

JOINT EVENT

11th International Conference on **Vascular Dementia**
&
27th Euro-Global Neurologists Meeting

July 23-25, 2018 | Moscow, Russia



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Implication of the multitarget therapies in the context of the aged associated oxidative stress induced cellular and subcellular hypoperfusion and mitochondrial DNA deletion during the development and maturation of Alzheimer disease: Past present future

Background & Hypothesis: Oxidative stress induced cerebral hypoperfusion and mitochondrial failure appears to be a key pathogenic factor in the development of age-associated diseases, triggering mild cognitive impairment and eventual conversion to Alzheimer disease (AD). Mitochondrial integrity is associated with cellular viability.

Aim: We studied cellular and subcellular features of hippocampal neurons and microvessel mitochondrial lesions, oxidative stress markers and protein immunoreactivity in animal models that mimic MCI and/or AD. In addition, we studied the effects of dietary antioxidant treatment on neuronal mitochondrial ultrastructure in rats. The goals of the proposed study are to determine the role of mitochondria failure and neuronal damage during the maturation of AD-like pathology in rats and transgenic mice overexpressing either amyloid β precursor protein (A β PP).

Methods: In this project, we applied the following methods: transmission electron microscopy (TEM) qualitative analysis; EM pre-embedding immunogold cytochemistry using probes for human wild type, 5 kb deleted and mouse mtDNA and antibodies against cytochrome c oxidase and quantitative morphometric analysis of the degree of mitochondrial lesions.

Results: There was a significantly higher degree of mitochondrial damage and mitochondrial DNA overproliferation and deletion in neurons and cerebrovascular wall cells in transgenic mice and aged untreated rats in comparison to age-matched controls and non-treated subjects. Mitochondrial abnormalities are associated with atherosclerotic lesions of brain microvessels.

Conclusion: based on this results our conclusion is that changes in mitochondrial morphology and mitochondrial DNA coexist with metabolic dysfunction in AD mice and age-associated neurodegeneration and may serve as diagnostic markers and treatment targets.

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.

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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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