

Multitasking:

Alzheimer's disease is also caused by a disruption of mitochondrial proteostasis.

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Keywords: Alzheimer's disease, multitasking, tau protein, amyloid, mitochondria, mitochondrial proteostasis, ubiquitin, autophagy (mitophagy).

Goal of the study: To find other causes than tau protein and amyloid accumulation in brain.

Introduction: Alzheimer disease (AD) and mechanisms underlying its etiology and progression are complex and multifactorial. The higher AD risk in women may serve as a clue to better understand these complicated processes [1]. Lifestyle behaviors such as poor diet and reduced physical activity, as well as environmental and metabolic risk factors. Described negatively tau protein impacts mitochondria, with provocation of AD and tauopathies [2,3]. Beta amyloid is present on the outside of cells in the form of plaques. Tau protein, inside cells in the form of glomeruli.

Material and methods: Were analyses articles from Google Scholar database, from the last 5 years 2020-2025, mentioned such words as ”Alzheimer's disease”, ”multitasking”, ”tau protein”, ”amyloid”, ”mitochondria”, ”mitochondrial proteostasis”, ”ubiquitin”, ”autophagy (mitophagy)”.

Results: Three types of task were administered to patients and 30 healthy controls: 1) informant –based scales and questionnaires, 2) a neuropsychological assessment exploring executive functions, episodic and semantic memory, and 3) a new original tests featuring multi-step naturalistic actions and multitasking: The Sequential Daily Life multitasking (SDLM). We predicted that patients with AD would mainly exhibit

task perplexity. The accumulation of soluble and insoluble aggregated amyloid-beta may potentiate pathologic processes in AD [4,5].

Conclusion: According to Burgess (200a), multitasking is a common characteristic of most daily life activities and relates to situations involving several tasks of carrying difficulty or priority. Performances on these tasks need to be dovetailed. A large amount of evidence supports the amyloid cascade hypothesis, which stated that amyloid-beta accumulation triggers tau hyperphosphorylation and aggregation [6]. In the context of which, eu-ergic mitochondria with the normalization of mitochondrial uniporter-Ca⁺⁺ and mitochondrial permeability pore transition, productively inactivating the active oxygen species and reactive nitrogen species, rejects the necrosis / apoptosis, cellular hypo- (an) ergic and proves the mitochondrial eu-energetic metabolic remodeling with the elimination of the hypo (an) ergic mitochondria performed by clearance lysosomal (mitophagy) [7-9]. Thus improving the functional activity of mitochondria, which is very important for AD. Accumulation of damaged mitochondria is a hallmark of aging and age-related neurodegeneration, including AD. Mitochondrial dysfunction (MD) is being considered as a potential therapeutic target in the treatment of AD and other neurodegenerative diseases. MD contributes to the development of many chronic, fatal diseases, such as Leigh syndrome and mitochondrial myopathy. These include the most severe forms of diabetes mellitus with deafness. MD also contribute to the development of neurodegenerative diseases, including Parkinson's and AD. Scientific research, supports the relevance of enhancing mitochondrial proteostasis to delay amyloid- β proteotoxic diseases, such as AD. Mitochondrial proteostasis is a localized form of mitochondrial homeostasis within general systemic homeostasis. It is a quality control system for intramitochondrial proteins. 1. Ensuring the proper formation of the unique three-dimensional structure of the protein molecule from a linear amino acid chain (proper folding), in which the protein assumes the most energetically favorable shape necessary for its cellular function, which is critical for protein activity. 2. The functioning. 3. Accumulation of toxic endogenous substances (degradation of damaged proteins), in the event of disruption of the ubiquitin-proteasome system (for short-lived proteins) and autophagy (for long-lived, aggregated proteins)[10-14].

It is important to note that life-saving successes in critically ill patients were discussed in our published studies with targeting therapies Lysosomal Mitochondrial Clearance of Autophagy (Mitophagy) and Abnormal/Extreme Myelopoiesis of the non-functional and incapable mitochondria to generate the energy needed by the body [15-48] and in the case of AD mental energy.

Scientific coverage of the relationship between gender and refractive error of the eye: problems and prospects, was considered as a localized disorder of mitochondrial dysfunction of mitochondrial proteostasis, a localized form of ocular mitochondrial homeostasis within general systemic homeostasis. With a description of sex-specific differences in the expression of mitochondrial genes and mitochondrial target proteins (MTPs) involved in oxidative phosphorylation (OXPHOS), in contrast to women, who showed increased expression of non-OXPHOS MTPs, indicating a pronounced sexual dimorphism of energy metabolism at the whole-body level. In energy homeostasis, the role of glucose as an energy substrate in the regulation of the menstrual cycle deserves special attention [49,50].

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