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The association between alcohol use and the progression of Alzheimer's disease

Devorah Heymann
The University of Melbourne, Australia

Background: Mild-moderate alcohol intake is widely considered to be associated with decreased risk of developing Alzheimer disease (AD), while heavy drinking increases the risk. There is little information about how alcohol affects the cognitive profile among those already diagnosed with AD.

Objective: To examine the relationship between alcohol, both the amount and type, and cognitive decline in a cohort of AD patients.

Methods: A cohort of 360 patients with early AD in New York, Boston, Baltimore and Paris were followed-up biannually for up to 19.28 years. At each visit, the cognitive profile of the patients was assessed using the modified Mini-Mental State Examination (mMMSE), and patients' alcohol intake, including beverage type, was reported by patients' primary caregivers. General estimating equation analysis was used to determine whether baseline alcohol use was associated with the rate of cognitive decline.

Findings: Heavy drinkers (8 or more alcoholic drinks/week) had a faster cognitive decline, deteriorating 2.625 more points on their mMMSE score annually compared to abstainers (P≤0.0001), or 3.429 more points compared to mild-moderate drinkers (1-7 alcoholic drinks/week) (P=0.006). There was no significant difference when comparing mild-moderate drinkers to abstainers. Increasing standard drinks of hard liquor, but not beer or wine, was also associated with a faster rate of cognitive decline (β=-0.165 P=≤0.0001).

Conclusions: Heavy alcohol consumption and more hard liquor are associated with a faster rate of cognitive decline in AD patients, suggesting that they may hasten progression of AD. Our results suggest that alcohol drinking habits might alter the course of AD.

devorah.haymann@hotmail.com

Disease-modifying neuroprotective action of the agent Dimebon (latrepirdine) in animal models of neurodegeneration

Bachurin S
RAS, Russian Federation

Dimebon launched in early 80s in Russia as antihistamine agent belongs to a fast-growing group of "old" medicines that were suggested to be effective for therapy of pathological conditions different from their original targets. Here, we overview most recent Dimebon studies on the pathological process in the brain involving *in vivo* models of proteinopathies where neurodegenerative effects are envisaged by a group of aggregate-prone proteins such as gamma-synuclein, mimic of hyperphosphorylated tau, and FUS which are engaged in numerous neurological diseases. The main focus is on *in vitro* models comprised of cultured SH-SYS5 neuroblastoma cells expressing mutant forms of TDP-43 and showing reduced number of inclusion-containing cells upon Dimebon treatments with activation of autophagy markers. The Dimebon effect on stabilization of mitochondrial functions by increasing the threshold to nonselective mitochondrial pores opening and increasing the calcium retention capacity of mitochondria, and reducing lipid peroxidation is also discussed. The results presented as well as data from other laboratories in the past year provide grounds for an inquest for re-evaluation of therapeutic potential of Dimebon and its newly designed analogs as promising disease-modifying agents in battle with neurodegenerative disorders. Meanwhile, new data emerges in favor of possible antiaging effect and application of Dimebon for treatments of depression, anxiety and ischemia.The most pronounced effect of Dimebon is observed in treatments starting at an early stage of the disease onset and this is the major fact that should be accounted in planning for clinical trials.

fakeralani2000@yahoo.com