Ghazaryan, J Tradit Med Clin Natur 2017, 6:2
DOI: 10.4172/2573-4555.1000e131

Journal of
Traditional Medicine & Clinical Naturopathy

Editorial Open Access

Study of Lipid Specters and Antioxidation Activity in Neurodegenerative Diseases

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In numerous works published in recent years and related to the behavior of cells in various damaging conditions (both *in vitro* and *in vivo*, including the pathological state of the organism and external influences), damage to the membrane systems of the cell occupies an essential place either as the first target to be damaged or As a system that determines in many ways the further fate of the cell.

Studying the properties of the neuron membrane for various brain diseases, changing the composition of membrane lipids and their fatty acid composition are of primary importance in the development of neuronal dysfunction [1]. In this connection, the study undertaken by us concerns quantitative changes in different categories of lipids and proteins in various neurodegenerative diseases (Parkinson's disease, Glaucoma, and Alzheimer's disease).

Lipids (neutral and acidic glycolipids, phospholipids) are the most important biological effectors, regulators, and mediators that participate in almost all important physiological processes (immune response, neuronal information transfer, vascular and muscle tone regulation, homeostasis, inflammation, etc.). There are numerous data on the involvement of various lipid categories in signaling processes (as secondary messengers), as well as in processes leading to the modulation of the activity of various protein kinases leading to the induction of apoptosis or necrosis. Of particular importance are studies of the functional role of acid and neutral glycolipids in the development of the pathogenesis of diseases of the nervous system due to the uniqueness of the chemical composition and the rich content of lipids (phospholipids, gangliosides, cerebrosides) insss the brain. The study of the glycolipid composition revealed a decrease in the content of acidic and neutral glycolipids in the brain of rats with simulated Parkinson's disease [2].

While it is difficult to interpret the biological significance of the changes in the various categories of lipids occurring in the brain tissue in the experimental PS, the existence of interfractional changes in the content of glycolipids and phospholipids remains indisputable [3].

The study of the content of the product of their hydrolytic decomposition, sphingosine, revealed its increase, which indicates the activation of ceramidase and sphingomyelinase enzymes, leading to the accumulation of ceramide and sphingosine. Sphingosine is able to enhance apoptosis by inhibiting protein kinase C. Using against the background of the described disorders, the lithium salt of the amino acid cysteine is accompanied by a marked increase in the content of the studied glycolipids [4].

The changes in the phospholipid composition and membranes revealed by us indicate their structural disorganization, which undoubtedly affects the physicochemical properties of the cell membrane. The physico-chemical state of the lipid bilayer of the plasma membrane controls the activity of membrane enzymes both by changing their conformation and by changing the diffusion of the substrate and the activator ions to the active sites. Mechanisms of damage to membrane enzymes in violation of the lipid composition concern either damage to the enzyme protein itself or damage to the microenvironment of the enzyme that affects the activity of the enzyme.

One of the mechanisms of disturbance of intracellular metabolism of lipids is peroxide oxidation of lipids and proteins. The intensification of this process leads to the formation of an excessive amount of free radicals, which disturbs the state of the cell membranes and the colloidal state of the protoplasm. In connection with the foregoing, the intensification of LPO in the simulation of the PS is of particular importance. Intensification of LPO was detected by us both in the enzymatic-NADPH-dependent, and in non-enzymatic-ascorbate-dependent oxidation systems. The triggering mechanism of LPO activation is the activation of the oxidative metabolism of dopamine and other neurotransmitters, in which free radical compounds are generated that aggravate NMDA mediated neurotoxicity and deplete the endogenous antioxidant defense system. To date, defects in mitochondrial DNA, increased apoptotic cascade and chronic oxidative stress are considered to be the main pathological processes leading to progressive and irreversible death of affected neurons [5-8].

Neurons are the most highly specialized cells of the body and perform the most complex functions that provide consciousness, movement, sensitivity and adaptation to the constantly changing conditions of the external and internal environment. Such intense activity requires extremely high energy costs.

Excessive reactions of LPO in pathological conditions damage, in the first place, membranes of neurons and their intracellular organelles (mitochondria, nuclei, lysosomes, endoplasmic reticulum). Taking into account the importance of biological membranes for the vital activity of any cellular structures, it becomes clear why oxidative stress is accompanied by catastrophic consequences for the cell, up to its death. Especially this applies to excitable cells (neurons, muscular fibrils of skeletal muscles and heart muscle), in the implementation of the functions of which the important point is the generation of the action potential-a change in the charge of the cell membrane in response to certain stimuli.

As a result of activation of lipid peroxidation and proteins, the membrane lipid layer becomes more permeable for protons (or hydroxyl ions) and for Ca^{2+}. An increase in the proton permeability of

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Received April 07, 2017; **Accepted** April 10, 2017; **Published** April 14, 2017

Citation: Ghazaryan G (2017) Study of Lipid Specters and Antioxidation Activity in Neurodegenerative Diseases. J Tradit Med Clin Natur 6: e131. doi: 10.4172/2573-4555.1000e131

membranes leads to an energy deficiency of the oxidative process in the cell, which leads to a disruption in the functions of the mitochondria. The neuron's need for energy is higher than other cells, the energy deficit leads to a neuron dystrophy [9,10].

Thus, it is obvious that the development of the pathogenesis of the pathological process studied by us is accompanied by molecular changes in the membrane of cells that are both an immediate target of the damaging effect of pathogenic factors and those involved in the pathological process in connection with the initiation of universal mechanisms of cell damage (deficit of energy production, intensification of processes of free radical oxidation, Activation of phospholipases, proteases, disruption of ion homeostasis, etc.).

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