. Clerk Maxwell in his eponymous *Treatise on Electricity and Magnetism*, creating a magnetic stress in the medium leads to an increase in the inductance potential between projected field surfaces from an external source and any electrically susceptible circuital surfaces in the region [15], differing from other interpretations such as beaming modes to receiver circuits [6, 9, 14, 16], or other simplistic models of resonance-character phenomenon [17-23]. In Maxwell's interpretation, magnetic stresses yield energy transfer by absorptive interactions via the field itself and its displacement in the medium, rather than the concept that the presence of the energy in the region jumps from transmitter to receiver and is somehow forced upon it.

Apart from modern interpretations of how the mechanism of non-thermal radio-frequency waves are transmitted and received, magnetic stress is a concept where the forces present in a changing magnetic field cause a strain in the medium in which it is present [57]. In terms of free-space, stress in the medium is so small that it only influences ordinary solutions when the circuit becomes sufficiently small [17]. However, in terms of the human body, the electrical tissues encompassed by the field will respond more dramatically to the phenomenon of magnetic stress, which is facilitated by the presence of a metallic disc placed between the antenna and the surface of the skin, as illustrated in Fig.1, B. Otherwise, without the use of the metallic disk, the magnetic field will pass through the electrical tissues with very little interaction, as illustrated in Fig.1, A.

3 Hypothesis of the application of non-thermal RF to the human body

Consider a human multipolar neuron in a very general way as having two planes, x and y, which bisect it forming a contour, S, of its surface extending in the z-direction, which is the same as the field emitted by the antenna. Planes tangential to the contour are more susceptible to an external magnetic force since the electrical capacity of the neuron's ion channels are also along the z-direction. The magnetic field is at right angles to it. For a stress in a region due to the presence of a magnetic field, let P_{hk} represent the stress where h indicates that the normal to the surface where the stress is impacting is parallel to the axis of h and k indicates the direction the positive side is acting on the negative side parallel to the axis of k. The condition that the stress will not produce a change in the angular momentum in elementary regions of the neuron is $P_{hk} = P_{kh}$.

Consider the region encompassed by the field, Ω , enclosing the contour as indicative of the forcive effect on the cell body by the field as dx dy dz with the origin at its center of mass. On the positive

face, dx dy, for which the value of $x = \frac{1}{2}dx$, the forcive expression of motion is,

Normal to
$$S_{xx}$$
:
Normal to S_{yy} :
In the z-direction:

$$\begin{pmatrix}
P_{xx} + \frac{1}{2} \frac{dP_{xx}}{dx} dx \\
P_{xy} + \frac{1}{2} \frac{dP_{xy}}{dx} dx \\
P_{xy} + \frac{1}{2} \frac{dP_{xy}}{dx} dx \\
P_{xz} + \frac{1}{2} \frac{dP_{xz}}{dx} dx \\
P_{xz} + \frac{1}{2}$$

as a mapping of the contour in the presence of a momentum with Z as its axis. Forces acting on the opposite side, $-X_{-x}$, $-Y_{-y}$, $-Z_{-z}$, are found by changing the sign of dx. For any neuron in the field, the entire forcive expression, $X \, dx \, dy \, dz$ is simultaneous with the momentum of the eddy,

$$X \, dx \, dy \, dz = X_{+x} + X_{+y} + X_{+z} + X_{-x} + X_{-y} + X_{-z},$$

$$= \left(\frac{dP_{xx}}{dx} + \frac{dP_{yx}}{dx} + \frac{dP_{zx}}{dx}\right) dx \, dy \, dz,$$
(2)

where $X = \frac{d}{dx}P_{xx} + \frac{d}{dy}P_{yx} + \frac{d}{dz}P_{zx}$. Since there is a momentum present along *Z*, the field is creating a force of rotation around the cell body. L dx dy dz is the moment of forces about the axis of the contour $S_{xx,yy}$ along the *z*-direction, so that the tension on the surface normal as the field rotates about *z* at *x* and *y*,

$$L \, dx \, dy \, dz = \frac{1}{2} dy \left(Z_{+y} - Z_{-y} \right) - \frac{1}{2} dz \left(Y_{+z} - Y_{-z} \right),$$

= $\left(P_{yz} - P_{xy} \right) dx \, dy \, dz$ (3)
 $L = P_{yz} - P_{xy}.$

Quantitatively, the contour experiences the field as quantities of magnetic force **H** and magnetic induction **B** with the angle, 2ε , between their motions, as,

$$P_{xx} = \frac{1}{4\pi} \left(\mathbf{B} \cdot \mathbf{H} \cos^{2} \varepsilon - \frac{1}{2} \mathbf{H}^{2} \right),$$

$$P_{yy} = \frac{1}{4\pi} \left(-\mathbf{B} \cdot \mathbf{H} \sin^{2} \varepsilon - \frac{1}{2} \mathbf{H}^{2} \right),$$

$$P_{zz} = \frac{1}{4\pi} \left(-\frac{1}{2} \mathbf{H}^{2} \right),$$

$$P_{yz} = 0,$$

$$P_{xy} = \frac{1}{4\pi} \mathbf{B} \cdot \mathbf{H} \cos \varepsilon \sin \varepsilon,$$

$$P_{yx} = -\frac{1}{4\pi} \mathbf{B} \cdot \mathbf{H} \cos \varepsilon \sin \varepsilon.$$
(4)

The energy presented to the neuron is described using the Hodgkin-Huxley model [24], where the neuron, an excitable cell, is treated as an electrical circuit. The lipid bilayer is represented as a

capacitance, C_m , and the voltage-gated ion channels contain both a conductance, whose maximum value is $\overline{g}_K, \overline{g}_{Na}, \overline{g}_l$, and a potential, V_K, V_{Na}, V_l , for potassium, sodium, and leak, respectively, per unit area. Over a single area, the total membrane current, I_m , is

$$I_m = C_m \frac{dV_m}{dt} + \overline{g}_K n^4 \left(V_m - V_K \right) + \overline{g}_{Na} m^3 \left(V_m - V_{Na} \right) + \overline{g}_l \left(V_m - V_l \right), \tag{5}$$

where n,m are dimensionless quantities between zero and one associated with channel activation and sodium channel deactivation. The average current, I_e , moving into the region, Ω , with a permeability, μ_e , is

$$I_e = \frac{1}{\Omega} \frac{d\Phi_B}{dt} \frac{\omega_0}{\mu_e},\tag{6}$$

where ω_0 is the angular momentum of the eddy field Φ_B . The energy stored in the region between the field and the surface, Γ , of the contour is

$$\int_{\Omega} \left(\boldsymbol{\sigma}_{0} \mathbf{E}_{0} \frac{\partial \mathbf{E}_{0}}{\partial t} + \boldsymbol{\mu}_{0} \mathbf{H}_{0} \right) d\Omega + \int_{\Omega} \frac{\left| \mathbf{J}_{0} \right|^{2}}{\boldsymbol{\sigma}_{0}} d\Omega - \int_{\Omega} \mathbf{E}_{0} \mathbf{J}_{0} d\Omega + \oint_{\Gamma} \left(\mathbf{E}_{0} \times \mathbf{H}_{0} \right) d\Gamma.$$
(7)

The neuronal somata will experience the energy as a pressure equal in all directions $\frac{1}{8\pi}$ **H**². It will only absorb energy in quantities sufficient to elevate power level to its steady state, and will feedback along P_{yx} to satisfy $P_{xy} = P_{yx}$ attaining a new equilibrium. A very detailed account of this theory is provided by Tucker [57].

4 Discussion of the potential benefit and risks

The scheme outlined in the previous sections and the hypothesis which supports it can be centered on the idea of a compact device wherein to test this notion. The methodology proposed in this paper is a direct application of magnetic resonant fields to subcutaneous neural tissues by mathematically describing the phenomenon as the creation of a link between potential of the coil and that of the neural tissues, or more generally, a potential link, which is the locus of a magnetic stress. A successful link is observed to be the field affecting neuronal somata resulting in an increased output of action potential signals consequential to the absorption of electrical energy [33, 34, 41, 42]. While this work is examining the problem in a very general way, it is an important first step to establish whether energy can be absorbed by neurons and neuroglia from artificial sources.

Some questions to answer are as follows. Can the energy be used by the composition of nerves and ganglia create chemical reactions due to the interaction of induced electrical currents and stimulate the production of neurotransmitters? Can the absorption be suggestive of new neural pathways, and perhaps even new neurons using this method in tandem with other treatment regimens? The detection and mapping of new pathways is outside the scope of this work but is the most important question to answer in future research. If it could be answered succinctly, it would imply a novel method to aid in the treatment of very serious neurodegenerative disorders.

Treatment of disorders in the somatic nervous system due to progressive neurodegeneration, while dependent upon a clear aetiology, can be addressed symptomatically [25, 26, 27] while in search of possible treatments [28, 29, 30, 31, 32, 41]. While this work does suggest a pathway to treatment of such severe debilitation disorders [36, 37, 38], it only does so by its approach to the problem from a mechanistic interpretation of the clinical evidence surrounding the epidemiology of fundamental degradation characteristics surrounding such disorders [39, 40]. While this is a very critical problem at this point in the research, it is also clear such applications indicate an underlying set of conditions that can be altered reversing the causality of symptoms of the disorder. A set of experiments is proposed to test the hypothesis proposed in this paper where an application of energy is presented to electrically capable tissues and its reaction to the presence of the field measured by an EEG. It is postulated that the neurons subjected to the magnetic stresses in the region would respond with spiking behavior, the observation being they had absorbed the energy in the manner described by [24] and such behavior is indicative of feedback.

Much more specifically, such an observation would suggest that the neurons responding to such energy transfer by the appearance of stress are employing it to remedy damage having occurred sometime in the past, the length of storage indicative of their capacity. One difficulty is distinguishing healthy from damaged tissues as the model suggests that nearly any neuron with ion channels would not only absorb energy but once converted, would increase ionic activity across the network. The increase in activity implies a measure of feedback, which reduces the absorption by the network by changing the cell's inductance potential.

One conclusion to be drawn from the experiments is that spiking behaviour and energy distribution of the magnetic potential [32, 33, 46] form a corollary to the effectiveness of energy transfer between the external device and neural tissues similar to direct electrical probe stimulation without surgical invasion. Given the indication of an ability of the body to grow neuronal tissues under special circumstances [25, 35, 42, 44, 45], it is important to understand not only how to apply energy to damaged or atrophied neural tissues [43, 47], but to attempt to stimulate neurogenesis [1, 48, 56] for exposure times and subsequent cognitive training for patients who have lost neurons due to health or accident. A successful conceptual model for such treatments would imply the following assumptions to be true:

- 1 The level of energy within the tissues affected by the disorder, including those atrophying, reflects a lack of self-repair. For example, certain catalytic drugs are administered which excite the chemical structures of the neural tissues with either an increase of sodium for enhanced firing and linking or an increase of potassium for enhanced suppression [49].
- 2 The amount of neural activity in tissue is indicated by the degree of neuronal firing and that which occurs between neurons. It illustrates which energy has been absorbed individually verses one that has been transferred intra-cell. This implies the connectedness between pools of neurons, arbitrarily bounded over a given area and demonstrates the transfer of energy from one set to the next and the degree of merit [50].
- 3 The number of pooled neurons is represented by the amount of spiking relative to the field potential, defined by the magnetic stress, and electrical current passing through the ion conductance. At the limit of conductance, new extraneous linking by dendrite growth includes not only new links but also new neurons.

While the benefit is rather easy to predict using the methods outlined herein, there is also the probability of grave risks in energy transfer to undesirable tissues. Outside of those target neurons in

the body, what about electrical tissues of microorganisms which are also within the exposed area? One observation is in the case of a bacterial infection. If an infection is present in the subject there is a chance that the bacterial colony can become exposed to the energy yielded by the magnetic stress which, like neurons, will be expected to grow and expand. Increases in the presence of energy increases the possibility for them to signal through the ion channels in the cell [51], due to the higher potential. While there are sources in the literature that discuss the link between bacterial and the creation of brain cells [52], it would be an interesting aspect of expansive research if neural dendrite and soma proliferation in terms of bacteria in the host following exposure were causal. Cancer may or may not be treatable by the same means as it is hypothesized that the resonance frequency of renegade cells are different from the healthy ones.

One idea to reduce high-level effects of exposure is to apply energy to a dielectric element, such as a lemon. Since the lemon is subject to a complex permittivity, dependent upon the resonance frequency [53], it was interesting to discover if it could be used to store charge—say, in terms of its energy signature—and then applied to the subject by placing it in their hand, rather than direct application.

Regarding the more complex forms of necrosis, the symptoms of amyotrophic lateral sclerosis are caused by degeneration of motor neurons in the spinal cord, brainstem, and motor cortex. Median survival from first symptoms is a little more than two years. The exact cause of this degeneration is not known presently but thought to be consequent of environmental exposures and genetic factors playing a role in the susceptibility of the disease. The hallmark of this disease is the selective death of motor neurons in the brain and spinal cord, leading to paralysis of voluntary muscles. The paralysis begins focally and disseminates in a pattern that suggests that degeneration is spreading among contiguous pools of motor neurons. If the mechanism that causes ALS can be disturbed by the contribution of replenished neural energy directly transferred to the nerve cells and glial tissues, it is proposed that it could arrest the progress of ALS not merely affecting the spread of the disorder [42, 54]. Nevertheless, one must be very considerate of the resonance frequency, potential, and magnitude of stress exhibited by the oscillator in conjunction with tissues [13, 55].

The hypothetical implication presented in this paper of the causality of energy transfer by nonthermal, magnetic-resonant, radio-frequency waves is in agreement with the results posited by Pacini, et. al, albeit with smaller machines. No tomography equipment was used, only small-scale devices. The expectation is that a healthy neuron cell after fifteen minutes exposure exhibits changes of morphology: a formation of vortexes of cells and exposed branched neurite featuring a synaptic bouton. Simultaneously, there is a reduction in thymidine incorporation and inositol lipid signaling. Pacini, et. al stipulates there is no synthesis of neither DNA proteins or transmission of the transformation to other cells, that each cell requires the same external influence. Vortexes imply a clustering of neurons, which are electrically chained in response to the presence of the energy, provided the creation of the magnetic stress.

Weighing the benefit and risks, if a carefully controlled experiment were conducted in tandem with a substantive pathology, this method could contribute strongly to a regimen of treatment. Additionally, in cases where the patient has not been diagnosed with a neurodegenerative disorder, there is an equal probability that neuron counts could be doubled and synapses expanded in an exponential manner—insuring the proper pathology is equally applied. This implies the possibility of the enhancement of human performance by artificial means; however, those changes would affect the subject in a holistic manner. Moreover, perhaps this is a point to be taken where nature has provided us a means to overcome our shortcomings as well as our maladies.

References

- S. Pacini, G.B. Vannelli, T. Barni, M. Ruggiero, I. Sardi, P. Pacini, and M. Gulisano, "Effect of 0.2 T static magnetic field on human neurons: remodeling and inhibition of signal transduction without genome instability," *Neuroscience letters*, 267(3), 185-88, 1999.
- [2] S. Pacini, et al. "Effects of 0.2 T static magnetic field on human skin fibroblasts," Cancer detection and prevention, vol. 27, No. 5, pp. 327-32, Jan 2003.
- [3] D. Formica and S. Silvestri, "Biological effects of exposure to magnetic resonance imaging: an overview," *Biomedical engineering online*, 3(1), 11, 2004.
- [4] C.A. Tucker, K. Warwick, and W. Holderbaum, "Efficient wireless power delivery for biomedical implants," *IET Wireless Sensor Systems*, Vol. 2, Iss. 3, pp. 176-82, Sep. 2012.
- [5] K. Warwick, M. Gasson, and B. Hutt, "The application of implant technology for cybernetic systems," *Arch. Neurology*, Vol. 60, No. 10, pp. 1369-73, 2003.
- [6] A. RamRakhyani, S. Mirabbasi, and M. Chiao, "Design and optimization of resonance-based efficient wireless power delivery systems for biomedical implants," *IEEE Transactions on Biomedical Circuits and Systems*, Vol. 5, Iss. 1, pp. 48-63, Jan. 2011.
- [7] K. Warwick and M. Gasson, "Practical Experimentation with Human Implants," *Uberveillance and the Social Implications of Microchip Implants: Emerging Technologies*, IGI Global, 2013.
- [8] Q. Yuan, C. Qiang, and S. Kunio, "Effect of Human Body on Near-Field Resonant Coupling Wireless Power Transmission System," *Proceedings Of the 2009 International Symposium on EMC*, pp. 25-28. 2009.
- [9] A. Poon, S. O'Driscoll, and T. H. Meng. "Optimal operating frequency in wireless power transmission for implantable devices," *Engineering in Medicine and Biology Society, EMBS 2007, 29th Annual International Conference of the IEEE*, pp. 5673-78. Aug. 2007.
- [10] P.K. Sharma and S. K. Guha, "Transmission of time varying magnetic field through body tissue," *Journal of Biological Physics*, Vol. 3, No. 2, pp. 95-102, 1975.
- [11] G. Reddy and G.J. Reddy, "Effects of Wireless Electricity on Human Bodies," *International Journal of Engineering Trends and Technology*, Vol. 4, Iss. 6, Jun. 2013.
- [12] F. Zhang, S.A. Hackworth, W. Fu, C. Li, Z. Mao, and M. Sun, "Relay Effect of Wireless Power Transfer Using Strongly Coupled Magnetic Resonances," *IEEE Transactions on Magnetics*, Vol. 47, No. 5, pp. 1478-81, May 2011.
- [13] C.A. Tucker, Transmission of wireless power by magnetic resonance, Ph.D. thesis, University of Reading, Reading, U.K., 2013.
- [14] J. Ho et al., "Wireless power transfer to deep-tissue microimplants," *Proceedings of the National Academy of Sciences*, 2014, doi: 10.1073/pnas.1403002111.
- [15] J. Clerk Maxwell, A Treatise on Electricity and Magnetism, Oxford at the Clarendon Press, Vol. 2, 1873.
- [16] D. Ahn and S. Hong, "A study on magnetic field repeater in wireless power transfer," *IEEE Transactions* on *Industrial Electronics*, pp. 360-71, Jan. 2013.
- [17] C.A. Tucker, U. Muehlmann, and M. Gebhart, "Contactless power transmission for NFC antennas in credit-card size format," *IET Circuits, Devices, and Systems*, pp. 18 36, 2016.
- [18] C. Saha, et al. "Wireless power transfer using relay resonators," *Applied Physics Letters*, Vol. 112, No. 26, 2018.
- [19] A. Beppu and K. Seiichiro, "Realization of transmitter for wireless power transfer to multi loads," 2018 IEEE International Conference on Industrial Technology, IEEE, 2018.

- [20] S.R. Khan, S.K. Pavuluri, and M.P. Desmulliez, "Accurate Modeling of Coil Inductance for Near-Field Wireless Power Transfer," *IEEE Transactions on Microwave Theory and Techniques*, Vol. 66, No. 9, pp. 4158-69, 2018.
- [21] M. Najjarzadegan, et. al, "Improved Wireless Power Transfer Efficiency Using Reactively Terminated Resonators," *IEEE Antennas and Wireless Propagation Letters*, Vol. 17, No. 5, pp.803-7, 2018.
- [22] Y. Luo, et. al, "Power waves-based analysis of magnetically coupled resonant wireless power transfer," In Technology, Networking, Electronic and Automation Control Conference, 2017 IEEE 2nd Information, pp. 878-82, Dec. 2017.
- [23] S. Go and A. Abramowicz, "Wireless power transfer system based on magnetic resonant coupling with directional coupler," In Antennas and Propagation in Wireless Communications (APWC), 2017 IEEE-APS Topical Conference on, pp. 197-8, Sep. 2017.
- [24] A.L. Hodgkin and A.F. Huxley, "A quantitative description of membrane current and its application to conduction and excitation in nerve," *The Journal of Physiology*, Vol. 177, No. 4, pp. 500-44, 1952.
- [25] Walton C, Pariser E, Nottebohm F., "The Zebra Finch Paradox: Song Is Little Changed, But Number of Neurons Doubles," *Journal of Neuroscience*, Vol. 32, Iss. 3, pp. 761-74, Jan. 2012.
- [26] Piemonte and Valle d'Aosta Register for Amyotrophic Lateral Sclerosis (PARALS). "Incidence of ALS in Italy: evidence for a uniform frequency in Western countries," *Neurology*, Vol. 56, pp. 239-44, 2001.
- [27] H. Hodkinson. "More favourable prognosis of motor neurone disease in old age," Age Ageing, Vol. 1, pp. 182-84, 1972.
- [28] E. Cellura, R. Spataro, A.C. Taiello, V. La Bella. "Factors affecting the diagnostic delay in amyotrophic lateral sclerosis," *Clinical Neurology and Neurosurgery*, Vol. 114, pp. 550-54, 2012.
- [29] J.R. Williams, D. Fitzhenry, L. Grant, D. Martyn, and D.A. Kerr. "Diagnosis pathway for patients with amyotrophic lateral sclerosis: retrospective analysis of the U.S. Medicare longitudinal claims database," *BioMedCentral Neurology*, Vol. 13, No. 1, pp. 160-67, 2013.
- [30] L.C. Wijesekera and P.N. Leigh. "Amyotrophic lateral sclerosis," *Orphanet Journal of Rare Diseases*, Vol. 4, No. 1, pp. 1-22, 2009.
- [31] C. Armon. Epidemiology of ALS/MND, In: Shaw P and Strong M, eds. *Motor Neuron Disorders*, Elsevier Sciences, pp. 167-206, 2003.
- [32] C. Armon. ALS 1996 and Beyond: New Hopes and Challenges. A manual for patients, families and friends, 4th Edition. California: LLU Department of Neurology, 2007.
- [33] B. Granula, K. Porzig, H. Toepfer, and M. Gacanovic. "A Comparison between an A-V and V Formulation in Transcranial Magnetic Stimulation," in *Comsol Conference, Rotterdam, Netherlands*, pp. 23-25, 2013.
- [34] K. Porzig, Konstantin, H. Brauer, and H. Toepfer. "The electric field induced by transcranial magnetic stimulation: A comparison between analytic and fem solutions," *Serbian Journal of Electrical Engineering* Vol. 11, No. 3, pp. 403-18, 2014.
- [35] M. Kaplan, "Environment complexity stimulates visual cortex neurogenesis: death of a dogma and a research career," *Trends in Neuroscience*, Vol. 24, Iss. 10, pp.617-20, Oct. 2001.
- [36] M.R. Turner, J. Scaber, J.A. Goodfellow, M.E. Lord, R. Marsden, and K. Talbot. "The diagnostic pathway and prognosis in bulbar-onset amyotrophic lateral sclerosis," *Journal of the Neurological Sciences*, Vol. 294, No. 1, pp. 81-85, 2010.
- [37] D.W. Mulder, "Clinical limits of amyotrophic lateral sclerosis," Advances in Neurology, Vol. 36, pp. 15-22, 1981.
- [38] J.F. Kurtzke. "Epidemiology of amyotrophic lateral sclerosis," Advances in Neurology, Vol. 36, pp. 281-302, 1981.

- [39] R.B. Forbes, S. Colville, and R.J. Swingler. "The epidemiology of amyotrophic lateral sclerosis (ALS/MND) in people aged 80 or over," *Age and Ageing*, Vol. 33, No. 2, pp. 131-4, 2004.
- [40] C. Wood-Allum and P.J. Shaw. "Motor neurone disease: a practical update on diagnosis and management," *Clinical Medicine*, Vol. 10, No. 3, pp. 252-8, 2010.
- [41] G. Null. Biomagnetic healing, http://www.garynull.com/Documents/magnets.htm, 1998.
- [42] A. Bellossi and R. Berget. "Pulsed Magnetic Fields: A Glimmer of Hope for Patient Suffering from Amyotrophic Lateral Sclerosis," *In Electricity and Magnetism in Biology and Medicine*, pp. 891-93. Springer, 1999.
- [43] X. Zhang, Y. Chen, C.L. Wang, and L.-Y.M. Huang. "Neuronal somatic ATP release triggers neuronsatellite glial cell communication in dorsal root ganglia," *Proceedings of the National Academy of Sciences*, Vol. 104, No. 23, pp. 9864-9, 2007.
- [44] P. Fromherz, "Interfacing neurons and silicon by electrical induction," Berichte der Bunsengesellschaft für physikalische Chemie, Vol. 100, Iss. 7, pp. 1093–1102, Jul. 1996.
- [45] J. Altman, "Are new neurons formed in the brains of adult mammals?" Science, Vol. 30, No. 135, pp.1127-28, Mar. 1962.
- [46] E. Izhikevich, "Simple model of spiking neurons," *IEEE Transactions on Neural Networks*, Vol. 14, No. 6, pp. 1569-72, Nov. 2003.
- [47] J.A. Connor and C.F. Stevens, "Voltage clamp studies of a transient outward membrane current in gastropod neural somata," *The Journal of Physiology*, Vol. 213, No. 1, pp. 21-30, 1971.
- [48] P.S. Eriksson, E. Perfilieva, T. Björk-Eriksson, A.M. Alborn, C. Nordborg, D.A. Peterson, and F.H. Gage, "Neurogenesis in the adult human hippocampus," Nature medicine, 4(11), pp.1313-17, 1998.
- [49] B. Göbel, K.M. Oltmanns, and M. Chung, "Linking neuronal brain activity to the glucose metabolism," *Theoretical Biology and Medical Modelling*, Vol. 10, No. 1, 2013.
- [50] S. Sasaki and M. Iwata, "Mitochondrial alterations in the spinal cord of patients with sporadic amyotrophic lateral sclerosis," *Journal of Neuropathology & Experimental Neurology*, Vol. 66, No. 1, pp. 10-16, 2007.
- [51] A. Prindle, L. Jintao, A. Munehiro, L. San, G.O. Jordi, and G.M. Süel. "Ion channels enable electrical communication in bacterial communities," *Nature*, Vol. 527, No. 7576, pp. 59-63, 2015.
- [52] I.M. Chiu, A.H. Balthasar, G. Nader, C.A. Von Hehn, Z. Fan, J. Tran, and B. Wainger, "Bacteria activate sensory neurons that modulate pain and inflammation," *Nature*, Vol. 501, No. 7465, pp. 52-57, 2013.
- [53] A. Kaewrawang, S. Swatdiponphallop and A. Siritaratiwat, "Study on Complex Permittivity of Tropical Thai Fruits," *Journal of Applied Sciences*, Vol. 7, pp. 1009-12, 2007.
- [54] M.P. Liebl, W. Johannes, A.S. Besemer, A.K. Schäfer, H. Reber, C. Behl, and A.M. Clement, "Low-frequency magnetic fields do not aggravate disease in mouse models of Alzheimer's disease and amyotrophic lateral sclerosis," *Scientific reports*, Vol. 5, 2015.
- [55] A. Huss, A. Spoerri, M. Egger, H. Kromhout, and R. Vermeulen, "Occupational exposure to magnetic fields and electric shocks and risk of ALS: The Swiss National Cohort," *Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration*, Vol. 16, No. 1-2, pp. 80-85, 2015.
- [56] J. Beers, "Biological effects of weak electromagnetic fields from 0 Hz to 200 MHz: a survey of the literature with special emphasis on possible magnetic resonance effects," *Magnetic Resonance Imaging*, Vol. 7, pp. 309-31, 1989.
- [57] C.A. Tucker, "Ruminations on the concept of magnetic stress," doi: 10.6084/m9.figshare.7064762.v3.



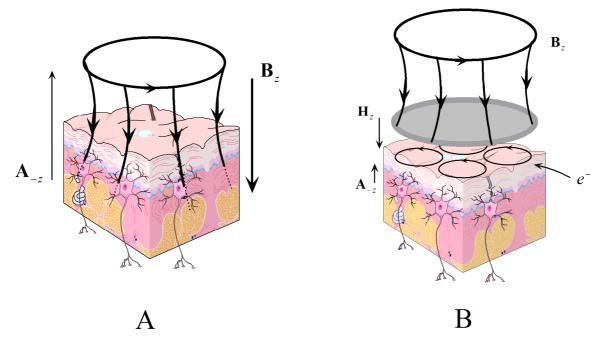


Fig.1. Depiction of interaction of a non-thermal magnetic-resonant field against a cross-section of human skin.