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Statistical Approach to Describing the Processes of Transport of Lipoproteins and Other Components in Blood Vessels – I

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To describe atherosclerotic processes in the intima of blood vessels, a statistical approach to describing non-equilibrium processes of blood component transport in the lumen-endothelium-intima system of blood vessels has been proposed, which involves taking into account the nature of interactions between blood components. Using the method of non-equilibrium statistical operator for the parameters of the abbreviated description, a system of transport equations has been obtained, which, within the framework of the selected model of component interaction, can describe non-Markovian in time and non-local in space processes of blood transport in vessels, taking into account possible reaction-diffusion processes in the vessel walls. A viscous reaction-diffusion description was used in the lumen, and a reaction-diffusion description in the endothelium-intima subsystem.

Keywords: *atherosclerosis; blood vessels; erythrocytes; lipoproteins; low-density lipoproteins; diffusion processes of ions and water molecules; non-equilibrium statistical operator; Maxwell's equations; peroxide free radicals; nitric oxide; macrophages.*

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1. Introduction

Atherosclerosis, as is known, is the accumulation of cholesterol and other fatty deposits in the intima of blood vessels, which leads to narrowing of their lumens. The consequences of this are the restriction of blood flow to vital organs, in particular, blood flow to the heart, lungs, liver, which can ultimately lead to various syndromes such as heart attacks, strokes.

In simple words, this is associated with thickening of the arterial wall and, as a result, blockage of the lumen of the arteries. Atherosclerotic fatty plaque consists of lipids, monocytes, foam cells and smooth muscle cells. The development of plaques begins with the accumulation of lipids, especially (low-density lipoproteins) LDL in the arterial wall. Then LDL oxidizes and stimulates inflammatory processes of macrophage recruitment to the lesion. Foam cells are formed from monocytes that have had endothelial oxidized LDL, and proinflammatory cytokines are activated in the process, which further stimulate macrophages to the lesion. Thus, it is now clear that endothelial dysfunction contributes to atherosclerosis, while normal function has an atheroprotective capacity. The endothelium plays an important role in the release of vasodilators, such as nitric oxide (NO), to counteract vasoconstrictors. NO is an important vasodilator responsible for maintaining vascular tone in the human body [1–12].

The current situation related to human cardiovascular diseases requires a more detailed understanding of the processes involving cholesterol (production and conversion to bile acids in the liver), free radicals, oxidants, antioxidants in the endothelium and intima, and the immune system. It is now firmly established that plaque formation in the intima of the circulatory system is caused by oxidized low-density lipoproteins (LDL) resulting from interaction with free radicals. The appearance of free radicals (as macromolecules, protein fragments, cell decay products, characterized by the presence of one, i.e. uncompensated electron in the shell) in the intima of blood vessels is mainly associated with inflammatory processes that can occur as a result of endothelial dysfunction [1–10, 12, 13], cell death,

biochemical reactions, the influence of viruses (influenza, etc.) and other factors that have entered the blood, and from it through the endothelium into the intima. In a recent paper [14], the authors conducted a detailed analysis of the influence of erythrocytes on the processes of vascular atherosclerosis and noted that circulating erythrocytes can, under appropriate circumstances, have a detrimental effect on the vascular endothelium [3, 6, 7, 13], participating in the development of plaques. This is largely due to the non-Newtonian nature of blood flow in vessels, which is one of the problems of mathematical modeling of such processes [15]. Mathematical models [16] of the cardiovascular system allow for detailed and quantitative investigation of both physiological and pathological conditions due to their ability to combine clinical data with physical knowledge of the processes underlying the function of this system.

In the works [17–21] a thorough review of the achievements of mathematical modeling of the processes of atherosclerotic cardiovascular diseases is given. Various mathematical models, their generalizations and important directions of further research are analyzed. The importance of mathematical modeling in revealing the complexities of lipid accumulation in macrophages during the formation of atherosclerotic plaque depending on the lipid load was noted in the works [22–24].

In the works [19, 21, 25–29] mathematical models describing the primary mechanisms that control the early stages of atherosclerosis are presented. They consider interactions between oxidized low-density lipoproteins (ox-LDL), monocytes/macrophages, cytokines, and foam cells at different spatial and temporal scales. The fluid-structure interaction problems used to describe the cardiovascular mechanics that occur between the blood and the arterial wall are related to a set of differential equations that describe the changing concentration of solutes. These models assume that there is an initial inflammatory phase associated with the development of the atherosclerotic lesion, and a longer, quasi-static process of plaque development within the arterial wall that follows the initial temporal process. In [19] it was shown how different concentrations of LDL in the blood and different immune responses can influence plaque development. The mathematical simulations used macroscopic reaction-diffusion transport equations for components and Navier-Stokes equations for averaged blood flow in vessels, in which the diffusion coefficients of components, chemical reaction coefficients, and viscosity coefficient are constants. At the same time, we are dealing with a complex, significantly spatially inhomogeneous system with different characteristic spatial and temporal scales. In addition, in particular, the diffusion coefficients of the components and the viscosity coefficient are related by the Kubo formulas to the time correlation functions "flow–flow" of the components $\langle \hat{\boldsymbol{j}}_\alpha(\boldsymbol{r};t)\hat{\boldsymbol{j}}_\xi(\boldsymbol{r}';t')\rangle$ and the "stress tensor-stress tensor" $\langle \hat{T}^{\lambda\nu}_\alpha(\boldsymbol{r};t)\hat{T}^{\lambda\nu}_\xi(\boldsymbol{r}';t')\rangle$, $\lambda,\nu = x,y,z$, which contain the mechanisms of the corresponding processes. In principle, in spatially inhomogeneous systems, the transport coefficients are functions of coordinates, and in the case of blood as a suspension, a non-Newtonian fluid, time memory effects with anomalous behavior of the coefficients are important transport [30–37]. The description of non-equilibrium processes in suspensions, non-Newtonian fluids is a complex problem [15, 38–40], in particular blood [14, 20, 41–46] taking into account the processes of erythrocyte aggregation. In addition, hemoglobin (hemo contains iron ions Fe^{2+}), present in erythrocytes, causes blood to behave as a magnetic fluid [47]. The magnetic characteristics of blood in arteries are of great hemodynamic interest in influencing the processes of atherosclerosis with potential clinical consequences [48–51].

An important issue is the kinetics of cytokines (pro- and anti-inflammatory) in the intima in the presence of inflammatory processes [52–55]. Anti-inflammatory cytokines play an important role in controlling the spread of atherosclerosis. At the same time, cytokines are produced by all types of cells involved in atherosclerosis, which act on different targets, exerting numerous effects, and are mainly responsible for the interaction between endothelial, leukocytes, smooth muscle cells and other living cells of the vessels. The work [55] shows that when a high density of ox-LDL and a moderate density of pro- and anti-inflammatory cytokines are present in the intima, inflammation will increase and reach the stage of chronic atherosclerosis. Another possibility is that if a high density of oxidized LDL, a moderate density of procytokines, and a high density of anticytokines are available in the intima, inflammation will increase and approach a state of coexistence equilibrium, but will not reach the stage of chronic atherosclerosis.

One of the important tasks in the processes of plaque formation in the endothelium-intima subsystem is to study the very slow diffusion of oxidized LDL and their uptake by macrophages. In the next section, we will consider this problem within the framework of a statistical model.

2. Statistical model

To describe atherosclerotic processes in the intima of blood vessels, we will use a statistical approach that takes into account the nature of the interactions of blood components that to one degree or another participate in these processes.

In a recent work [14], a detailed analysis of the influence of erythrocytes on the processes of vascular atherosclerosis was carried out. In particular, erythrocytes captured by a plaque feed the damaged vessel with lipids and prooxidant molecules, such as hemoglobin, heme, and iron. At the same time, iron captured by macrophages, which aggregate into plaques due to lysis and phagocytosis of erythrocytes, strongly contributes to the formation of foam cells and destabilization of the plaque and its subsequent rupture. In previous studies of atherosclerotic processes in the walls of blood vessels, it was believed that the influence of erythrocyte circulation was not important, although the non-Newtonian nature of blood flow is mainly associated with erythrocytes. The blood velocity shift (WSS) on the endothelial surface is important, since we have a non-Newtonian fluid and problems of erythrocyte aggregation, adhesion and aggregation of platelets [20]. In particular, in the works [56, 57] it was noted that atherosclerosis mainly occurs in areas with low/reduced WSS, and the authors of the work [58], as a result of a detailed analysis of the influence of WSS on healthy endothelial function, gave an important estimate that a healthy physiological value of WSS varies between $1.2 - 7\,\text{Pa}$ during the cardiac cycle. Wall shear stresses below approximately $1 - 1.2\,\text{Pa}$ lead to stagnant and recirculating processes [59] in the vessels, which is a disruption of healthy physiological blood flow and catalyzes the initiation and progression of atherosclerosis with increased residence time of low-density lipoproteins (LDL) [60, 61].

From this point of view, the most important barrier to atherosclerotic processes is the normal functioning of the vascular endothelium and therefore the study of problems with endothelial dysfunction is important [6, 7]. It is important to note here that the shear stress [62], created by blood flow on the endothelial layer, is the physiologically most important stimulus for the continuous formation of nitric oxide (NO), produced by endothelial NO synthase (eNOS), and is a fundamental factor in cardiovascular homeostasis. It regulates systemic blood pressure, vascular remodeling and angiogenesis [63]. In particular, NO regulates vascular tone by interacting with the enzyme guanylate cyclase (in the active site of which it binds to an iron atom and thus increases enzymatic activity) in smooth muscles and vascular permeability, and also inhibits platelet adhesion and aggregation, initiating a series of reactions that lead to vasodilation. It is already well known that NO production in endothelial cells is regulated by an increase in the concentration of calcium ions Ca^{2+} in the cytoplasm and by the shear stress caused by blood flow. The increase in the cytoplasmic concentration of calcium ions Ca^{2+} is mainly activated by adenosine triphosphate (ATP), which is released from erythrocytes and endothelial cells. In this case, erythrocytes can absorb NO, reducing the bioavailability of NO in blood vessels. In addition, NO, due to its reactivity, can react with O_2 [6] or free radicals, bind to heme-containing proteins, or interact with iron-sulfur centers:

$$\text{L-arg} + O_2 + 1.5\text{NADPH} \rightarrow \text{L-citr} + 1.5\text{NADP}^+ + \text{NO} + 2H_2O, \tag{1}$$

where L-arg is L-arginine, L-citr is L-citrulline, NADPH is an electron transport transmembrane molecular machine that catalyzes the transfer of electrons from NADP^+, which is being oxidized, to undergo this catalytic reaction with the formation of nitric oxide NO. From this point of view, mathematical modeling of diffusion and reactions [6, 7, 64, 65] involving nitric oxide is of great importance.

Blood exhibits complex rheological behavior, which is mainly related to its components, including erythrocytes, leukocytes, platelets and plasma. In general, as is known, blood is a liquid suspension (connective tissue) consisting of plasma $(50 - 60\%)$ — a liquid intercellular substance and $40 - 45\%$ of erythrocytes, leukocytes, in particular monocytes and platelets. Plasma contains $90 - 92\%$ of water, $7-8\%$ of proteins (albumins, globulins, fibrogens), 0.12% of glucose, up to $0.8-1\%$ of fats (triglycerides,

phospholipids, cholesterol), 0.9% of salts, in particular sodium, potassium, magnesium and calcium, as well as biologically active substances (hormones, cytokines, vitamins). The flow of blood, as a non-Newtonian fluid, interacts with the vascular endothelium, the macromolecules of which are elongated in the direction of blood movement and have the property of allowing the necessary blood components, LDL, HDL, monocytes, water molecules, etc., to pass through the intima layers through reverse osmotic processes.

From the point of view of statistical description, blood is a complex ion-molecular-macromolecular system. Erythrocytes, leukocytes, monocytes, platelets, LDL, HDL have a complex internal structure associated with their functions. From the theoretical description, we have the interaction of two subsystems: blood flow, which interacts with the endothelium–intima in the vessels. As structural elements of the model of such a system, we will take charged particles (positively and negatively charged ions, uncompensated electrons in free radicals), polar water molecules, macromolecular structures, which we will consider with the allocation of certain active centers (for example, vitamins E in LDL, uncompensated electrons in free radicals). The endothelium-intima subsystem, with which blood interacts, will be considered as a dynamic elastic membrane with a thickness of h, a porous structure through which all components necessary for functioning can penetrate (diffuse): oxygen O_2, CO_2, NO, LDL, HDL, monocytes (which in the intima transform into macrophages), vitamins C (ascorbate), etc. An important component of the endothelium-intima subsystem is the presence of free radicals as fragments of macromolecules, proteins, and cell decay products, characterized by the presence of uncompensated electrons in the structure, which are mainly associated with inflammatory processes. It is important to note that free radicals are practically absent in the lumen of blood vessels, due to the large number of antioxidants in the bloodstream. At the same time, in the intima, free radicals, interacting with molecules of α-tocopherol (vitamin E) on LDL (can be from 3 to 15, on average 6) convert it, respectively, into a radical [66]. And if the inflammatory process is present in the intima, then the process of oxidation of molecules of E by radicals continues. In the structure of oxidized vitamins E of LDL, localized negatively charged centers of uncompensated electrons appear, the value of which can be from -3, -4, -5, -6 and higher. Obviously, such large macroions of LDL cause electrical polarization of surrounding molecules, macromolecules, certain solvation processes. However, when all of the vitamin E molecules in a lipoprotein are oxidized, the lipid moiety becomes susceptible to lipid peroxidation and eventually the LDL is completely oxidized [25].

As a result of such processes, their properties change, they are characterized by subdiffusion (very slow diffusion), they cannot return from the intima to the lumen and are actively captured by macrophages. At the same time, the available vitamin C in the aqueous environment of the intima, which restores vitamin E, can slow down the process of oxidation of LDL by free radicals [67–70]. Moreover, HDL (which can have only one vitamin E and is practically not oxidized and when captured by macrophages it is able to leave such localization, taking cholesterol from plaques) in the intima also affects free radicals and can take low-density cholesterol from plaques and transform it from the intima into the lumen into the blood, and then to the liver. The importance of vitamin C and high plasma concentrations of high-density lipoproteins in the fight against plaque formation and its destruction was noted in the work [25]. In addition, the role of pro-inflammatory and anti-inflammatory cytokines [52–55], which are present in the endothelium-intima areas where the inflammatory process takes place, is important. Cytokines signal monocytes about the presence of inflammatory processes, and they in turn move from the lumen through the endothelium into the intima, where they transform into macrophages.

In such a blood–endothelium–intima system, we have a complex motion of translational, rotational, and vibrational components on different spatial and temporal scales. The Hamiltonian of such a system can be represented in the following form:

$$H(t) = \sum_{f}\sum_{l=1}^{N_f}\frac{m_f v_l^2}{2} + \frac{1}{2}\sum_{ff'}\sum_{l\neq l'}^{N_f N_{f'}}\Phi_{ff'}(\boldsymbol{r}_l,\boldsymbol{r}_{l'}) + \sum_{\alpha}\sum_{i=1}^{N_\alpha}\left(\frac{m_\alpha v_i^2}{2} + \frac{\overleftrightarrow{J}_\alpha : \boldsymbol{w}_i\boldsymbol{w}_i}{2}\right)$$

$$+\frac{1}{2}\sum_{\alpha\beta}\sum_{i\neq j}^{N_\alpha N_\beta}\Phi_{\alpha\beta}(\boldsymbol{r}_i,\boldsymbol{r}_j)+\sum_{\alpha f}\sum_{il}^{N_\alpha N_f}\Phi_{\alpha f}(\boldsymbol{r}_i,\boldsymbol{r}_l)+\sum_{f}\sum_{l=1}^{N_f}Z_f e\varphi(\boldsymbol{r}_l;t)-\sum_{\alpha}\sum_{i=1}^{N_\alpha}\boldsymbol{d}_i\cdot\boldsymbol{E}(\boldsymbol{r}_i;t)$$
$$+\sum_{\xi}\sum_{i=1}^{N_\xi}\sum_{a=1}^{M_\xi}\frac{m_a v_{ia}^2}{2}+\frac{1}{2}\sum_{\xi\zeta}\sum_{i\neq j}^{N_\xi N_\zeta}\sum_{a,b}^{M_\xi M_\zeta}\varphi_{ab}(|\boldsymbol{r}_{ia},\boldsymbol{r}_{jb}|)+\sum_{\xi}\sum_{i=1}^{N_\xi}\sum_{a\neq b}^{M_\xi}\phi_{ab}(\boldsymbol{r}_{ia},\boldsymbol{r}_{ib})$$
$$+\sum_{\xi}\sum_{i=1}^{N_\xi}\sum_{a}^{M_\xi}Z_a e\varphi(\boldsymbol{r}_{ia};t)+\sum_{\alpha\xi}\sum_{i,j}^{N_\alpha N_\xi}\sum_{a}^{M_\xi}\Phi_{\alpha a}(\boldsymbol{r}_i,\boldsymbol{r}_{aj})+\sum_{f\xi}\sum_{l,j}^{N_f N_\xi}\sum_{a}^{M_\xi}\Phi_{fa}(\boldsymbol{r}_l,\boldsymbol{r}_{aj})$$
$$+\sum_{s}\sum_{l=1}^{N_s}\sum_{a=1}^{M_s}\frac{m_a v_{ia}^2}{2}+\frac{1}{2}\sum_{ss'}\sum_{l\neq l'}^{N_s N_{s'}}\sum_{a,b}^{M_s M_{s'}}\varphi_{ab}(|\boldsymbol{r}_{la},\boldsymbol{r}_{l'b}|)+\sum_{s}\sum_{l=1}^{N_s}\sum_{a\neq b}^{M_s}\phi_{ab}(\boldsymbol{r}_{la},\boldsymbol{r}_{lb})$$
$$+\sum_{\alpha s}\sum_{i,l}^{N_\alpha N_s}\sum_{a}^{M_s}\Phi_{\alpha a}(\boldsymbol{r}_i,\boldsymbol{r}_{al})+\sum_{fs}\sum_{l,l'}^{N_f N_s}\sum_{a}^{M_s}\Phi_{fa}(\boldsymbol{r}_l,\boldsymbol{r}_{al'}),\tag{2}$$

where N_f is the number of ions (positively and negatively charged) of species f, their mass m_f and $\boldsymbol{v}_l$ is the velocity vector, $\Phi_{ff'}(\boldsymbol{r}_l,\boldsymbol{r}_{l'})$ is the pair interaction potential of ions of species f, f'; N_α is the number of molecules of the type α with mass m_α and moment of inertia tensor $\overleftrightarrow{J}_\alpha$, their $\boldsymbol{v}_i$ and $\boldsymbol{\omega}_i$ are the velocity and angular velocity vectors, $\Phi_{\alpha\beta}(\boldsymbol{r}_i,\boldsymbol{r}_j)$ and $\Phi_{\alpha f}(\boldsymbol{r}_i,\boldsymbol{r}_l)$ are the pair interaction potentials between molecules of the types α and β and between molecules α and ions of the type f in the lumen; $Z_f e\varphi(\boldsymbol{r}_l;t)$ is the effect on ions of the $Z_f e$ variety of the $\varphi(\boldsymbol{r}_l;t)$ potential of the electric field created by all ions, dipole molecules (water) of the blood, and $\boldsymbol{d}_i\cdot\boldsymbol{E}(\boldsymbol{r}_i;t)$ is the effect of this electric field on the dipole moment of molecule i of the α variety; N_ξ is the number of macromolecular structures of the order ξ, in which M_ξ of force centers — neutral or charged atoms are isolated (in particular, erythrocytes, in hemoglobins, in the hemoglobin of which the active center is the iron ion Fe^{2+}, in free radicals — active centers — uncompensated electrons, oxidized vitamins E in LDL in the intima region), $\boldsymbol{r}_{ia}$ and $\boldsymbol{v}_{ia}=\mathrm{d}\boldsymbol{r}_{ia}(t)/\mathrm{d}t$ denote, respectively, the position and velocity of atom a belonging to molecule i and having mass m_a, while φ_{ab} and ϕ_{ab} are atom-atom potentials between different molecules and intramolecular potentials related to atomic interactions within the molecule. The intermolecular atom–atom potential may consist of some short–range part, for example the Lennard–Jones potential, and the Coulomb interaction of point atomic charges, i.e.

$$\varphi_{ab}(r)=4\varepsilon_{ab}\left[\left(\frac{\sigma_{ab}}{r}\right)^{12}-\left(\frac{\sigma_{ab}}{r}\right)^{6}\right]+\frac{Z_a Z_b e^2}{r},\tag{3}$$

where the intensity of short–range interactions is determined by the parameters ε_{ab}, and the scale of distances is given by the diameters of atoms σ_a, so that $\sigma_{ab}=(\sigma_a+\sigma_b)/2$. The intramolecular atom–atom potential is responsible for the vibrational oscillations of atoms in the molecule and can be approximately replaced by the potential of a harmonic oscillator, namely $\phi_{ab}(r)=k_{ab}r^2$, where k_{ab} is the coefficient of the stiffness of the interaction between atoms a and b. For most real polar liquids, the coefficients k_{ab} are quite large and the effects of deformation of the molecule and changes in its dipole moments in electric fields can be neglected. For rigid models of polar liquids, a convenient representation is one in which the atomic phase variables $\boldsymbol{r}_{ia}(t)$ and $\boldsymbol{v}_{ia}(t)$ at each time t are expressed in terms of the translational and rotational motions of the molecule as a whole, i.e.

$$\boldsymbol{r}_{ia}(t)=\boldsymbol{r}_i(t)+\boldsymbol{\delta}_{ia}(t),\qquad \boldsymbol{v}_{ia}(t)=\boldsymbol{v}_i(t)+\boldsymbol{w}_i(t)\times\boldsymbol{\delta}_{ia}(t),$$

where $\boldsymbol{\delta}_{ia}$ is the position of atom a belonging to molecule i relative to its center of mass $\boldsymbol{r}_i=\sum_a m_a\boldsymbol{r}_{ia}/\sum_a m_a$, which has a translational velocity $\boldsymbol{v}_i$ and rotates with an angular velocity $\boldsymbol{w}_i$. N_s is the number of macromolecular structures of the endothelium–intima subsystem, which we consider as a dynamic porous structure with vibrational movements of components with pair interaction potentials $\Phi_{ss'}$ between them. Endothelium macromolecules are elongated in the direction of blood movement and have the property of passing the necessary blood components LDL, HDL, monocytes,

water molecules, etc. into the intima layers during reverse osmotic processes. The permeability of the endothelium of the arterial wall depends on the shear stress of the blood velocity on the endothelial surface. Therefore, we assume that in the near-surface active layer of the blood flow-endothelium, the diffusion of LDL, HDL, monocytes, water molecules and other necessary components diffuse in the perpendicular direction to the endothelium. For a theoretical description of non-equilibrium transport processes in the blood flow system in the vessels, we will choose a certain set of parameters of the abbreviated description, which can also be measured by experimental methods. The main parameters of the abbreviated description of non-equilibrium processes in the lumen subsystem (lumen volume V_1) will be chosen

$$\langle \hat{n}_f^{(1)}(\boldsymbol{r})\rangle^t, \quad \langle \hat{n}_\alpha^{(1)}(\boldsymbol{r},\bar{\boldsymbol{d}})\rangle^t, \quad \langle \hat{n}_{\xi a}^{(1)}(\boldsymbol{r})\rangle^t, \quad \langle \hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r})\rangle^t \tag{4}$$

are non-equilibrium average values of the densities of blood components, ions, water molecules, LDL, HDL, monocytes, and other components (erythrocytes, leukocytes, platelets) and the non-equilibrium value of the total blood momentum density, where the averaging $\langle(\ldots)\rangle^t = \int d\Gamma_N(\ldots)\varrho(x^N;t)$ is performed by the non-equilibrium distribution function (non-equilibrium statistical operator) $\varrho(x^N;t)$, N is the total number of particles in the system, $d\Gamma_i = dx_i = d\boldsymbol{r}_i d\boldsymbol{p}_i d\bar{\boldsymbol{d}}_i$ is an element of the phase space of the system, $\boldsymbol{r}_i$ $\boldsymbol{p}_i$ are the coordinate vector and momentum vector of the i-th particle, $\bar{\boldsymbol{d}}_j = \frac{\boldsymbol{d}_j}{|\boldsymbol{d}_j|}$ is the unit vector of the dipole orientation of molecule j. The non-equilibrium distribution function $\varrho(x^N;t)$ satisfies the Liouville equation with the Liouville operator corresponding to the Hamiltonian of the system (2). The index "1" will denote the lumen subsystem. Note that the system has a cylindrical geometry, the value of the coordinate z varies along the axis of the vessels, and the value of the radius vector $\bar{\boldsymbol{r}}$ varies across the vessels, from the middle of the vessels to the endothelium border $|\bar{\boldsymbol{r}}_{end}|$ and further to the intima border $|\bar{\boldsymbol{r}}_{int}|$, $|\bar{\boldsymbol{r}}_{int}| - |\bar{\boldsymbol{r}}_{end}| = h$ — the thickness of the endothelium-intima with a volume of V_2). The average values (4) correspond to the microscopic densities:

$$\hat{n}_f^{(1)}(\boldsymbol{r}) = \sum_{l=1}^{N_f} \delta(\boldsymbol{r} - \boldsymbol{r}_l) \tag{5}$$

is the microscopic number density of ions of type f,

$$\hat{n}_\alpha^{(1)}(\boldsymbol{r},\bar{\boldsymbol{d}}) = \sum_{j=1}^{N_\alpha} \delta(\boldsymbol{r} - \boldsymbol{r}_j)\,\delta(\bar{\boldsymbol{d}} - \bar{\boldsymbol{d}}_j) \tag{6}$$

is the microscopic number density of molecules (macromolecules) of the type α,

$$\hat{n}_{\xi a}^{(1)}(\boldsymbol{r}) = \sum_{j=1}^{N_\xi}\sum_{a}^{M_\xi} \delta(\boldsymbol{r} - \boldsymbol{r}_{ja}) \tag{7}$$

is the microscopic density of the number of active centers (atoms, charges) of the variety a with the total number of them M_ξ in the macromolecular structure of the type ξ,

$$\hat{n}_{er}^{(1)}(\boldsymbol{r}) = \sum_{j=1}^{N_{er}}\sum_{a}^{M_{er}} \delta(\boldsymbol{r} - \boldsymbol{r}_{ja}) + \sum_{j=1}^{N_{er}}\sum_{f}^{S_{er}} \delta(\boldsymbol{r} - \boldsymbol{s}_{jf}) \tag{8}$$

is the microscopic density of the number of active negatively charged centers of the f variety with Lagrangian coordinates $\boldsymbol{s}_{jf}$ with the total number of their S_f on the surface of the erythrocyte membrane and a of positively charged and magnetically active centers Fe^{2+} in the hemoglobin heme in the internal macromolecular structure of the erythrocyte,

$$\hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r}) = \sum_f \hat{\boldsymbol{p}}_f^{(1)}(\boldsymbol{r}) + \sum_\alpha \hat{\boldsymbol{p}}_\alpha^{(1)}(\boldsymbol{r},\bar{\boldsymbol{d}}) + \sum_\xi \hat{\boldsymbol{p}}_\xi^{(1)}(\boldsymbol{r}) + \sum_{er} \hat{\boldsymbol{p}}_{er}^{(1)}(\boldsymbol{r}) \tag{9}$$

is the microscopic density of the total pulse of blood components,

$$\hat{\boldsymbol{p}}_f(\boldsymbol{r}) = \sum_{l=1}^{N_f} \boldsymbol{p}_l\,\delta(\boldsymbol{r} - \boldsymbol{r}_l) \tag{10}$$

is the microscopic momentum density of ions of type f in the blood,

$$\hat{\boldsymbol{p}}_{\alpha}(\boldsymbol{r},\bar{\boldsymbol{d}}) = \sum_{i=1}^{N_{\alpha}} \boldsymbol{p}_i\,\delta(\boldsymbol{r}-\boldsymbol{r}_i)\,\delta(\bar{\boldsymbol{d}}-\bar{\boldsymbol{d}}_j) \tag{11}$$

is the microscopic momentum density of molecules (macromolecules) of the type α,

$$\hat{\boldsymbol{p}}_{\xi}(\boldsymbol{r}) = \sum_{i=1}^{N_{\xi}}\sum_{a=1}^{M_{\xi}} \boldsymbol{p}_a\,\delta(\boldsymbol{r}-\boldsymbol{r}_{ia}) \tag{12}$$

is the microscopic momentum density of the number of active centers (atoms, charges) of the type a with the total number of them M_{ξ} in the macromolecular structure of the type ξ.

$$\hat{\boldsymbol{p}}^{(1)}_{er}(\boldsymbol{r}) = \sum_{i=1}^{N_{er}}\sum_{a=1}^{M_{er}} \boldsymbol{p}_a\,\delta(\boldsymbol{r}-\boldsymbol{r}_{ia}) + \sum_{i=1}^{N_{er}}\sum_{f=1}^{S_{er}} \boldsymbol{p}_f\,\delta(\boldsymbol{r}-\boldsymbol{s}_{if}) \tag{13}$$

is the microscopic momentum density of the number of active negatively charged centers of the type f with Lagrangian coordinates $\boldsymbol{s}_{jf}$ with the total number of their S_f on the surface of the erythrocyte membrane and a of positively charged and magnetically active centers Fe^{2+} in the hemoglobin heme in the internal macromolecular structure of the erythrocyte.

In addition, macromolecular complexes, such as erythrocyte-erythrocyte, may form in the lumen, leading to aggregation processes. Such processes will be described by non-equilibrium density–density functions, in particular:

$$\left\langle \hat{G}^{(11)}_{\xi a,\zeta b}(\boldsymbol{r},\boldsymbol{r}')\right\rangle^t, \tag{14}$$

where

$$\hat{G}^{(11)}_{\xi a,\zeta b}(\boldsymbol{r},\boldsymbol{r}') = \hat{n}^{(1)}_{\xi a}(\boldsymbol{r})\,\hat{n}^{(1)}_{\zeta b}(\boldsymbol{r}'). \tag{15}$$

It is important to note that such paired non-equilibrium distribution functions of macromolecular structures (suspensions) through the Fourier transform in space and time are associated with dynamic structural factors that can be measured experimentally by neutron scattering [39].

In the endothelium-intima region, the parameters of the abbreviated description are the non-equilibrium average value of the densities of the components LDL, ox-LDL, HDL, macrophages, water molecules, ions, free radicals, oxidants, vitamin C, pro-inflammatory and anti-inflammatory cytokines, T-cells, macromolecular structures of the endothelium–intima subsystem:

$$\left\langle \hat{n}^{(2)}_{\gamma}(\boldsymbol{r})\right\rangle^t, \tag{16}$$

where

$$\left\langle G^{(22)}_{\gamma\zeta}(\boldsymbol{r},\boldsymbol{r}')\right\rangle^t = \left\langle \hat{n}^{(2)}_{\gamma}(\boldsymbol{r})\hat{n}^{(2)}_{\zeta}(\boldsymbol{r}')\right\rangle^t, \tag{17}$$

are paired non-equilibrium correlation functions that describe processes depending on the components, in particular oxidation by radicals of LDL, HDL; processes of reduction of vitamin E in LDL under the action of vitamin C; processes of LDL capture by macrophages, formation of pro-inflammatory and anti-inflammatory cytokines; processes of macrophage aggregation with formation and growth of plaques. In addition, paired non-equilibrium functions of particles of two subsystems are important

$$\left\langle \hat{G}^{(12)}_{\gamma\zeta}(\boldsymbol{r},\boldsymbol{r}')\right\rangle^t = \left\langle \hat{n}^{(1)}_{\gamma}(\boldsymbol{r})\hat{n}^{(2)}_{\zeta}(\boldsymbol{r}')\right\rangle^t, \tag{18}$$

where $\hat{n}^{(1)}_{\gamma}(\boldsymbol{r})$ is the microscopic density of the number of lumen components, and $\hat{n}^{(2)}_{\zeta}(\boldsymbol{r})$ is the microscopic density of endothelium–intima components, in particular, when $\zeta = s$, and s are the macromolecular structures of the endothelium-intima, then we actually have correlations at the boundary of the blood–endothelium–intima subsystems.

To find the non-equilibrium distribution function of the components of the system $\varrho(x^N;t)$, it is necessary to solve the Liouville equation with the Liouville operator corresponding to the Hamiltonian (2), which can be represented in the form:

$$i\hat{L}_N(t) = \sum_{f}\sum_{l}^{N_f} \boldsymbol{v}_l\cdot\frac{\partial}{\partial \boldsymbol{r}_l} - \sum_{ff'}\sum_{ll'}^{N_fN_{f'}} \left(\frac{1}{m_f}\frac{\partial}{\partial \boldsymbol{r}_l}\Phi_{ff'}(\boldsymbol{r}_l,\boldsymbol{r}_{l'})\cdot\frac{\partial}{\partial \boldsymbol{v}_l} + \frac{1}{m_{f'}}\frac{\partial}{\partial \boldsymbol{r}_{l'}}\Phi_{ff'}(\boldsymbol{r}_l,\boldsymbol{r}_{l'})\cdot\frac{\partial}{\partial \boldsymbol{v}_{l'}}\right)$$

$$
\begin{aligned}
&+\sum_{f}\sum_{l}^{N_f}\frac{Z_f e}{m_f}\frac{\partial}{\partial \boldsymbol{r}_l}\varphi(\boldsymbol{r}_l;t)\cdot\frac{\partial}{\partial \boldsymbol{v}_l}+\sum_{\alpha}\sum_{i}^{N_\alpha}\left(\boldsymbol{v}_i\cdot\frac{\partial}{\partial \boldsymbol{r}_i}+(\boldsymbol{w}_i\times\bar{\boldsymbol{d}}_i)\cdot\frac{\partial}{\partial \bar{\boldsymbol{d}}_i}\right)\\
&+\sum_{\alpha}\sum_{i}^{N_\alpha}\frac{1}{m_\alpha}\frac{\partial}{\partial \boldsymbol{r}_i}(\boldsymbol{E}(\boldsymbol{r}_i;t)\cdot\boldsymbol{d}_i)\cdot\frac{\partial}{\partial \boldsymbol{v}_i}+\sum_{\alpha}\sum_{i}^{N_\alpha}\boldsymbol{d}_i\times\boldsymbol{E}(\boldsymbol{r}_i;t)\cdot\frac{\partial}{\partial \overleftrightarrow{J}_i\boldsymbol{\omega}_i}\\
&-\sum_{\alpha\beta}\sum_{ij}^{N_\alpha N_\beta}\left(\frac{1}{m_\alpha}\frac{\partial}{\partial \boldsymbol{r}_i}\Phi_{\alpha\beta}(\boldsymbol{r}_i,\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \boldsymbol{v}_i}+\frac{1}{m_\beta}\frac{\partial}{\partial \boldsymbol{r}_j}\Phi_{\alpha\beta}(\boldsymbol{r}_i,\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \boldsymbol{v}_j}\right)\\
&-\sum_{\alpha\beta}\sum_{ij}^{N_\alpha N_\beta}\left(\bar{\boldsymbol{d}}_i\times\frac{\partial}{\partial \bar{\boldsymbol{d}}_i}\Phi_{\alpha\beta}(\boldsymbol{r}_i,\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \overleftrightarrow{J}_i\boldsymbol{\omega}_i}+\bar{\boldsymbol{d}}_j\times\frac{\partial}{\partial \bar{\boldsymbol{d}}_j}\Phi_{\alpha\beta}(\boldsymbol{r}_i,\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \overleftrightarrow{J}_j\boldsymbol{\omega}_j}\right)\\
&-\sum_{\alpha f}\sum_{il}^{N_\alpha N_f}\left(\frac{1}{m_\alpha}\frac{\partial}{\partial \boldsymbol{r}_i}\Phi_{\alpha f}(\boldsymbol{r}_i,\boldsymbol{r}_l)\cdot\frac{\partial}{\partial \boldsymbol{v}_i}+\frac{1}{m_f}\frac{\partial}{\partial \boldsymbol{r}_l}\Phi_{\alpha f}(\boldsymbol{r}_i,\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \boldsymbol{v}_l}\right)\\
&-\sum_{\alpha f}\sum_{il}^{N_\alpha N_f}\bar{\boldsymbol{d}}_i\times\frac{\partial}{\partial \bar{\boldsymbol{d}}_i}\Phi_{\alpha f}(\boldsymbol{r}_i,\boldsymbol{r}_l)\cdot\frac{\partial}{\partial \overleftrightarrow{J}_i\boldsymbol{\omega}_i}+\sum_{\xi}\sum_{i}^{N_\xi}\boldsymbol{v}_i\cdot\frac{\partial}{\partial \boldsymbol{r}_i}+\sum_{\xi}\sum_{i}^{N_\xi}\sum_{a}^{M_\xi}\boldsymbol{w}_i\times\boldsymbol{\delta}_{ia}\cdot\frac{\partial}{\partial \boldsymbol{\delta}_{ia}}\\
&-\sum_{\xi\zeta}\sum_{ij}^{N_\xi N_\zeta}\sum_{a,b}^{M_\xi M_\zeta}\left(\frac{1}{m_\xi}\frac{\partial}{\partial \boldsymbol{\delta}_{ia}}\varphi_{ab}(\boldsymbol{r}_i,\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \boldsymbol{v}_i}+\frac{1}{m_\zeta}\frac{\partial}{\partial \boldsymbol{\delta}_{jb}}\varphi_{ab}(\boldsymbol{r}_i,\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \boldsymbol{v}_j}\right)\\
&-\sum_{\xi}\sum_{i}^{N_\xi}\mathbf{I}_i{}^{-1}\boldsymbol{w}_i\times(\mathbf{I}_i\boldsymbol{w}_i)\cdot\frac{\partial}{\partial \boldsymbol{w}_i}\\
&-\sum_{\xi\zeta}\sum_{ij}^{N_\xi N_\zeta}\sum_{a,b}^{M_\xi M_\zeta}\left(\mathbf{I}_i{}^{-1}\boldsymbol{\delta}_{ia}\times\frac{\partial}{\partial \boldsymbol{\delta}_{ia}}\varphi_{ab}(\boldsymbol{r}_i,\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \boldsymbol{w}_i}+\mathbf{I}_j{}^{-1}\boldsymbol{\delta}_{jb}\times\frac{\partial}{\partial \boldsymbol{\delta}_{jb}}\varphi_{ab}(\boldsymbol{r}_i,\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \boldsymbol{w}_j}\right)\\
&-\sum_{\xi f}\sum_{il}^{N_\xi N_f}\sum_{a}^{M_\xi}\left(\frac{1}{m_\xi}\frac{\partial}{\partial \boldsymbol{\delta}_{ia}}\Phi_{af}(\boldsymbol{r}_{ia},\boldsymbol{r}_l)\cdot\frac{\partial}{\partial \boldsymbol{v}_i}+\frac{1}{m_f}\frac{\partial}{\partial \boldsymbol{r}_l}\Phi_{af}(\boldsymbol{r}_{ia},\boldsymbol{r}_l)\cdot\frac{\partial}{\partial \boldsymbol{v}_l}\right)\\
&-\sum_{\xi\alpha}\sum_{ij}^{N_\xi N_\alpha}\sum_{a}^{M_\xi}\left(\frac{1}{m_\xi}\frac{\partial}{\partial \boldsymbol{\delta}_{ia}}\Phi_{a\alpha}(\boldsymbol{r}_{ia},\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \boldsymbol{v}_i}+\frac{1}{m_\alpha}\frac{\partial}{\partial \boldsymbol{r}_j}\Phi_{a\alpha}(\boldsymbol{r}_{ia},\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \boldsymbol{v}_j}\right.\\
&\left.+\mathbf{I}_i^{-1}\boldsymbol{\delta}_{ia}\times\frac{\partial}{\partial \boldsymbol{\delta}_{ia}}\Phi_{a\alpha}(\boldsymbol{r}_{ia},\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \boldsymbol{\omega}_i}+\bar{\boldsymbol{d}}_j\times\frac{\partial}{\partial \bar{\boldsymbol{d}}_j}\Phi_{a\alpha}(\boldsymbol{r}_{ia},\boldsymbol{r}_j)\cdot\frac{\partial}{\partial \overleftrightarrow{J}_i\boldsymbol{\omega}_i}\right),
\end{aligned}
\tag{19}
$$

where $\mathbf{I}_i=\sum_a m_a[(\boldsymbol{\delta}_{ia}\cdot\boldsymbol{\delta}_{ia})\mathbf{1}-\boldsymbol{\delta}_{ia}\boldsymbol{\delta}_{ia}]$ denotes the matrix of moments of inertia of a molecule with mass $m_\alpha=\sum_a^{M_\alpha}m_a$, and $\mathbf{1}$ is the identity matrix, $\bar{\boldsymbol{d}}_j=\frac{\boldsymbol{d}_j}{|\boldsymbol{d}_j|}$ is the unit vector of the dipole orientation of molecule j. By substituting in the corresponding sums $\xi\zeta\to ss'$, $\xi\zeta\to\xi s'$, $\xi f\to sf$, $\xi\alpha\to s\alpha$ in (19) we obtain the contributions to the Liouville operator from the macromolecular structures of the endothelium-intima subsystem.

To solve the Liouville equation for the non-equilibrium distribution function $\varrho(t)$ with the Liouville operator (19), we will use the method of the non-equilibrium statistical operator [71–73] with reference to the description of diffusion-reaction processes. The general solution of the Liouville equation taking into account the design can be presented in the form:

$$
\varrho(t)=\varrho_{rel}(t)-\int_{-\infty}^{t}e^{\varepsilon(t'-t)}T_{rel}(t,t')\big(1-P_{rel}(t')\big)\,iL_N(t')\,\varrho_{rel}(t')\,dt',
\tag{20}
$$

where $T_{rel}(t,t')$ is the time evolution operator

$$
T_{rel}(t,t')=\exp\left\{-\int_{t'}^{t}(1-P_{rel}(t''))\,iL_N\,dt''\right\}
\tag{21}
$$

with the Kawasaki–Gunton projection operator, the structure of which depends on the relevant distribution function $\varrho_{rel}(t)$, which is determined from the extremum of the Gibbs information entropy, while maintaining the normalization conditions $\varrho_{rel}(t)$ and with fixed values of the main set of parameters of the abbreviated description (4), (14), (16)–(18) and a fixed pressure P. In this case, $\varrho_{rel}(t)$ has the form:

$$\varrho_{rel}(x^N;t) = \exp\Bigg(- \Phi(t) - \beta\Bigg(H(t) - PV_1 - \sum_f \sum_{\kappa=1}^{2} \int_{V_\kappa} d\boldsymbol{r}\, \nu_f^\kappa(\boldsymbol{r};t)\, \hat{n}_f^\kappa(\boldsymbol{r}) - \sum_\alpha \sum_{\kappa=1}^{2} \int_{V_\kappa} d\boldsymbol{r} \int d\bar{\boldsymbol{d}}\, \nu_\alpha^\kappa(\boldsymbol{r},\bar{\boldsymbol{d}};t)\, \hat{n}_\alpha^\kappa(\boldsymbol{r},\bar{\boldsymbol{d}}) - \sum_\xi \sum_a^{M_\xi} \sum_{\kappa=1}^{2} \int_{V_\kappa} d\boldsymbol{r}\, \mu_{\xi a}^\kappa(\boldsymbol{r};t)\, \hat{n}_{\xi a}^\kappa(\boldsymbol{r}) - \sum_{\gamma\zeta} \sum_{\kappa\lambda=1}^{2} \int_{V_\kappa} d\boldsymbol{r} \int_{V_\lambda} d\boldsymbol{r}'\, \mu_{\gamma\zeta}^{\kappa\lambda}(\boldsymbol{r},\boldsymbol{r}';t)\, \hat{G}_{\gamma\zeta}^{\kappa\lambda}(\boldsymbol{r},\boldsymbol{r}') - \int_{V_1} d\boldsymbol{r}\, \boldsymbol{v}^{(1)}(\boldsymbol{r};t)\, \hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r}) \Bigg)\Bigg), \tag{22}$$

where

$$\Phi(t) = \ln \int d\Gamma_N \exp\Bigg(- \beta\Bigg(H(t) - PV_1 - \sum_f \sum_{\kappa=1}^{2} \int_{V_\kappa} d\boldsymbol{r}\, \nu_f^\kappa(\boldsymbol{r};t)\, \hat{n}_f^\kappa(\boldsymbol{r}) - \sum_\alpha \sum_{\kappa=1}^{2} \int_{V_\kappa} d\boldsymbol{r} \int d\bar{\boldsymbol{d}}\, \nu_\alpha^\kappa(\boldsymbol{r},\bar{\boldsymbol{d}};t)\, \hat{n}_\alpha^\kappa(\boldsymbol{r},\bar{\boldsymbol{d}}) - \sum_\xi \sum_a^{M_\xi} \sum_{\kappa=1}^{2} \int_{V_\kappa} d\boldsymbol{r}\, \mu_{\xi a}^\kappa(\boldsymbol{r};t)\, \hat{n}_{\xi a}^\kappa(\boldsymbol{r}) - \sum_{\gamma\zeta} \sum_{\kappa\lambda=1}^{2} \int_{V_\kappa} d\boldsymbol{r} \int_{V_\lambda} d\boldsymbol{r}'\, \mu_{\gamma\zeta}^{\kappa\lambda}(\boldsymbol{r},\boldsymbol{r}';t)\, \hat{G}_{\gamma\zeta}^{\kappa\lambda}(\boldsymbol{r},\boldsymbol{r}') - \int_{V_1} d\boldsymbol{r}\, \boldsymbol{v}^{(1)}(\boldsymbol{r};t)\, \hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r}) \Bigg)\Bigg) \tag{23}$$

is the Massier–Planck functional, the indices κ, λ take the values 1 — the lumen subsystem and 2 — the endothelium–intima subsystem, $\nu_f(\boldsymbol{r};t) = \mu_f(\boldsymbol{r};t) + Z_f e\varphi(\boldsymbol{r};t)$ are the electrochemical and $\mu_f(\boldsymbol{r};t)$ are the chemical potentials of ions of the f type, depending on the coordinates and time in the corresponding subsystem, $\nu_\alpha(\boldsymbol{r};t) = \mu_\alpha(\boldsymbol{r};t) + \boldsymbol{d}_\alpha \cdot \boldsymbol{E}(\boldsymbol{r};t)$ are the dipole–chemical and chemical $\mu_\alpha(\boldsymbol{r};t)$ potentials of molecules of the α type, depending on coordinates and time in the corresponding subsystem, $\mu_{\gamma\zeta}^{\kappa\lambda}(\boldsymbol{r},\boldsymbol{r}';t)$ is the chemical potential of the complex of macromolecular structures of the $\gamma\zeta$ varieties depending on coordinates and time in the corresponding subsystems, in particular erythrocyte–erythrocyte in the first subsystem (lumen), cholesterol–macrophage in the endothelium-intima subsystem, $\boldsymbol{v}^{(1)}(\boldsymbol{r};t)$ is the hydrodynamic velocity of blood in the lumen. $\varphi(\boldsymbol{r};t)$ is the scalar potential of the electric field $\boldsymbol{E}(\boldsymbol{r};t)$ created by ions and dipoles in the system. The electric field $\boldsymbol{E}(\boldsymbol{r};t) = \langle \hat{\boldsymbol{E}}(\boldsymbol{r}) \rangle^t$ satisfies the averaged Maxwell's equations:

$$\boldsymbol{\nabla} \times \langle \hat{\boldsymbol{E}}(\boldsymbol{r}) \rangle^t = -\frac{1}{c} \frac{\partial}{\partial t} \langle \hat{\boldsymbol{B}}(\boldsymbol{r}) \rangle^t, \tag{24}$$

$$\boldsymbol{\nabla} \times \langle \hat{\boldsymbol{H}}(\boldsymbol{r}) \rangle^t = \frac{1}{c} \frac{\partial}{\partial t} \langle \hat{\boldsymbol{D}}(\boldsymbol{r}) \rangle^t + \frac{4\pi}{c} \Bigg(\sum_f \frac{Z_f e}{m_f} \langle \hat{\boldsymbol{p}}_f(\boldsymbol{r}) \rangle^t + \sum_\alpha \frac{1}{m_\alpha} \boldsymbol{d}_\alpha \cdot \frac{\partial}{\partial \boldsymbol{r}} \langle \hat{\boldsymbol{p}}_\alpha(\boldsymbol{r}) \rangle^t \Bigg), \tag{25}$$

$$\boldsymbol{\nabla} \cdot \langle \hat{\boldsymbol{B}}(\boldsymbol{r}) \rangle^t = 0, \tag{26}$$

$$\boldsymbol{\nabla} \cdot \langle \hat{\boldsymbol{D}}(\boldsymbol{r}) \rangle^t = 4\pi \Bigg(\sum_f Z_f e \langle \hat{n}_f(\boldsymbol{r}) \rangle^t + \sum_\alpha \boldsymbol{d}_\alpha \cdot \frac{\partial}{\partial \boldsymbol{r}} \langle \hat{n}_\alpha(\boldsymbol{r}) \rangle^t \Bigg), \tag{27}$$

where $\nabla = \frac{\partial}{\partial \boldsymbol{r}}$, with boundary conditions at the boundary of the separation of two phases at $k = (1)$ are for the lumen subsystem, $k = (2)$ are for the endothelium–intima subsystem:

$$\begin{aligned} &\mathbf{n}_1 \cdot (\langle \hat{\boldsymbol{B}}(\boldsymbol{r}_2) \rangle^t - \langle \hat{\boldsymbol{B}}(\boldsymbol{r}_1) \rangle^t) = 0, \\ &\mathbf{n}_1 \cdot (\langle \hat{\boldsymbol{D}}(\boldsymbol{r}_2) \rangle^t - \langle \hat{\boldsymbol{D}}(\boldsymbol{r}_1) \rangle^t) = Q_1(\mathbf{S}_{w1};t), \\ &\mathbf{n}_1 \times (\langle \hat{\boldsymbol{E}}(\boldsymbol{r}_2) \rangle^t - \langle \hat{\boldsymbol{E}}(\boldsymbol{r}_1) \rangle^t) = 0, \\ &\mathbf{n}_1 \times (\langle \hat{\boldsymbol{H}}(\boldsymbol{r}_2) \rangle^t - \langle \hat{\boldsymbol{H}}(\boldsymbol{r}_1) \rangle^t) = \sum_a Q_a(\mathbf{S}_{w1};t) \boldsymbol{v}_a(\mathbf{S}_{w1};t), \end{aligned} \tag{28}$$

at the corresponding values of the coordinates $z_1 = z_2 = 0$ at the interface of the subsystems, where $Q_a(\mathbf{S}_{w1};t)$ is the surface charge of ions of the type a at the interface of the lumen-endothelium-intima

phases, $Q_1(\mathbf{S}_{w1};t)=\sum_a Q_a(\mathbf{S}_{w1};t)$ is the total surface charge. Moreover, from the law of conservation of charge it follows:

$$\mathbf{n}_1\cdot\frac{z_a e}{m_a}\langle\hat{\boldsymbol{p}}_a(\boldsymbol{r}_s)\rangle^t=\frac{\partial}{\partial t}Q_a(\mathbf{S}_{w1};t),$$

where $\boldsymbol{v}_a(\boldsymbol{r};t)=\frac{\langle\hat{\boldsymbol{p}}_a(\boldsymbol{r})\rangle^t}{m_a\langle\hat{n}_a(\boldsymbol{r})\rangle^t}$ is the average velocity of ions of type a, $\hat{\boldsymbol{p}}_a(\boldsymbol{r})=\sum_{j=1}^{N_a}\boldsymbol{p}_j\delta(\boldsymbol{r}-\boldsymbol{r}_j)$ is the microscopic density of the number of ions of type a; $\mathbf{n}_1$ is a unit vector directed perpendicular to the plane of the phase separation "lumen – endothelium–intima". The boundary conditions on the surface S_{w1} are obtained by integrating Maxwell's equations over an infinitesimal volume surrounding a small region of S_{w1}.

The microscopic intensities of the electric $\hat{\boldsymbol{E}}(\boldsymbol{r})$ and magnetic $\hat{\boldsymbol{H}}(\boldsymbol{r})$ fields and the corresponding inductions $\hat{\boldsymbol{D}}(\boldsymbol{r})$, $\hat{\boldsymbol{B}}(\boldsymbol{r})$ satisfy the microscopic Lorentz–Maxwell equations. The known integral relations between $\langle\hat{\boldsymbol{D}}(\boldsymbol{r})\rangle^t$ and $\langle\hat{\boldsymbol{E}}(\boldsymbol{r})\rangle^t$ and $\langle\hat{\boldsymbol{B}}(\boldsymbol{r})\rangle^t$ and $\langle\hat{\boldsymbol{H}}(\boldsymbol{r})\rangle^t$ determine the spatially inhomogeneous dielectric function $\varepsilon(\boldsymbol{r},\boldsymbol{r}';t,t')$ and the magnetization $\chi(\boldsymbol{r},\boldsymbol{r}';t,t')$, which describe the polarization processes in the system:

$$\left\langle\hat{\boldsymbol{D}}(\boldsymbol{r}_l)\right\rangle^t=\int_0^\infty\int_{V_f}d\boldsymbol{r}_f\,\varepsilon(\boldsymbol{r}_l,\boldsymbol{r}_f;\tau)\left\langle\hat{\boldsymbol{E}}(\boldsymbol{r}_l)\right\rangle^{t-\tau}d\tau, \tag{29}$$

$$\left\langle\hat{\boldsymbol{B}}(\boldsymbol{r}_l)\right\rangle^t=\int_0^\infty\int_{V_f}d\boldsymbol{r}_f\chi(\boldsymbol{r}_l,\boldsymbol{r}_f;\tau)\left\langle\hat{\boldsymbol{H}}(\boldsymbol{r}_l)\right\rangle^{t-\tau}d\tau. \tag{30}$$

The parameters $\nu_f^{(\kappa)}(\boldsymbol{r};t)$, $\nu_\alpha^{(\kappa)}(\boldsymbol{r};t)$, $\mu_{\xi a}^{(\kappa)}(\boldsymbol{r};t)$, $\mu_{\gamma\zeta}^{(\kappa\lambda)}(\boldsymbol{r},\boldsymbol{r}';t)$ and $\boldsymbol{v}^{(1)}(\boldsymbol{r};t)$ are determined from the corresponding self-consistency conditions:

$$\begin{gathered}\left\langle\hat{n}_f^\kappa(\boldsymbol{r})\right\rangle^t=\left\langle\hat{n}_f^\kappa(\boldsymbol{r})\right\rangle_{rel}^t,\quad\left\langle\hat{n}_\alpha^\kappa(\boldsymbol{r})\right\rangle^t=\left\langle\hat{n}_\alpha^\kappa(\boldsymbol{r})\right\rangle_{rel}^t,\\ \left\langle\hat{n}_{\xi a}^\kappa(\boldsymbol{r})\right\rangle^t=\left\langle\hat{n}_{\xi a}^\kappa(\boldsymbol{r})\right\rangle_{rel}^t,\quad\left\langle\hat{G}_{\gamma\zeta}^{\kappa\lambda}(\boldsymbol{r},\boldsymbol{r}')\right\rangle^t=\left\langle\hat{G}_{\gamma\zeta}^{\kappa\lambda}(\boldsymbol{r},\boldsymbol{r}')\right\rangle_{rel}^t,\\ \left\langle\hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r})\right\rangle^t=\left\langle\hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r})\right\rangle_{rel}^t.\end{gathered} \tag{31}$$

To reveal the structure of the non-equilibrium distribution function $\varrho(t)$ according to (20), we reveal the action of the operators $(1-P_{rel}(t'))$ and $iL_N(t)$ on $\varrho_{rel}(x^N;t)$ (22), as a result we obtain:

$$\begin{aligned}\left(1-P_{rel}(t)\right)iL_N\varrho_{rel}(t)&=\sum_f\sum_{\kappa=1}^2\int_{V_\kappa}d\boldsymbol{r}\,\beta\,\nu_f^{(\kappa)}(\boldsymbol{r};t)^{(\kappa)}\,I_n^f(\boldsymbol{r};t)\\&+\sum_\alpha\sum_{\kappa=1}^2\int_{V_\kappa}d\boldsymbol{r}\int d\bar{\boldsymbol{d}}\,\beta\,\nu_\alpha^{(\kappa)}(\boldsymbol{r},\bar{\boldsymbol{d}};t)^{(\kappa)}\,I_n^\alpha(\boldsymbol{r},\bar{\boldsymbol{d}};t)+\sum_\xi\sum_a^{M_\xi}\sum_{\kappa=1}^2\int_{V_\kappa}d\boldsymbol{r}\,\beta\,\mu_{\xi a}^{(\kappa)}(\boldsymbol{r};t)^{(\kappa)}\,I_n^{\xi a}(\boldsymbol{r};t)\\&+\sum_{\gamma\zeta}\sum_{\kappa\lambda=1}^2\int_{V_\kappa}d\boldsymbol{r}\int_{V_\lambda}d\boldsymbol{r}'\mu_{\gamma\zeta}^{(\kappa\lambda)}(\boldsymbol{r},\boldsymbol{r}';t)^{(\kappa\lambda)}\,I_G^{\gamma\zeta}(\boldsymbol{r},\boldsymbol{r}';t)+\int_{V_1}d\boldsymbol{r}\,\boldsymbol{v}^{(1)}(\boldsymbol{r};t)^{(1)}\,I_p(\boldsymbol{r};t),\end{aligned} \tag{32}$$

where the generalized flows that form the transfer processes in the system have the following form:

$${}^{(\kappa)}I_n^f(\boldsymbol{r};t)=(1-P(t))\,iL_N\hat{n}_f^{(\kappa)}(\boldsymbol{r})=-(1-P(t))\,\frac{1}{m_f}\frac{\partial}{\partial\boldsymbol{r}}\cdot\hat{\boldsymbol{p}}_f^{(\kappa)}(\boldsymbol{r}), \tag{33}$$

$${}^{(\kappa)}I_n^\alpha(\boldsymbol{r},\bar{\boldsymbol{d}};t)=(1-P(t))\,iL_N\hat{n}_\alpha^{(\kappa)}(\boldsymbol{r},\bar{\boldsymbol{d}})=-(1-P(t))\frac{1}{m_\alpha}\frac{\partial}{\partial\boldsymbol{r}}\cdot\hat{\boldsymbol{p}}_\alpha^{(\kappa)}(\boldsymbol{r},\bar{\boldsymbol{d}})-(1-P(t))\frac{\partial}{\partial\bar{\boldsymbol{d}}}\cdot\hat{\boldsymbol{L}}_\alpha^{(\kappa)}(\boldsymbol{r},\bar{\boldsymbol{d}}), \tag{34}$$

where

$$\hat{\boldsymbol{L}}_\alpha^{(\kappa)}(\boldsymbol{r},\bar{\boldsymbol{d}})=\sum_i^{N_\alpha}(\omega_i\times\bar{\boldsymbol{d}}_i)\,\delta(\boldsymbol{r}-\boldsymbol{r}_i)\,\delta(\bar{\boldsymbol{d}}-\bar{\boldsymbol{d}}_i) \tag{35}$$

is the microscopic density of the rotational velocity vector of molecules of the type α,

$$\begin{aligned}{}^{(\kappa)}I_n^{\xi a}(\boldsymbol{r};t)&=(1-P(t))\,iL_N\hat{n}_{\xi a}^{(\kappa)}(\boldsymbol{r})\\&=-(1-P(t))\frac{1}{m_\xi}\frac{\partial}{\partial\boldsymbol{r}}\cdot\hat{\boldsymbol{p}}_{\xi a}^{(\kappa)}(\boldsymbol{r})-(1-P(t))\sum_i^{N_\xi}\frac{\partial}{\partial(\boldsymbol{r}-\boldsymbol{r}_i)}\cdot\hat{\boldsymbol{L}}_i^{(\kappa)}(\boldsymbol{r}-\boldsymbol{r}_i),\end{aligned} \tag{36}$$

where

$$\hat{\boldsymbol{L}}_i^{(\kappa)}(\boldsymbol{r}-\boldsymbol{r}_i)=\sum_a^{M_\xi}\boldsymbol{\omega}_i\times\boldsymbol{\delta}_{ia}\,\delta(\boldsymbol{r}-\boldsymbol{r}_i-\boldsymbol{\delta}_{ia}) \tag{37}$$

is the microscopic density of the rotational velocity vector of the active centers of the i-th molecule.

$$\begin{aligned}{}^{(\kappa\lambda)}I_G^{ab}(\boldsymbol{r};t)&=(1-P(t))iL_N\hat{G}_{ab}^{(\kappa\lambda)}(\boldsymbol{r},\boldsymbol{r}')\\&=(1-P(t))\big(iL_N\hat{n}_a^{(\kappa)}(\boldsymbol{r})\big)\hat{n}_b^{(\lambda)}(\boldsymbol{r}')+(1-P(t))\hat{n}_a^{(\kappa)}(\boldsymbol{r})\big(iL_N\hat{n}_b^{(\lambda)}(\boldsymbol{r})\big),\end{aligned} \tag{38}$$

$${}^{(1)}I_p(\boldsymbol{r};t)=(1-P(t))iL_N\hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r}) \tag{39}$$

is a generalized blood momentum density flow. The generalized flows include the generalized Mori projection operator, which has the following structure:

$$\begin{aligned}P(t)\hat{A}=\langle\hat{A}\rangle_{rel}^t&+\sum_f\int d\boldsymbol{r}\frac{\delta\langle\hat{A}\rangle_{rel}^t}{\delta\big\langle\hat{n}_f^{(\kappa)}(\boldsymbol{r})\big\rangle^t}\big(\hat{n}_f^{(\kappa)}(\boldsymbol{r})-\langle\hat{n}_f^{(\kappa)}(\boldsymbol{r})\rangle^t\big)\\&+\sum_\alpha\int d\boldsymbol{r}\int d\bar{\boldsymbol{d}}\frac{\delta\langle\hat{A}\rangle_{rel}^t}{\delta\langle\hat{n}_\alpha^{(\kappa)}(\boldsymbol{r},\bar{\boldsymbol{d}})\rangle^t}(\hat{n}_\alpha^{(\kappa)}(\boldsymbol{r},\bar{\boldsymbol{d}})-\langle\hat{n}_\alpha^{(\kappa)}(\boldsymbol{r},\bar{\boldsymbol{d}})\rangle^t)\\&+\sum_\xi\sum_a^{M_\xi}\int d\boldsymbol{r}\,\frac{\delta\langle\hat{A}\rangle_{rel}^t}{\delta\big\langle\hat{n}_{\xi a}^{(\kappa)}(\boldsymbol{r})\big\rangle^t}\big(\hat{n}_{\xi a}^{(\kappa)}(\boldsymbol{r})-\big\langle\hat{n}_{\xi a}^{(\kappa)}(\boldsymbol{r})\big\rangle^t\big)\\&+\sum_{ab}\int d\boldsymbol{r}\int d\boldsymbol{r}'\frac{\delta\langle\hat{A}\rangle_{rel}^t}{\delta\big\langle\hat{G}_{ab}^{(\kappa\lambda)}(\boldsymbol{r},\boldsymbol{r}')\big\rangle^t}\big(\hat{G}_{ab}^{(\kappa\lambda)}(\boldsymbol{r},\boldsymbol{r}')-\langle\hat{G}_{ab}^{(\kappa\lambda)}(\boldsymbol{r},\boldsymbol{r}')\rangle^t\big)\\&+\sum_a\int d\boldsymbol{r}\frac{\delta\langle\hat{A}\rangle_{rel}^t}{\delta\big\langle\hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r})\big\rangle^t}\big(\hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r})-\langle\hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r})\rangle^t\big).\end{aligned} \tag{40}$$

Now, substituting (32) into (20), for the non-equilibrium distribution function we obtain:

$$\begin{aligned}\varrho(t)=\varrho_{rel}(t)-\int_{-\infty}^t e^{\varepsilon(t'-t)}T_{rel}(t,t')\Big(&\sum_f\sum_{\kappa=1}^2\int_{V_\kappa}d\boldsymbol{r}\,\beta\,\nu_f^{(\kappa)}(\boldsymbol{r};t')^{(\kappa)}\,I_n^f(\boldsymbol{r};t')\\&+\sum_\alpha\sum_{\kappa=1}^2\int_{V_\kappa}d\boldsymbol{r}\,d\bar{\boldsymbol{d}}\,\beta\,\nu_\alpha^{(\kappa)}(\boldsymbol{r},\bar{\boldsymbol{d}};t')^{(\kappa)}\,I_n^\alpha(\boldsymbol{r},\bar{\boldsymbol{d}};t')+\sum_\xi\sum_a^{M_\xi}\sum_{\kappa=1}^2\int_{V_\kappa}d\boldsymbol{r}\,\beta\,\mu_{\xi a}^{(\kappa)}(\boldsymbol{r};t')^{(\kappa)}\,I_n^{\xi a}(\boldsymbol{r};t')\\&+\sum_{\gamma\zeta}\sum_{\kappa\lambda=1}^2\int_{V_\kappa}d\boldsymbol{r}\int_{V_\lambda}d\boldsymbol{r}'\beta\mu_{\gamma\zeta}^{(\kappa\lambda)}(\boldsymbol{r},\boldsymbol{r}';t')^{(\kappa\lambda)}I_G^{\gamma\zeta}(\boldsymbol{r},\boldsymbol{r}';t')+\int_{V_1}d\boldsymbol{r}\beta\boldsymbol{v}^{(1)}(\boldsymbol{r};t')^{(1)}I_p(\boldsymbol{r};t')\Big)\varrho_{rel}(t')\,dt'.\end{aligned} \tag{41}$$

It contains a non-dissipative part $\varrho_{rel}(t)$ and a dissipative part associated with the generalized flows ${}^{(\kappa)}I_n^f(\boldsymbol{r};t')$, ${}^{(\kappa)}I_n^\alpha(\boldsymbol{r},\bar{\boldsymbol{d}};t')$, ${}^{(\kappa)}I_n^{\xi a}(\boldsymbol{r};t')$, ${}^{(\kappa\lambda)}I_G^{\gamma\zeta}(\boldsymbol{r},\boldsymbol{r}';t')$, ${}^{(1)}I_p(\boldsymbol{r};t')$ in the corresponding subsystems. Using the non-equilibrium distribution function for the main set of parameters of the abbreviated description (4), (14), (16)–(18), we obtain the following system of equations:

$$\begin{aligned}\frac{\partial}{\partial t}\big\langle\hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r})\big\rangle^t=\big\langle iL_N(t)\hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r})\big\rangle_{rel}^t&-\int_{V_1}d\boldsymbol{r}'\int_{-\infty}^t e^{\varepsilon(t'-t)}\varphi_{pp}^{11}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\,dt'\\&-\sum_{f'}\sum_{\kappa'=1}^2\int_{V_{\kappa'}}d\boldsymbol{r}'\int_{-\infty}^t e^{\varepsilon(t'-t)}\varphi_{pn}^{1f'\kappa'}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\nu_{f'}^{(\kappa')}(\boldsymbol{r}';t')\,dt'\\&-\sum_{\alpha'}\sum_{\kappa'=1}^2\int_{V_{\kappa'}}d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\int_{-\infty}^t e^{\varepsilon(t'-t)}\varphi_{pn}^{1\alpha'\kappa'}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\nu_{\alpha'}^{(\kappa')}(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')\,dt'\\&-\sum_{\xi'}\sum_{a'}^{M_{\xi'}}\sum_{\kappa'=1}^2\int_{V_{\kappa'}}d\boldsymbol{r}'\int_{-\infty}^t e^{\varepsilon(t'-t)}\varphi_{pn}^{1\xi'a'\kappa'}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{\xi'a'}^{(\kappa')}(\boldsymbol{r}';t')\,dt'\end{aligned}$$

$$-\sum_{\gamma'\zeta'}\sum_{\kappa'\lambda'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int_{V'_{\lambda}}d\boldsymbol{r}''\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{pG}^{1\gamma'\zeta'\kappa'}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\,\beta\,\mu_{\gamma'\zeta'}^{(\kappa'\lambda')}(\boldsymbol{r}',\boldsymbol{r}'';t')\,dt', \quad (42)$$

$$\begin{aligned}\frac{\partial}{\partial t}\big\langle\hat{n}_{f}^{(\kappa)}(\boldsymbol{r})\big\rangle^{t} &= -\sum_{f'}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nn}^{f\kappa f'\kappa'}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\nu_{f'}^{(\kappa')}(\boldsymbol{r}';t')\,dt'\\ &-\sum_{\alpha'}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nn}^{f\kappa\alpha'\kappa'}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\nu_{\alpha'}^{(\kappa')}(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')\,dt'\\ &-\sum_{\xi'}\sum_{a'}^{M_{\xi'}}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nn}^{f\kappa\xi'a'\kappa'}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{\xi'a'}^{(\kappa')}(\boldsymbol{r}';t')\,dt'\\ &-\sum_{\gamma'\zeta'}\sum_{\kappa'\lambda'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int_{V'_{\lambda}}d\boldsymbol{r}''\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nG}^{f\kappa\gamma'\zeta'\kappa'}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\,\beta\,\mu_{\gamma'\zeta'}^{(\kappa'\lambda')}(\boldsymbol{r}',\boldsymbol{r}'';t')\,dt'\\ &-\int_{V_1}d\boldsymbol{r}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{np}^{f\kappa 1}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\,dt', \end{aligned} \quad (43)$$

$$\begin{aligned}\frac{\partial}{\partial t}\big\langle\hat{n}_{\alpha}^{(\kappa)}(\boldsymbol{r},\bar{\boldsymbol{d}})\big\rangle^{t} &= -\sum_{f'}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nn}^{\alpha\kappa f'\kappa'}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')\,\beta\,\nu_{f'}^{(\kappa')}(\boldsymbol{r}';t')\,dt'\\ &-\sum_{\alpha'}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nn}^{\alpha\kappa\alpha'\kappa'}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\nu_{\alpha'}^{(\kappa')}(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')\,dt'\\ &-\sum_{\xi'}\sum_{a'}^{M_{\xi'}}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nn}^{\alpha\kappa\xi'a'\kappa'}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')\,\beta\,\mu_{\xi'a'}^{(\kappa')}(\boldsymbol{r}';t')\,dt'\\ &-\sum_{\gamma'\zeta'}\sum_{\kappa'\lambda'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int_{V'_{\lambda}}d\boldsymbol{r}''\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nG}^{\alpha\kappa\gamma'\zeta'\kappa'}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}'\boldsymbol{r}'';t,t')\,\beta\,\mu_{\gamma'\zeta'}^{(\kappa'\lambda')}(\boldsymbol{r}',\boldsymbol{r}'';t')\,dt'\\ &-\int_{V_1}d\boldsymbol{r}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{np}^{\alpha\kappa 1}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\,dt', \end{aligned} \quad (44)$$

$$\begin{aligned}\frac{\partial}{\partial t}\big\langle\hat{n}_{\xi a}^{(\kappa)}(\boldsymbol{r})\big\rangle^{t} &= -\sum_{f'}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nn}^{\xi a\kappa f'\kappa'}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\nu_{f'}^{(\kappa')}(\boldsymbol{r}';t')\,dt'\\ &-\sum_{\alpha'}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nn}^{\xi a\kappa\alpha'\kappa'}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\nu_{\alpha'}^{(\kappa')}(\boldsymbol{r}'\bar{\boldsymbol{d}}';t')\,dt'\\ &-\sum_{\xi'}\sum_{a'}^{M_{\xi'}}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nn}^{\xi a\kappa\xi'a'\kappa'}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{\xi'a'}^{(\kappa')}(\boldsymbol{r}';t')\,dt'\\ &-\sum_{\gamma'\zeta'}\sum_{\kappa'\lambda'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\int_{V'_{\lambda}}d\boldsymbol{r}''\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{nG}^{\xi a\kappa\gamma'\zeta'\kappa'}(\boldsymbol{r},\boldsymbol{r}'\boldsymbol{r}'';t,t')\,\beta\,\mu_{\gamma'\zeta'}^{(\kappa'\lambda')}(\boldsymbol{r}',\boldsymbol{r}'';t')\,dt'\\ &-\int_{V_1}d\boldsymbol{r}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{np}^{\xi a\kappa 1}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\,dt', \end{aligned} \quad (45)$$

$$\begin{aligned}\frac{\partial}{\partial t}\big\langle\hat{G}_{\gamma\zeta}^{(\kappa\lambda)}(\boldsymbol{r},\boldsymbol{r}')\big\rangle^{t} &= -\sum_{f'}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}''\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{Gn}^{\gamma\zeta\kappa\lambda f'\kappa'}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\,\beta\,\nu_{f'}^{(\kappa')}(\boldsymbol{r}'';t')\,dt'\\ &-\sum_{\alpha'}\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}''\int d\bar{\boldsymbol{d}}'\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\varphi_{Gn}^{\gamma\zeta\kappa\lambda\alpha'\kappa'}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}';t,t')\,\beta\,\nu_{\alpha'}^{(\kappa')}(\boldsymbol{r}'',\bar{\boldsymbol{d}}';t')\,dt' \end{aligned}$$

$$
\begin{aligned}
&- \sum_{\xi'} \sum_{a'}^{M_{\xi'}} \sum_{\kappa'=1}^{2} \int_{V_{\kappa'}} d\boldsymbol{r}'' \int_{-\infty}^{t} e^{\varepsilon(t'-t)} \varphi_{Gn}^{\gamma\zeta\kappa\lambda\kappa\xi'a'\kappa'}(\boldsymbol{r}, \boldsymbol{r}', \boldsymbol{r}''; t, t')\, \beta\, \mu_{\xi'a'}^{(\kappa')}(\boldsymbol{r}''; t')\, dt' \\
&- \sum_{\gamma'\zeta'} \sum_{\kappa'\lambda'=1}^{2} \int_{V_{\kappa'}} d\boldsymbol{r}'' \int_{V'_{\lambda}} d\boldsymbol{r}''' \int_{-\infty}^{t} e^{\varepsilon(t'-t)} \varphi_{GG}^{\gamma\zeta\kappa\lambda\gamma'\zeta'\kappa'\lambda'}(\boldsymbol{r}, \boldsymbol{r}', \boldsymbol{r}'', \boldsymbol{r}'''; t, t')\, \beta\, \mu_{\gamma'\zeta'}^{(\kappa'\lambda')}(\boldsymbol{r}'', \boldsymbol{r}'''; t')\, dt' \\
&- \int_{V_1} d\boldsymbol{r}'' \int_{-\infty}^{t} e^{\varepsilon(t'-t)} \varphi_{Gp}^{\gamma\zeta\kappa\lambda\kappa 1}(\boldsymbol{r}, \boldsymbol{r}', \boldsymbol{r}''; t, t')\, \beta\, \boldsymbol{v}^{(1)}(\boldsymbol{r}''; t')\, dt', \qquad (46)
\end{aligned}
$$

where $\varphi_{pp}^{11}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ is the transfer kernel (memory function) that determines the generalized blood viscosity coefficient taking into account the translational, rotational motions of the components and polarization processes associated with the change in the microscopic density of the electric field created by ions and dipole components of the blood. $\varphi_{pp}^{11}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ has the structure of the time correlation function of the generalized blood momentum density flows in the first subsystem:

$$
\varphi_{pp}^{11}(\boldsymbol{r}, \boldsymbol{r}'; t, t') = \left\langle I_p^{(1)}(\boldsymbol{r}; t) T_{rel}(t, t') I_p^{(1)}(\boldsymbol{r}'; t') \right\rangle_{rel}^{t'}. \qquad (47)
$$

Transfer kernels of the type $\varphi_{nn}^{AB}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ determine generalized diffusion coefficients for components including in various subsystems, in particular for ions:

$$
\varphi_{nn}^{f\kappa f'\kappa'}(\boldsymbol{r}, \boldsymbol{r}'; t, t') = \frac{\partial}{\partial \boldsymbol{r}} \cdot D_{ff'}^{\kappa\kappa'}(\boldsymbol{r}, \boldsymbol{r}'; t, t') \cdot \frac{\partial}{\partial \boldsymbol{r}'}, \qquad (48)
$$

where

$$
D_{ff'}^{\kappa\kappa'}(\boldsymbol{r}, \boldsymbol{r}'; t, t') = \left\langle (1 - P(t)) \frac{1}{m_f} \hat{\boldsymbol{p}}_f^{(\kappa)}(\boldsymbol{r}) T_{rel}(t, t') (1 - P(t')) \frac{1}{m_{f'}} \hat{\boldsymbol{p}}_{f'}^{(\kappa)}(\boldsymbol{r}')) \right\rangle_{rel}^{t'} \qquad (49)
$$

is the generalized diffusion coefficient of ions of the types f, f' in the subsystems κ κ' and $f = f'$, $\kappa = \kappa'$ we have the generalized diffusion coefficient of ions of the type f in the subsystem κ, and also, when $f = f'$, but $\kappa = 1$, $\kappa' = 2$, then $D_{ff}^{12}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ describes the dynamic correlation of the generalized fluxes of ions of the type f between the two subsystems and this is very important, since it shows how ions from one subsystem pass into another. Transfer kernels $\varphi_{GG}^{\gamma\zeta\kappa\lambda\gamma'\zeta'\kappa'\lambda'}(\boldsymbol{r}, \boldsymbol{r}', \boldsymbol{r}'', \boldsymbol{r}'''; t, t')$

$$
\varphi_{GG}^{abcd}(\boldsymbol{r}, \boldsymbol{r}', \boldsymbol{r}'', \boldsymbol{r}'''; t, t') = \left\langle I_G^{ab}(\boldsymbol{r}, \boldsymbol{r}'; t)\, T_{rel}(t, t')\, I_G^{cd}(\boldsymbol{r}'', \boldsymbol{r}'''; t') \right\rangle_{rel}^{t'} \qquad (50)
$$

determine the coefficients of the corresponding biochemical reactions between components in different subsystems, in particular, oxidation of LDL by free radicals, reduction of vitamins E in LDL when interacting with vitamins C, processes of capture of peroxidized LDL by macrophages in the endothelium-intima subsystem. Data of the transport kernel in the Markov approximation in time and neglecting spatial dependence:

$$
\varphi_{GG}^{AB} \sim K_{AB}\, \delta(\boldsymbol{r} - \boldsymbol{r}'')\, \delta(\boldsymbol{r}' - \boldsymbol{r}''')\, \delta(t - t')
$$

can be compared with the corresponding reaction constants of the works [19,21,25–29]. The transport kernels of the type φ_{nG}^{abc}, φ_{pn}^{ab}, φ_{pG}^{abc} describe the cross-dissipative correlations of the generalized flows of blood components in different subsystems:

$$
\varphi_{pG}^{abc}(\boldsymbol{r}, \boldsymbol{r}', \boldsymbol{r}''; t, t') = \left\langle I_p^{a}(\boldsymbol{r}; t)\, T_{rel}(t, t')\, I_G^{bc}(\boldsymbol{r}', \boldsymbol{r}''; t') \right\rangle_{rel}^{t'}, \qquad (51)
$$

$$
\varphi_{pn}^{a}(\boldsymbol{r}, \boldsymbol{r}'; t, t') = \left\langle I_p^{a}(\boldsymbol{r}; t)\, T_{rel}(t, t')\, I_n(\boldsymbol{r}'; t') \right\rangle_{rel}^{t'} \qquad (52)
$$

The obtained system of transport equations (42)–(46) within the framework of the selected model of component interaction can describe non-Markovian in time and non-local in space processes of blood transport in vessels, taking into account possible processes in the vessel walls. All its equations for the corresponding transport kernels include the hydrodynamic velocity $\boldsymbol{v}^{(1)}(\boldsymbol{r}'; t')$, as one of the important parameters of the studies. In addition, this system of transport equations is open–ended, nonlinear and can be applied to the description of both laminar and turbulent processes. It is important to note that in the first subsystem — the lumen — a viscous reaction-diffusion description is used, and in the endothelium–intima subsystem — a reaction–diffusion description.

3. Diffusion-reaction transport processes in the dynamics of the circulatory system

Important from the point of view of atherosclerosis processes are the processes when in the intima vitamins E (ArOH) in the structure of LDL are oxidized by peroxyl free radicals $ROO^{\bullet}$ [67, 68, 70]

$$(ROO^{\bullet})_{aq} + (ArOH)_{lip} \to (ROOH)_{aq} + (ArO^{\bullet})_{lip}, \tag{53}$$

$ArO^{\bullet}$ are radicals of α-tocopherol. Peroxyl free radicals $ROO^{\bullet}$ arise from the oxidation of free radicals $R^{\bullet}$:

$$R^{\bullet} + O_2 \to ROO^{\bullet}. \tag{54}$$

Since LDL can have from 3 to 15 vitamins E, in the presence of inflammatory processes, the process of oxidation of vitamins E by free radicals is triggered. Along with this, in the aqueous environment of the intima, vitamin C (ascorbic acid AH_2), which is in the form of ascorbate anion $(AH^{-})_{aq}$, can actively reduce vitamin E in the structure of LDL by the reaction [67, 68, 70]:

$$(ArO^{\bullet})_{lip} + (AH^{-})_{aq} \to (ArOH)_{lip} + (A^{-\bullet})_{aq}. \tag{55}$$

Therefore, according to this reaction, vitamin E is reduced and the ascorbic radical $A^{-\bullet}$ is formed, which can again neutralize the radicals $ROO^{\bullet}$ with the participation of protons H^{+}:

$$A^{-\bullet} + ROO^{\bullet} + H^{+} \to A + ROOH. \tag{56}$$

At the same time, dehydroascorbic acid A is also formed. And therefore, there may be competition between the processes of oxidation and reduction of vitamin E in the structure of LDL. Under the condition of inflammatory processes and insufficient amounts of vitamin C in the intima, the process of oxidation of vitamins E will progress. In this case, an interesting situation arises, the oxidation of vitamins E leads to the appearance of negatively charged radicals $(ArO^{\bullet})_{lip}$ of the order of 3 to 15 in the structure of LDL. These macroions ofL with negative charges polarize the surrounding macromolecules of the intima s, water molecules, and others, leading to solvation formations. Such solvation coats around ofL can play the role of some protection against the action of free radicals. It is also important to note here that vitamins C, as strong antioxidants, can directly fight free radicals $ROO^{\bullet}$ in the presence of oxygen molecules O_2 and protons H^{+} through a series of chemical reactions [68]:

$$ROO^{\bullet} + AH^{-} \to ROOH + A^{-\bullet}, \tag{57}$$

$$AH^{-} + O_2 \to A^{-\bullet} + O_2^{-\bullet} + H^{+}, \tag{58}$$

$$A^{-\bullet} + O_2 \to A + O_2^{-\bullet},$$

$$O_2^{-\bullet} + ROO^{\bullet} + H^{+} \to O_2 + ROOH, \tag{59}$$

$$O_2^{-\bullet} + AH^{-} + H^{+} \to H_2O_2 + A^{-\bullet}, \tag{60}$$

in which radicals $A^{-\bullet}$ according to (56) and radicals $O_2^{-\bullet}$ neutralize free radicals $ROO^{\bullet}$, forming ROOH. In all these redox reactions, we have processes of proton transfer bound to electrons on the corresponding components. Bound proton–electron transfer in biological systems is very important and well studied from the point of view of reaction constants based on quantum-statistical calculations [58, 74, 75]. It is important to note that a change in proton concentration affects the activity and conformation of proteins, as well as the properties of other macromolecular structures. From this point of view, the kinetics of proton diffusion–controlled transport is a very important area of research and requires the application of methods of non-equilibrium statistical mechanics. In the redox processes of macromolecular structures (53), (55), (56), (57)–(60) we are dealing with correlated proton-electron charge transfer. For a deeper understanding of these processes in order to influence them, it will be necessary to take into account models of charge transfer dynamics along hydrogen bonds [76–78] between macromolecular structures. In particular, to describe the oxidation processes of vitamins E in the structure of LDL by free radicals, according to [76, 77] in the Hamiltonian of the problem it is necessary to take into account proton-electron transfer based on the pseudospin–electron model:

$$\sum_{ROO^{\bullet}(j),LE\,ArOH(l)} \sum \Big(\Phi_{ROO^{\bullet},ArOH}(\boldsymbol{r}_j, \boldsymbol{r}_l) + \sum_{\sigma} \big[E_o(n_\sigma(\boldsymbol{r}_j) + n_\sigma(\boldsymbol{r}_l))$$

$$+ gS^z(n_\sigma(\boldsymbol{r}_l) - n_\sigma(\boldsymbol{r}_j)) - \Omega(a^+_\sigma(\boldsymbol{r}_j)a_\sigma(\boldsymbol{r}_l)S^+ + a^+_\sigma(\boldsymbol{r}_l)a_\sigma(\boldsymbol{r}_j)S^-)\big]\Big), \quad (61)$$

where $\Phi_{\mathrm{ROO}^\bullet,\mathrm{ArOH}}(\boldsymbol{r}_j,\boldsymbol{r}_l)$ is the potential of interaction between free radicals with vitamins E in the structure of LDL, $E_0 = \varepsilon - \triangle_0/2$, ε is the energy of electronic levels, $\triangle_0$ corresponds to the minimum of the adiabatic proton potential, $g = \varepsilon - \triangle_0$ is the proton-electron coupling constant, Ω is the proton tunneling energy, the jumps of which are covered by pseudospins S^+, S^-. $n_\sigma(\boldsymbol{r}_j) = a^+_\sigma(\boldsymbol{r}_j)a_\sigma(\boldsymbol{r}_j)$ is the electron population operator, in particular for free radicals, $\sigma = \uparrow\downarrow$ is the electron spin. And corresponding to the reactions of vitamin E reduction in the structure of LDL by ascorbic anions AH^- in the Hamiltonian of the problem, it is necessary to take into account proton-electron transfer based on the pseudospin–electron model, i.e. proton transfer from AH^- to $\mathrm{ArO}^\bullet$:

$$\sum_{(\mathrm{ArO}^\bullet)_{lip}(j),\mathrm{AH}^-(l)} \Big(\Phi_{\mathrm{ArO}^\bullet,\mathrm{AH}^-}(\boldsymbol{r}_j,\boldsymbol{r}_l)$$
$$+ \sum_\sigma \big[E_o(n_\sigma(\boldsymbol{r}_j) + n_\sigma(\boldsymbol{r}_l)) + gS^z(n_\sigma(\boldsymbol{r}_l) - n_\sigma(\boldsymbol{r}_j)) - \Omega(a^+_\sigma(\boldsymbol{r}_j)a_\sigma(\boldsymbol{r}_l)S^+ + a^+_\sigma(\boldsymbol{r}_l)a_\sigma(\boldsymbol{r}_j)S^-)\big]\Big). \quad (62)$$

In the intimate area, there are actually three types of LDL: LDL — non-oxidized, which we will denote LE (have unoxidized vitamins E); LDL, in which vitamins E are oxidized by free radicals, we will denote them ofL, and from vitamin E a hydrogen ion H^+ is transferred to a free radical, as a result ofL has negatively charged active centers; and LDL, in which fully oxidized vitamins E are oxidized by oxygen (peroxidized LDL), which we will denote — pL, which are actively captured by macrophages. Charged active centers in ofL obviously cause polarization around themselves and create electromagnetic fields, which also affect polarization processes according to Maxwell's equations, or at the microlevel of the Lorentz–Maxwell equations. pL when interacting with macrophages mc are captured, forming complexes $\langle \hat{G}_{pL,mc}(\boldsymbol{r},\boldsymbol{r}')\rangle^t$.

In addition, macrophages in the intima must be divided by function, in particular, macrophages that have just formed in the intima from monocytes, active macrophages that absorb pL, HDL, erythrocytes that have entered the intima and other products of the decay of macromolecular structures, as well as saturated (loaded) macrophages, which apparently with subdiffusion movements become centers of their aggregation and plaque formation. HDL are practically not oxidized and HDL captured by macrophages can take cholesterol from them and remove it from macrophages with subsequent passage into the lumen, and then into the liver, in particular for the conversion of transported cholesterol into bile acids.

In the case of high pressures at the branches, tortuous arteries, and especially inflammatory processes (as a result of stress, viral diseases, etc.) of the endothelium-intima subsystem (loss of elasticity), weakly turbulent processes may occur, in which the interaction of blood with the endothelial surface is of great importance. To study the influence of such processes on non-equilibrium processes in the endothelium-intima subsystem (plaque formation), it is necessary to take into account the equation for the transfer of the non-equilibrium average value of the blood pulse density. Such an equation, taking into account diffusion-reaction processes in the endothelium-intima subsystem, has the form:

$$\frac{\partial}{\partial t}\big\langle \hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r})\big\rangle^t = \big\langle iL_N(t)\hat{\boldsymbol{p}}^{(1)}(\boldsymbol{r})\big\rangle^t_{rel} - \int_{-\infty}^t e^{\varepsilon(t'-t)}\Big(\int_{V_1} d\boldsymbol{r}'\varphi_{pp}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')$$
$$+ \sum_s \int_{V_2} d\boldsymbol{r}' \int d\bar{\boldsymbol{d}}'\varphi^{1-s}_{pn}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_s(\boldsymbol{r}'\bar{\boldsymbol{d}}';t')$$
$$+ \sum_\kappa \sum_{f'} \int_{V_\kappa} d\boldsymbol{r}'\varphi^{1-f'}_{pn}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{f'}(\boldsymbol{r}';t') + Z_{f'}e\varphi(\boldsymbol{r}';t)\big)$$
$$+ \int_{V_2} d\boldsymbol{r}' \int d\bar{\boldsymbol{d}}'\varphi^{1-w}_{pn}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\big(\mu_w(\boldsymbol{r}',\bar{\boldsymbol{d}}';t') + \boldsymbol{d}\cdot\boldsymbol{E}(\boldsymbol{r}';t')\big)$$
$$+ \int_{V_2} d\boldsymbol{r}'\varphi^{1-LE}_{pn}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{LE}(\boldsymbol{r}';t') + \int_{V_2} d\boldsymbol{r}'\varphi^{1-HL}_{pn}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{HL}(\boldsymbol{r}';t')\Big)$$

$$+\sum_{f'}\int_{V_2} d\boldsymbol{r}'\varphi_{pn}^{1-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{of'L}(\boldsymbol{r}';t')+Z_{f'}e\varphi(\boldsymbol{r}';t)\big)$$

$$+\int_{V_2} d\boldsymbol{r}'\int \bar{\boldsymbol{d}}'\varphi_{pn}^{1-macr}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_{macr}(\boldsymbol{r}',\bar{\boldsymbol{d}}';t'))\,dt', \tag{63}$$

where $\varphi_{pn}^{1-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ are the transport kernels that describe the dynamic correlations between viscous blood flows with "diffusion" flows of macromolecules of the endothelium-intima subsystem. By "diffusion" flows of macromolecules of the endothelium-intima subsystem we will understand vibrations (within a certain frequency interval), conformational changes, and displacements of macromolecules, through the structure of which the necessary blood components diffuse from the lumen to the intima, which occurs continuously in time; $\varphi_{pn}^{1-f'}(\boldsymbol{r},\boldsymbol{r}';t,t')$ are the transport kernels, which at $\kappa=1$ describe the dynamic correlations between viscous blood flows with diffusion flows of charged particles with valence $Z_{f'}$, in particular H^+, OH^-, AH^-, etc. in the lumen, and at $\kappa=2$ describe dynamic correlations between viscous blood flows with diffusion flows of charged components H^+, $O_2^{-\bullet}$, AH^-, $A^{-\bullet}$, etc., $\mu_{f'}(\boldsymbol{r}';t')$ are non-equilibrium values of the chemical potential of components f', $\varphi(\boldsymbol{r}';t)$ is the scalar potential of the electric field formed by all charged components; $\varphi_{pn}^{1-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ describes the dynamic correlations between viscous blood flows with diffusion (translational and rotational) flows of dipolar water molecules in the intima, and especially the correlations with the stress tensor of water molecules and blood plasma ions included in (39) taking into account (9), $\mu_w(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')$ are the non-equilibrium values of the chemical potential of dipolar water molecules; $\varphi_{pn}^{1-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')$ and $\varphi_{pn}^{1-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ are transport kernels that describe dynamic correlations between viscous blood flows and the corresponding diffusion flows of low- and high-density lipoproteins, and they are important from the point of view of cholesterol transport as a "building" material for cells in areas of vascular inflammation, as well as for the transport of unused cholesterol by high-density lipoproteins from the intima into the lumen, and then into the liver; $\varphi_{pn}^{1-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel that describes the dynamic correlations between viscous blood flows and the diffusion flow of low-density lipoproteins, in which a certain amount of f' vitamins E is oxidized in the structure, and $of'L$ contains negatively charged centers, $\nu_{of'L}(\boldsymbol{r}';t')=\mu_{ofL}(\boldsymbol{r}';t')+Z_{f'}e\varphi(\boldsymbol{r}';t')$ is the electrochemical potential, and $\mu_{of'L}(\boldsymbol{r}';t')$ is the chemical potential of low-density lipoproteins in which vitamins E are oxidized with a localized charge $Z_{f'}e$, the sum over f' means how many vitamins E are oxidized in lipoproteins; the effect of viscous blood flows on the movement of macrophages with chemical potentials $\mu_{macr}(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')$ in the intima can be described by the transport kernel $\varphi_{pn}^{1-macr}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$.

The activity and concentration of macrophages increases with the increase in the concentration of free radicals $ROO^\bullet$ in areas of inflammatory processes in the intima. In these areas of inflammation, the concentration of low- and high-density lipoproteins also increases. The transport equation for the non-equilibrium value of the density of free radicals (in which there is an uncompensated electron) in the intima, taking into account redox processes, has the form:

$$\frac{\partial}{\partial t}\langle\hat{n}_{\mathrm{ROO}^\bullet}(\boldsymbol{r})\rangle^t = -\int_{-\infty}^{t} e^{\varepsilon(t'-t)}\Bigg(\sum_s\int_{V_2} d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\varphi_{nn}^{\mathrm{ROO}^\bullet-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_s(\boldsymbol{r}'\bar{\boldsymbol{d}}';t')$$

$$+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{\mathrm{ROO}^\bullet-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{LE}(\boldsymbol{r}';t')+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{\mathrm{ROO}^\bullet-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{HL}(\boldsymbol{r}';t')$$

$$+\sum_{f'}\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{\mathrm{ROO}^\bullet-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{of'L}(\boldsymbol{r}';t')+Z_{f'}e\varphi(\boldsymbol{r}',t')\big)$$

$$+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{\mathrm{ROO}^\bullet-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{pL}(\boldsymbol{r}';t')$$

$$+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{\mathrm{ROO}^\bullet-\mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{H^+}(\boldsymbol{r}';t')+Z_{H^+}e\varphi(\boldsymbol{r}',t')\big)$$

$$+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{\mathrm{ROO}^\bullet-\mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}';t,t')\beta(\mu_{\mathrm{AH}^-}(\boldsymbol{r}';t')+Z_{\mathrm{AH}^-}e\varphi(\boldsymbol{r}',t'))$$

$$+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{\mathrm{ROO}^\bullet-\mathrm{A}^{-\bullet}}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{\mathrm{A}^{-\bullet}}(\boldsymbol{r}';t')+Z_{\mathrm{A}^{-\bullet}}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\int_{V_2} d\boldsymbol{r}'\int_{V_2} d\boldsymbol{r}''\varphi_{nG}^{\mathrm{ROO}^\bullet-\mathrm{O}_2^{-\bullet},\mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$$
$$\times\beta\big(\mu_{\mathrm{O}_2^{-\bullet},\mathrm{H}^+}(\boldsymbol{r}',\boldsymbol{r}'';t')+Z_{\mathrm{O}_2^{-\bullet}}e\varphi(\boldsymbol{r}';t')+Z_{\mathrm{H}^+}e\varphi(\boldsymbol{r}'';t')\big)$$
$$+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{\mathrm{ROO}^\bullet-\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{\mathrm{ROO}^\bullet}(\boldsymbol{r}';t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\int_{V_2} d\boldsymbol{r}'\int_{V_2} d\boldsymbol{r}''\varphi_{nG}^{\mathrm{ROO}^\bullet-\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$$
$$\times\beta\big(\mu_{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r}',\boldsymbol{r}'';t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}',t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}'',t')\big)$$
$$+\int d\boldsymbol{r}'d\bar{\boldsymbol{d}}'\varphi_{nn}^{\mathrm{ROO}^\bullet-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\big(\mu_w(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')+\boldsymbol{d}\cdot\boldsymbol{E}(\boldsymbol{r}',t')\big)$$
$$+\int_{V_1} d\boldsymbol{r}'\varphi_{np}^{\mathrm{ROO}^\bullet-1}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t'))\,dt', \tag{64}$$

where $\varphi_{nn}^{\mathrm{ROO}^\bullet-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$, $\varphi_{nn}^{\mathrm{ROO}^\bullet-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nn}^{\mathrm{ROO}^\bullet-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ are the transfer nuclei that describe the interdiffusion processes between free radical flows and macromolecules s in the intima and low and high density lipoproteins. When $\mathrm{ROO}^\bullet$ interacts with LE, the oxidation of vitamins E in the structure of LE (LDL). Since the structure of HL (HDL) can contain one vitamin E, which can also be oxidized when interacting with $\mathrm{ROO}^\bullet$, this contribution does not make a fundamental contribution compared to LDL. $\varphi_{nn}^{\mathrm{ROO}^\bullet-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')$ are the transfer kernels that describe the interdiffusion processes between the flows of free radicals and low-density lipoproteins, which contain f' oxidized vitamins E. In this case, it is necessary to take into account the proton-electron transport between $\mathrm{ROO}^\bullet$ and $(\mathrm{ArOH})_{lip}$ (53). The oxidation process of $(\mathrm{ArOH})_{lip}$ in the LDL structure can be described by the pairwise non-equilibrium distribution function $\langle\hat{G}_{\mathrm{ROO}^\bullet,LE}(\boldsymbol{r},\boldsymbol{r}')\rangle^t$ and at the same time, ROOH (free radicals are neutralized by proton-electron transport) and $of'L$ — low-density lipoproteins with oxidized f' vitamins E are formed. $\varphi_{nn}^{\mathrm{ROO}^\bullet-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel that describes the interdiffusion of free radicals and low-density lipoproteins, which have f' oxidized vitamins E. In this process, due to proton–electron transport, further oxidation of vitamins E in the LDL structure may occur. Such processes can be described by a pair non-equilibrium distribution function $\langle\hat{G}_{\mathrm{ROO}^\bullet,of'L}(\boldsymbol{r},\boldsymbol{r}')\rangle^t$, and at the same time ROOH are formed again and further oxidation of LDL occurs. $\varphi_{nn}^{\mathrm{ROO}^\bullet-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describing the interdiffusion of free radicals and peroxidized low-density lipoproteins, in which fully oxidized vitamin E and such peroxidized LDL are actively taken up by macrophages, which can be described by the pairwise nonequilibrium distribution function $\langle\hat{G}_{pL,macr}(\boldsymbol{r},\boldsymbol{r}')\rangle^t$. $\varphi_{nn}^{\mathrm{ROO}^\bullet-\mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nn}^{\mathrm{ROO}^\bullet-\mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nn}^{\mathrm{ROO}^\bullet-\mathrm{A}^{-\bullet}}(\boldsymbol{r},\boldsymbol{r}';t,t')$ and $\varphi_{nG}^{\mathrm{ROO}^\bullet-\mathrm{O}_2^{-\bullet},\mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$ are the transfer kernels that describe the interdiffusion of free radicals and protons H^+, anions AH^-, radicals $\mathrm{A}^{-\bullet}$, $\mathrm{O}_2^{-\bullet}$ together with H^+ taking into account the influence of the scalar potential of the electric field, which as a result of the reactions (57)–(60) lead to the neutralization of $\mathrm{ROO}^\bullet$, which is very important from the point of view of reducing the oxidation processes of LDL in the intima, while the collective diffusion of free radicals and the mutual diffusion of the complexes $\mathrm{ROO}^\bullet$, $\mathrm{ROO}^\bullet$ and free radicals are described by the transfer nuclei $\varphi_{nn}^{\mathrm{ROO}^\bullet-\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nG}^{\mathrm{ROO}^\bullet-\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$, respectively. At the same time, chemical reactions can occur in the complexes $\mathrm{ROO}^\bullet$, $\mathrm{ROO}^\bullet$:

$$\mathrm{ROO}^\bullet+\mathrm{ROO}^\bullet\to\mathrm{ROO\text{-}OOR}, \tag{65}$$

which is the neutralization of free radicals due to the emergence of a covalent bond between free radicals $\mathrm{ROO}^\bullet$. In this case, it is important to prescribe in the Hamiltonian of the problem the characteristic interactions $\Phi_{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}')$, which lead to ROO-OOR. $\varphi_{nn}^{\mathrm{ROO}^\bullet-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel, which describes the mutual diffusion of free radicals and dipolar water molecules,

taking into account the influence of the electric field $\boldsymbol{E}(\boldsymbol{r}',t')$ created by all charges of the system; $\varphi_{np}^{\mathrm{ROO}^\bullet-1}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel that describes the dynamic correlations between the diffusion fluxes of free radicals in the intima with the viscous blood flows in the lumen. Analysis of the transport equation for the non-equilibrium average value of the free radical density shows that all processes are important from the point of view of neutralizing free radicals and that vitamins C and high-density lipoproteins play an important role in this. Also important is the equation for the pair non-equilibrium distribution function for the complex $\mathrm{ROO}^\bullet$, $\mathrm{ROO}^\bullet$ with the chemical potential $\mu_{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r}',\boldsymbol{r}'';t')$, which is included in the equation (64).

$$
\begin{aligned}
\frac{\partial}{\partial t}\langle \hat{G}_{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}')\rangle^t = -\int_{-\infty}^{t} e^{\varepsilon(t'-t)}\Bigg(\sum_s \int_{V_2} d\boldsymbol{r}'' \int d\bar{\boldsymbol{d}}''\, \varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-s}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')\,\beta\,\mu_s(\boldsymbol{r}'\bar{\boldsymbol{d}}';t') \\
+\int_{V_2} d\boldsymbol{r}''\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-LE}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\,\beta\,\mu_{LE}(\boldsymbol{r}'';t') \\
+\int_{V_2} d\boldsymbol{r}''\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-HL}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\beta\mu_{HL}(\boldsymbol{r}'';t') \\
+\sum_{f'}\int_{V_2} d\boldsymbol{r}''\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-of'L}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\beta\big(\mu_{of'L}(\boldsymbol{r}'';t')+Z_{f'}e\varphi(\boldsymbol{r}'',t')\big) \\
+\int_{V_2} d\boldsymbol{r}''\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-pL}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\,\beta\,\mu_{pL}(\boldsymbol{r}'';t') \\
+\int_{V_2} d\boldsymbol{r}''\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-H^+}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\,\beta\big(\mu_{H^+}(\boldsymbol{r}'';t')+Z_{H^+}e\varphi(\boldsymbol{r}'',t')\big) \\
+\int_{V_2} d\boldsymbol{r}''\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-AH^-}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\,\beta\big(\mu_{AH^-}(\boldsymbol{r}'';t')+Z_{AH^-}e\varphi(\boldsymbol{r}'',t')\big) \\
+\int_{V_2} d\boldsymbol{r}''\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-A^{-\bullet}}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\,\beta\big(\mu_{A^{-\bullet}}(\boldsymbol{r}'';t')+Z_{A^{-\bullet}}e\varphi(\boldsymbol{r}'',t')\big) \\
+\int_{V_2} d\boldsymbol{r}''\int_{V_2} d\boldsymbol{r}'''\varphi_{GG}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-\mathrm{O}_2^{-\bullet},H^+}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\boldsymbol{r}''';t,t') \\
\quad\times\beta(\mu_{\mathrm{O}_2^{-\bullet},H^+}(\boldsymbol{r}'',\boldsymbol{r}''';t')+Z_{\mathrm{O}_2^{-\bullet}}e\varphi(\boldsymbol{r}'';t')+Z_{H^+}e\varphi(\boldsymbol{r}''';t')) \\
+\int_{V_2} d\boldsymbol{r}''\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\,\beta\big(\mu_{\mathrm{ROO}^\bullet}(\boldsymbol{r}'';t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}'',t')\big) \\
+\int_{V_2} d\boldsymbol{r}''\int_{V_2} d\boldsymbol{r}'''\varphi_{GG}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\boldsymbol{r}''';t,t') \\
\quad\times\beta\big(\mu_{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r}'',\boldsymbol{r}''';t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}'',t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}''',t')\big) \\
+\int_{V_2} d\boldsymbol{r}''\int_{V_2} d\boldsymbol{r}'''\varphi_{GG}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-\mathrm{ROO\text{-}OOR}}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\boldsymbol{r}''';t,t')\beta\mu_{\mathrm{ROO\text{-}OOR}}(\boldsymbol{r}'',\boldsymbol{r}''';t') \\
+\int_{V_1} d\boldsymbol{r}''\varphi_{Gp}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-1}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}'';t') \\
+\int d\boldsymbol{r}''d\bar{\boldsymbol{d}}''\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-w}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}';t,t')\,\beta\big(\mu_w(\boldsymbol{r}'',\bar{\boldsymbol{d}}';t')+\boldsymbol{d}\cdot\boldsymbol{E}(\boldsymbol{r}'',t')\big)\Bigg)dt', \quad (66)
\end{aligned}
$$

where $\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$, $\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ are the transfer kernels describing the interdiffusion processes between the flows of free radical complexes $\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet$ and macromolecules s intima and low and high density lipoproteins. When one of $\mathrm{ROO}^\bullet$ and LE interact, the oxidation of vitamins E in the structure of LE (LDL) occurs. $\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')$ are the transfer kernels that describe the interdiffusion processes between the flows of free radical complexes and low-density lipoproteins, which contain f' of oxidized vitamins E. In this case, it is necessary to take into account the proton-electron transport between $\mathrm{ROO}^\bullet$

and $(\mathrm{ArOH})_{lip}$ (53), (61), and the oxidation process of $(\mathrm{ArOH})_{lip}$ in the LDL structure can be described by the pair non-equilibrium distribution function $\langle \hat{G}_{\mathrm{ROO}^\bullet,LE}(\boldsymbol{r},\boldsymbol{r}')\rangle^t$, and at the same time, ROOH (free radicals are neutralized by proton-electron transport) and $of'L$ — low-density lipoproteins with oxidized f' vitamins E are formed. $\varphi_{nn}^{\mathrm{ROO}^\bullet - of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel that describes the interdiffusion of free radicals and low-density lipoproteins, which contain f' of oxidized vitamins E. In this process, due to proton–electron transport, further oxidation of vitamins E in the LDL structure can occur. Such processes can be described by the pair non-equilibrium distribution function $\langle \hat{G}_{\mathrm{ROO}^\bullet,of'L}(\boldsymbol{r},\boldsymbol{r}')\rangle^t$ and at the same time ROOH are formed again in the complex and further oxidation of LDL occurs. $\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet - pL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel that describes the interdiffusion of free radical complexes and peroxidized low-density lipoproteins in which vitamin E is completely oxidized, and such peroxidized LDLs are actively taken up by macrophages, which can be described by the pairwise nonequilibrium distribution function $\langle \hat{G}_{pL,macr}(\boldsymbol{r},\boldsymbol{r}')\rangle^t$. $\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet - \mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet - \mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet - \mathrm{A}^{-\bullet}}(\boldsymbol{r},\boldsymbol{r}';t,t')$ and $\varphi_{GG}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet - \mathrm{O}_2^{-\bullet},\mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$ are the transfer kernels describing the interdiffusion of free radical complexes and protons H^+, anions AH^-, radicals $\mathrm{A}^{-\bullet}$, $\mathrm{O}_2^{-\bullet}$ together with H^+ taking into account the influence of the scalar potential of the electric field, which as a result of reactions (57)–(60) lead to the neutralization of $\mathrm{ROO}^\bullet$ in the complex, which is very important from the point of view of reducing the oxidation processes of LDL in the intima, while the collective diffusion of free radicals and the mutual diffusion of complexes $\mathrm{ROO}^\bullet$, $\mathrm{ROO}^\bullet$ and complexes of free radicals are described by the transfer nuclei $\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet - \mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{GG}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet - \mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$. The transfer kernel $\varphi_{GG}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet - \text{ROO-OOR}}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\boldsymbol{r}'';t,t')$ describes the interdiffusion of the complex $\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet$ and the products ROO-OOR (with chemical potential $\mu_{\text{ROO-OOR}}(\boldsymbol{r}'',\boldsymbol{r}''';t')$) of reactions between the radicals $\mathrm{ROO}^\bullet$. The influence of viscous blood flows in the lumen on the diffusion processes of the $\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet$ complexes in the intima is described by the transport kernel $\varphi_{Gp}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet - 1}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$, and $\varphi_{Gn}^{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet - w}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel describing the mutual diffusion between the $\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet$ complexes and water molecules, taking into account the influence of the electric field $\boldsymbol{E}(\boldsymbol{r}'',t')$. In the equation for the non-equilibrium distribution function for radicals $\mathrm{ROO}^\bullet$, we have taken into account almost all the interdiffusion and reaction processes between the main components in the intima, which is important from the point of view of inflammatory processes and the formation of aggregates ROO-OOR in the intima. $\langle \hat{G}_{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}')\rangle^t$ can be related (by Laplace transforms in time and Fourier transforms in coordinates) to the non-equilibrium dynamic structure factor $S_{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{k},\omega)$, which is important from the point of view of experimental studies on light scattering and theoretical methods for their calculations.

In the redox processes of macromolecular structures (53), (55), (56), (57)–(60) protons H^+ actively participate. The change in the non-equilibrium average proton density is described by the following equation:

$$
\begin{aligned}
\frac{\partial}{\partial t}\langle \hat{n}_{H^+}(\boldsymbol{r})\rangle^t = &- \int_{-\infty}^{t} e^{\varepsilon(t'-t)} \Bigg(\int d\boldsymbol{r}' \varphi_{nn}^{H^+ - H^+}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{H^+}(\boldsymbol{r}';t') + Z_+ e\varphi(\boldsymbol{r}',t')\big) \\
&+ \sum_s \int_{V_2} d\boldsymbol{r}' \int d\bar{\boldsymbol{d}}' \varphi_{nn}^{\mathrm{H}^+ - s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_s(\boldsymbol{r}'\bar{\boldsymbol{d}}';t') \\
&+ \int d\boldsymbol{r}' d\bar{\boldsymbol{d}}' \varphi_{nn}^{\mathrm{H}^+ - w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\big(\mu_w(\boldsymbol{r}',\bar{\boldsymbol{d}}';t') + \boldsymbol{d}\cdot\boldsymbol{E}(\boldsymbol{r}',t')\big) \\
&+ \sum_{f'} \int d\boldsymbol{r}' \varphi_{nn}^{\mathrm{H}^+ - of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{of'L}(\boldsymbol{r}';t') + Z_{f'} e\varphi(\boldsymbol{r}',t')\big) \\
&+ \int d\boldsymbol{r}' \varphi_{nn}^{\mathrm{H}^+ - \mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{\mathrm{ROO}^\bullet}(\boldsymbol{r}';t') + Z_{\mathrm{ROO}^\bullet} e\varphi(\boldsymbol{r}',t')\big) \\
&+ \int_{V_2} d\boldsymbol{r}' \varphi_{nn}^{\mathrm{H}^+ - pL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{pL}(\boldsymbol{r}';t')
\end{aligned}
$$

$$+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{\mathrm{H}^+-AH^-}(\boldsymbol{r},\boldsymbol{r}';t,t')\beta(\mu_{\mathrm{AH}^-}(\boldsymbol{r}';t')+Z_{\mathrm{AH}^-}e\varphi(\boldsymbol{r}',t'))$$
$$+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{\mathrm{H}^+-\mathrm{A}^{-\bullet}}(\boldsymbol{r},\boldsymbol{r}';t,t')\beta\big(\mu_{\mathrm{A}^{-\bullet}}(\boldsymbol{r}';t')+Z_{\mathrm{A}^{-\bullet}}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\int_{V_2} d\boldsymbol{r}'\int_{V_2} d\boldsymbol{r}''\varphi_{nG}^{\mathrm{H}^+-\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$$
$$\times\beta\big(\mu_{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r}',\boldsymbol{r}'';t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}',t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}'',t')\big)$$
$$+\int_{V_1} d\boldsymbol{r}'\varphi_{np}^{\mathrm{H}^+-1}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\Big)\,dt', \tag{67}$$

where $\varphi_{nn}^{\mathrm{H}^+-\mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel that describes the collective diffusion of protons in the intima and is related to the proton diffusion coefficient $D_{jj}^{\mathrm{H}^+-\mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}';t,t')$:

$$\varphi_{nn}^{\mathrm{H}^+-\mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{\mathrm{H}^+}(\boldsymbol{r}';t')+Z_+e\varphi(\boldsymbol{r}',t')\big)$$
$$=-\frac{\partial}{\partial\boldsymbol{r}}\cdot D_{jj}^{\mathrm{H}^+-\mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}';t,t')\cdot\beta\left(\frac{\partial}{\partial\boldsymbol{r}'}\mu_{\mathrm{H}^+}(\boldsymbol{r}';t')+Z_+e\boldsymbol{E}(\boldsymbol{r}',t')\right) \tag{68}$$

taking into account the influence of the electric field $\boldsymbol{E}(\boldsymbol{r}',t')$. $\varphi_{nn}^{\mathrm{H}^+-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$, $\varphi_{nn}^{\mathrm{H}^+-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ are the transfer kernels that describe the interdiffusion processes of protons with macromolecules s of the intima and dipolar water molecules in the intima, while it is important to take into account the solvation processes. $\varphi_{nn}^{H^+-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transfer kernel that describes the interdiffusion processes of protons and low-density lipoproteins, in which a certain amount f' of vitamins E are oxidized. This is an important contribution because in these processes vitamin E can be reduced and such interaction of protons with f' LDL should be described in detail in the Hamiltonian of the system. The following transfer nuclei included in the equation (67) $\varphi_{nn}^{\mathrm{H}^+-\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nn}^{\mathrm{H}^+-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nn}^{\mathrm{H}^+-\mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nn}^{\mathrm{H}^+-\mathrm{A}^{-\bullet}}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nG}^{\mathrm{H}^+-\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$ are important from the point of view of neutralization of free radicals, radicals and description of redox processes of LDL. Since pH in arterial blood is about 7.4 in venous and tissue fluid 7.35, in the cytosol of cells about 7.0, then $\varphi_{np}^{\mathrm{H}^+-1}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the core of the transfer describing the dissipative processes between the diffusion flows of protons and the viscous alkaline flows of blood. Important in these processes is the transfer equation for the non-equilibrium value of the average density of ascorbic anions $\langle\hat{n}_{\mathrm{AH}^-}(\boldsymbol{r})\rangle^t$, which has the following form:

$$\frac{\partial}{\partial t}\langle\hat{n}_{\mathrm{AH}^-}(\boldsymbol{r})\rangle^t q=-\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\Big(\int d\boldsymbol{r}'\varphi_{nn}^{AH^--AH^-}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{\mathrm{AH}^-}(\boldsymbol{r}';t')+Z_{\mathrm{AH}^-}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\sum_s\int_{V_2}d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\varphi_{nn}^{AH^--s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\beta\mu_s(\boldsymbol{r}'\bar{\boldsymbol{d}}';t')$$
$$+\int d\boldsymbol{r}'d\bar{\boldsymbol{d}}'\varphi_{nn}^{AH^--w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\beta(\mu_w(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')+\boldsymbol{d}\cdot\boldsymbol{E}(\boldsymbol{r}',t'))$$
$$+\sum_{f'}\int d\boldsymbol{r}'\varphi_{nn}^{AH^--of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')\beta(\mu_{of'L}(\boldsymbol{r}';t')+Z_{f'}e\varphi(\boldsymbol{r}',t'))$$
$$+\int d\boldsymbol{r}'\varphi_{nn}^{AH^--ROO^\bullet}(\boldsymbol{r},\boldsymbol{r}';t,t')\beta(\mu_{ROO^\bullet}(\boldsymbol{r}';t')+Z_{ROO^\bullet}e\varphi(\boldsymbol{r}',t'))$$
$$+\int_{V_2}d\boldsymbol{r}'\varphi_{nn}^{AH^--pL}(\boldsymbol{r},\boldsymbol{r}';t,t')\beta\mu_{pL}(\boldsymbol{r}';t')$$
$$+\int_{V_2}d\boldsymbol{r}'\varphi_{nn}^{AH^--H^+}(\boldsymbol{r},\boldsymbol{r}';t,t')\beta(\mu_{AH^-}(\boldsymbol{r}';t')+Z_{AH^-}e\varphi(\boldsymbol{r}',t'))$$
$$+\int_{V_2}d\boldsymbol{r}'\int_{V_2}d\boldsymbol{r}''\varphi_{nG}^{AH^--ROO^\bullet,ROO^\bullet}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')\times$$

$$\beta(\mu_{\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r}',\boldsymbol{r}'';t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}',t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}'',t'))$$
$$+\int_{V_1}d\boldsymbol{r}'\varphi_{np}^{\mathrm{AH}^--1}(\boldsymbol{r},\boldsymbol{r}';t,t')\beta\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\Big)dt', \tag{69}$$

where $\varphi_{nn}^{\mathrm{AH}^--\mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel that describes the collective diffusion of ascorbine anions in the intima and is related to the diffusion coefficient of ascorbine anions $D_{jj}^{\mathrm{AH}^--\mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}';t,t')$:

$$\varphi_{nn}^{\mathrm{AH}^--\mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{AH^-}(\boldsymbol{r}';t')+Z_{AH^-}e\varphi(\boldsymbol{r}',t')\big)$$
$$=-\frac{\partial}{\partial\boldsymbol{r}}\cdot D_{jj}^{\mathrm{AH}^--\mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}';t,t')\cdot\beta\left(\frac{\partial}{\partial\boldsymbol{r}'}\mu_{\mathrm{AH}^-}(\boldsymbol{r}';t')+Z_{\mathrm{AH}^-}e\boldsymbol{E}(\boldsymbol{r}',t')\right) \tag{70}$$

taking into account the influence of the electric field $\boldsymbol{E}(\boldsymbol{r}',t')$. $\varphi_{nn}^{\mathrm{AH}^--s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$, $\varphi_{nn}^{\mathrm{AH}^--w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ are the transfer kernels that describe the interdiffusion processes of ascorbine anions with macromolecules s of the intima and dipolar water molecules in the intima, while it is important to take into account the solvation processes. $\varphi_{nn}^{\mathrm{AH}^--of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel that describes the interdiffusion processes of ascorbic anions and low-density lipoproteins, in which a certain amount of f' of vitamin E is oxidized. This is an important contribution because in these processes vitamin E can be reduced and such interaction of ascorbic anions with f' of LDL should be described in detail in the Hamiltonian of the system. The following transfer nuclei included in the equation (67) $\varphi_{nn}^{\mathrm{AH}^--\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nn}^{\mathrm{AH}^--pL}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nn}^{\mathrm{AH}^--H^+}(\boldsymbol{r},\boldsymbol{r}';t,t')$, $\varphi_{nG}^{\mathrm{AH}^--\mathrm{ROO}^\bullet,\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$ are important from the point of view of neutralizing free radicals and describing LDL redox processes; $\varphi_{np}^{\mathrm{AH}^--1}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describing the dissipative processes between the diffusion flows of ascorbic anions and the viscous alkaline blood flows.

Note that the diffusion movement of peroxidized LDL (pL) is primarily influenced by the macromolecular structure of the endothelium and intima, as well as macrophages that capture them. Therefore, from the system of equations (42)–(46), we extract the equations for non-equilibrium average values for the three types of LDL, taking into account dissipative correlations with macrophages and the macromolecular structure of the endothelium–intima, which is considered as a dynamic porous system of components of the type s:

$$\frac{\partial}{\partial t}\langle\hat{n}_{LE}^{\kappa}(\boldsymbol{r})\rangle^t=-\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\Bigg(\sum_s\int_{V_2}d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\varphi_{nn}^{LE-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_s(\boldsymbol{r}'\bar{\boldsymbol{d}}';t')$$
$$+\int_{V_2}d\boldsymbol{r}'\varphi_{nn}^{LE-\mathrm{ROO}^\bullet}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{\mathrm{ROO}^\bullet}(\boldsymbol{r}';t')+Z_{\mathrm{ROO}^\bullet}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\varphi_{nn}^{LE-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{LE}(\boldsymbol{r}';t')+\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'\varphi_{nn}^{LE-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{HL}(\boldsymbol{r}';t')$$
$$+\sum_{f'}\int_{V_2}d\boldsymbol{r}'\varphi_{nn}^{LE-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{of'L}(\boldsymbol{r}';t')+Z_{f'}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\sum_{\kappa'=1}^{2}\int_{V_{\kappa'}}d\boldsymbol{r}'d\bar{\boldsymbol{d}}'\varphi_{nn}^{LE-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\big(\mu_w(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')+\boldsymbol{d}\cdot\boldsymbol{E}(\boldsymbol{r}',t')\big)$$
$$+\int_{V_2}d\boldsymbol{r}'\varphi_{nn}^{LE-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{pL}(\boldsymbol{r}';t')$$
$$+\int_{V_2}d\boldsymbol{r}'\int_{V_2}d\boldsymbol{r}''d\bar{\boldsymbol{d}}''\varphi_{nG}^{LE-pL,mc}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')\,\beta\,\mu_{pL,mc}(\boldsymbol{r}',\boldsymbol{r}''\bar{\boldsymbol{d}}'';t')$$
$$+\int_{V_1}d\boldsymbol{r}'\varphi_{np}^{LE-1}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\Bigg)dt', \tag{71}$$

where at $\kappa=1$ the equation describes the change in time $\langle\hat{n}_{LE}^{\kappa}(\boldsymbol{r})\rangle^t$ in the lumen, and $\varphi_{nn}^{LE-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel that describes the mutual diffusion motion of lipoproteins LE and macromolecules s of the intima taking into account their rotational motion, $\varphi_{nn}^{LE-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel at $\kappa'=1$ describes the mutual diffusion of lipoproteins LE in the lumen,

and at $\kappa'=2$ describes the mutual diffusion of lipoproteins LE between the lumen – endothelium–intima; $\varphi_{nn}^{LE-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel at $\kappa'=1$ describes the interdiffusion of low-density lipoproteins LE and high-density lipoproteins HL in the lumen, and at $\kappa'=2$ describes the interdiffusion of low-density lipoproteins LE and high-density lipoproteins HL between the lumen – endothelium–intima subsystems; $\varphi_{nn}^{LE-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describing the interdiffusion of low-density lipoproteins LE and low-density lipoproteins in which vitamins E are oxidized, i.e. $of'L$ between the subsystems lumen – endothelium–intima; $\varphi_{nn}^{LE-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ – the transport kernel describing the interdiffusion of low-density lipoproteins LE and low-density lipoproteins in which vitamins E are oxidized and fully peroxidized – pL between the subsystems lumen – endothelium–intima; $\varphi_{nn}^{LE-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel describing the interdiffusion of low-density lipoproteins LE with dipolar water molecules at $\kappa'=1$ in the lumen, and at $\kappa'=2$ between the lumen – endothelium–intima subsystems taking into account the influence of the electric field; $\varphi_{np}^{LE-1}(\boldsymbol{r},\boldsymbol{r}';t,t')$ – the transport kernel reflects the dissipative correlations of the generalized low-density lipoprotein flow and the generalized flow of the total blood momentum density in the lumen of the vessel (which is related to the blood viscous stress tensor, on which the blood viscosity coefficient is built according to the Kubo formulas).

At $\kappa=2$ the equation describes the change in time $\langle\hat{n}_{LE}^{\kappa}(\boldsymbol{r})\rangle^t$ in the endothelium–intima subsystem, and $\varphi_{nn}^{LE-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel that describes the mutual diffusion motion of lipoproteins LE and macromolecules s of the intima taking into account their rotational motion, $\varphi_{nn}^{LE-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel at $\kappa'=1$ describes the mutual diffusion of lipoproteins LE between the endothelium–intima – lumen subsystems, and at $\kappa'=2$ describes the mutual diffusion lipoproteins LE in the endothelium-intima subsystems; $\varphi_{nn}^{LE-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel at $\kappa'=1$ describes the interdiffusion of low-density lipoproteins LE and high-density lipoproteins HL between the lumen–endothelium – intima subsystems, at $\kappa'=2$ describes the interdiffusion of low-density lipoproteins LE and high-density lipoproteins HL in the intima, $\varphi_{nn}^{LE-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describes the interdiffusion of low-density lipoproteins LE and low-density lipoproteins in which vitamins E are oxidized, i.e. $of'L$ in the intima; $\varphi_{nn}^{LE-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describing the interdiffusion of low-density lipoproteins LE and low-density lipoproteins in which vitamins E are oxidized and fully peroxidized – pL; $\varphi_{nn}^{LE-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ – the transport kernel describing the interdiffusion of low-density lipoproteins LE with dipolar water molecules at $\kappa'=1$ in the endothelium-intima and lumen subsystem, and at $\kappa'=2$ in the endothelium–intima subsystem taking into account the influence of the electric field; $\varphi_{nG}^{LE-pL,mc}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')$ is the transport kernel describing the interdiffusion of low-density lipoproteins LE and complexes of peroxidized LDL – pL and macrophages mc; $\varphi_{np}^{LE-1}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel reflecting the dissipative correlations of the generalized flow of low-density lipoproteins and the generalized flow of the total momentum density of blood in the lumen of the vessel (which is related to the blood viscous stress tensor, on which the blood viscosity coefficient is built according to the Kubo formulas). When blood viscosity changes, this transfer core will transmit the effect of changes in the concentration of LDL in the lumen to LDL in the intima. Thus, for $\kappa=1$ and $\kappa=2$, the equations (71) describing changes in time and space of non-equilibrium mean value of low-density lipoproteins take into account the main diffusion, reaction–diffusion and viscous processes between the main components of blood in the lumen and the endothelium–intima.

However, in conditions of inflammatory processes in the intimate area due to the activity of free radicals, the oxidation process of vitamins E in the LDL structure will continue. The change in the non-equilibrium average value of LDL density with a certain amount of oxidized vitamins E is described by the following equation:

$$\frac{\partial}{\partial t}\langle\hat{n}_{ofL}(\boldsymbol{r})\rangle^t=-\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\Bigg(\sum_s\int_{V_2}d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\varphi_{nn}^{ofL-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_s(\boldsymbol{r}'\bar{\boldsymbol{d}}';t')$$
$$+\sum_{\kappa=1}^{2}\int_{V_\kappa}d\boldsymbol{r}'\varphi_{nn}^{ofL-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\mu_{LE}(\boldsymbol{r}';t')+\sum_{\kappa=1}^{2}\int_{V_\kappa}d\boldsymbol{r}'\varphi_{nn}^{ofL-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\mu_{HL}(\boldsymbol{r}';t')$$

$$+\sum_{f'}\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{ofL-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{of'L}(\boldsymbol{r}';t')+Z_{f'}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\int d\boldsymbol{r}'\varphi_{nn}^{ofL-\mathrm{H}^+}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{\mathrm{H}^+}(\boldsymbol{r}';t')+Z_{+}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{ofL-\mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{AH^-}(\boldsymbol{r}';t')+Z_{AH^-}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\int_{V_2} d\boldsymbol{r}'d\bar{\boldsymbol{d}}'\varphi_{nn}^{ofL-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\big(\mu_{w}(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')+\boldsymbol{d}\cdot\boldsymbol{E}(\boldsymbol{r}',t')\big)$$
$$+\int_{V_2} d\boldsymbol{r}'\varphi_{nn}^{ofL-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{pL}(\boldsymbol{r}';t')$$
$$+\sum_{f'}\int_{V_2} d\boldsymbol{r}'\int_{V_2} d\boldsymbol{r}''\varphi_{nG}^{ofL-of'L,\mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$$
$$\times\,\beta\big(\mu_{f',AH^-}(\boldsymbol{r}',\boldsymbol{r}'';t')+Z_{f'}e\varphi(\boldsymbol{r}',t')+Z_{AH^-}e\varphi(\boldsymbol{r}'',t')\big)$$
$$+\int_{V_1} d\boldsymbol{r}'\varphi_{np}^{ofL-1}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\Big)dt', \tag{72}$$

where $\varphi_{nn}^{ofL-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel describing the inter-modification motion of lipoproteins ofL, in which vitamins E and macromolecules s of the intima are oxidized, taking into account their rotational motion, $\varphi_{nn}^{ofL-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel at $\kappa=1$ describing the inter-diffusion of lipoproteins ofL and LE between the lumen-endothelium-intima subsystems, at $\kappa=2$ describing the inter-diffusion of lipoproteins ofL and LE in the intima; $\varphi_{nn}^{ofL-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel at $\kappa=1$ describes the interdiffusion of low-density lipoproteins ofL and high-density lipoproteins HL between the lumen–endothelium – intima subsystems, at $\kappa=2$ describes the inter-diffusion of low-density lipoproteins with oxidized vitamins E – ofL and high-density lipoproteins HL in the intima, $\varphi_{nn}^{ofL-ofL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describes the interdiffusion of low-density lipoproteins with oxidized vitamins E, i.e. ofL in the intima; $\varphi_{nn}^{ofL-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describing the interdiffusion of low-density lipoproteins ofL and fully peroxidized LDL – pL; $\varphi_{nG}^{ofL-of'L,\mathrm{AH}^-}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'';t,t')$ – the transport kernel describing the interdiffusion of low-density lipoproteins ofL and complexes $of'L$ – ascorbate anion AH^-; $\varphi_{np}^{ofL-1}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel reflecting the dissipative correlations of the generalized flow of low-density lipoproteins with oxidized vitamins E and the generalized flow of the total pulse density of blood in the lumen of the vessels. After the complete oxidation of vitamins E in the structure of low-density lipoproteins under the action of oxygen, ofL are further oxidized and become vulnerable to the processes of their capture by macrophages. The transport equation describing the change in time of the non-equilibrium value of the average density of peroxidized LDL – pL has the following structure:

$$\frac{\partial}{\partial t}\langle\hat{n}_{pL}(\boldsymbol{r})\rangle^t=-\int_{-\infty}^{t}e^{\varepsilon(t'-t)}\Big(\sum_{s}\int_{V_2}d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\varphi_{nn}^{pL-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_s(\boldsymbol{r}'\bar{\boldsymbol{d}}';t')$$
$$+\sum_{\kappa=1}^{2}\int_{V_\kappa}d\boldsymbol{r}'\varphi_{nn}^{pL-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{LE}(\boldsymbol{r}';t')+\sum_{\kappa=1}^{2}\int_{V_\kappa}d\boldsymbol{r}'\varphi_{nn}^{pL-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{HL}(\boldsymbol{r}';t')$$
$$+\sum_{f'}\int_{V_2}d\boldsymbol{r}'\varphi_{nn}^{pL-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{of'L}(\boldsymbol{r}';t')+Z_{f'}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\int_{V_2}d\boldsymbol{r}'\varphi_{nn}^{pL-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{pL}(\boldsymbol{r}';t')$$
$$+\int_{V_2}d\boldsymbol{r}'d\bar{\boldsymbol{d}}'\varphi_{nn}^{pL-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\big(\mu_{w}(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')+\boldsymbol{d}\cdot\boldsymbol{E}(\boldsymbol{r}',t')\big)$$
$$+\int_{V_2}d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\varphi_{nn}^{pL-mc}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_{mc}(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')$$

$$+\int_{V_2} d\boldsymbol{r}' \int_{V_2} d\boldsymbol{r}'' d\bar{\boldsymbol{d}}'' \varphi_{nG}^{pL-pL,mc}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')\,\beta\,\mu_{pL,mc}(\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t')$$
$$+\int_{V_2} d\boldsymbol{r}' d\bar{\boldsymbol{d}}' \int_{V_2} d\boldsymbol{r}'' d\bar{\boldsymbol{d}}'' \varphi_{nG}^{pL-mc,mc}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')\,\beta\,\mu_{mc,mc}(\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t')$$
$$+\int_{V_1} d\boldsymbol{r}' \varphi_{np}^{pL-1}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\Big) dt', \tag{73}$$

where $\varphi_{nn}^{pL-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel describing the self-diffusion movement of peroxidized low-density lipoproteins pL and macromolecules s in the intima, studies show that such lipoproteins can only perform subdiffusion (very slow) movements in the intima and cannot return to the lumen, becoming susceptible to capture by macrophages; $\varphi_{nn}^{pL-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describing the self-diffusion movement of peroxidized low-density lipoproteins pL and non-oxidized low-density lipoproteins; $\varphi_{nn}^{pL-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describing the intermodiffusion motion of peroxidized low-density lipoproteins pL and high-density lipoproteins; $\varphi_{nn}^{pL-of'L}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describing the intermodiffusion motion of peroxidized low-density lipoproteins pL and low-density lipoproteins containing f' of oxidized vitamins E; $\varphi_{nn}^{pL-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel describing the very slow collective diffusion of peroxidized low-density lipoproteins pL; $\varphi_{nn}^{pL-mc}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is a transport kernel that describes the mutual diffusion movement of peroxidized low-density lipoproteins pL and macrophages, this is one of the key processes in the capture of pL by macrophages, in which a similar role is played by the transport kernels $\varphi_{nG}^{pL-pL,mc}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')$, $\varphi_{nG}^{pL-mc,mc}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}'',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')$, which describe the dissipative correlations between the flows lipoproteins pL and complexes pL,mc, mc,mc; $\varphi_{np}^{pL-1}(\boldsymbol{r},\boldsymbol{r}';t,t')$ – the transport kernel reflects the dissipative correlations of the generalized flow of peroxidized low-density lipoproteins and the generalized flow of the total pulse density of blood in the lumen of the vessels.

Extremely important role in the fight against atherosclerosis is played by high-density lipoproteins, which transport unused cholesterol (excess) to the liver for the formation of fatty acids, which is important for the digestive system. It is very important to maintain a stable amount of high-density lipoproteins in the blood. The equation for changing the non-equilibrium value of the average density of high-density lipoproteins, taking into account viscous-diffusion-reaction processes, has the form:

$$\frac{\partial}{\partial t}\langle \hat{n}_{HL}^{\kappa}(\boldsymbol{r})\rangle^t = -\int_{-\infty}^{t} e^{\varepsilon(t'-t)}\Big(\sum_s \int_{V_2} d\boldsymbol{r}' \int d\bar{\boldsymbol{d}}' \varphi_{nn}^{HL-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_s(\boldsymbol{r}'\bar{\boldsymbol{d}}';t')$$
$$+\sum_{\kappa'=1}^{2}\int_{V_\kappa} d\boldsymbol{r}' \varphi_{nn}^{HL-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{LE}(\boldsymbol{r}';t') + \sum_{\kappa'=1}^{2}\int_{V_\kappa} d\boldsymbol{r}' \varphi_{nn}^{HL-HL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{HL}(\boldsymbol{r}';t')$$
$$+\int_{V_2} d\boldsymbol{r}' \varphi_{nn}^{HL-ofL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\big(\mu_{of'L}(\boldsymbol{r}';t') + Z_{f'}e\varphi(\boldsymbol{r}',t')\big)$$
$$+\sum_{\kappa'=1}^{2}\int_{V_2} d\boldsymbol{r}' d\bar{\boldsymbol{d}}' \varphi_{nn}^{HL-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\big(\mu_w(\boldsymbol{r}',\bar{\boldsymbol{d}}';t') + \boldsymbol{d}\cdot\boldsymbol{E}(\boldsymbol{r}',t')\big)$$
$$+\int_{V_2} d\boldsymbol{r}' \varphi_{nn}^{HL-pL}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\mu_{pL}(\boldsymbol{r}';t')$$
$$+\int_{V_2} d\boldsymbol{r}' \int_{V_2} d\boldsymbol{r}'' d\bar{\boldsymbol{d}}'' \varphi_{nG}^{HL-pL,mc}(\boldsymbol{r},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')\,\beta\,\mu_{pL,mc}(\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t')$$
$$+\int_{V_1} d\boldsymbol{r}' \varphi_{np}^{HL-1}(\boldsymbol{r},\boldsymbol{r}';t,t')\,\beta\,\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\Big) dt', \tag{74}$$

where at $\kappa = 1$ the equation describes the change in time $\langle \hat{n}_{LH}^{\kappa}(\boldsymbol{r})\rangle^t$ in the lumen, and $\varphi_{nn}^{LH-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel that describes the interdiffusion motion of lipoproteins LH and macromolecules s of the intima taking into account their rotational motion, $\varphi_{nn}^{LH-LE}(\boldsymbol{r},\boldsymbol{r}';t,t')$ is the transport kernel at $\kappa' = 1$ describes the interdiffusion of high-density lipoproteins LH and

low-density lipoproteins LE in the lumen, and at $\kappa' = 2$ describes the interdiffusion of lipoproteins high-density lipoproteins LH and low-density lipoproteins LE between the lumen – endothelium–intima subsystems; $\varphi_{nn}^{LH-LH}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ is the transport kernel at $\kappa' = 1$ describes the interdiffusion of lipoproteins LH in the lumen, and at $\kappa' = 2$ describes the interdiffusion of lipoproteins LH between the lumen – endothelium–intima subsystems; $\varphi_{nn}^{LH-of'L}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ is the transport kernel describes the interdiffusion of high-density lipoproteins LH and low-density lipoproteins in which vitamins E are oxidized, i.e. $of'L$ between the lumen – endothelium–intima subsystems; $\varphi_{nn}^{L-pL}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ is the transport kernel describing the interdiffusion of high-density lipoproteins LH and low-density lipoproteins, in which vitamins E are oxidized and fully peroxidized – pL between the subsystems lumen – endothelium–intima; $\varphi_{nn}^{LH-w}(\boldsymbol{r}, \boldsymbol{r}', \bar{\boldsymbol{d}}'; t, t')$ is the transport kernel describing the interdiffusion of high-density lipoproteins LH with dipolar water molecules at $\kappa' = 1$ in the lumen, and at $\kappa' = 2$ between the subsystems lumen – endothelium–intima, taking into account the influence of the electric field; $\varphi_{np}^{LH-1}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ is the transport kernel reflecting the dissipative correlations of the generalized lipoprotein flux high-density and the generalized flux of the total blood momentum density in the lumen of the vessel (which is related to the blood viscous stress tensor, on which the blood viscosity coefficient is constructed according to the Kubo formulas).

At $\kappa = 2$ the equation describes the change in time $\langle \hat{n}_{LH}^{\kappa}(\boldsymbol{r}) \rangle^t$ in the endothelium–intima subsystem, and $\varphi_{nn}^{LH-s}(\boldsymbol{r}, \boldsymbol{r}', \bar{\boldsymbol{d}}'; t, t')$ is the transport kernel that describes the inter-modification motion of lipoproteins LH and macromolecules s of the intima taking into account their rotational motion, $\varphi_{nn}^{LH-LH}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ is the transport kernel at $\kappa' = 1$ describes the inter-diffusion of lipoproteins LH between the endothelium–intima – lumen subsystems, and at $\kappa' = 2$ describes the inter-diffusion of lipoproteins LH in endothelium–intima subsystems; $\varphi_{nn}^{LH-LE}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ is the transport kernel at $\kappa' = 1$ describes the interdiffusion of high-density lipoproteins LH and low-density lipoproteins LE between the lumen – endothelium–intima subsystems, at $\kappa' = 2$ describes the interdiffusion of high-density lipoproteins LH and low-density lipoproteins LE in the intima, $\varphi_{nn}^{LH-of'L}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ is the transport kernel describes the interdiffusion of high-density lipoproteins LH and low-density lipoproteins in which vitamins E are oxidized, i.e. $of'L$ in the intima; $\varphi_{nn}^{LH-pL}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ is the transport kernel describing the interdiffusion of high-density lipoproteins LH and low-density lipoproteins in which vitamins E are oxidized and fully peroxidized – pL; $\varphi_{nn}^{LH-w}(\boldsymbol{r}, \boldsymbol{r}', \bar{\boldsymbol{d}}'; t, t')$ – the transport kernel describing the interdiffusion of high-density lipoproteins LH with dipolar water molecules at $\kappa' = 1$ in the endothelium-intima and lumen subsystem, and at $\kappa' = 2$ in the endothelium–intima subsystem taking into account the influence of the electric field; $\varphi_{nG}^{LH-pL,mc}(\boldsymbol{r}, \boldsymbol{r}', \boldsymbol{r}'', \bar{\boldsymbol{d}}''; t, t')$ is the transport kernel describing the interdiffusion of high-density lipoproteins LH and complexes of peroxidized LDL – pL and macrophages mc; $\varphi_{np}^{LH-1}(\boldsymbol{r}, \boldsymbol{r}'; t, t')$ is the transport kernel reflecting the dissipative correlations of the generalized flow of high-density lipoproteins and the generalized flow of the total momentum density of blood in the lumen of the vessel (which is related to the blood viscous stress tensor, on which the blood viscosity coefficient is built according to the Kubo formulas).

Thus, the set of equations (71)–(74) describes the change in time of the average density of low- and high-density lipoproteins in the lumen and in the endothelium-intima subsystem of vessels. They do not take into account the processes of maturation of LDL from very low-density lipoproteins (produced in the liver) with the participation of HDL, as well as the maturation of HDL itself (produced in the liver). In the absence of inflammatory processes in the endothelium–intima subsystem, normal functioning of the immune system, the movement of low- and high-density lipoproteins is balanced and excess cholesterol that was not used in cellular processes is removed by high-density lipoproteins, which transport it to the liver with subsequent processing into bile acids for digestion processes in the gastrointestinal tract. However, the presence of inflammatory processes is accompanied by an increased concentration of peroxide free radicals, while the concentration of lipoproteins $\langle \hat{n}_{ofL}(\boldsymbol{r}) \rangle^t$ and $\langle \hat{n}_{pL}(\boldsymbol{r}) \rangle^t$ increases, and macrophages, which are constantly in the intima (their lifespan is from 45 days to several months), enter the protective processes. Macrophages are constantly transformed from monocytes (large white blood cells, their share does not exceed 3–8% of the total number of leukocytes, which are formed in

the bone marrow and play an important role in protecting the body from infections and inflammatory processes) during their transition from the lumen to the endothelium-intima subsystem. Monocytes remain in the bloodstream for only one to three days, after which they migrate to the tissues, where they transform into macrophages or dendritic cells. These transformed cells actively participate in the fight against microorganisms, the elimination of damaged tissues and the regulation of inflammatory reactions. After activation, macrophages secrete a variety of cytokines and chemokines, which "signal" to monocytes about the presence of inflammatory processes in the corresponding area in the lumen–endothelium – intima system. This causes the directed movement of monocytes to the area of inflammatory processes and their transition from the lumen to the endothelium–intima and their transformation into macrophages, which again secrete many cytokines and chemokines and the process takes on an avalanche–like character. At the same time, pro- and anti-inflammatory cytokines are released, and as noted in the introduction, in the work [55] it was shown that when a high concentration of $\langle \hat{n}_{pL}(\boldsymbol{r})\rangle^t$ and a moderate concentration of pro- and anti-inflammatory cytokines are present in the intima, inflammation will increase and reach the stage of chronic atherosclerosis. However, if the concentration of anti-inflammatory cytokines in the intima is sufficiently high, inflammation may increase but will not reach the stage of chronic atherosclerosis. Since the life spans of monocytes and macrophages are finite, although very large compared to the times of intermolecular interactions, it is important to consider that macrophages disintegrate after the end of their life span, and the pigments themselves that the cell has absorbed are released together with the decay cells and are again absorbed by "new" macrophage cells. This process is very complex and free radicals arise again in it, and therefore a high concentration of antioxidants is important, in particular vitamin C, according to the reactions in the redox processes of macromolecular structures (53), (55), (56), (57)–(60). In the area of inflammatory processes in the intima, with an increase in the concentration of macrophages, especially those loaded with peroxidized low-density lipoproteins, as life experience shows (my personal experience, since I suffered a heart attack with subsequent stenting), they can aggregate into foamy structures, which subsequently form plaques. Therefore, the next important step from the point of view of mathematical modeling of atherosclerosis processes is to obtain equations for the non-equilibrium average value of macrophage density $\langle \hat{n}_{mc}(\boldsymbol{r},\bar{\boldsymbol{d}})\rangle^t$, and correlation functions $\langle \hat{G}^{pL,mc}_{nn}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}')\rangle^t$, $\langle \hat{G}^{mc,mc}_{nn}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}')\rangle^t$. The equation for $\langle \hat{n}_{mc}(\boldsymbol{r},\bar{\boldsymbol{d}})\rangle^t$ has the following form:

$$
\begin{aligned}
\frac{\partial}{\partial t}\langle \hat{n}_{mc}(\boldsymbol{r},\bar{\boldsymbol{d}})\rangle^t = &-\int_{-\infty}^{t} e^{\varepsilon(t'-t)}\Bigg(\sum_s \int_{V_2} d\boldsymbol{r}' \int d\bar{\boldsymbol{d}}'\,\varphi^{mc-s}_{nn}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_s(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')\\
&+\int_{V_2} d\boldsymbol{r}'\varphi^{mc-LE}_{nn}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')\beta\mu_{LE}(\boldsymbol{r}';t')+\int_{V_2} d\boldsymbol{r}'\varphi^{mc-HL}_{nn}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')\beta\mu_{HL}(\boldsymbol{r}';t')\\
&+\int_{V_2} d\boldsymbol{r}'\varphi^{mc-ofL}_{nn}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')\,\beta\big(\mu_{of'L}(\boldsymbol{r}';t')+Z_{f'}e\varphi(\boldsymbol{r}',t')\big)\\
&+\int_{V_2} d\boldsymbol{r}'\varphi^{mc-pL}_{nn}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')\,\beta\,\mu_{pL}(\boldsymbol{r}';t')\\
&+\sum_{\kappa'=1}^{2}\int_{V_2} d\boldsymbol{r}'d\bar{\boldsymbol{d}}'\varphi^{mc-w}_{nn}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\big(\mu_w(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')+\boldsymbol{d}\cdot\boldsymbol{E}(\boldsymbol{r}',t')\big)\\
&+\int_{V_2} d\boldsymbol{r}'\int d\bar{\boldsymbol{d}}'\varphi^{mc-mc}_{nn}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')\,\beta\,\mu_{mc}(\boldsymbol{r}',\bar{\boldsymbol{d}}';t')\\
&+\int_{V_2} d\boldsymbol{r}'d\bar{\boldsymbol{d}}'\int_{V_2} d\boldsymbol{r}''d\bar{\boldsymbol{d}}''\varphi^{mc-mc,mc}_{nG}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')\,\beta\,\mu_{mc,mc}(\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t')\\
&+\int_{V_2} d\boldsymbol{r}'d\bar{\boldsymbol{d}}'\int_{V_2} d\boldsymbol{r}''d\bar{\boldsymbol{d}}''\varphi^{mc-pL,mc}_{nG}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')\,\beta\,\mu_{pL,mc}(\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t')\\
&+\int_{V_1} d\boldsymbol{r}'\varphi^{mc-1}_{np}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')\beta\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')\Bigg)dt',
\end{aligned}
\tag{75}
$$

where $\varphi^{mc-s}_{nn}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel describing the self-modification movement of macrophages and macromolecules s of the intima, $\varphi^{mc-LE}_{nn}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')$, $\varphi^{mc-LH}_{nn}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')$ are

the transport kernels describing the self-modification movements of macrophages and low- and high-density lipoproteins; $\varphi_{nn}^{mc-ofL}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')$ is the transport kernel describing the self-modification movement of macrophages and low-density lipoproteins in which f of vitamins E are oxidized, $\varphi_{nn}^{mc-pL}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')$ is the transport kernel describing the self-modification movement of macrophages and peroxidized low-density lipoproteins pL, which are in favorable states for their capture by macrophages; $\varphi_{nn}^{mc-w}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel that describes the self-diffusion motion of macrophages and dipolar water molecules taking into account the influence of the electric field; $\varphi_{nn}^{mc-mc}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}';t,t')$ is the transport kernel describing the collective very slow diffusion of macrophages, $\varphi_{nG}^{mc-mc,mc}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')$ is the transport kernel describing the self-diffusion movement of macrophages and the mc,mc complex, which can be an essential signal in the processes of macrophage aggregation, $\varphi_{nG}^{mc-pL,mc}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')$ is the transport kernel describing the self-modification motion of macrophages and the pL,mc complex, which can also be an essential signal in the processes of macrophage aggregation, $\varphi_{np}^{mc-1}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}';t,t')$ is the transport kernel reflecting the dissipative correlations of the generalized subdiffusion flow of macrophages and the generalized flow of the total blood momentum density in the lumen of the vessel (which is related to the viscous stress tensor of blood). This contribution can be considered small from the point of view of the subdiffusion motions of macrophages.

$$
\begin{aligned}
&\frac{\partial}{\partial t}\big\langle \hat{G}_{nn}^{pL,mc}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}')\big\rangle^t = \big\langle iL_N(t)\hat{G}_{nn}^{pL,mc}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}')\big\rangle_{rel}^t\\
&-\int_{-\infty}^{t} e^{\varepsilon(t'-t)}\Bigg(\sum_s\int_{V_2} d\boldsymbol{r}''\int d\bar{\boldsymbol{d}}''\varphi_{Gn}^{pL,mc-s}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')\,\beta\,\mu_s(\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t')\\
&+\int_{V_2} d\boldsymbol{r}''\varphi_{Gn}^{pL,mc-pL}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'';t,t')\,\beta\,\mu_{pL}(\boldsymbol{r}'';t')\\
&+\int_{V_2} d\boldsymbol{r}''\int_{V_2} d\boldsymbol{r}'''d\bar{\boldsymbol{d}}'''\varphi_{GG}^{pL,mc-pL,mc}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\boldsymbol{r}''',\bar{\boldsymbol{d}}''';t,t')\,\beta\,\mu_{pL,mc}(\boldsymbol{r}'',\boldsymbol{r}''',\bar{\boldsymbol{d}}''';t')\\
&+\int_{V_2} d\boldsymbol{r}''d\bar{\boldsymbol{d}}''\int_{V_2} d\boldsymbol{r}'''d\bar{\boldsymbol{d}}'''\varphi_{GG}^{pL,mc-mc,mc}(\boldsymbol{r},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\bar{\boldsymbol{d}}',\boldsymbol{r}''',\bar{\boldsymbol{d}}''';t,t')\beta\mu_{mc,mc}(\boldsymbol{r}'',\bar{\boldsymbol{d}}'',\boldsymbol{r}''',\bar{\boldsymbol{d}}''';t')\Bigg)dt', \quad (76)
\end{aligned}
$$

$$
\begin{aligned}
&\frac{\partial}{\partial t}\big\langle \hat{G}_{nn}^{mc,mc}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}')\big\rangle^t = \big\langle iL_N(t)\hat{G}_{nn}^{mc,mc}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}')\big\rangle_{rel}^t\\
&-\int_{-\infty}^{t} e^{\varepsilon(t'-t)}\Bigg(\sum_s\int_{V_2} d\boldsymbol{r}''\int d\bar{\boldsymbol{d}}''\varphi_{Gn}^{mc,mc-s}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'';t,t')\,\beta\,\mu_s(\boldsymbol{r}'',\bar{\boldsymbol{d}}';t')\\
&+\int_{V_2} d\boldsymbol{r}''\varphi_{Gn}^{mc,mc-pL}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'';t,t')\,\beta\,\mu_{pL}(\boldsymbol{r}'';t')\\
&+\int_{V_2} d\boldsymbol{r}''\int_{V_2} d\boldsymbol{r}'''d\bar{\boldsymbol{d}}'''\varphi_{GG}^{mc,mc-pL,mc}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\boldsymbol{r}''',\bar{\boldsymbol{d}}''';t,t')\,\beta\,\mu_{pL,mc}(\boldsymbol{r}'',\boldsymbol{r}''',\bar{\boldsymbol{d}}''';t') \quad (77)\\
&+\int_{V_2} d\boldsymbol{r}''d\bar{\boldsymbol{d}}''\int_{V_2} d\boldsymbol{r}'''d\bar{\boldsymbol{d}}'''\varphi_{GG}^{mc,mc-mc,mc}(\boldsymbol{r},\bar{\boldsymbol{d}},\boldsymbol{r}',\bar{\boldsymbol{d}}',\boldsymbol{r}'',\bar{\boldsymbol{d}}'',\boldsymbol{r}''',\bar{\boldsymbol{d}}''';t,t')\,\beta\,\mu_{mc,mc}(\boldsymbol{r}'',\bar{\boldsymbol{d}}'',\boldsymbol{r}''',\bar{\boldsymbol{d}}''';t')\Bigg)dt'.
\end{aligned}
$$

It is important to note that a special problem is the calculations of generalized transport kernels, which contain the main mechanisms of viscous, diffusion–reaction, redox processes in the lumen–endothelium – intima system in the obtained transport equations. Also important is the presence of polarization processes associated with the transport of ions, electrons, water molecules, macromolecular structures in the electromagnetic fields they create. The electromagnetic fields themselves undoubtedly affect viscous, diffusion-reaction, redox processes and this must be taken into account when calculating the transport kernels. The exact calculation of the transport nuclei is currently impossible, therefore approximate calculations based on physical, chemical, biological assumptions are necessary. In this case, it is important to understand the spatial–temporal correlations of processes and their mutual influence, self-organization. Analysis of viscous, diffusion–reaction, redox processes already at the level

of the obtained transport equations indicates that for the normal functioning of the cardiovascular system, the balance of ions K^+, Na^+, Ca^{2+} (in particular, ion channels), H^+, OH^-, Cu^+, Fe^{2+}, Mg^{2+}, molecules H_2O, O_2, NO, vitamins E, C, A, B-group, amino acids L-arginine, L-carnitine and others is important. Their deficiency or excess leads to imbalance and, in particular, at the level of low-density lipoproteins, to the gradual formation of plaques – the formation of atherosclerotic processes in the intima of blood vessels.

4. Conclusion

A statistical approach to the description of non-equilibrium processes of blood component transport in the lumen–endothelium–intima system of blood vessels is proposed. This approach involves taking into account the nature of the interactions of blood components, which to one degree or another participate in atherosclerotic processes. It is based on the parameters of the abbreviated description: non-equilibrium average values of the densities of blood components, ions, water molecules, in particular LDL, HDL, monocytes, and other components (erythrocytes, leukocytes, platelets) and the non-equilibrium value of the total blood pulse density for the vascular lumen subsystem; in the endothelium-intima region, the parameters of the abbreviated description are non-equilibrium average values of the densities of the components LDL, ox-LDL, HDL, macrophages, water molecules, ions, free radicals, oxidants, vitamin C, pro-inflammatory and anti-inflammatory cytokines, T-cells, macromolecular structures of the endothelium – intima. Using the method of the non-equilibrium statistical operator for the parameters of the abbreviated description, a system of transport equations (42)–(46) was obtained, which, within the framework of the selected model of component interaction, can describe non-Markovian in time and non-local in space blood transport processes in vessels, taking into account possible reaction–diffusion processes in the vessel walls. All its equations for the corresponding transport kernels include the hydrodynamic blood velocity $\boldsymbol{v}^{(1)}(\boldsymbol{r}';t')$, as one of the important parameters of the studies. In addition, this system of transport equations is open-ended, nonlinear and can be applied to the description of both laminar and turbulent processes. It is important to note that in the first subsystem – the lumen, a viscous reaction-diffusion description was used, and in the endothelium-intima subsystem – a reaction–diffusion description. In addition, this approach takes into account the electromagnetic fields created by ions and dipole molecules, for example water, the intensities of which satisfy the averaged Maxwell equations.

Based on such a statistical model, it is important to consider two important tasks: the description of the processes of subdiffusion of oxidized LDL and macrophages in the endothelium-intima subsystem, and the possibility of describing the influence of turbulent (weak) blood processes in the lumen on subdiffusion processes in the endothelium–intima subsystem.

In the following works, we will consider one of the ways of approximate calculation of generalized transport kernels and conduct a study of subdiffusion processes for ox-LDL and macrophages in the intima, using the methodology of fractional derivatives [73, 79].

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Статистичний підхід до опису процесів транспорту ліпопротеїнів та інших компонент у кровоносних судинах – I

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Для опису атеросклеротичних процесів у інтимі судин кровотоку запропоновано статистичний підхід опису нерівноважних процесів переносу компонент крові у системі просвіт–ендотелій–інтима кровоносних судин, який передбачає врахування характеру взаємодій між компонентами крові. Використавши метод нерівноважного статистичного оператора для параметрів скороченого опису, отримано систему рівнянь переносу, яка в рамках вибраної моделі взаємодії компонент може описувати немарковські у часі та нелокальні у просторі процеси переносу крові у судинах з врахуванням можливих реакційно-дифузійних процесів у стінках судин. У просвіті застосовувався в'язко реакційно-дифузійний опис, а у підсистемі ендотелій–інтима — реакційно-дифузійний.

Ключові слова: *атеросклероз; кровоносні судини; еритроцити; ліпопротеїни; ліпопротеїни низької щільності; процеси дифузії іонів та молекул води; нерівноважний статистичний оператор; рівняння Максвелла; вільні пероксидні радикали; оксид азоту; макрофаги.*