ARTICLE

Active body, healthy mind

Jadwiga Nazimek on whether exercise can delay – or even prevent – dementia

Exercise is a good treatment for many health problems, both physical and mental. It reduces physical frailty and might prolong the lifespan, but (in synergy with other factors, such as lifestyle or genetics) it can improve the quality of later life by preserving healthy intellectual functioning and preventing – or at least delaying – dementia.

This knowledge is exceedingly valuable in the face of the growing proportion of older adults in modern society and the growing burden of dementia. So how can exercise reduce the risk of dementia, and what can psychologists do to encourage people who fail to exercise?

questions

How would lack of exercise affect the cognitive development of children?

Is there a difference in levels of exercise between men and women, and if so, does it translate into differences in the risk of dementia?

resources

http://nihseniorhealth.gov/stories/stories.html

Kramer, A., Erickson, K.I. & Colcombe, S.J. (2006). Exercise, cognition, and the aging brain. *Journal of Applied Physiology, 101*, 1243–1251.

It's a normal part of ageing that many will be familiar with: as we get older, memory, visuospatial ability and executive function (planning, scheduling, working memory, inhibition and multitasking) tend to suffer. Indeed, their impairment is one of the first symptoms of Alzheimer's disease. But could a healthy lifestyle be the key to keeping a healthy mind?

It appears that older adults who exercise regularly and enjoy higher level of physical fitness perform better on cognitive tasks involving these functions than do their sedentary peers (e.g. Kramer et al., 2006). What's more, aerobic exercise seems to be considerably more beneficial than non-aerobic activity (Jedrziewski et al., 2007). Thus, aerobic exercise appears to help to counterbalance the effects of ageing on higher-order cognitive function.

It is not only the normal effects of ageing that can be reduced by regular physical activity: some studies (e.g. Rockwood & Middleton, 2007) suggest the risk of mild cognitive impairment can be reduced, with others finding that the risk of Alzheimer's disease can be lowered by up to 30 per cent (Jedrziewski et al., 2007). Evidence, however, is still inconsistent: in some studies physical activity has no effects on the risk of vascular dementia, where the blood flow to the brain is insufficient to maintain healthy cells (Rockwood & Middleton, 2007). In others, exercise is associated with a lower risk of vascular dementia, but not that of Alzheimer's disease (Ravaglia et al., 2007). This might be related to definitions of vascular dementia (e.g. those relying on the presence of stroke are less sensitive), or perhaps physical activity affects differently different types of dementia. Similarly, there is evidence that exercise can counteract genetic susceptibility to Alzheimer's disease (Rovio et al., 2005), although this result is lacking replication.

Overall, there is evidence showing that regular exercise reduces risk of dementia, but the area is plagued by methodological problems and incomplete knowledge about biological mechanisms of the effects of physical activity on cognitive functioning.

How does exercising improve thinking?

One mechanism is the growth of new neurons (neurogenesis) and the plasticity of the connections between the neurons (synaptic plasticity). Mice who exercise are better at learning spatial and memory task than their sedentary counterparts (Cotman & Berchtold, 2007). This effect, however, takes place only if a signalling molecule called brain-derived neurotrophic factor is present. The neurotrophic factor supports survival and plasticity of existing neurons. 'Long-term potentiation', thought to be the cellular mechanism of learning, occurs when repetitive stimulation results in increased potentiation of the synapse – the synaptic response to the stimuli is stronger and lasts longer. This phenomenon is impaired in mice lacking the neurotrophic factor (Korte et al., 1995). Therefore, by supporting long-term potentiation, the neurotrophic factor appears to play a role in memory and learning (Cotman & Berchtold, 2007). The neurotrophic factor is also involved in the growth and survival of new neurons and synapses in the hippocampus (the seat of memory and learning) and cortex (responsible for thinking).

The fact that levels of the neurotrophic factor are 30–80 per cent lower in people suffering from Alzheimer's disease suggests its involvement in the pathology of the illness (Murray et al., 1994). In rodents,

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exercise increases the levels of the neurotrophic factor (Berchtold et al., 2005), as well as the growth and flexibility of neurons (van Praag et al., 1999). The two processes – neurogenesis and synaptic plasticity – might interact, so that the growth of new cells contributes to the improved plasticity of the synapses (van Praag et al., 1999), and learning supports the survival of the newborn cells (Tashiro et al., 2007).

The effect of exercise on the birth and survival of new neurons appears to be related to the presence of a protein hormone similar to insulin (the insulin-like growth factor 1 or IGF-I), which regulates metabolism, as well as growth, multiplication and death of cells. Physical activity increases the uptake of IGF-I into to the brain, including the hippocampus (Trejo et al., 2001). Mice in whose brains IGF-I is insufficient suffer from reduced neurogenesis and problems with spatial learning. In these animals moderate treadmill running exercise has no effect on the deficits, but they can be eliminated by the administration of the insulin-like growth factor (Trejo et al., 2007).

The immune system is another party in the interaction between exercise and thinking. Increased level of inflammatory factors is highly associated with cognitive impairment in Alzheimer's disease, where brain cells die as a result of the build-up of abnormal protein in plaques and tangles (Parachikova et al., 2007). Sedentary, aged mice, in whose brains the Alzheimer's disease pathology is already present, have higher levels of inflammation compared to healthy mice the same age. After three weeks of voluntary wheel running, however, the diseased mice regain normal working and spatial memory (Parachikova et al., 2008). This might be because exercise significantly increases levels of certain chemokines in the brain. These chemokines protect neurons in the hippocampus from death induced by the plaques, as well as improve communication between brain cells (Bezzi et al., 2001). However, the exact mechanism of the effect of exercise on inflammatory response in the brain remains unclear (Parachikova et al., 2008).

Exercise also improves blood flow in the brain, therefore influencing the amount and concentration of nutrients such as oxygen and glucose, as well as the expanding and contracting movement of the cerebral vessels, which in turn affects brain cells. Blood delivers nutrients such as oxygen and glucose, essential for the neurons to function. In addition, blood carries factors that affect neural activity, and their concentration might depend on the volume of the blood flow. Brain cells might react to the expanding and contracting movement of the blood vessels. Finally, blood affects neurons by regulating the temperature of the brain tissue ('Blood may help us think', 2007). Thus, by improving the cerebral blood flow, exercise is likely to improve the function of the brain. Moreover, regular physical activity lowers the risk of cerebrovascular disease and helps to fight the vascular risk factors (such as hypertension) which increase the risk of incidence and progression of Alzheimer's disease and vascular dementia. By increasing cerebral blood flow, exercise helps to reduce damage to the neurons resulting from oxidative stress (excess of toxic free radicals) present in the early stages of Alzheimer's disease (Lange-Asschenfeldt & Kojda, 2008). Finally, angiogenesis (the growth of new capillaries) as a result of exercise protects neurons via substances such as vascular endothelial growth factor. The latter is involved in production and release of the brain-derived neurotrophic factor from one of its sources, the brain microvascular endothelial cells.

Therefore, neurogenesis interacts with angiogenesis, and by improving the health of the vascular system, exercise helps to protect neurons and might delay the occurrence of dementia.

By exercising, young men can protect their future cognitive ability

How much and how often?

As a guide, exercise appears to reduce the risk of dementia by 32 per cent provided it takes place three or more times a week (Rockwood & Middleton, 2007). Each session should last at least 20 minutes, be vigorous, and cause breathlessness and sweating (Jedrziewski et al., 2007).

Some studies suggest there is a dose–response relationship, in the sense that the group with lowest levels of exercise is at the greatest risk, while the group with highest levels of exercise is at lowest risk (e.g. Rockwood & Middleton, 2007). Other studies suggest that the benefits from exercise, in terms of risk of dementia, increase in the dose–response manner up to the moderate level, and then the benefits cease (e.g. Larson et al., 2006). Finally, exercising at the age of 15–25 years can protect cognitive ability in later life, at least in men

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(Dik et al., 2003), and exercising in midlife can be just as beneficial as that in later life (Rockwood & Middleton, 2007).

Preventing or delaying?

Even though the evidence that physical activity has good effect on cognitive function is robust, there are some inconsistencies and methodological problems. Some studies find that physical activity is unrelated to cognitive function (e.g. Broe et al., 1998; Yamada et al., 2003). This might be due to using self-report data concerning exercise, not distinguishing aerobic from non-aerobic activity, lack of assessment of duration, intensity and frequency of activities and failure to eliminate participants with subclinical dementia (Kramer et al., 2006). Other common difficulties include lack of agreed definitions and criteria of physical fitness and dementia (e.g. single disease entity versus categories; Jedrziewski et al., 2007; Rockwood & Middleton, 2007).

Finally, the lack of an accepted definition of 'prevented dementia' makes it difficult to determine whether a particular method is successful or not in this endeavour. It has been suggested that delaying dementia equals preventing it, since dementia occurs in late stages of life – delaying it by two years would reduce prevalence by 25 per cent (Brookmeyer et al., 1998). However, physical activity reduces physical frailty in older people and thus might prolong lifespan, therefore actually serving to increase the prevalence of dementia!

What can psychologists do?

Neurocognitive knowledge of how exercise affects the brain can help work on techniques that would lower the threshold of exercise for the elderly, who can find it difficult to get the appropriate levels of activity (Cotman & Berchtold, 2007). Occupational psychologists investigate how high-strain work prevents exercise in even those employees who intend to be more physically active, by affecting their confidence and self-efficacy at motivational stage, as well putting their good intentions into practice (Payne et al., 2002). Suggested solutions include flexible working hours and convenient facilities, such as an onsite gym.

In order to encourage exercise in the frame of health promotion (usually cardiovascular), exercise and health psychologists analyse factors that influence motivation and perseverance in keeping fit, as well as barriers and ways of promoting physical activity. Sometimes those younger adults who are willing to engage in physical activity might only need some simple encouraging triggering messages ('Just do it', 'Don't think about it'), others need help in getting started and reminders (O'Brien et al., 2005). However, the elderly should seek appropriate medical advice before undertaking exercise.

More difficult to tackle can be barriers such as unsafe neighbourhood, perceived illness and physical disability and gender, but also cultural stereotypes of older adults as frail and dependent (Brawley et al., 2003). Gaining confidence and increasing self-efficacy via programmes such as the group-mediated cognitive-behavioural approach, can help older adults overcome these barriers (Rejeski et al., 2003). Other group strategies, described by Brawley and colleagues (2003), include lifestyle intervention, which involves stages of change model and group teaching of cognitive and behavioural strategies. Participants, encouraged to spend less time on sedentary activities such as watching television, often choose to replace them with more intense activities, such as walking. Teaching the skill of self-monitoring can also successfully increase physical activity. Combining individual interventions with those aimed at the community (e.g. via healthcare providers) and those directed by the state (tax incentives, constructing neighbourhood sidewalks) can create a social-ecologic, multiple-level approach. In fact, some exercise psychologists work as consultants for initiatives of the government and NHS in promoting exercise and providing training to those involved.

Challenges ahead

It appears that regular physical activity can reduce the risk of dementia, provided that it takes the appropriate form and is undertaken with sufficient frequency and duration. At this stage of research, however, many issues are not clear; for example, inconsistencies of evidence concerning effects of exercise on different types of dementia might be due to methodological problems, but also lack of knowledge about the biological processes evoked by exercise in the brain. These processes might, again, depend on the type of exercise and affect individual brains differently. Psychologists investigate factors related to exercise and on the basis of these can suggest solutions. Challenges include linking the individual strategies to population-wide programmes that promote healthy lifestyle and to group programmes of social problem-solving.

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