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Alzheimer disease: Past present future

Neurodegenerative disorders are characterized by a loss of cognitive function and inappropriate death of nerve cells in areas of the brain that control such functions as memory and language. The trigger for nerve cell death is unknown in the AD, as well as in other neurodegenerative conditions, in which memory decline is a prominent feature. A rapidly growing body of evidence indicates that increased oxidative stress resulting from reactive oxygen radicals is associated with the aging process and age-related degenerative disorders such as atherosclerosis, ischemia/reperfusion, and arthritis, stroke, and neurodegenerative diseases. Reactive oxygen species (ROS) are generated at sites of inflammation and injury, and at low levels, they can function as signaling intermediates in the regulation of fundamental cell activities such as growth and adaptation responses. At higher concentrations, ROS can cause cell injury and death. The vascular endothelium, which regulates the passage of macromolecules and circulating blood to cells and tissue, is a major target of oxidative stress, playing a critical role in the pathophysiology of vascular diseases. Since the vascular endothelium, neurons, and glia are all able to synthesize, store and release ROS and vascular active substances in response to certain stimuli, their contribution to the pathophysiology of atherosclerosis, stroke, other non-atherosclerotic cerebrovascular disease and neurodegenerative syndrome such as mild cognitive impairment (MCI) and the AD is extremely important. In addition, abnormalities in cholesterol metabolism, oxidative stress, and vascular lesions are important factors in the pathogenesis of late-onset forms of the AD, forms of mental retardation, stroke and MCI. This idea is based on the positive correlations found between stroke, MCI, AD and cardiovascular diseases. New evidence indicates that continuous formation of free ROS induces cellular damage and decreases antioxidant defenses. Specifically, oxidative stress increases vascular endothelial permeability and promotes leukocyte adhesion, all of which are coupled with alterations in endothelial signal transduction and redox-regulated transcription factors. We theorize that the cellular and molecular mechanisms, by which cholesterol metabolism abnormalities induce the formation of large amounts of ROS, decrease endothelial barrier function via the overexpression of inducible nitric oxide synthase (iNOS) and promote leukocyte adhesion. Chronic injury stimuli have the action of inducing decompensation and or alterations in normal vascular function, which results in the development of cerebrovascular arterio- and atherosclerosis that further manifest as stroke, MCI and/or AD.

Recent Publications

1. Bachurin SO, Gavrilova SI, Samsonova A, Barreto GE, Aliev G. Mild cognitive impairment due to Alzheimer disease: Contemporary approaches to diagnostics and pharmacological intervention. Pharmacol Res. 2017 Nov 21. pii: S1043-6618(17)30937-4. doi: 10.1016/j.phrs.2017.11.021. [Epub ahead of print].
2. Dzyurkevich MS, Babkov DA, Shtyrlin NV, Mayka OY, Iksanova AG, Vassiliev PM, Balakin KV, Spasov AA, Tarasov VV, Barreto G, Shtyrlin YG, Aliev G. Pyridoxine dipharmacophore derivatives as potent glucokinase activators for the treatment of type 2 diabetes mellitus. Sci Rep. 2017 Nov 22;7(1):16072. doi:10.1038/s41598-017-16405-2.
3. Kosenko EA, Tikhonova LA, Montoliu C, Barreto GE, Aliev G, Kaminsky YG. Metabolic Abnormalities of Erythrocytes as a Risk Factor for Alzheimer's Disease. Front Neurosci. 2018 Jan 5;11:728. doi: 10.3389/fnins.2017.00728. eCollection 2017.

Biography

Gjumrakch Aliev is the President of GALLY International Biomedical Research Institute Inc., San Antonio, Texas, USA. He also holds appointment with the University of Atlanta, Georgia, USA as a Professor of Cardiovascular, Neuropathology, Gerontology, Health Science and Healthcare Administration and as a Leading Scientist, Institute of Physiologically Active Compounds, Russian Academy of Sciences, Moscow Region, Chernogolovka, Russian Federation. He completed his MD in 1982 at the Baku Medical University (former USSR) with cum laude. Then he completed his PhD in Cardiovascular Diseases at the prestigious Russian Academy of the Medical Sciences, Moscow, Russia in 1988 with cum laude. He received postdoctoral training with Professor G Burnstock at University College of the London. He is an outstanding teacher, scholar, and a renowned scientist in the area of cellular molecular physiology, and cardiovascular, and neurodegeneration-mediated pathologies including Alzheimer disease (AD). He is nationally and internationally reputed in his area. He is one of the most cited authors in his fields with high impact factors.

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