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Editorial Note on Age-related Neurogenic Decline

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EDITOIRAL

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Aging is often seen as a steady loss in the function of many tissues and organs, resulting in disturbance of homeostasis and increased frailty. A rise in the frequency of senescent cells in most organs is one of the hallmarks of ageing. Senescence occurs in response to a variety of stresses, such as telomere attrition and other forms of DNA damage, oncogene activation, epigenetic remodelling, hypoxia, oxidative stress, or mitochondrial malfunction. Cell senescence is an irreversible cell cycle arrest characterised by an increase in heterochromatin and foci of DNA damage-responsive proteins, often known as DNA scars, as well as elevated levels of cyclin-dependent kinase inhibitor (CKI). Although these cells do not grow, they remain in the tissues and exhibit a high level of secretory activity, which is known as the senescence-associated secretory phenotype (SASP).

Their secretome is made up of a complex mix of molecules, including pro-inflammatory cytokines and chemokines, as well as proteases, that are released into the extracellular space, creating a pro-inflammatory environment that can have a negative impact on neighbouring cells and overall tissue biology. The build-up of senescent cells in tissues with increasing age implies a loss in cell repair mechanisms and the immune system's ability to eliminate both injured and senescent cells. This is owing in part to the fact that these cells adopt pro-survival pathways in order to avoid detection, which results in immune clearance. Increased senescent cell burden causes functional cell loss and a wide range of non-autonomous disruptions, which coincide with a decline in tissue regeneration and performance, favouring organ malfunction. As a result, in recent years, senescence has emerged as a driver of ageing and age-related disorders.

Neurogenesis persists in two limited niches: the sub-ventricular zone near the lateral ventricles and the sub-granular zone within the dentate gyrus. Neural stem/progenitor cells (NSPCs) are maintained in these areas throughout adulthood and give rise to daughter cells that differentiate into neurons and glia. Adult neurogenesis appears to have a role in normal brain function, and evidence suggests that neurological diseases and neuro degeneration may be caused, at least in part, by decreased neuronal output of adult NSPCs.

In the adult mammalian brain, neural progenitor cells that retain the ability to create new neurons are found largely in the sub-ventricular zone and the sub-granular zone of the hippocampal dentate gyrus. Normal ageing causes a decrease in neuron development in both locations. This phenomenon is linked to cognitive deterioration. The molecular mechanisms behind age-related neurogenic deterioration, however, remain unknown. Neurogenesis is a complex, multistep process, and the observed age-related decline could be due to a reduced pool of neural stem cells, slower cell cycle progression, a lower survival rate, a deficit in migration capacity, or an inability to undergo neuronal differentiation and develop functional neurons.

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