



Faculty Of physical Therap,
Cairo University



Cairo University

Effect of Electromagnetic Therapy on Mechanoreceptor Number as A risk Factor of Low Back Pain

Mahmoud Mohamed Mahmoud Nabhan¹, Haitham Sharaf El-Din Mohammed²,
Alaa Eldin Abd Elhakeem Balbaa¹.

¹Orthopaedic Department, Faculty of Physical Therapy, Cairo University, Giza, Egypt

²Biophysics Department, Faculty of Science, Cairo University, Giza, Egypt

ABSTRACT

Back ground: Low back pain (LBP) refers to pain at the lower back or lumbosacral region. It is a very common symptom and is associated with a high rate of disability. Spinal instability is believed to be an independent risk factor for the development and progression of LBP. Patients with LBP showed defective proprioception, especially position sense, compared to healthy individuals. Proprioceptive defect was attributed to ischemia and depletion of mechanoreceptors within thoracolumbar fascia. Thus, therapeutic interventions that may help in reversing proprioception deficits such as electromagnetic field can be used as preventive and therapeutic methods. Purpose: The present study aimed at investigating the effects of Electromagnetic field (EMF) on the number of mechanoreceptors in thoracolumbar fascia. Methods: Sixteen healthy adult Wister male rats weighing between 250 to 300 gm were randomly divided into two groups: (I) EMF exposed (experimental) and (II) sham exposed animals. Each one of the EMF exposed animals was placed in a separate plastic cage inside electromagnetic set up cylinder coil (42cm diameter) that was connected to a main power supply (AC -50 Hz) and adjusted via variac to produce EMF of 0.3 mT. The EMF intensity was monitored with a probe connected to a digital tesla meter. Rats were exposed for 1h/day at the same time every day, six days/week for two weeks. Sham exposed animals went through the same procedures except that no EMF was generated. Six-hours following the last session, all animals were sacrificed. Tissues were harvested and the thoracolumbar fascia stained with 1% gold chloride solution for histological examination. Results: The number of Pacinian corpuscles significantly increased in the experimental group compared to that of the sham exposed group ($P < 0.05$). The Golgi tendon organs number and Ruffini corpuscles number were statistically not different between the two groups ($p > 0.05$). Conclusion: EMF is effective in increasing the number of Pacinian corpuscles in thoracolumbar fascia but not the number of Golgi tendon organs or Ruffini corpuscles in healthy Wister rats. Key words: Electromagnetic Therapy, fascia, mechanoreceptor, rats.

INTRODUCTION

Low back pain (LBP) refers to pain at the lower back or lumbosacral

region. It is a very common symptom and is associated with a high rate of disability. For example, a 1-year disability score of 50% or more in UK

showed the prevalence of disability to be 5.4% in males and 4.5% for females. Furthermore, time off work has been reported as 11% for males and 7% for females with a lifetime prevalence of 34% and 23% respectively that accounted for about half of the lost work time (Walsh and Cruddas, 1992; Waddell, 2002). Therefore, the overall cost associated with LBP was substantial (Maniadakis and Gray, 2000). Prevention and treatment of LBP is not easy as it is not a self-limiting disease but it may present with recurrent attacks (Hestbaek et al., 2003). That is why there is a need for more understanding of its etiology, pathomechanics, risk factors and pathogenesis in order to better develop new treatment interventions (Adamset al., 2007).

Spinal instability is believed to be an independent risk factor for the development and progression of LBP (Byland Sinnott, 1991; Leetun et al., 2004). Instability may result in a large intervertebral motion and subsequent overstress and deformation of surrounding tissues that are rich in nociceptors. This eventually may cause pain and substitution (Wyke, 1970; Schleip et al., 2007). Spinal instability is counteracted by three systems: active, passive and neural systems. The passive system is formed of ligaments and fascia and acts as a signal transducer to the neural system that conveys messages about vertebral position or motion (proprioception) to the central nervous system. In addition, it produces forces that restrict or control the motion at the end of

available range (Panjabi, 1992). Fascia is a major player in executing these functions because it has a relatively great excursion. This makes it more liable to deformation, and thus stimulates proprioceptors more intensely (Schleip et al., 2007).

Unfortunately, fascia and its regulatory role have been traditionally underestimated. Evidence suggested the continuity of the fascial system between regions as well as the fascial role as a body-wide proprioceptive/communicating organ (Kassolik et al., 2009). This role is mediated by its numerous mechanoreceptors. Thus, factors affecting the proprioceptive and signaling functions of fascia are recommended to be investigated (Chaudhry et al., 2007). patients with LBP showed defective proprioception, especially position sense, compared to healthy individuals (Byland Sinnott, 1991; Brumagneet al., 2000). Such finding has also been shown in other regions as hip (MoraesMRB et al., 2011), knee after ACL injury (Dhillonet al., 2012) and ankle (Konradsen et al., 1993).

Proprioceptive defect was attributed to ischemia and depletion of mechanoreceptors within thoracolumbar fascia (TLF) (Bednar et al., 1995) as well as alteration in proprioception afferent and central processing of this sensory input. As a result, the local muscle control and subsequently segmental stability decreases. This increases spine vulnerability to subfailure injuries and

recurrence of LBP (Brumagne et al., 2000). Therapeutic exercise can enhance proprioception significantly (Beard et al., 1994). However, exercise is an active intervention in which the patient has to exert effort. With pain and dysfunction, patient may refrain from such interventions, and thus, chronicity may follow. Thus, there is a need to investigate potential preventive or therapeutic interventions that may help in reversing proprioception deficits such as electromagnetic field (Markov, 2007a).

Electromagnetic field (EMF) is a passive, noninvasive, safe, and easy therapeutic intervention (Markov, 2007a). Experimental studies showed that EMF increases neural tissue healing rate by 22% (Sisken, et al., 1989). Moreover, It increases neuron function and diameter by two folds (Ito and Bassett, 1983). Effects of EMF can be attributed to induced vasodilatation of small capillaries; as it produces up to 8.7% capillary dilatation after one hour of exposure (Smith et al., 2004), acceleration of intracellular signaling pathway (Markov, 2007b), stimulation of gene expression, stabilization of cytosolic Ca^{+2} (Blank and Goodman, 2004), acceleration of axon growth rate, up regulation of neurotrophic factors in the neurons (Gordon et al., 2007), increasing protein synthesis and decreasing of H_2O_2 induced apoptosis (Programmed cell death) (Grassi et al., 2004). The present study aimed at investigating the effects of EMF on the number of mechanoreceptors, specifically Pacinian and Ruffini

corpuscles as well as Golgi tendon organs in TLF.

MATERIAL AND METHODS

Experimental animals

Sixteen healthy adult Wistar male rats weighing between 250 to 300 gm were obtained from the Research Institute of Ophthalmology, Egypt. Animals were housed at the Department of Biophysics, Faculty of Science, Cairo University. Animals housing, care and experimental procedures were approved and were done in accordance to guidelines of animal handling and care of the Institutional Animal Care and Use Committee (IACUC) of the Faculty of Medicine, Cairo University. All procedures were performed to minimize the number of animals used and their suffering.

Experimental design

Animals were randomly divided by computerized generation using excel software into two groups with each group containing eight animals: Group I (electromagnetic field exposed), animals were subjected to electromagnetic therapy with the following parameters (biphasic symmetrical sine wave AC-type, Frequency: 50 Hz, Intensity: 0.3 mT, and Duration: 1 h/day, 6 days/week for 2 weeks). Group II (Sham exposed) Animals were placed inside the electromagnetic set up for the same duration as their exposed counterparts. However, the generator supplying the

solenoid was turned off and no EMF was generated.

Electromagnetic set up

The EMF was generated within a solenoid carrying current of 18 Ampere at 50 Hz from the main supply (220–230 Volts) via a Variac (KAR 0303 k No. 000420 manufactured in Yugoslavia). It consists of a coil with 320 turns made of electrically insulated 0.8 mm copper wire. The coil was wound around a copper cylinder of 2 mm thickness, 42 cm diameter, and 40 cm length. The cylinder wall was earthed to eliminate the static charges. The magnetic field intensity was measured at different locations to find out the most homogenous zone inside the solenoid core. This was done using Gauss/Tesla meter with probe manufactured by Bell Technologies Inc. (Orlando, FL, USA) for placing rats during magnetic exposure. Plastic cages with dimensions of 15 cm length 9.5 cm width and 10 cm height were used to house the animals inside the magnetic field. Each cage has multiple openings of 0.75 cm diameter each in the anterior and posterior wall for animal ventilation.

Tissue harvesting

Six hours after last session of EMF exposure rats were sacrificed by cervical dislocation after narcosis with chloroform vapor for at least 5 min. The skin covering the back was then cut open, and TLF was harvested along the insertions of latissimusdorsi muscles and origin of gluteus maximus muscles. A surgical blade was used to remove all visible muscle fibers from the fascia. This was performed and

checked by visual inspection. The effective sample size that was harvested had a length of between 12 mm and 18 mm. All TLF samples were then kept fully immersed in Krebs-Ringer solution (Ringer solution + glucose for cell survival) at room temperature. Air exposure time without immersion was kept to below 2 min.

Histological processing and analysis

Tissue was stained using a modified gold-chloride method (Gairns, 1930). This technique stains the mechanoreceptors and allows each structure to be distinguished. It is also employed to identify cells, collagens, fascicular regions and conjunctive tissue (Moraes et al., 2011). Each specimen was placed in a separate plastic container covered by a solution of 3:1 lemon juice to 88 % formic acid. The container was placed in darkness for 10-20 minutes or until the tissue color turned transparent. Then, the used solution was replaced by a 1 % solution of gold chloride, and the container was placed again in darkness for additional 10 to 60 minutes (Koch et al., 1995) or until the tissue turns to a uniform golden yellow. Then, the gold chloride solution was poured out and collected for future use. Tissues were then immersed in a 25 % solution of formic acid for from 3 to 8 hours in darkness (block staining) before tissues were rinsed in 3 changes of 70 % ethanol, each for 10 minutes. Tissues were placed in glycerol for 24 hours. Finally, tissues were dehydrated and paraffin-embedded according to standard histological procedures.

Longitudinal serial 5 microns sections were cut (LEICA RM2125RTS, USA) at 30 micron sampling rate and prepared for bright field examination. The lemon juice-formic acid and the gold chloride baths were initially kept at about 4 °C, but were allowed to warm to room temperature after the tissues have been placed in them. During processing, once tissues were placed in the acid solution, they were not touched by any metal implement until they have been in glycerol for 24 hours (O'Connor and Gonzales, 1979).

Stained sections were assessed by a blind assessor under light microscope (Olympus U-MDOB3, Japan) with a magnification of 100X and 400X. Three slides with four sections were visualized and the number of each mechanoreceptor was

counted. Areas of interest were captured using a standard microscope camera (Olympus E.330, Japan) and was analyzed using histologist visual inspection. Maximum number of each type of mechanoreceptor in each section was then counted per (400X) magnification power field to be used for further statistical analysis.

Statistical analysis

The main outcomes measurements for this study were the number of Pacinian corpuscles, Ruffini corpuscles and Golgi tendon organ. All data are presented as medians and ranges. Between groups, comparison was done using the Mann-Whitney test with significance level set at $p < 0.05$. All statistical analyses were done using the SPSS version 21.0 (IBM incorporation, Chicago, IL, USA).

RESULTS

The number of Pacinian corpuscles (Figure 4), for EMF exposed animal group ranged from 1 to 11 with a median of (4), whereas the sham exposed animal group ranged from 0 to 2 with a median of (1). Between groups comparisons showed a significant increase in corpuscles number in the EMF compared to the sham exposed group ($p = 0.007$) (Figure1).

The number of Ruffini corpuscle (Figure 5), for EMF exposed animal group ranged from 0 to 2 with a median of (1), whereas the sham exposed animal group ranged from 0 to

3 with a median of (0). Comparing the animals in EMF and sham exposed groups showed a non-significant difference in corpuscles number ($p = 0.505$) (Figure2).

The number of Golgi tendon organ (Figure 6), for EMF exposed animal group ranged from 0 to 1 with a median of (0), whereas the sham exposed animal group ranged from 0 to 6 with a median of (0). Comparing the animals in EMF and sham exposed groups showed a non-significant difference in Golgi tendon organ number ($p = 0.645$) (Figure3).

DISCUSSION

The present study aimed at investigating the effect of EMF exposure on thoracolumbar fascia content of Pacinian corpuscles, Ruffini corpuscles and Golgi tendon organs. Results showed a significant increase in Pacinian corpuscle number in EMF exposed group with respect to the sham exposed animals. For Ruffini corpuscle and Golgi tendon organ, EMF exposed animals were not different from sham exposed animals. It was reported that, when Wister rats were exposed to EMF with a frequency of 60 Hz and intensity of 0.7 mT for 21 days, an up regulation of neurotrophic growth factors gene expression and increase resistance to oxidative stress were occurred (Tasset et al., 2012). Vasodilation was also reported as a potential factor for EMF effects (Okano et al., 1999; Gmitrov et al., 2002; Leociet al. 2014). As the mechanoreceptors in TLF are surrounded and adjacent to blood vessels (Yahia et al., 1992) it is the most structure that benefits from vasodilation. Based on the present findings, the EMF used in the present study could have positive effect on the number of Pacinian corpuscles that may further enhance proprioception function.

Furthermore, the increase in Pacinian corpuscles could be attributed to increased polypeptide synthesis which enhances nerve anabolism (Sisken et al., 1990). Also, the increase in DNA expression without mutation (

Pacini et al., 1999; Blank and Goodman, 2004) in association with up regulation of neurotrophic factors in the neurons (Tasset et al., 2012) and inhibition of puromycin which is a protein synthesis inhibitor that stops DNA ribosome translation and inhibition of H₂O₂ induced apoptosis (Grassi et al., 2004), and the up regulated expression of mRNAs transcriptional factors that play pivotal roles in new cells differentiation in to neuronal rather than a glial phenotype. Also, increased levels of mRNA for Ca_v1.2 channel subunits which mediate neurogenesis (Cuccurazzu et al., 2010).

To the best of authors' knowledge, there are no reports on the effect of EMF on mechanoreceptors were done in vivo. Yet, previous studied support the role of EMF stimulation on neurite outgrowth. For example Pilla et al. (1999) used EMF with the following parameters: 65 μ s, 200 μ T and 500 μ s, 20 μ T on chick dorsal root ganglia culture and found that neurite length was doubled. Also, Mcfarlane et al. (2000) found that stimulation using EMF (4.5–15.8 μ T, 50 Hz AC) on rat PC12 pheochromocytoma culture for 23 h significantly stimulated neurite outgrowth by 16.9%. Fan et al. (2004) used EMF (0.23 mT, 50 Hz) for 96 h on rat PC12 pheochromocytoma culture. They found that average number of neurites per cell increased to 2.38 compared to 1.91 neurites/cell in the control dishes. Furthermore, Zhang et al. (2005) used EMF

stimulation (1.36 mT, 50Hz) for 96h with 10% duty cycle and found increased average length of neurites. Falone et al. (2007) studied the effect of 50 Hz and 1.0 mT EMF applied for 96h on human neuroblastoma (SH-SY5Y) cell culture and found an increase in the neurite outgrowth. Cruces-solis et al. (2010) applied EMF of 6.4 mT and frequencies of 7 and 10 Hz produced by rotating permanent magnet for 2 h daily on Westar rat chromaffin cells for 7-days. His results showed increased neurite outgrowth.

The present study has few limitations. First, changes seen after exposure to EMF were reported after only two weeks. Furthermore, only histological analyses were done as end point assessment. The study is descriptive in nature and future studies are recommended to study the underlying mechanism.

CONCLUSION

Within the limitation of this study, it could be concluded that the extremely low frequency (50 Hz) EMF was effective in increasing the number of Pacinian corpuscles in normal thoracolumbar fascia of adult Wistar rats after one hour, six days/week for 2 weeks of exposure to EMF.

REFERENCES

1. Adams MA, Burton AK, Dolan P, and Bogduk N. 2007. The

- Biomechanics of Back Pain. Churchill Livingstone.
2. Beard DJ, Dodd CAF, Trundle HR, and Simpson FARW. 1994. "Proprioception Enhancement in Cruciate Ligament Deficiency FO" : 654–59.
3. Bednar DA, Orr FW, and Simon GT. 1995. "Observations on the Pathomorphology of the Thoracolumbar Fascia in Chronic Mechanical Back Pain. A Microscopic Study." *Spine (Phila Pa 1976)*: 15;20(10):1161–64.
4. Blank M, and Goodman R. 2004. "Initial Interactions in Electromagnetic Field-Induced Biosynthesis." *Journal of cellular physiology* 199(3): 359–63.
5. Brumagne s, Cordo P, Verschueren S, Lysens R, and Swinnen S. 2000. "The Role of Paraspinal Muscle Spindles in Lumbosacral Position Sense in Individuals with and without Low Back Pain." *Spine* 25(8): 989–94.
6. Byl NN, and Sinnott P. 1991. "Variations in Balance and Body Sway in Middle-Aged Adults: Subjects with Healthy Backs Compared with Subjects with Low-Back Dysfunction." *Spine* 16(3).
7. Chaudhry H, Huang C, Schleip R, Ji Z, Bukiet B, and Findley T. 2007. "Viscoelastic Behavior of Human Fasciae under Extension in Manual Therapy." *Journal of Bodywork and Movement Therapies* 11(2): 159–67.

8. Cruces-solis H, Eli D, Verdugo-di L, and Herna H. 2010. "Neurite Outgrowth on Chromaffin Cells Applying Extremely Low Frequency Magnetic Fields by Permanent Magnets." 40: 545–50.
9. Cuccurazzu B, Leone L, Podda MV, Piacentini R, Elisa R, Ripoli C, Azzena GB, and Grassi C. 2010. "Exposure to Extremely Low-Frequency (50 Hz) Electromagnetic Fields Enhances Adult Hippocampal Neurogenesis in C57BL / 6 Mice." *Experimental Neurology* 226(1): 173–82.
10. Dhillon MS, Bali K, and Prabhakar S. 2012. "Differences among Mechanoreceptors in Healthy and Injured Anterior Cruciate Ligaments and Their Clinical Importance." *Muscles, ligaments and Tendons Journal* 2: 38–43.
11. Falone S, Grosside MR, Cinque B, D'Angelo B, Tettamanti E, Cimini A, Di Ilio C, and Amicarelli F. 2007. "Fifty Hertz Extremely Low-Frequency Electromagnetic Field Causes Changes in Redox and Differentiative Status in Neuroblastoma Cells." *The international journal of biochemistry & cell biology* 39(11): 2093–2106.
12. Fan W, Ding J, Duan W, and Zhu YM. 2004. "The Influence of Magnetic Fields Exposure on Neurite Outgrowth in PC12 Rat Pheochromocytoma Cells." 282: 325–28.
13. Gairns FW. 1930. "A Modified Gold Chloride Method for the Demonstration of Nerve Endings." *QJ Microsc Sci* 74: 151–55.
14. Gmitrov J, Ohkubo C, and Okano H. 2002. "Effect of 0.25 T Static Magnetic Field on Microcirculation in Rabbits." 229(September 2001): 224–29.
15. Gordon GA. 2007. "Designed Electromagnetic Pulsed Therapy :Clinical Applications." (December 2006): 579–82.
16. Gordon T, Brushart TM, Amirjani N, and Chan km. 2007. "The Potential of Electrical Stimulation to Promote Functional Recovery after Peripheral Nerve Injury — Comparisons between Rats and Humans" eds. H Millesi and R Schmidhammer. *How to Improve the Results of Peripheral Nerve Surgery SE - 1* 100: 3–11.
17. Grassi C, D'Ascenzo M, Torsello A, Martinotti G, Wolf F, Cittadini A, and Azzena G. 2004. "Effects of 50 Hz Electromagnetic Fields on Voltage-Gated Ca²⁺ Channels and Their Role in Modulation of Neuroendocrine Cell Proliferation and Death." *Cell Calcium* 35(4): 307–15.
18. Hestbaek L, Leboeuf-Yde C, Engberg M, Lauritzen T, Bruun NH, and Manniche C. 2003. "The Course of Low Back Pain in a General Population. Results from a 5-Year Prospective Study." *Journal of Manipulative*

- and Physiological Therapeutics 26(4): 213–19.
19. Ito H and Bassett CA. 1983. “Effect of Weak, Pulsing Electromagnetic Fields on Neural Regeneration in the Rat.” *Clinical orthopaedics and related research* 181: 283–90.
 20. Kassolik K, Jaskólska A, Kisiel-Sajewicz K, Marusiak J, Kawczyński A, and Jaskólski A. 2009. “Tensegrity Principle in Massage Demonstrated by Electro- and Mechanomyography.” *Journal of bodywork and movement therapies* 13(2): 164–70.
 21. Koch B, Kurriger G, and Brand RA. 1995. “Characterisation of the Neurosensory Elements of the Feline Cranial Cruciate Ligament.” *Journal of anatomy* 187 (Pt 2: 353–59.
 22. Konradsen L, Ravn JB, and Sorensen AI. 1993. “Proprioception at the Ankle: Anaesthetic Blockade of Ligament Receptors.” *British Editorial Society of Bone and Joint Surgery* 75(3).
 23. Leetun DT, Ireland ML, Willson JD, Ballantyne BT, and Davis IM. 2004. “Core Stability Measures as Risk Factors for Lower Extremity Injury in Athletes.” *Medicine & Science in Sports & Exercise* 36(6): 926–34.
 24. Leoci R, Aiudi G, Silvestre F, Lissner E, and Lacalandra GM. 2014. “Effect of Pulsed Electromagnetic Field Therapy on Prostate Volume and Vascularity in the Treatment of Benign Prostatic Hyperplasia: A Pilot Study in a Canine Model.” *The Prostate* 74(11): 1132–41.
 25. Maniadakis N, and Gray A. 2000. “The Economic Burden of Back Pain in the UK.” *Pain* 84(1): 95–103.
 26. Markov MS. 2007a. “Expanding Use of Pulsed Electromagnetic Field Therapies.” *Electromagnetic biology and medicine* 26(3): 257–74.
 27. ———. 2007b. “Pulsed Electromagnetic Field Therapy History, State of the Art and Future.” *The Environmentalist* 27(4): 465–75.
 28. McFarlane EH, Dawe GS, Marks M, and Campbell IC. 2000. “Changes in Neurite Outgrowth but Not in Cell Division Induced by Low EMF Exposure: Influence of Field Strength and Culture Conditions on Responses in Rat PC12 Pheochromocytoma Cells.” *Bioelectrochemistry and Bioenergetics*.
 29. Moraes MRB, Cavalcante MLC, Leite JAD, Macedo JN, and Sampaio MLB. 2011. “The Characteristics of the Mechanoreceptors of the Hip with Arthrosis.” *Journal of Orthopaedic Surgery and Research* 6(1): 58.
 30. O’Connor BL, and Gonzales J. 1979. “Mechanoreceptors of the Medial Collateral Ligament of the Cat Knee Joint.” *Journal of anatomy* 129(Pt 4): 719–29.

31. Okano H, Gmitrov J, and Ohkubo C. 1999. "Biphasic Effects of Static Magnetic Fields on Cutaneous Microcirculation in Rabbits." *Bioelectromagnetics* 20(3): 161–71.
32. Pacini S, Vannelli GB, Barni T, Ruggiero M, Sardi I, Pacini P, and Gulisano M. 1999. "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.
33. Panjabi MM. 1992. "The Stabilizing System of the Spine. Part I. Function, Dysfunction, Adaptation, and Enhancement." *Journal of spinal disorders* 5(4): 383–89; discussion 397.
34. Pilla AA, Muehsam DJ, Markov MS, and Sicken BF. 1999. "EMF Signals and Ion/ligand Binding Kinetics: Prediction of Bioeffective Waveform Parameters." *Bioelectrochemistry and Bioenergetics* 48(1): 27–34.
35. Schleip R, Vleeming A, Lehmann-Horn F, and Klingler W. 2007. "Letter to the Editor Concerning 'A Hypothesis of Chronic Back Pain: Ligament Subfailure Injuries Lead to Muscle Control Dysfunction' (M. Panjabi)." *European spine journal: official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society* 16(10): 1733–35; author reply 1736.
36. Sicken BF, Kanje M, Lundborg G, and Kurtz W. 1990. "Pulsed Electromagnetic Fields Stimulate Nerve Regeneration in Vitro and in Vivo." *Restorative Neurology and Neuroscience* 1(3): 303–9.
37. Sicken BF, Kanje M, Lundborg G, Herbst E, and Kurtz W. 1989. "Stimulation of Rat Sciatic Nerve Regeneration with Pulsed Electromagnetic Fields." *Brain Research* 485(2): 309–16.
38. Smith TL, Wong-Gibbons D, and Maultsby J. 2004. "Microcirculatory Effects of Pulsed Electromagnetic Fields." *Journal of Orthopaedic Research* 22(1): 80–84.
39. Tasset, I et al. 2012. "Neuroprotective Effects of Extremely Low-Frequency Electromagnetic Fields on a Huntington's Disease Rat Model: Effects on Neurotrophic Factors and Neuronal Density." *Neuroscience* 209: 54–63.
40. Waddell G. 2002. *The Back Pain Revolution (cross from Biomechanics of Back Pain)*. 2nd editio. edinburgh.
41. Walsh K, Cruddas M, and Coggon D. 1992. "Low Back Pain in Eight Areas of Britain." *J epidemiol community health* 49: 227–30.
42. Wyke B. 1970. "The Neurological Basis of Thoracic Spinal Pain." *Rheumatology* 10(7): 356–59.

43. Yahia L, Rhalmi S, Newman N, and Isler M. 1992. "Sensory Innervation of Human Thoracolumbar Fascia. An Immunohistochemical Study." *Acta orthopaedica Scandinavica* 63(2): 195–97.
44. Zhang Y, Ding J, Duan W, and Fan W. 2005. "Influence of Pulsed Electromagnetic Field with Different Pulse Duty Cycles on Neurite Outgrowth in PC12 Rat Pheochromocytoma Cells." *Bioelectromagnetics* 26(5): 406–11.