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Healthy Aging Research

Commentary

Neuroscience of Aging and its Functional Changes

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ABOUT THE STUDY

The neurology of ageing is the study of the adjustments that the ageing neural system makes. The central nervous system experiences various changes as we age, including modest atrophy of the cortex, which is thought to be non-pathological. Numerous neurological and neurodegenerative diseases, including amyotrophic lateral sclerosis, dementia, moderate cognitive impairment, Parkinson's disease, and Creutzfeldt-Jakob disease, are also linked to aging.

Normal structural and neural changes

Adult neurogenesis, a process that only slightly affects the hypothalamus and striatum, is a process that very little neurogenesis happens in adults. Age-related brain volume loss may have a number of causes, including cell death, a reduction in cell volume, and changes in synapse topology. The exact reason for this decline is still unknown. The changes in brain volume are uneven across areas, with the prefrontal cortex experiencing the greatest volume loss, followed by the striatum, the temporal lobe, the cerebellar vermis, the cerebellar hemispheres, and the hippocampus, in that order.

The amygdala and ventromedial prefrontal cortex, according to one review, remained mostly unatrophied, which is consistent with the fact that emotional stability occurs with non-pathological ageing. In non-pathological ageing, enlargement of the ventricles, sulci, and fissures is also typical.

Neuroplasticity, synaptic functioning, and voltage-gated calcium channels may also be linked to changes. Neurons fire less frequently and are less flexible as a result of increased hyperpolarization, which may be caused by abnormal calcium regulation. This impact is especially noticeable in the hippocampus of elderly animals and may play a significant role in the memory problems linked to aging.

Three stages the fast, medium, and slow hyperpolarization's can be distinguished in the hyperpolarization of a neuron. Aged neurons experience longer opening of calcium-dependent potassium channels during the medium and slow hyperpolarization phases. Deregulated calcium and hypo activity of cholinergic, dopaminergic, serotonergic, and glutaminergic pathways have been proposed as causes of the extension of this phase.

Normal functional changes

From middle age, episodic memory gradually starts to deteriorate, whereas semantic memory remains stable until early old age and then starts to degrade. Working memory activities cause older adults to engage their prefrontal cortex more frequently, potentially to make up for it with executive functions. Aging-related cognitive deficits can include a slowdown in processing speed and difficulty focusing.

According to a paradigm, put forth to explain altered activation, decreased dopamine functioning and lower neuronal efficiency brought on by amyloid plaques cause compensatory activation. Aging causes a decrease in processing of negative stimuli compared to positive stimuli, which can be shown even in autonomic nerve reactions to emotionally charged stimuli. Reduced Achilles and plantar reflex responses are also related to ageing. Normal ageing also causes a decrease in nerve conductivity.

DNA damage: After age 40, and particularly after age 70, some human frontal brain genes have lower transcriptional expression. Particularly, ageing reduces the expression of genes that are essential for synaptic plasticity. In elderly people, there is a significant increase in DNA damage, most likely oxidative DNA damage, in the promoters of genes with decreased expression in the cortex.

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