New evidence caffeine may slow Alzheimer's disease and other dementias, restore cognitive function

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Although caffeine is the most widely consumed psychoactive drug worldwide, its potential beneficial effect for maintenance of proper brain functioning has only recently begun to be adequately appreciated. Substantial evidence from epidemiological studies and fundamental research in animal models suggests that caffeine may be protective against the cognitive decline seen in dementia and Alzheimer's disease (AD). A special supplement to the *Journal of Alzheimer's Disease*, "Therapeutic Opportunities for Caffeine in Alzheimer's Disease and Other Neurodegenerative Diseases," sheds new light on this topic and presents key findings.

Guest editors Alexandre de Mendonça, Institute of Molecular Medicine and Faculty of Medicine, University of Lisbon, Portugal, and Rodrigo A. Cunha, Center for Neuroscience and Cell Biology of Coimbra and Faculty of Medicine, University of Coimbra, Portugal, have assembled a group of international experts to explore the effects of caffeine on the brain. The resulting collection of original studies conveys multiple perspectives on topics ranging from molecular targets of caffeine, neurophysiological modifications and adaptations, to the potential mechanisms underlying the behavioral and neuroprotective actions of caffeine in distinct brain pathologies.

"Epidemiological studies first revealed an inverse association between the chronic consumption of caffeine and the incidence of Parkinson's disease," according to Mendonça and Cunha. "This was paralleled by animal studies of Parkinson's disease showing that caffeine prevented motor deficits as well as neurodegeneration "Later a few epidemiological studies showed that the consumption of moderate amounts of caffeine was inversely associated with the cognitive decline associated with aging as well as the incidence of Alzheimer's disease. Again, this was paralleled by animal studies showing that chronic caffeine administration prevented memory deterioration and neurodegeneration in animal models of aging and of Alzheimer's disease."

Key findings presented in "Therapeutic Opportunities for Caffeine in Alzheimer's Disease and Other Neurodegenerative Diseases":

- Multiple beneficial effects of caffeine to normalize brain function and prevent its degeneration
- Caffeine's neuroprotective profile and its ability to reduce amyloid-beta production
- Caffeine as a candidate disease-modifying agent for Alzheimer's disease
- Positive impact of caffeine on cognition and memory performance
- Identification of adenosine A2A receptors as the main target for neuroprotection afforded by caffeine consumption
- Confirmation of data through valuable meta-analyses presented
- Epidemiological studies corroborated by meta-analysis suggesting that caffeine may be protective against Parkinson's disease
- Several methodological issues must be solved before advancing to decisive clinical trials

Mendonça and Cunha also observe that "the daily follow-up of patients with AD has taught us that

improvement of daily living may be a more significant indicator of amelioration than slight improvements in objective measures of memory performance. One of the most prevalent complications of AD is depression of mood, and the recent observations that caffeine might be a mood normalizer are of particular interest."

More information: The entire issue has been made available on a no-fee basis at iospress.metapress.com/content/t13614762731/

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