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Commentary

Neurodegeneration and Clinical Neurophysiology of the Ageing Brain

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DESCRIPTION

Aging is associated with cognitive decline in a large proportion of the population and is the primary risk factor for Alzheimer's disease and other common neurodegenerative disorders. Despite its importance in disease pathogenesis and morbidity, brain ageing is poorly understood at the molecular level. The purpose of this paper is to integrate what is known about age-related cognitive and neuroanatomical changes with recent advances in understanding basic molecular mechanisms that underpin ageing. An important question is how normal brain ageing progresses to pathological ageing, resulting in neurodegenerative disorders. Toxic protein aggregates, such as amyloid protein in Alzheimer's disease, tau in frontotemporal dementia, and synuclein in Parkinson's disease, have been identified as potential contributory factors.

Human data show that executive functions associated with the frontal lobe and hippocampal regions of the brain may be selectively maintained or enhanced in fitter people. Similarly, improved performance is observed in aged animals subjected to increased physical and mental demand, and it appears that the vascular component of the brain response is driven by physical activity, whereas the neuronal component is influenced by learning. Recent research has linked neurogenesis, at least in the hippocampus, to the brain's response to exercise, with learning increasing the survival of these neurons.

The mature brain's non-neuronal tissues respond to experience as well, indicating that the brain reflects both recent and long-term experience. Neurodegenerative disorders are distinguished by extensive neuron death, which results in functional decline; however, the neurobiological correlates of functional decline in normal ageing are less well defined. For decades, it was widely assumed that widespread neuron death in the neocortex and hippocampus was an unavoidable consequence of brain ageing. However, recent quantitative studies suggest that neuron death is limited in normal ageing and is unlikely to account for age-related impairment of neocortical and hippocampal functions.

Development, genetic defects, the environment, disease, and an inborn process the ageing process can all be blamed for ageing changes. The chance of death at a given age is a measure of the average number of ageing changes accumulated by people of that age, i.e., of physiologic age, and the rate of change of this measure is referred to as the rate of ageing. The qualitative and quantitative differences in neuron loss between ageing and Alzheimer's disease are discussed, and age-related changes in functional and biochemical attributes of hippocampal circuits that may mediate functional decline in the absence of neuron death are investigated. Physiological brain ageing is characterized by synaptic contact loss and neuronal apoptosis, which results in age-dependent declines in sensory processing, motor performance, and cognitive function. Neural redundancy and plastic remodelling of brain networking, also secondary to mental and physical training, promotes maintenance of brain activity in healthy elderly for everyday life and fully productive affective and intellectual capabilities. However, age is the most important risk factor for neurodegenerative disorders that affect cognition, such as Alzheimer's disease (AD).

Oscillatory electromagnetic brain activity is a defining feature of neuronal network function in a variety of brain regions. Electro Encephalo Graphy (EEG), Event-Related Potential (ERP), Magneto Encephalo Graphy (MEG), and Transcranial Magnetic Stimulation (TMS) are modern neurophysiological techniques that can accurately index normal and abnormal brain ageing and allow for non-invasive analysis of cortico-cortical connectivity.

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