



## Study of Lipid Specters and Antioxidation Activity in Neurodegenerative Diseases

Gayane Ghazaryan\*

Institute of Molecular Biology, National Academy of Sciences of the Republic of Armenia (NASRA), Yerevan 0019, Armenia

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) in 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  $\text{Ca}^{2+}$ . An increase in the proton permeability of

\*Corresponding author: Gayane Ghazaryan, Institute of Molecular Biology, National Academy of Sciences of the Republic of Armenia (NASRA), 24 Marshal Baghramyan Ave, Yerevan 0019, Armenia, Tel: +37477267539; E-mail: [gaykaz70@mail.ru](mailto:gaykaz70@mail.ru)

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.

Copyright: © 2017 Ghazaryan G. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

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.).

## References

1. Haag M (2003) Essential fatty acids and the brain. *Can J Psychiatry* 48: 195-203
2. Ghazaryan GS, Hovsepyan LM, Zakaryan (2009) Phospholipids in dynamics of free radical oxidation of lipids in biological membranes brain under conditions of Parkinson's disease. Abs. 9th World Congress of Biological Psychiatry, Paris, France.
3. Veldhuizen R, Possmayer F (2004) Phospholipid metabolism in lung surfactant. *Subcell Biochem* 37: 359-388.
4. Ghazaryan G, Hovsepyan L, Zanginyan H (2013) Oxidative Modification of Proteins in Experimental Parkinsons Syndrome, 7<sup>th</sup> Neurodegenerative Conditions Research Development, Boston, MA, USA.
5. Büeler H (2010) Mitochondrial dynamics, cell death and the pathogenesis of Parkinson's disease. *Apoptosis* 15: 101-108.
6. Stadtman ER, Levine RL (2000) Protein oxidation. *Ann N Y Acad Sci* 899: 191-208.
7. Vlajinac HD, Sipetic SB, Maksimovic JM, Marinkovic JM, Dzoljic ED, et al. (2010) Environmental factors and Parkinson's disease: a case-control study in Belgrade, Serbia *Int J Neurosci* 120: 361-367.
8. Martínez A, Portero-Otin M, Pamplona R, Ferrer I (2010) Protein targets of oxidative damage in human neurodegenerative diseases with abnormal protein aggregates. *Brain Pathol* 20: 281-297
9. Tampo Y (2000) Studies on membrane Factors in iron-supported lipid peroxidation. *Yakugaku Zasshi* 120: 387-396.
10. Uttara B, Singh AV, Zamboni P, Mahajan RT (2009) Oxidative stress and neurodegenerative diseases: a review of upstream and downstream antioxidant therapeutic options. *J Curr Neuropharmacol* 7: 65-74.

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

## OMICS International: Open Access Publication Benefits & Features

### Unique features:

- Increased global visibility of articles through worldwide distribution and indexing
- Showcasing recent research output in a timely and updated manner
- Special issues on the current trends of scientific research

### Special features:

- 700+ Open Access Journals
- 50,000+ Editorial team
- Rapid review process
- Quality and quick editorial, review and publication processing
- Indexing at major indexing services
- Sharing Option: Social Networking Enabled
- Authors, Reviewers and Editors rewarded with online Scientific Credits
- Better discount for your subsequent articles

Submit your manuscript at: [www.omicsonline.org/submit](http://www.omicsonline.org/submit)