Putting the brakes on Alzheimer’s disease: Exercise and its effects on cognitive decline, neurogenesis and inflammation in the APP/PS-1 mouse

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Putting the brakes on Alzheimer's disease: Exercise and its effects on cognitive decline, neurogenesis and inflammation in the APP/PS-1 mouse

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Research Abstract
Alzheimer’s disease (AD) is a progressive, neurodegenerative condition characterised by accumulation of toxic ?eta-amyloid (A?) plaques[1, 2] and is one of the leading causes of dementia globally. The cognitive impairment that is a hallmark of AD may be caused by

inflammation in the brain resulting from accumulation of A? protein that ultimately leads to neuronal dysfunction and loss. The hippocampus is a key brain region associated with learning and memory. In addition to being particularly vulnerable to AD pathology, it is one of just two regions in the adult brain in which new neurons are produced continuously throughout life, a process termed adult hippocampal neurogenesis(AHN). AHN is believed to be critical for learning and memory in the healthy brain[3, 4] and evidence suggests that reduced AHN contributes to age-related cognitive deficits in disorders such as AD[5, 6]. Moreover, neuroinflammation, as is seen in AD, is a potent negative modulator of AHN[7] thus indicating a causal link between neuroinflammation, impaired AHN and AD-associated deficits in brain function.

Accordingly, strategies that boost AHN may have protective or therapeutic potential in disorders associated with impaired AHN, such as AD. Exercise is a well-established promoter of AHN and is also a well-established mechanism for cognitive enhancement in humans and animals[8, 9]. Exposure of AD mouse models to exercise improves their cognitive abilities and reduces AD-associated neuropathology[10]. Recent evidence indicates that exercise may also act to dampen neuroinflammation, potentially via the chemokine protein CX3CL1[11]. These data suggest that the anti-inflammatory and AHN-stimulating effects of exercise may be of benefit in understanding the pathological development of AD and in identifying potential therapeutic targets. Here, we propose to examine whether regular exercise protects against the cognitive impairment, neuroinflammation and deficits in AHN observed in a validated transgenic mouse model of AD.

Further information available at:

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