

Exercise can slow onset of Alzheimer's memory loss, study reports

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(Medical Xpress)—Keeping active can slow down the progression of memory loss in people with Alzheimer's disease, a study has shown. A team of researchers from The University of Nottingham has identified a stress hormone produced during moderate exercise that may protect the brain from memory changes related to the disease.

The work, funded by Research into Ageing (Age UK) and the University and published in the *Journal of Alzheimer's Disease*, may also explain why people who are susceptible to stress are at more risk of developing the disease.

Alzheimer's disease is the most common cause of [dementia](#) affecting almost 500,000 people in the UK, the majority of who are over the age of 65. Symptoms can include [memory loss](#), [mood changes](#) and problems with communicating and reasoning.

There is no cure for Alzheimer's and, although there are a few treatments available that can reduce the symptoms in some people, they cannot halt the progression of the disease.

Increasingly, there is evidence that physical and mental activity can reduce people's chances of developing the disease or can slow down its progression but up until now it has been unclear how this happens.

Protective effect

The Nottingham team, led by Dr Marie-Christine Pardon in the School of Biomedical Sciences, has discovered that the stress hormone CRF—or corticotrophin-releasing factor—may have a protective effect on the [brain](#) from the memory changes brought on by Alzheimer's disease.

CRF is most associated with producing stress and is found in high levels in people experiencing some forms of anxiety and depressive diseases. Normal levels of CRF, however, are beneficial to the brain, keeping the mental faculties sharp and aiding the survival of [nerve cells](#). Unsurprisingly then, studies have shown that people with Alzheimer's disease have a reduced level of CRF.

The researchers used an [experimental drug](#) to prevent the hormone from binding to a brain receptor called CRFR1 in mice with Alzheimer's disease that were free from memory impairments, therefore blocking the effects of the hormone. They discovered that the mice had an abnormal stress response with reduced [anxiety](#) but increased behavioural inhibition when confronted by a stressful situation—in this case being placed in a new environment—and this was due to the abnormal functioning of the CRFR1. This abnormal stress response before the onset of symptoms may explain why people susceptible to stress are more at risk of developing Alzheimer's.

Memory enhancing

Dr Pardon and her team also found that interrupting the hormone from binding on to the CRFR1 receptor blocked the improvement of memory normally promoted by exercise. However, in mice with Alzheimer's a repeated regime of [moderate exercise](#) restored the normal function of the CRF system allowing its memory enhancing effects. The results are in line with the idea that regular exercise is a means of improving one's ability to deal with everyday stress in addition to keeping mental abilities keen.

Finally, their study showed that the switching on of this particular brain receptor during exercise increased the density of synapses, which makes the connection between nerve cells, the loss of which is thought to be responsible for the early memory loss seen in Alzheimer's patients.

Dr Pardon said: "This is the first time that researchers have been able to identify a brain process directly responsible for the beneficial effects of exercise in slowing down the progression of the early memory decline characteristics of Alzheimer's disease.

"Overall, this research provides further evidence that a healthy lifestyle involving [exercise](#) slows down the risk of Alzheimer's disease and opens avenues for the new interventions targeting the altered CRFR1 function associated with the early stages of the disease."

More information: An early online version of the paper—Corticotropin-Releasing Factor Receptor 1 Activation During Exposure to Novelty Stress Protects Against Alzheimer's Disease-Like Cognitive Decline in A β PP/PS1 Mice—detailing the findings has been published online at

iospress.metapress.com/content/75m787746365k55g/

Provided by University of Nottingham

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