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ABSTRACT

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Abstracts

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Liposome membranes with magnetite nanoparticles localized in the hydrophobic region can be considered as a magnetoelastic.
We studied an effect of external magnetic fields on liposomes containing hydrophobized magnetite nanoparticles in their membranes; carboxyfluorescein dye was used as a model substance loaded inside. The solution containing nanocomposite vesicles was kept in a constant magnetic field of 1.9 kOe for an hour. Before and after exposure a fluorescence intensity of the sample was measured.
A change in the fluorescence intensity of carboxyfluorescein indicates the release of a dye from the carrier vesicles into the solution under the influence of an external magnetic field. As in the case of electric field experiments, the vesicles exposed to the magnetic field were further characterized by TEM. Analysis of micrographs obtained by this method indicates a change in the shape of liposomes from quasi-spherical to ellipsoidal.
Theoretical calculations based on the analogy with an electrostatic model, as well as the numerical solution of the Laplace equation for a spherical ferrofluid layer in an external magnetic field, indicate that the shape of an ellipsoid extended along the direction of the external magnetic field strength is the most energetically favorable. Nanocomposite magnetic liposomes change their shape from spherical to ellipsoidal under the influence of an external magnetic field, the membranes of such vesicles are deformed, which leads to an increase in their permeability to dye molecules.
The effects we have discovered give us a possibility of creating new biomimetic biocompatible colloidal systems for encapsulating drugs that have a capabilities of controlled non-thermal release of encapsulated substances by using external physical influences.
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S9.587. Biophysical investigations nerve cell functions

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Using the modern optical methods (Raman spectroscopy, laser interference microscopy, confocal microscopy), molecular and cellular changes during the functioning of the nerve cell were studied. It was found that when a series of action potentials or receptor activation is commanded, not only the membrane potential changes, but the viscosity of the plasma membrane, as well as the state of the mitochondria and the cytoskeleton of the cell.

S9.588. Biophysical basis of epileptic activity: the hypothesis of membrane contamination

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The commonly accepted electrophysiological hallmark of epileptic activity is the paroxysmal depolarization shift (PDS). More than fifteen years ago it was hypothesized that epilepsy is associated with membrane pollution. This hypothesis includes the following assumptions: 1) PDS arises as result of the transition of pacemaker potentials into hypertrophied long-term high-amplitude depolarizations; 2) disruption of the order of the lipid bilayer (for example, as a result of the inclusion of amphiphilic substances) causes the transformation of pacemaker potentials into PDS [1].
Many well-known facts about epilepsy can be explained in the framework of this hypothesis, such as the fact that refractory epilepsy can be treated with a ketogenic diet [2] and vagus nerve stimulation [3], the antiepileptic effects of valproate, cholesterol and other membrane fluid stabilizers.
Additional studies are needed to clarify points 1 and 2. Previously, it was shown that by changing the parameters of the pacemaker potential model, one can trace the transition from the pacemaker potential to paroxysmal depolarization [2]. But the question of determining the channels involved in the generation of the pacemaker potential remains open. To model the described processes, both experimentally and mathematically [4], a new biophysical model is needed. We develop approaches to such a model in [5].
Indirect evidence supports the idea that bilayer contamination with amphiphilic substances can enhance epileptogenicity, but there is no clear demonstration that membrane pollution promotes transformation the pacemaker potential into paroxysmal depolarization [6].
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S9.589. Biophysical regularities of nitrogen monoxide and copper content changes in rat brain during simulated cerebral ischemia

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Nitric oxide (NO) is one of the key signaling molecules that regulate the body's physiological functions, including the nervous system,

both normal and pathological [1]. Studies of the role of NO in the vital activity of organisms began shortly after the discovery of the regulatory role of NO in normal vascular tone as a mediator of vasodilation [2,3,4]. Through synthases, the level of NO is controlled in neurons, neuroglia and microglia of the brain in normal and pathological conditions by enzymatic and non-enzymatic reactions. Under physiological conditions, the function of NO is consistent and in coordination with many other regulatory systems in the nervous tissue. Of great interest is the involvement of NO in the underlying mechanisms of the development of various pathological conditions in the body [5]. It was found that in some pathological processes NO plays both a protective role and a destructive one, which is governed by many factors [6]. The development of pathological processes in the brain (hypoxia and ischemia)is associated with an increase in the activity of the regulatory systems of the brain (including the NO system) This is naturally accompanied by an increase in oxygen consumption (which exacerbates hypoxia) and an increase in underoxidized products in brain tissue [4,7].

Although, the role of the NO system in these conditions is systematically across the globe, there are still many ambiguities in this fundamental and applied problem. One of the reasons for such a pessimistic situation is the technical complexity of regulating the NO level since NO is formed during rapid chemical reactions involving a wide range of molecules and intermediaries, including metals, thiols, free radicals, amino acids, calcium, and oxygen. It is relevant to study the biophysical patterns of changes in the NO content during ischemic processes in the brain. Therefore, the matter of using modern methods for detecting and quantifying the NO content in the tissues of living organisms in normal and experimental models of pathologies became relevant. One of the most effective methods for identification and quantifying NO in biological tissues is the electron paramagnetic resonance (EPR) method [8]. The authors attempted to detail some biophysical patterns of nitrogen monoxide formation in cerebral ischemia. This method is based on a technique developed by prof. Vanin et al that depends on the reaction of a radical (in this case, NO) with a spin trap. . As a result of the reaction, an adduct with a characteristic EPR spectrum is formed. The authors applied the Fe2+ complex with diethyldithiocarbamate (DETC) to capture NO and form a stable triple complex (DETC)2-Fe2+-NO in animal tissues. These complexes are characterized by an easily recognizable EPR spectrum with a g-factor value of g=2.035 – 2.040 and a triplet hyperfine structure [8,9]. The method has a sensitivity of 0.04–0.4 nM, allows direct measurements, and is highly sensitive due to the use of spin traps [8]. The work aimed o study the effects of experimental ischemic brain damage on the intensity of NO production and copper content (as an indicator of superoxide dismutase) in the hippocampus of rats using EPR spectroscopy with spin trap technique. The results show a significant decrease in NO content in the hippocampus 1 day after modeling ischemia by carotid artery ligation. When modeling ischemia with simultaneous intranasal administration of mesenchymal stem cells (MSCs), no significant difference in NO content was found between ischemic and control rats. After 2 days, the NO content in the hippocampus of ischemic rats was restored. In the hippocampus of rats in which ischemia was modeled with simultaneous intranasal administration of MSCs, no significant difference in NO content relative to ischemic rats was found after 2 days. The copper content in the rat hippocampus decreased unreliably 1 day after modeling ischemia caused by carotid artery ligation and there was a unreliably tendency to increase 2 days later. All in all, NO measurements indicate a tendency to restore the level of NO characteristic to the intact animals. So was found a tendency to increase the effectiveness of the antioxidant system both 1 and 2 days after ischemia.

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S9.590. Brownian-yet-not-Gaussian diffusion in brain's parenchyma: experimental evidence and mathematical modeling

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Brownian-yet-not-Gaussian (BnG) diffusion discovered about ten years ago [1] in some complex biophysical media is characterized by the nontrivial combination features: when one explores the mead-squared displacement of random walkers, it follows the usual linear time dependence but the spatial profile of their probability density function does not correspond to the normal distribution.

Among the different physical origins of such a process, it has been shown [2] that the BnG can emerge as a consequence of the quenched disorder when a marker diffuses in a random medium with a local correlation of inhomogeneities. Such a structure is typical for the extracellular space in the brain's parenchyma where local gaps uniformly filled by the interstitial fluid form a complex random porous structure at a large scale. The targeted search for features typical for the BnG already confirms their existence [3].

Thus, this work explores the data obtained using the MRI mapping of the Gd-based contrast agent concentrations in rat's brain in vivo. It is demonstrated that fitting the dynamic sequence of radial cross-sections of the concentration's spatiotemporal distribution follows the Laplacian probability. density function at intermediate time scales. Further, it is followed by its transition to the Gaussian one at large time scales (but supplied with narrowing localised central peak in the point of injection) that is in line with the theory of BnG homogenization process.

In addition to the direct processing of the biophysical experimental data, the modelling approach based on the master equation will be presented and discussed with a special focus on the perspectives for distinguishing between interpretations of underlying stochastic processes. In addition, outlooks for the usage of such models for quantification of the brain extracellular space's structure and topology from results of macroscopic dynamical pictures of the solute transport in the parenchyma will be discussed.

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