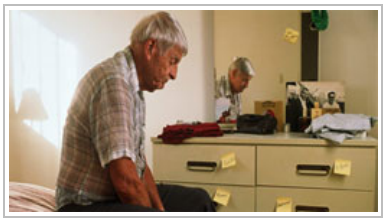
Exercise, melatonin help reduce Alzheimer's symptoms

Last Updated: Thursday, September 27, 2012,13:01

Tags: Exercise , Melatonin , Alzheimer's symptoms



Washington: In a new study, researchers have shown the combined effect of neuroprotective therapies - voluntary physical exercise and daily intake of melatonin, against Alzheimer's.

According to a group of researchers from the Barcelona Biomedical Research Institute (IIBB), in collaboration with the University of Granada and the Autonomous University of Barcelona, daily voluntary exercise and daily intake of melatonin, both of which are known for the effects they have in regulating circadian rhythm, show a synergistic effect against brain deterioration in the 3xTg-AD mouse, which has three mutations of Alzheimer's disease.

"For years we have known that the combination of different anti-aging therapies such as physical exercise, a Mediterranean diet, and not smoking adds years to one's life," Coral Sanfeliu, from the IIBB, told SINC.

"Now it seems that melatonin, the sleep hormone, also has important anti-aging effects," Sanfeliu said.

For the study, the experts analysed the combined effect of sport and melatonin in 3xTg-AD mice which were experiencing an initial phase of Alzheimer's and presented learning difficulties and changes in behaviour such as anxiety and apathy.

The mice were divided into one control group and three other groups which would undergo different treatments: exercise –unrestricted use of a running wheel–, melatonin –a dose equivalent to 10 mg per kg of body weight–, and a combination of melatonin and voluntary physical exercise. In addition, a reference group of mice were included which presented no mutations of the disease.

"After six months, the state of the mice undergoing treatment was closer to that of the mice with no mutations than to their own initial pathological state. From this we can say that the disease has significantly regressed," Sanfeliu said.

The results show a general improvement in behaviour, learning, and memory with the three treatments.

These procedures also protected the brain tissue from oxidative stress and provided good levels of protection from excesses of amyloid beta peptide and hyperphosphorylated TAU protein caused by

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the mutations. In the case of the mitochondria, the combined effect resulted in an increase in the analysed indicators of improved performance which were not observed independently.

"Transferring treatments which are effective in animals to human patients is not always consistent, given that in humans the disease develops over several years, so that when memory loss begins to surface, the brain is already very deteriorated," Sanfeliu added.

The study has been published in the journal Neurobiology of Aging.

ANI

First Published: Thursday, September 27, 2012, 13:01

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