Title

Mitochondria-tau association promotes cognitive decline and hippocampal bioenergetic deficits during the aging

Abstract

Current studies indicate that pathological modifications of tau are associated with mitochondrial dysfunction, synaptic failure, and cognitive decline in neurological disorders and aging. We previously showed that caspase-3 cleaved tau, a relevant tau form in Alzheimer's disease (AD), affects mitochondrial bioenergetics, dynamics and synaptic plasticity by the opening of mitochondrial permeability transition pore (mPTP). Also, genetic ablation of tau promotes mitochondrial function boost and increased cognitive capacities in aging mice. However, the mechanisms and relevance of these alterations for the cognitive and mitochondrial abnormalities during aging, which is the primary risk factor for AD, has not been explored. Therefore, in this study we used aging C57BL/6 mice (2–15 and 28-month-old) to evaluate hippocampus-dependent cognitive performance and mitochondrial function. Behavioral tests revealed that aged mice (15 and 28-month-old) showed a reduced cognitive performance compared to young mice (2 month). Concomitantly, isolated hippocampal mitochondria of aged mice showed a significant decrease in bioenergetic-related functions including increases in reactive oxygen species (ROS), mitochondrial depolarization, ATP decreases, and calcium handling defects. Importantly, full-length and caspase-3 cleaved tau were preferentially present in mitochondrial fractions of 15 and 28-month-old mice. Also, aged mice (15 and 28-month-old) showed an increase in cyclophilin D (CypD), the principal regulator of mPTP opening, and a decrease in Opa-1 mitochondrial localization, indicating a possible defect in mitochondrial dynamics. Importantly, we corroborated these findings in immortalized cortical neurons expressing mitochondrial targeted full-length (GFP-T4-OMP25) and caspase-3 cleaved tau (GFP-T4C3-OMP25) which

resulted in increased ROS levels and mitochondrial fragmentation, along with a decrease in Opa-1 protein expression. These results suggest that tau associates with mitochondria and this binding increases during aging. This connection may contribute to defects in mitochondrial bioenergetics and dynamics which later may conduce to cognitive decline present during aging. © 2024 Elsevier Inc.

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Aging; Alzheimer Disease; Animals; Caspase 3; Cognitive Dysfunction; Hippocampus; Mice; Mice, Inbred C57BL; Mitochondria; Reactive Oxygen Species; adenosine triphosphate; caspase 3; cyclophilin D; fusion protein; mitochondrial permeability transition pore; mitochondrial protein; optic atrophy protein 1; reactive oxygen metabolite; tau protein; unclassified drug; caspase 3; reactive oxygen metabolite; aging; animal cell; animal experiment; animal model; animal tissue; Article; behavioral test parameters; bioenergy; brain cell; calcium metabolism; cell membrane depolarization; cellular distribution; cognitive defect; comparative study; controlled study; disease course; female; hippocampus; male; mental performance; mitochondrial respiration; mouse; nonhuman; protein expression; aging; Alzheimer disease; animal; C57BL mouse; cognitive defect; genetics; hippocampus; metabolism; mitochondrion; pathology

Chemicals/CAS

adenosine triphosphate, 15237-44-2, 56-65-5, 987-65-5; caspase 3, 169592-56-7; cyclophilin A, ; cyclophilin B, ; cyclophilin C, ; cyclophilin D, ; cyclophilin F, ; peptidylprolyl isomerase, ; Caspase 3, ; Reactive Oxygen Species,

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