By using electron microscopic techniques we have found that the mitochondrial lesions appeared to be the primary hallmark of the glioblastoma. Vessel endothelium from tumor tissues shows the damage of mitochondrion cristae. The mitochondria derived lysosomes appeared to be permanent feature of the glial cells derived tumor cells. The lipid laden tumor cells and surrounding cells often show a different degree of mitochondria abnormality (such as mitochondria with broken cristae, presence of edema in their matrix, disruption of inner and external mitochondrial membrane). Moreover, giant mitochondria also appeared to be permanent features of tumor growth and metastases. Comparative characteristics of marginal and central portion of tumor tissues obtained from patients undergoing surgery with diagnosis of the primary glioblastoma showed that distance area of tumor tissue characterized heterogeneous distribution of damage in the structure of the mitochondria. Central regions of tumor tissues in almost all of area shows astrocytes with clusters of mitochondria derived lysosomes. The same patterns of cellular and subcellular damage were seen in spinal cord tumor.

One of the big challenges for treatment of neurodegenerative diseases and cancer appeared to be delivering drugs into the injury affected tissues. Our future studies are aimed to show that injection of silver nanoparticles in the brain lead to leaking on the inter endothelial contact and luminal plasma membrane, and therefore elucidate the possibility of penetrating into the cerebrovascular, neuronal, and glial cell which are especially damaged in AD and/or brain cancer.

Our clinical study showed the preservation and improvement of cognitive tasks in depressed and demented patients after 24, 36 and 60 month follow up of combined pharmacological (especially the combination of the diseases and mitochondrion specific compounds) and non-pharmacological treatment. The study group consisted of 156 medically ill and physically disabled patients with mild to moderate dementia and depression. Patients were treated with antidepressants, cholinesterase inhibitors, and NMDA antagonists, along with their regular medication regimen. Non-pharmacological intervention was centered on a home-based program of physical and cognitive exercises as well as with vitamins and supplements (multivitamins, vitamin E, L-methylfolate, alpha-lipoic acid, acetyl-L-carnitine, omega-3, and coenzyme Q-10) and diet modification. Cognitive assessments were performed yearly. After 60 months of treatment, performance of all tasks remained at or above baseline. The MMSE, Cognistat–Attention, Cognistat–Judgment, and RFFT - Total Unique Designs demonstrated significant improvement. Our results also demonstrate the arrest in cognitive decline in demented/depressed patients with multiple medical comorbidities for 60 months.

Our study, for the first time, demonstrated the pattern of oxidative stress induced mitochondrial DNA overproliferation and/or deletion as well as mitochondrial enzyme activities during the development of human AD, and animals that mimic human AD, colorectal cancer in liver metastasis, and malignant brain cancers. We conclude that mitochondrial lesions, especially mitochondrial DNA abnormalities, are responsible for cell viability which can be used as new diagnostic tools and/or criteria for the earlier detection of diseases and future considerations for this approach will enable us to open new pathways, not only for the better understanding of BBB homeostasis which most likely plays a key role in the development of AD, but also for the development of new and more specific treatment strategies that will be more powerful and effective in bringing a cure for this devastating disease. Thus, our research involving the conjugation of the silver nanoparticles with mitochondrially-specific drugs would help to diminish the lesions that occur in AD and/or tumor tissues. Future investigations addressing the application of a combined, integrative treatment models in clinical practices are warranted.

Biography

Gjumrakch Aliev, MD&Ph.D., President “GALLY” International Biomedical Research Institute Inc., San Antonio, Texas, USA. He also hold appointment with the University of Atlanta, Atlanta, Georgia, USA as a Professor of Cardiovascular, Neuropathology, Gerontology, Health Science and Healthcare Administration. Associate Director, Doctor of Science Program in Health Science and Healthcare Administration; Member, International Advisory Council/Board, University of Atlanta Monograph Series within School of Health Science and Healthcare Administration. He received his MD in 1982, from the Baku Medical University (former USSR) with cum laude. Then he accomplished his Ph.D. in Cardiovascular Diseases from the prestigious Russian Academy of the Medical Sciences, Moscow, Russia in 1988 with cum laude. He received postdoctoral training with Professor G. Burnstock in the University College of the London. He authored and coauthored more than 500 publications in the fields of neurodegenerative diseases research (Alzheimer disease), as well as cardio- and cerebrovascular disease, cancer, and electron microscopy. He also has several patents and rationalizations. Dr. Aliev's accomplishments in the area of biochemistry and cellular biology have tremendous implications for drug design towards cancer, AD, and cerebrovascular and neurodegeneration related pathologies. He is world-renowned expert in electron microscopy. His work has been published in numerous prestigious journals such as Nature Clinical Cardiology, J. Neuroscience, Circulation Research, Blood, J. Cellular and Molecular Medicine, Atherosclerosis and many others which reflect his leading role in his research areas. He is currently the Editor in Chiefs for "Applied Cell Biology", "World Journal of Neuroscience", "Open Journal of Psychiatry" and "Journal of Aging Science" which by itself shows the voluminous and outstanding work he has accomplished in the area of cellular and molecular biology as well as aged associated clinical sciences.

galiev03@gmail.com, GAliev@uofa.edu, cobalt55@gallyinternational.com