

Structural Changes of Aging Brain

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Description

Most prevalent neurodegenerative disorders, such as mild cognitive impairment, dementias such as Alzheimer's disease, cerebrovascular disease, Parkinson's disease, and Lou Gehrig's disease, are all linked to ageing. While there has been a lot of study on ageing illnesses, there have been few studies on the molecular biology of the ageing brain in the absence of neurodegenerative disease or the neuropsychological profile of healthy older persons. However, research shows that ageing is linked to a variety of anatomical, chemical, and functional changes in the brain, as well as a variety of neurocognitive alterations. According to recent research in model organisms, there are unique changes in gene expression at the single neuron level as animal's age. This page is dedicated to discussing the changes that occur as a result of healthy ageing.

Structural Changes

Many physical, biochemicals, chemical and psychological changes accompany ageing. As a result, it is reasonable to conclude that the brain is not immune to this phenomenon. The cerebral ventricles grow as people become older, according to CT scans [1].

Age-related regional declines in brain volume have been found in more recent MRI investigations. Regional volume loss is not uniform; certain brain regions shrink at up to 1% per year, while others remain relatively steady till the end of life. The brain is a complicated organ that is made up of many distinct types of tissue and substances. The various functions of various brain tissues may be more or less vulnerable to age-related alterations. Grey matter and white matter are the two types of brain matter that can be found in the brain. White matter consists of tightly packed myelinated axons connecting the neurons of the cerebral cortex to each other and to the periphery, whereas grey matter consists of cell bodies in the cortex and subcortical nuclei [2].

Loss of Neural Circuits and Brain Plasticity

The ability of the brain to modify form and function is referred to as brain plasticity. This is related to the idiom "if you don't use it, you lose it," which means that if you don't use it, your brain will allocate less somatotopic space to it. The outcome of age-induced changes in calcium regulation is one proposed reason

for the reported age-related plasticity impairments in animals. Changes in our ability to handle calcium will eventually disrupt neuronal firing and the ability to transmit action potentials, affecting the brain's ability to modify shape and function [3].

Because of the brain's complexity, with all of its structures and activities, it's reasonable to believe that some sections are more susceptible to ageing than others. The hippocampus and neocortical circuits are two circuits worth highlighting. It has been proposed that age-related cognitive impairment is caused in part by synaptic changes rather than neuronal death. Animal research has also suggested that this cognitive loss is caused by functional and biochemical aspects in cortical networks, such as alterations in enzyme activity, chemical messengers, or gene expression.

Thinning of the Cortex

Advances in MRI technology have made it possible to see the brain structure in great detail *in vivo* in a simple and non-invasive manner. The insula and superior parietal gyri have been found as being particularly sensitive to age-related grey matter reductions in older individuals in studies using Voxel-based morphometric [4]. It's also worth mentioning that areas around the calcarine sulcus, such as the cingulate gyrus and the occipital cortex, appear to be immune to this loss of grey matter density over time.

Grey matter density in the posterior temporal cortex was shown to be more prevalent in the left hemisphere than the right, and was limited to posterior language cortices as people got older [5]. Certain language activities, including as word retrieval and production, have been discovered to be located in the more anterior language cortices, which decline with age. The width of the sulcus has also been reported to increase with age, as well as with cognitive deterioration in the elderly.

Age-related Neuronal Morphology

Age-related cognitive deficiencies may not be the result of neuronal loss or cell death, but rather of modest region-specific changes in the morphology of neurons, according to a growing body of research from cognitive neuroscientists throughout the world [6]. Humans over the age of 50 have a 46 percent reduction in spine number and density when compared to younger people. According to an electron microscopy study in

monkeys, spines on the apical dendritic tufts of pyramidal cells in the prefrontal cortex of senior animals (27–32 years old) had lost 50% of their spines compared to young animals (6–9 years old).

Neurofibrillary Tangles

Alzheimer's disease, Parkinson's disease, diabetes, hypertension and arteriosclerosis are all age-related neuropathologies that make it difficult to discriminate between normal ageing patterns. The placement of neurofibrillary tangles is one of the key variations between normal and pathological ageing. Pairs of helical filaments make up neurofibrillary tangles [7].

The amount of tangles in each damaged cell body in normal, non-demented ageing is generally minimal and limited to the olfactory nucleus, parahippocampal gyrus, amygdala and entorhinal cortex. There is a general rise in the density of tangles as the non-demented individual matures, but no significant change in where tangles are detected. Amyloid plaques are another prominent neurodegenerative factor observed in the brains of Alzheimer's sufferers [8].

Role of Oxidative Stress

Oxidative stress, inflammatory responses and alterations in the cerebral microvasculature have all been linked to cognitive impairment. It is uncertain what effect each of these pathways has on cognitive ageing. The most manageable and well-understood risk factor is oxidative stress.

"Physiological stress on the body induced by the accumulated damage done by free radicals insufficiently mitigated by antioxidants and that is to be connected with ageing," according to the Merriam-Webster Medical Dictionary online. As a result, oxidative stress is defined as the damage caused to cells by free radicals generated during the oxidation process [9].

Humans have many different types of memory, including declarative memory (which includes episodic and semantic memory), working memory, spatial memory, and procedural memory. Memory skills, particularly those connected with the medial temporal lobe, have been reported to be particularly

prone to age-related decrease in studies. A number of studies using a range of methodologies, including histology, structural imaging, functional imaging, and receptor binding, have found that age-related processes resulting in memory alterations are particularly affecting the frontal lobes and frontal-striatal dopaminergic networks [10].

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