

Mechanism of the Biological Impact of Weak Electromagnetic Fields and the In Vitro Effects of Blood Degassing

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Abstract—The physical validity of the mechanism proposed by the author is discussed. According to the theory, prolonged exposure to weak electromagnetic fields leads to enlargement of microbubbles and degassing of bioliquid. Degassing alters the physical and chemical properties of bioliquid that affect some medical and biological indicators. The following changes in some blood parameters during degassing in vitro were analyzed: decrease in the glucose concentration, abnormal activation of blood clotting, increase in the rate of blood cell aggregation, decrease in the effectiveness of aspirin as an inhibitor of platelet aggregation and the slowing of indirect anticoagulants. All this is evidence of possible connection between environmental electromagnetic pollution and the risk of cardiovascular disease.

Keywords: electromagnetic field, water, microbubbles, degassing, plasma, cardiovascular disease

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INTRODUCTION

The task of elucidating the mechanisms of biological action of weak electromagnetic fields (EMF) is called forth by the ever-growing electromagnetic pollution of the environment by industrial and domestic sources. The question of the influence of weak fields on human health remains quite topical [1] and there is yet not clear answer to it, first of all because of the absence, despite all efforts [2], of a clear mechanism of action of weak EMF. The lack of grounded theoretical models keeps back the laboratory research on the impact of fields in vivo and in vitro [2]. Epidemiological studies traditionally associate electromagnetic pollution with increasing risk of oncological diseases in population groups living or working near the EMF sources [2, 3]. Why particularly oncological ones, it is not clear, the more so that no reliable elevation of the risk of such diseases as a result of EMF exposure has thus far been disclosed [3].

A similar problem exists in the area of water structuring or activation by external influence. Thus the authors of [4], having investigated the effects of EMF in triply distilled and deionized water, come to a conclusion that the field may act on the water-dissolved gas. In [5, 6] it is shown that low-intensity EMF change the relationship between the amounts of dissolved air and of gaseous air in microbubbles. It is reported that the water temperature practically does

not change thereby; however, the author supposes that it is the local temperature gradients created by EMF that lead to bubble enlargement. The possible role of microbubbles in the transduction of the energy of external influences has been also discussed in other works (see, e.g., [7, 8]).

There is a body of experimental evidence confirming the existence of air microbubbles in blood plasma [9–12]. Obviously, bioliquids such as blood plasma, cytoplasm, or interstitial fluid contain small amounts (~2 vol %) of dissolved air gases, and part of these gases under the laws of thermodynamics gather into microbubbles stabilized by adsorbed ions [13]. Nevertheless, none of the known mechanisms of biological action of weak EMF [2] does consider this fact. An exception to be mentioned is a work [9] showing that EMF-induced growth of microbubbles may affect the mass transfer across the erythrocyte membrane.

Any explanation of the biological action of weak EMF must answer at least three key questions: (1) What is the primary target of EMF? (2) How does the impact accumulate? (3) What is the cause of bioeffects? Recently the author has advanced a hypothesis [14] whereby it is the air microbubbles that are the primary targets in the action of weak EMF on water and bioliquids. Prolonged exposure in weak EMF leads to growth of microbubbles and degassing of the bioliquid—this is how impact accumulation takes place. The degassing of bioliquid alters its physicochemical properties, which tells on the medicobiological indices [15]. In [16–19] it has been shown that in vitro degas-

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sing of blood plasma substantially influences the results of clinical and biochemical analyses; i.e., the bioeffect of degassing is obvious.

The aim of the present work is, uniting the earlier results, to show that the risk of cardiovascular diseases (CVD) may be increased by prolonged action of weak EMF on the human organism. By the WHO data [20], CVD are the main cause of mortality worldwide (about 30%). It is believed that the main factors of risk of heart disease and stroke are improper nutrition, physical inertness and tobacco consumption, and also quite a number of factors influencing the development of chronic diseases—these are globalization, urbanization, and aging of the population. No less important causes of CVD appear to be the socioeconomic status and the psychological condition of the population. The present work is meant to draw attention to one more factor—the ever-growing electromagnetic pollution of the environment. The existence of such association might explain the recently traced tendency to growth of CVD in industrially developed countries, where the problem of electromagnetic pollution is quite topical.

PHYSICS OF DEGASSING BY ELECTROMAGNETIC FIELD

Water contacting with air presents an open heterogeneous system. A part of dissolved air forms a colloid of microbubbles. The bubbles are thermodynamically metastable because of adsorption of ions on their surface [21]. They arise as thermal fluctuations of density. Air hydrophobicity predetermines high surface tension and (Laplace) bubble pressure, while ion adsorption creates a backpressure stabilizing the bubble size. Bubbles (as well as drops) present traps for ions; this phenomenon is the basis of the operation of the bubble chamber (Wilson's chamber). The estimates of bubble radius are equivocal, just as the experimental data on the surface charge, because the states are metastable. In [13, 22–24] the mean bubble radius is $a \approx 70, 180, 20, \text{ and } 300 \text{ nm}$ respectively. We suppose that these microbubbles may serve a primary target for the impact of EMF on water and bioliquids.

The water–gas interface is charged negatively and attracts a diffuse cloud of counter-ions from solution [25, 26]. Therewith Laplace pressure $P_L = -2\sigma/a$, where σ is surface tension coefficient, is balanced by Coulomb repulsion of adsorbed ions $P_Q = Q^2/4\pi\epsilon_1\epsilon_0a^4$, where Q is bubble surface charge, ϵ_1 is gas permittivity, ϵ_0 is dielectric constant. At a preset surface charge density $\chi = Q/4\pi a^2$ the electrostatic pressure does not depend on bubble size. Charge density χ can be expressed to a potential gradient at the bubble outer surface as $\chi = \epsilon_2\epsilon_0\zeta/\delta$, where ϵ_2 is water permittivity, ζ is surface potential, δ is the effective double layer thickness. According to [26], the surface potential of a

bubble does not depend on its size and in distilled water of pH 7 it is $\zeta = -35 \text{ mV}$.

The electrophoretic mobility of a bubble is determined by the Smoluchowski equation $v_E = \epsilon_2\epsilon_0\zeta/\eta$, where η is dynamic viscosity (for water at normal conditions, $\sim 0.001 \text{ kg m}^{-1} \text{ s}^{-1}$). For typical household electric fields of 220 V/cm at 50 Hz this formula yields an estimate of maximum displacement $\sim 0.05 \text{ }\mu\text{m}$. Note that the latter does not depend on and is comparable to the bubble size. Such substantial fluctuations in the external field cannot but tell on bubble stability via exciting characteristic oscillation, attended either by collapse or by growth of bubbles [27].

Apart from that, bubble as density jumps can be polarized by an electric field [14]. The bubble polarization vector $\mathbf{d} = (\epsilon_1 - \epsilon_2)(\epsilon_1 + 2\epsilon_2)^{-1}a^3\epsilon_0\mathbf{E}_0$ is directed against the $\mathbf{E}_0$ field inasmuch as $\epsilon_1 < \epsilon_2$, which gives an a -independent positive increment to external pressure $\Delta P = -\mathbf{d}\mathbf{E}_0/4\pi a^3/3$ and a force pushing the bubble towards smaller fields, $\mathbf{F}_g = -\text{grad}(-\mathbf{d}\mathbf{E}_0)$, and the force of dipole–dipole attraction (on average), the radial value of which is $F_r = -3d^2/8\pi\epsilon_2\epsilon_0r^4$. Note that all these forces are independent of the $\mathbf{E}_0$ field direction.

It may be expected that in an alternating field the pressure surplus would lead to bubble growth by a mechanism of one-way diffusion known from acoustics [27] and, as a consequence, to growth of the gas phase volume in the «water plus air» colloid. Displacement against the field gradient would lead to reduction of the equilibrium concentration of bubble colloid in the corresponding region, to formation and leaving of new bubbles. Approach and coalescence also lead to bubble enlargement and flotation. Eventually we get a decrease in the quantity of gas molecules in solution—degassing, which is known to alter the physicochemical properties of liquid (conductivity, pH, transparency, surface tension). There are a number of hard-to-explain experiments with EMF action on aqueous solution, the results of which might be interpreted as EMF-induced degassing [4, 7, 8, 23, 28].

Admittedly, estimates of polarization effects prove to be negligible because of the smallness of the characteristic bubble size [14]. However, taking into account the conducting «coat» of counter-ions [21, 26], which is also polarized, one may expect more appreciable effects [29].

In an alternating magnetic field, there may be one more effect of the primary impact of EMF on water [29]. By the Faraday law, an alternating magnetic field induces an eddy current of counter-ions about the bubble and a current of rotation of the negatively charged bubble in the opposite direction. As a result, all bubbles acquire unidirectional magnetic moments, whereby they will mutually attract and be pulled into the region of larger fields.

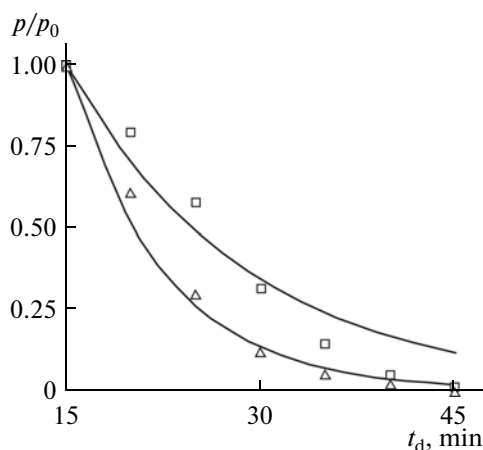


Fig. 1. Partial pressure of dissolved oxygen (squares) and carbon dioxide (triangles) in blood plasma samples as dependent on the time t_d of centrifugation at 3000 rpm, relative to that in control p_0 (data of [18]).

Thus, there are clear physical grounds for regarding microbubbles as a primary target of the EMF impact on water and bioliquids. Accumulation of the impact proceeds through liquid degassing as a result of bubble growth, fusion, and exit. Degassing in its turn alters the physicochemical properties of liquid—conductivity, acidity, transparency, etc. [14, 30]. The biological action of EMF is determined by the changes in the physicochemical properties of bioliquid during and after degassing.

MEDICOBIOLOGICAL EFFECTS OF BLOOD DEGASSING

The bioeffects of degassing can be modeled apart from EMF exposure. It has been shown [16–19] that blood plasma degassing in vitro substantially influences the biochemical and rheological parameters of blood.

One of the main functions that blood performs in the animal and human organism is the respiratory function. Blood, transporting air oxygen from the lung alveolar capillaries to tissue capillaries, thereby ensures continuous delivery of oxygen indispensable for normal vital processes in the organism. Therewith it is known [31] that all this oxygen must go through the stage of dissolution in the plasma. The concentration of dissolved oxygen in plasma is modest, just some 0.3 vol%, but it is this quantity that «governs» the direction and rate of oxygen transfer via hemoglobin. According to the conclusions in [10, 21, 26], a part of gas dissolved in electrolyte—which blood plasma in essence is—forms a colloid of microbubbles. By Henry's law, the content of blood-dissolved air and the equilibrium between the gas phase (colloid) and solu-

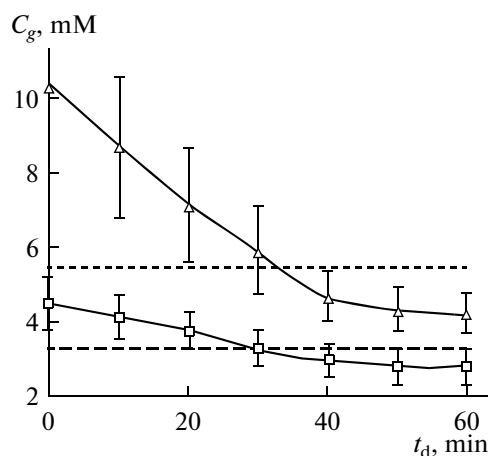


Fig. 2. Glucose concentration C_g in plasma of healthy donors (squares) and patients (triangles) after centrifugation for t_d min; dotted and dashed lines mark the upper and lower bounds of the norm (data of [17]).

tion in vivo can change upon variation of external conditions, e.g. atmospheric pressure, mountaineering or diving. To add, the gas phase may grow under the action of acoustic fields or EMF. As distinct from such possible degassing events in vivo, in the experiments in vitro, with the blood sample contacting the atmosphere, the rising bubbles take the air away from plasma, thereby more significantly degassing the entire liquid volume. Release of bubbles attending blood sedimentation has been observed microscopically [11].

During centrifugation of plasma samples, vibration causes rapid enlargement of microbubbles, while the centrifugal pressure accelerates their floatation to the surface. Figure 1 presents calibration curves [18] relating the duration of centrifugation with the extent of plasma degassing. The content of gases dissolved in plasma was determined in separate samples without access of air in a modular OMNI C analyzer (Roche, Switzerland).

Centrifugation of blood samples is an indispensable step in assaying the glucose content. Therewith it was shown [17] that the glucose content declines with increasing centrifugation time t_d (see Fig. 2). After 10 min centrifugation (by a standard protocol) the glucose content in plasma decreases by 10%. Longer duration or repeated centrifugation may take this index out of the normal range for healthy persons or put it within the norm in patients, which means that a diagnosis based on such data will be erroneous.

The level of glycosylated blood hemoglobin or its largest fraction HbA1c is the most exact and prognostic index among the biochemical indices of the degree of compensation and the course of diabetes mellitus [32]. The HbA1c content was determined [17] in

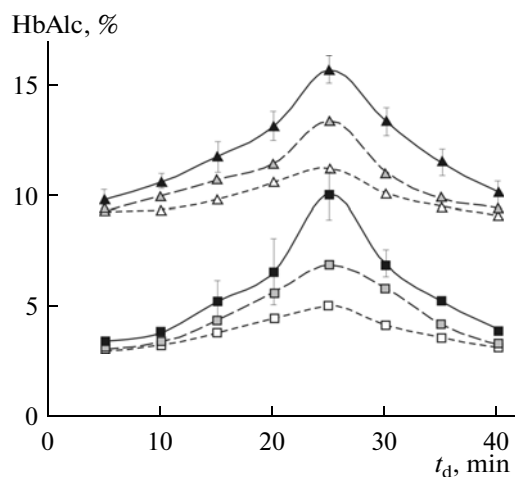


Fig. 3. Fraction of HbA1c in blood erythrocytes of healthy donors (squares) and patients (triangles) after (dotted) 10, (dashed) 20, and (solid lines) 30 min incubation in plasma pretreated in a centrifuge for t_d min (erythrocytes separated from plasma at $t_d = 5$ min) (data of [17]).

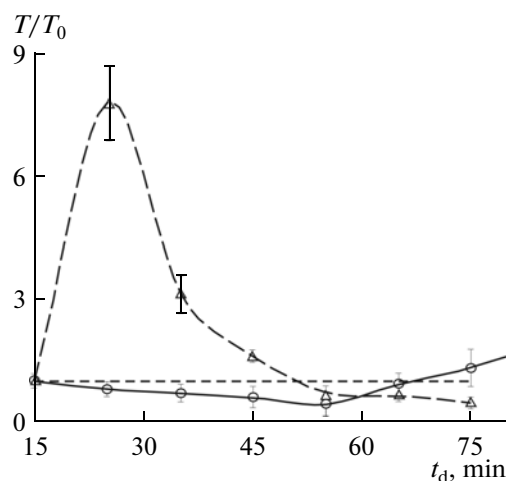


Fig. 4. Change in the clotting time (rel. un.) upon surplus centrifugation, $t_d > 15$ min. Solid line, extrinsic pathway, T_{ex}/T_{ex0} ($T_{ex0} = 0.4$ min at $t_d = 15$ min); dashed, intrinsic T_{in}/T_{in0} ($T_{in0} = 0.61$ min at $t_d = 15$ min); dotted line marks initial level (data of [16]).

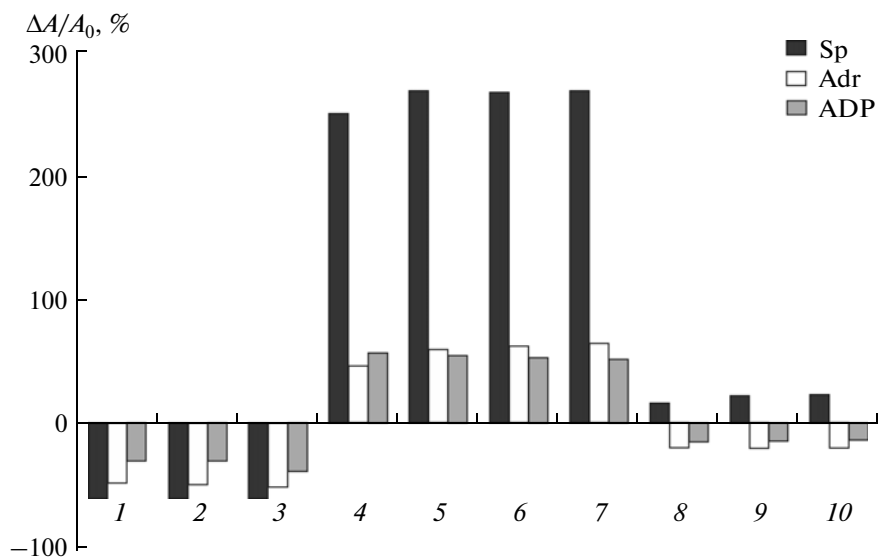


Fig. 5. Change in the extent of spontaneous (Sp), adrenaline-induced (Adr) and ADP-induced platelet aggregation (% of control) caused by: 1, 250 mg aspirin; 2, 325 mg aspirin; 3, 500 mg aspirin; 4, plasma degassing; 5, degassing + 250 mg aspirin; 6, degassing + 325 mg aspirin; 7, degassing + 500 mg aspirin; 8, normal + degassed plasma + 250 mg aspirin; 9, normal + degassed + 325 mg aspirin; 10, normal + degassed + 500 mg aspirin (data of [19]).

accordance with the diagnostic criteria [33, 34]. As evident from Fig. 3, upon increasing the centrifugation time to 25 min the share of HbA1c in plasma samples from healthy donors increased from 3 to 8%, and in diabetics from 10 to 15%. In this way, plasma degassing increases the glycosylation rate, which may lead to chemical modification of proteins inasmuch as it makes them more susceptible to oxidation even at a practically normal level of blood glucose.

In [16] we have examined the times of blood clotting by the intrinsic (T_{in}) and extrinsic (T_{ex}) pathways as dependent on centrifugation time t_d (see Fig. 4). Over the interval $15 < t_d < 25$ min, T_{in} grows sharply to a maximum at $t_d = 25$ min, and then more smoothly declines, crossing the initial normal value T_{in0} at $t_d = 50$ min (time of complete degassing according to Fig. 1). At $50 < t_d < 75$ min, T_{in} practically does not change, being half of T_{in0} . A totally different kinetics is

seen in clotting by the extrinsic pathway. Over the interval $15 < t_d < 25$ min, T_{ex} decreases roughly by half of the initial, and then grows at $t_d > 50$ min. Thus, maximal activity of blood and tissue prothrombinase is attained upon full degassing of plasma.

In [19] we studied the change in platelet function in vitro in response to removal of dissolved gases from plasma by centrifugation (Fig. 5). As inducers of aggregation, we used adrenaline and ADP, while as an inhibitor, aspirin. The rates of spontaneous and induced platelet aggregation decrease substantially upon adding aspirin (groups 1–3 in Fig. 5). However, plasma degassing leads to significant enhancement of both spontaneous and induced aggregation, and aspirin no longer changes the pattern regardless of dose (groups 4–7). At that, degassing reduces several times the effect of aggregation inducers inasmuch as it acts as an inducer by itself. To confirm the inhibitory action of dissolved gases, the degassed specimens were mixed with an equal amount of normal plasma (groups 8–10 in Fig. 5); in such conditions were observed substantial reduction of spontaneous aggregation and inhibition, albeit less efficient, of induced platelet aggregation by aspirin. Thus, upon removal of dissolved air in vitro the platelet aggregation rate grows while aspirin becomes ineffective as inhibitor of thrombogenesis. Deterioration of the rheological properties of blood upon a change in the concentration of dissolved gases in vivo may affect the microcirculation and substantially retard or completely stop the blood flow (stasis). A minor decrease in dissolved air concentration would be sufficient to cause appearance of thrombi. Degassing in vitro mimics a «pre-thrombotic state» in the blood of healthy persons.

Works [29, 35] report reliable correlation of the changes in blood indices caused by in vitro degassing with the changes usually taking place in CVD. Upon comparison of the indices of the hemostasis system in all cases we observe reduced efficiency of the natural clotting inhibitors, which manifests itself as significant reduction of T_{in} and T_{ex} [16]. In cardiological practice, for checking the efficiency and sufficiency of therapy with indirect anticoagulants (Warfarin, Syncoumar, etc.), widespread is a method that consists in calculating an international normalized ratio (INR) using reference (INR-standardized) thromboplastin [36]. Examination of blood from patients on Warfarin and Syncoumar has shown [35] that plasma degassing in vitro lowers the INR values 2–2.5 times. It is possible that a degassing effect of EMF in vivo would also block or hinder the action of these drugs. How then should one choose the dosage of anticoagulant and reduce the risk of overdosing, which is fraught with hemorrhages? Such a problem has not even been posed heretofore.

Thus, there are physical and medicobiological grounds for supposing that the nonthermal bioeffects of EMF are conditioned by degassing of bioliquids,

and that these effects may be regarded as a factor of added risk of CVD resulting from electromagnetic pollution of the environment.

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