

A Brief Review of the Current State of Research on the Biological Effects of Weak Magnetic Fields

I. A. Shaev^a, V. V. Novikov^{a,*}, E. V. Yablokova^a, and E. E. Fesenko^a

^a *Institute of Cell Biophysics, Russian Academy of Sciences, Pushchino, Moscow oblast, 142290 Russia*

**e-mail: docmag@mail.ru*

Received December 27, 2021 revised December 27, 2021 accepted December 30, 2021

Abstract—Some current trends in the development of research on the effects and mechanisms of the biological action of weak and ultra-weak static magnetic fields, low-frequency alternating magnetic fields, combined magnetic fields, and radio frequency fields in combination with a static magnetic field are presented. Experimental studies in which interesting and somewhat unexpected effects of magnetic fields with strength significantly lower than the magnetic field of the Earth (including those with intensities close to zero) were observed, are considered. The data are given taking into account the materials of the joint annual meeting of the Society of Bioelectromagnetism and the European Association of Bioelectromagnetism “BioEM 2021” (September 26–30, 2021, Ghent, Belgium).

Keywords: weak magnetic fields, free radicals, reactive oxygen species, blood, neutrophils, malignant neoplasms

DOI: 10.1134/S0006350922020191

It has been repeatedly noted that changes in the activity of free radicals, such as intracellular reactive oxygen species (ROS) and reactive nitrogen species (RNS), endogenous antioxidant enzymes, and compounds that maintain the physiological concentrations of free radicals in cells, are among the most noticeable effects of exposure to a weak magnetic field (MF) [1–3]. These changes affect many physiological functions [1], can lead to DNA damage [4, 5], modify immune and inflammatory responses [6], may affect cell proliferation and differentiation [7, 8], wound treatment results [9], neuro-electrical activity [10], and behavior [11]. However, until recently, no acceptable and reliable hypothesis or mechanism has been developed that could adequately explain all the observed effects of static and low-frequency alternating magnetic fields on free radical processes.

Reactive free radicals (mainly ROS and RNS) are formed as a result of cellular metabolism, especially in mitochondria, as well as by oxidases of cellular and intracellular membranes. ROS include mainly singlet oxygen, superoxide, peroxides and the hydroxyl radical. RNS include peroxyxynitrite, which is the product of a reaction between nitric oxide and superoxide, and nitrogen dioxide; nitric oxide is generated in the cell by the corresponding synthases. Under normal conditions, the level of free radicals is controlled by various

inducible antioxidant enzymes, primarily superoxide dismutase, catalase, and glutathione peroxidase.

Modern theoretical approaches that are currently being developed indicate that the theoretical threshold of 100 μT for triggering the mechanism of radical pairs in low-frequency alternating and static MFs, determined on the basis of previously accepted, but, as it turned out, oversimplified calculations [12], is overestimated by about two orders of magnitude [13]. It was found that these outdated calculations did not consider resonance transitions between states created by electronic and nuclear moments, which showed a strong connection in the case of weak external MFs of the order of the geomagnetic field (GMF), 30–65 μT [13, 14]. In a weak field, the coupling between nuclei, nuclei and electrons, and Zeeman shifts in the energy levels of electrons and nuclei, can lead to transitions with resonance frequencies spanning from several hertz into the megahertz region [13, 15]. It is assumed within the framework of this concept that the activity of an alternating MF in the frequency range from units to tens of Hz can be caused by a change in the nuclear magnetic moments of protons interacting with each other. The interaction of proton spins in large biological molecules is used in NMR experiments to help understand the structure of these molecules [16]. The resonance frequencies for these interactions are usually from 1 to 20 Hz [13]. The experimentally detected high sensitivity to changes in the growth rate of fibro-

Abbreviations: ROS, reactive oxygen species; RNS, reactive nitrogen species; MF, magnetic field; GMF, geomagnetic field; EMF, electromagnetic field; SMF, static magnetic field.

sarcoma cells under a magnetic field with a frequency of 16 and 17 Hz at 9.8 μT indicates a long lifetime of the interaction of protons in a large biological molecule. The activity of the field in the megahertz range may be associated with the level of hyperfine energy, which is generated due to the interaction of nuclear spins and the magnetic moments of electrons. For hyperfine transitions in the Earth's magnetic fields, the frequencies are usually in the range of 1–20 MHz; for nuclear spins, they are less than a few kilohertz [13, 17]. It is convenient to consider both electronic and nuclear spins having both singlet (S) and triplet (T) states. Quantum numbers characterizing spin states are usually multiples of $1/2$. The T^+ and T^- states are tuned in frequency with a magnetic field, while the T_0 and S states are not.

The dynamics of the experimentally detected effects of MFs is satisfactorily explained if considered from a biological point of view; that is, shifts in radical concentrations in the body always tend to be compensated by feedback due to the activation of antioxidant systems [18]. For different biological objects, the resulting time delays in compensating for the concentration of radicals have different durations; therefore, significant and reproducible effects of the action of MFs can apparently be recorded under certain time modes of the field action on identical objects and under identical experimental conditions. The results of experiments on cell cultures adapted for cultivation outside the body under rather harsh conditions of standard biological incubators, which themselves are sources of strong magnetic interference, may not always be indicative [19]. Such objects, by virtue of their adaptability to the action of external MFs, may have short compensation times for radical concentrations, especially if the levels of ROS production are near their physiological values. In this regard, studies of the effect of MFs on native tissues (adapted to life in conditions of weak GMF), in particular, on blood [20, 21] and on its cellular components, including phagocytic neutrophil cells (one of the main producers of ROS in the blood), during short-term incubation outside the body in certain (created and clearly controlled) magnetic conditions are promising, [22–24]. It was possible to obtain stable and pronounced results of the effect of changes in the parameters of the magnetic fields (combined, “zero”, static and pulsed MFs) on the production of ROS [25–28] with these objects, which makes it possible to investigate the biophysical molecular mechanisms of the action of this physical factor [29–31].

It is believed that one of the most likely targets of the physical mechanism of reception of weak MFs are the processes of recombination of radical pairs, the rate of which varies depending on the induction of the field in the presence of which these processes occur

[13, 32]. In connection with the above, it seems that this mechanism is widespread in biological objects and is implemented in various radical pairs, the detailed characteristics of which have yet to be studied. In particular, this widely accepted but not yet fully proven model is used to explain magnetosensitivity in birds [33, 34]. One of the weak points of this model is that the theory predicts that the effect is not strong enough for it to manifest itself at the macroscopic level, since its influence must be suppressed by the internal electron-electron dipole interaction [35, 36]. The classical model of this mechanism assumes the presence of two geminal molecular radicals that appear as a result of the decay of a molecular precursor, either as a result of photoexcitation, or a cascade of reactions that do not depend on light. This radical pair initially consists of two physically separated molecules with correlated spins in a common singlet state. From this singlet state, the pair can evolve over time under the influence of local magnetic interactions that modulate its multiplicity (transition from singlet to triplet state and vice versa) and, consequently, the probability of its recombination. This dynamics is mediated by the Zeeman effect (the appearance of new energy sublevels in MF) in combination with local changes in the magnetic field resulting from the action of hyperfine interactions. Thus, hyperfine interactions of electrons with nuclei are the main driver of singlet triplet interconversion in this model. The anisotropy of these interactions (electron-nuclear dipole interaction) leaves an imprint of directivity on the magnetically sensitive characteristics of this process; this provides a theoretical basis for the “compass effect”, which allows the required direction of movement in space to be found (orientation by magnetic field of birds, insects, etc.). According to this model, chemical sensitivity to the induction and direction of the surrounding magnetic field arises in the photoreceptor protein cryptochrome located in the retina of the eye, where spin dynamics modulates the proportion of radical pairs that recombine without affecting pairs that “avoid” recombination due to their adoption of a structurally different state (for example, by changing the molecular conformation). Thus, a sensor is formed that is insensitive to the polarity of the field, but reacts to a narrow (but adjustable) range of magnetic field strength. This sensor depends on light (although the stage of magnetoreception may be photon-independent), and fails when weak radio frequency electromagnetic fields act. Thus, for example, a special receptor can function in some migratory birds, which is combined with vision so that the bird is able to “see” changes in MF by an order of magnitude of $1/1000$ of GMF, and navigate by the magnetic relief of the Earth [32].

An alternative scenario for the development of magnetobiological effects was formulated in the theoretical works of V. N. Binh [37–39], who believed that

the main interest of magnetobiologists should be associated with non-specific (independent of special receptors) magnetic effects observed in many organisms, from protozoa and fungi to insects, plants, fish, animals and humans [40]. In this scenario, in contrast to the mechanism of radical pairs, the external MF changes the dynamics of a single magnetic moment relative to the selected direction set by its local biophysical environment. In this case, the most significant changes in the dynamics of magnetic moments can occur in the “zero” MF, when the gap between the split Zeeman sublevels of magnetic moments becomes comparable to the width of the levels themselves. There are a few possible targets with a single magnetic moment in the body; these are the electron, proton, magnetic nuclei and the orbital moment of the electron. The gyromagnetic ratios and lifetimes of all these potential targets of the MF action differ significantly and are often known by an order of magnitude for different molecular environments. Therefore, in the presence of a detailed experimental dependence of the magnitude of the effect on the magnitude of the attenuated residual static MF in the range close to “zero”, there is a probability of successful calculation and determination of the target responsible for a specific magnetobiological effect. In this regard, we can mention the results of our experiments on the effect of a weakened static MF on the production of ROS by neutrophils, in which the anisotropy of the response was indeed revealed depending on the magnitude of the residual static MF [27, 41, 42]. It is hoped that in the presence of a more detailed experimentally determined dependence, such calculations will be possible to make and determine the corresponding primary targets of this magnetobiological effect.

It should be borne in mind that free radicals cannot be considered the only mechanism by which static and alternating MFs affect cell viability. Other mechanisms may also be involved, for example activation of the ERK1/2 signaling pathway [43], or mechanisms associated with heat shock proteins [44]. It was assumed that the direct target of a weak electromagnetic field (EMF) is a voltage sensor, which in normal physiology controls the opening of voltage gated calcium channels (VGCC) in response to partial depolarization of the plasma membrane [45]. Four different classes of VGCC are activated in response to low-level EMF exposure, namely, L-type, T-type, N-type, and P/Q-type VGCCs [45]. Voltage-gated sodium, potassium and chloride channels, each of which is controlled by a similar voltage sensor, are also activated when exposed to low-intensity EMF. The protein molecule of the voltage-gated calcium channel contains a four-domain structure in which each domain carries an α -helix, designated as an S4 helix, containing five positive charges. These four charged α -helices act together as a so-called voltage sensor, a structure

that reacts to electrical changes on the plasma membrane, opening the channel. The structure and location of the voltage sensor, as well as the use of Coulomb and Ohm's laws predict that the EMF forces acting on the voltage sensor are large, about 7.2 million times stronger than the forces acting on uniquely electrically charged groups in the aqueous parts of our cells. Voltage-gated sodium, potassium and chloride channels seem to play only a secondary role in the creation of EMF effects, so that in the first approximation, the effects can be explained by the predominant activation of VGCC and the subsequent increase in intracellular calcium. This explains why voltage sensors may be the main direct target of EMF. A large number of non-thermal pathophysiological effects of EMF can be explained by the action of VGCC activation caused by two different pathways, the calcium signaling pathway and the peroxynitrite/free radicals/oxidative stress/inflammation pathway. Static MF can act through a voltage sensor to activate VGCC and, presumably, other potential-dependent ion channels. Static MF does not create forces on static electrically charged objects; however, plasma membranes are constantly moving, and therefore, the VGCC voltage sensors located in the plasma membrane are also moving, so that static MF can create forces that vary in time on the charges of the VGCC voltage sensor. These possibilities clearly increase the likelihood of the situation in which highly penetrating, varying in time magnetic fields derived from MM waves or EMFs with other frequency, including extremely high densities of EMF modulating pulses, can have very high activity when acting directly on the 20 positive charges in the VGCC voltage sensor; this will lead to the activation of this channel.

Another hypothesis about the biological aspect of the interaction of low-frequency magnetic fields and cells has been presented [46]. The membrane protein of the endoplasmic reticulum STIM1 protein, which functions as a sensor for several cellular states (low levels of Ca^{2+} , temperature rise, elevated levels of oxygen radicals, and hypoxia), was assumed to be a promising candidate for the role of an MF sensor. Such sensory function can either be direct (through a local temperature increase caused by intracellular electric fields), or indirect due to a reaction to elevated ROS levels. Activated STIM1 leads to subsequent effects due to activation of signal transmission processes and changes in gene expression leading to secondary events. The nature of these changes will depend on both the type of cell and the specific physiological state that the cell exhibits during STIM1 activation. It was assumed that the oxidative processes triggered by MF play a key role in the biological effectiveness of this. It is not yet known whether STIM1 contains epitopes that might have magnetic dipole moments and therefore determine its special magnetic sensitivity. This model,

apparently, could be used to explain the already discovered effect of the increase in the concentration of intracellular Ca^{2+} in neutrophils under weak combined MF [29, 30], which is realized due to the release of calcium ions from intracellular depots (for example, from the endoplasmic reticulum), and is not associated with an increase in permeability cell membrane for external Ca^{2+} ions.

Our recent papers have presented the results of the effect of hypomagnetic conditions [24, 27, 41, 42] and combined magnetic fields [22, 23] on the production of ROS by mouse peritoneal neutrophils. The cells of neutrophils that were not activated were used as a simple model responding to the effects of hypomagnetic conditions; in this case it was possible to exclude additional factors associated with the restructuring of the metabolic mode of neutrophils during chemical stimulation by respiratory burst activators from consideration [24, 41, 42]. A decrease in intracellular ROS production under hypomagnetic conditions was revealed according to the data obtained by two methods, namely, fluorescence analysis using 2,7-dichloro-4-hydroxyfluorescein and dihydrodamine 123, and activated chemiluminescence with lucigenin (a relatively selective probe for the superoxide anion) [27]. Incubation of the neutrophil suspension under hypomagnetic conditions (less than $0.02 \mu\text{T}$) led to a significant decrease in the intensity of its lucigenin-dependent chemiluminescence (by approximately 30%). With an increase in the induction of the static field to $2.5 \mu\text{T}$, this effect disappeared and reappeared at $7.0 \mu\text{T}$; it was observed up to 30 and $44 \mu\text{T}$, at which point the results no longer differed from the control (in GMF with a static MF of $44 \mu\text{T}$) [27, 41]. Similar data were obtained by fluorescence analysis using 2,7-dichloro-4-hydroxyfluorescein [27, 42].

In order to specify the cellular locus responsible for the magnet dependence of ROS production in neutrophils under hypomagnetic conditions, an inhibitory analysis was performed; it demonstrated the potential of considering mitochondria as the locus, since 2,4-dinitrophenol, an uncoupler of oxidative phosphorylation, in the concentration range of $5\text{--}200 \mu\text{M}$ led to almost complete leveling of differences between the control and experimental samples, in contrast to diphenyliodonium (NADPH oxidase inhibitor), which reduced ROS production both in the control and in the experiment at concentrations from 2.5 to $100 \mu\text{M}$ [42].

On the contrary, the possible role of NADPH oxidase as the final link in the system of magnetically sensitive ROS production was revealed by experiments with activated neutrophils under the action of the combined MF [22]. The luminol-dependent chemiluminescent signal was significantly amplified in a suspension of neutrophils previously exposed to a weak

static field ($42 \mu\text{T}$) and a collinear low-frequency field (the sum of frequencies 1.0, 4.4 and 16.5 Hz , induction $0.86 \mu\text{T}$, exposure 1 h) in response to stimulation with bacterial peptide fMLF or forbol ester PMA [22]. The same method also demonstrated the opposite effect of reduced respiratory burst intensity in a neutrophil suspension in response to the fMLF activator under combined MF with other parameters (a static component of $60 \mu\text{T}$ and a collinear alternating low-frequency magnetic field with a frequency of 49.5 Hz and with induction in the range of $60\text{--}180 \text{ nT}$, at 40 min exposure) [23].

Experiments in this field are important not only because of the significant number of “white spots” regarding the understanding of the effects and mechanisms of weak MF on neutrophils, but also because of the special role that these cells play in the immune response. Neutrophils are the most mobile fraction of immunocompetent cells; they are the first to appear in the focus of inflammation and they serve as an initial link in the chain of protective reactions of the body [47]. Therefore, a change in the main characteristics of nonspecific immunity, as well as general immunoreactivity, can be achieved by exposure to MFs on these cells; these changes may be aimed at optimizing the immune response.

Studies of the effects of static and low-frequency alternating magnetic fields on cancer cells were widely presented at a seminar of the joint annual meeting of the Society of Bioelectromagnetism and the European Association of Bioelectromagnetism “BioEM 2021” (September 26–30, 2021, Ghent, Belgium) [48]. Studies of the therapeutic potential of weak static MF (SMF) in relation to cancer cells were carried out [49]. In early experiments, the effect of acceleration of division of human neuroblastoma cells with a simultaneous decrease in their mobility caused by screening of GMF (induction was $<200 \text{ nT}$) was discovered [50, 51]. However, the mechanism of this effect was difficult to determine in the absence of a special molecular marker. Therefore, at the next stage, the role of telomerase was studied, since this enzyme is key in tumor cells; its work underlies the main feature of the latter, the absence of the Hayflick limit. In addition to its classical role of affecting the telomere length, telomerase is also associated with the migration of cancer cells; overexpression of its telomerase reverse transcriptase (TERT) subunit promotes cell migration, whereas a decrease in the expression leads to a decrease in migration and adhesion ability [52, 53]. An acceleration of cell proliferation, inhibition of migration, a decrease in telomerase activity, and TERT expression were found under moderate SMF (approximately 150 mT) on 4T1 breast cancer cells. In particular, the number of cells in the experiment was the same as in the control after 24 h of exposure, it slightly increased after 48 h (11.02%), and significantly

increased after 72 h (19.28%). The efficiency of cell migration in the experiment was 71.68% of control after 24 h of exposure. To explain this effect, the authors considered the possibility of an increase in the expression of *e2f1*, a TERT transcription repressor and a positive regulator of the mitotic cell cycle. Although SMF in earlier studies was considered to play the role of an accelerator of 4T1 cell proliferation [54, 55], this result is not paradoxical, since the effects of SMF largely depended on cell types and the parameters of the field itself [56]. A potential application of these results may be associated with the increase in the effectiveness of some chemotherapeutic drugs specifically against rapidly dividing cells. Thus, it has been shown that the telomerase network reacts to SMF and can act as a target in the magnetotherapy of breast cancer. It is expected that in further studies specific parameters of magnetic conditions will be determined for the reduction of telomeres to a critical length and stop division, which should lead to aging of tumor cells.

Effects of weak static magnetic fields on human HT-1080 fibrosarcoma cells have been detected [57]. Experimental samples were exposed to these fields for 4 consecutive days; the induction of a static MF varied from 0.5 to 600 μT ; for the control, this parameter was maintained at 45 μT (which corresponded to GMF). It has been shown that the relative rates of cell growth vary depending on the magnitude of magnetic induction. An increase in the induction of the field led to an increase in the concentration of mitochondrial calcium, an increase in the membrane potential and a decrease in intracellular pH. The concentration of hydrogen peroxide increased at 100 and 200 μT , decreased at 300 and 400 μT , and increased again at 500 and 600 μT . In general, the intensity of oxidative stress increased slightly with an increase in SMF induction, while the concentrations of the superoxide anion and NO decreased. These results show that weak static MF can accelerate or inhibit the growth rate of HT-1080 fibrosarcoma tumor cells and cause changes in the concentration of ROS, which ultimately affects various cell functions, while the influx of calcium into the mitochondria was one of the first steps in the corresponding changes.

It was reported that proteins with iron-sulfur clusters in the electron transport chain in mitochondria are probably one of the important molecules interacting with external static and radiofrequency magnetic fields according to the radical pair model [58]. The effect of radiofrequency MF at frequencies from 1 to 10 MHz in combination with SMF on cell growth, intracellular pH, membrane potential, concentrations of hydrogen peroxide, and mitochondrial calcium was studied. Human HT-1080 fibrosarcoma cells were exposed to these fields for 4 days. The results showed

that cell growth and membrane potential depended on the frequency of the field, while the concentration of mitochondrial calcium did not show significant changes. In addition, radio frequency fields tend to reduce the concentration of hydrogen peroxide. It was concluded that a radio frequency field in combination with a static MF can change the yield of the reaction product if it is in resonance with the splitting of energy levels resulting from hyperfine and Zeeman interactions. The compartment responsible for magnetoreception can be the mitochondrial network, which in this case functions as a signal transmission system with frequency and amplitude modulation, and is also sensitive to various physiological variables, such as the concentration of ROS. It is believed that complexes I and III of the electron transport chain in mitochondria are the main sources of production of these ROS. Complex I contains the largest number of iron-sulfur clusters, most of which have paramagnetic properties and have relatively high spin states. Although Fe-S clusters are mainly known as electron carriers, they are present in the active centers of many enzymes, also providing a number of functions such as initiation and stabilization of radical chain reactions. It has been shown that various Fe-S clusters integrated into the protein structure can provide hyperfine interaction in the frequency range from 2.15 MHz to 3.85 MHz [58].

Summarizing the results of the studies on the anti-tumor effects of MF presented at the seminar in Ghent, we can assume that there are grounds for moderate optimism about the antitumor potential of magnetotherapy methods, which is consistent with the conclusions made in other works in this field [6, 59–61].

In conclusion, it should be noted that the current prevalence of experimental and theoretical approaches to the analysis of the mechanisms of biological effects of weak MFs based on the principles of spin chemistry does not exclude, but rather complements other areas of research in this field [62–70].

COMPLIANCE WITH ETHICAL STANDARDS

Conflict of interest. The authors declare that they have no conflicts of interest.

Statement on the welfare of humans or animals. This article does not contain any studies involving animals performed by any of the authors.

REFERENCES

1. H. Lai, *Electromagn. Biol. Med.* **38**, 231 (2019).
2. H. Wang and X. Zhang, *Int. J. Mol. Sci.* **18**, 2175 (2017).
3. B. Zhang and L. Tian, *Bioelectromagnetics* **41**, 573 (2020).

4. G. Giorgi, C. Pirazzini, M. G. Bacalini, et al., *Radiat. Environ. Biophys.* **56**, 193 (2017).
5. V. V. Novikov, V. O. Ponomarev, G. V. Novikov, et al., *Biophysics (Moscow)* **55**, 565 (2010).
6. E. G. Novoselova, V. V. Novikov, S. M. Lunin, et al., *Electromagn. Biol. Med.* **38**, 74 (2019).
7. A. V. Van Huizen, J. M. Morton, L. J. Kinsey, et al., *Sci. Adv.* **5**, eaau7201 (2019).
<https://doi.org/10.1126/sciadv.aau7201>
8. V. V. Novikov, I. M. Sheiman, and E. E. Fesenko, *Bioelectromagnetics* **29**, 387 (2008).
9. M. Jedrzejczak-Silicka, M. Kordas, M. Konopacki, and R. Rakoczy, *Int. J. Mol. Sci.* **22**, 5785 (2021).
<https://doi.org/10.3390/ijms22115785>
10. F. Barnes and B. Greenebaum, *Bioelectromagnetics* **41**, 213 (2020).
11. N. V. Bobkova, V. V. Novikov, N. I. Medvinskaya, et al., *Electromagn. Biol. Med.* **37**, 127 (2018).
12. R. K. Adair, *Bioelectromagnetics* **20**, 255 (1999).
13. F. S. Barnes and B. Greenebaum, *Bioelectromagnetics* **36**, 45 (2015).
14. V. O. Ponomarev and V. V. Novikov, *Biophysics (Moscow)* **54**, 163 (2009).
15. F. Barnes and B. Greenebaum, *Bioelectromagnetics* **41**, 392 (2020).
16. C. A. G. Haasnoot, F. A. A. M. de Leeuw, and C. Altona, *Tetrahedron* **36**, 2783 (1980).
17. F. Barnes and B. Greenebaum, *Environ. Res.* **163**, 165 (2018).
18. F. Barnes and S. Kandala, *Bioelectromagnetics* **39**, 249 (2018).
19. L. Portelli, T. Schomay, and F. Barnes, *Bioelectromagnetics* **34**, 337 (2013).
20. V. V. Novikov, E. V. Yablokova, and E. E. Fesenko, *Biophysics (Moscow)* **60**, 429 (2015).
21. V. V. Novikov, E. V. Yablokova, and E. E. Fesenko, *Biophysics (Moscow)* **61**, 105 (2016).
22. V. V. Novikov, E. V. Yablokova, and E. E. Fesenko, *Biophysics (Moscow)* **61**, 429 (2016).
23. V. V. Novikov, E. V. Yablokova, and E. E. Fesenko, *Biophysics (Moscow)* **65**, 82 (2020).
24. V. V. Novikov, E. V. Yablokova, and E. E. Fesenko, *Biophysics (Moscow)* **63**, 365 (2018).
25. V. V. Novikov, E. V. Yablokova, and E. E. Fesenko, *Electromagn. Biol. Med.* **39**, 364 (2020).
26. V. V. Novikov, E. V. Yablokova, I. A. Shaev, E. E. Fesenko, *Biophysics (Moscow)* **66**, 434 (2021).
27. V. V. Novikov, Yablokova, and I. A. Shaev, *IOP Conf. Ser.: Earth Environ. Sci.* **2021**, 853, 012008.
28. V. V. Novikov, E. V. Yablokova, A. P. Kadyrkov, and E. E. Fesenko, *Biophysics (Moscow)* **66**, 614 (2021).
29. V. V. Novikov, E. V. Yablokova, and E. E. Fesenko, *Biophysics (Moscow)* **62**, 440 (2017).
30. V. V. Novikov, E. V. Yablokova, G. V. Novikov, and E. E. Fesenko, *Biophysics (Moscow)* **62**, 759 (2017).
31. V. V. Novikov, E. V. Yablokova, and E. E. Fesenko, *Biophysics (Moscow)* **63**, 193 (2018).
32. P. J. Hore and H. Mouritsen, *Annu. Rev. Biophys.* **45**, 299 (2016).
33. R. Wiltshcko and W. Wiltshcko, *BioEssays* **28**, 157 (2006).
34. R. Wiltshcko and W. Wiltshcko, *J. R. Soc. Interface* **16**, 20190295 (2019).
35. N. S. Babcock and D. R. Kattinig, *J. Phys. Chem. Lett.* **11**, 2414 (2020).
36. N. S. Babcock and D. R. Kattinig, *JACS Au* **11**, 2033 (2021).
<https://doi.org/10.1021/jacsau.1c00332>
37. V. N. Binhi, *Biophysics (Moscow)* **61**, 170 (2016).
38. V. N. Binhi and F. S. Prato, *PLoS One* **12**, e0179340 (2017).
39. V. N. Binhi and F. S. Prato, *Bioelectromagnetics* **38**, 41 (2017).
40. T. K. Breus, V. N. Binhi, and A. A. Petrukovich, *Phys.—Usp.* **59**, 480 (2016).
41. V. V. Novikov, E. V. Yablokova, I. A. Shaev, and E. E. Fesenko, *Biophysics (Moscow)* **65**, 443 (2020).
42. V. V. Novikov, E. V. Yablokova, I. A. Shaev, and E. E. Fesenko, *Biophysics (Moscow)* **65**, 625 (2020).
43. L. Qiu, L. Chen, X. Yang, et al., *J. Cell. Physiol.* **234**, 7734 (2019).
44. O. Zeni, M. Simko, and M. R. Scarfi, *Front. Public Health* **18**, 280 (2017).
45. M. L. Pall, *J. Cell. Mol. Med.* **17**, 958 (2013).
46. M. Simko and M. O. Mattsson, *Med. Hypotheses* **122**, 68 (2019).
47. A. N. Mayansky, *Tsitokiny Vospalenie* **6** (3), 3 (2007).
48. *Abstr. of the Joint Annu. Meet. of the Bioelectromagnetics Society and the European Bioelectromagnetics Association (BioEM 2021). Workshop 3: Ultraweak and Weak Static, ELF, and RF Field Effects on Biological Systems* (Ghent, Belgium. 2021), pp. 306–319.
49. Z. Fan, P. Hu, L. Xiang, et al., *BioMed Res. Int.* **2020**, art. 7472618 (2020).
50. W. Mo, Y. Liu, P. Bartlett, and R. He, *Sci. China: Life Sci.* **57**, 448 (2014).
51. W. Mo, Z. Zhang, D. Wang, et al., *Sci. Rep.* **6**, 22624 (2016).
52. H. Liu, Q. Liu, Y. Ge, et al., *Sci. Rep.* **6**, 22886 (2016).
53. V. Maggisano, M. Celano, G. Lombardo, et al., *Mol. Cell. Endocrinol.* **448**, 34 (2017).
54. S. Tofani, D. Barone, M. Cintorino, et al., *Bioelectromagnetics* **22**, 419 (2001).
55. L. Zhang, J. Wang, H. Wang, et al., *Oncotarget* **7**, 41527 (2016).
56. L. Zhang, X. Ji, X. Yang, and X. Zhang, *Oncotarget* **8**, 13126 (2017).
57. H. Gurhan, R. Bruzon, S. Kandala, et al., *Bioelectromagnetics* **42**, 212 (2021).

58. H. Gurhan and F. Barnes, in *Abstr. of the Joint Annu. Meet. of the Bioelectromagnetics Society and the European Bioelectromagnetics Association (BioEM 2021)* (Ghent, Belgium, 2021), pp. 309–310.
59. X. Aoshu, Q. Wang, I. Xin, and T. Lin, *Front. Oncol.* **11**, 638146 (2021).
<https://doi.org/10.3389/fonc.2021.638146>
60. V. V. Novikov, G. V. Novikov, and E. E. Fesenko, *Bioelectromagnetics* **30**, 343 (2009).
61. L. Makinistian and I. Belyaev, in *Pulsed Electromagnetic Fields for Clinical Applications*, Ed. by M. S. Markov, J. T. Ryaby and E. I. Waldorff (CRC, Boca Raton, 2020), pp. 137–157.
62. V. V. Novikov and A. V. Karnaukhov, *Bioelectromagnetics* **18**, 25 (1997).
63. M. N. Zhadin, V. V. Novikov, F. S. Barnes, and N. F. Pergola, *Bioelectromagnetics* **19**, 41 (1998).
64. V. V. Novikov, E. V. Yablokova, and E. E. Fesenko, *Appl. Sci. (Switzerland)* **10**, 3326 (2020).
65. A. R. Liboff, *Electromagn. Biol. Med.* **39**, 45 (2020).
66. A. R. Liboff, C. Poggi, and P. Pratesi, *Electromagn. Biol. Med.* **36**, 154 (2017).
67. N. A. Belova and V. A. Panchelyuga, *Biophysics (Moscow)* **55**, 661 (2010).
68. E. D’Emilia, L. Giuliani, M. Ledda, et al., *Electromagn. Biol. Med.* **36**, 55 (2017).
69. A. Pazur, *Electromagn. Biol. Med.* **37**, 100 (2018).
70. L. Makinistian, *Sci. Rep.* **9**, 7478 (2019).

Translated by E. Puchkov