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Link between cell signaling, cancer and Alzheimer disease via oxidative stress induced by nitric oxide-dependent mitochondrial DNA overproliferation and deletion in the context of the drug development for neurodegeneration and cancer

Gjumrakch Aliev
GALLY International Biomedical Research & Consulting LLC, USA

Neurodegeneration and cancer are fast becoming the leading causes of age-associated disability, dementia and ultimately death worldwide. Although oxidative stress has been intensely studied, little analysis has been done in chronic oxidative stress-induced mitochondrial models and cell signaling pathway. Moreover, DNA-overproliferation and/or deletion initiate mitochondrial deregulation causing energy failure, which has been implicated in the pathogenesis of Alzheimer disease (AD), tumor growth, and metastasis. In addition, the overexpression of the cascades initiates the formation and release of large amounts of reactive free radicals [mainly nitric oxide (NO) via the overexpression of NO synthases], which cause the oxidative stress, cellular alterations, and concomitant mitochondrial lesions and decline in normal organ function. One of the key features of tumors is the deficiency in tissue energy that accompanies mitochondrial lesions and formation of the hypoxic smaller sized mitochondria with ultrastructural abnormalities. We theorize that mitochondrial involvement may play a significant role in the etiopathogenesis of cancer. Recently we demonstrate a potential link between AD and cancer, and anticancer drugs are being explored for the inhibition of AD-like pathology in transgenic mice. Severity of the cancer growth, metastasis, and brain pathology in AD correlate with the degree of mitochondrial ultrastructural abnormalities. Recent advances in the cell-cycle re-entry of the terminally differentiated neuronal cells indicate that NO-dependent mitochondrial abnormal activities and mitotic cell division are not the only important pathogenic factors in pathogenesis of cancer and AD, but open a new window for the development of novel treatment strategies for these devastating diseases. The present study explores the intimate, i.e. direct relationship between chronic oxidative stress and mitochondrial damage as a vital life-supporter for cells and/or the microcirculatory systems whose damage occurs before the development of human AD. Further extension of this approach will enable us not only for the better understanding of the blood brain barrier (BBB) homeostasis, which most likely plays a key role in the development of AD and some of forms of the cancer, but also for the development of new and more specific treatment strategies.

Biography

Gjumrakch Aliev, M.D. & Ph.D. has completed his Ph.D. at the age of 29 years from Moscow State University and Institute of Human Morphology, Russian Academy of Medical Sciences, and postdoctoral studies from University College of London, United Kingdom. He is the President and CEO of "GALLY" International Biomedical Research Consulting LLC, San Antonio, TX, USA a premier Biomedical Research service organization. He also serves as a Professor of Cardiovascular Neuropathology, Geriatrics and Health Sciences and Healthcare Administration at the University of Atlanta, GA, USA. He has published more than 190 papers in reputed journals and serving as an Editors in Chief and Editorial Board Member of repute.

aliev03@gmail.com