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NEUROPHYSIOLOGICAL MECHANISMS AND ITS EFFECTS ON MEMORY IN AGING

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Memory, categorized as explicit, implicit, or procedural, is stored across varying durations, from transient working memory to enduring long-term retention (Squire, 2017). Aging disrupts neurophysiological mechanisms governing memory encoding, consolidation, and retrieval, necessitating deeper mechanistic insights to inform therapeutic strategies (Gallagher et al., 2019). Neuroanatomical studies using MRI reveal age-associated structural decline, including hippocampal and prefrontal cortex atrophy (Fjell et al., 2016), alongside white matter degradation marked by hyperintensities (WMHs) (Wardlaw et al., 2015). These changes disrupt neural connectivity, impairing memory networks and executive function (Ferreira et al., 2016).

Aging impairs synaptic plasticity mechanisms such as Long-Term Potentiation (LTP) and Long-Term Depression (LTD) (Kumar et al., 2019), while reduced dendritic complexity and synaptic density limit adaptive remodelling (Morrison & Baxter, 2016). “Mitochondrial dysfunction exacerbates these deficits, as aging neurons exhibit impaired energy metabolism, oxidative stress, and compromised ATP production, further destabilizing synaptic efficacy” (Picard et al., 2016, p. 312). Concurrently, neurotransmitter dysregulation–including cholinergic decline (Ballinger et al., 2016), heightened GABAergic inhibition (McQuail et al., 2015), and glutamatergic disruption (Bettio et al., 2017)–destabilizes the excitatory-inhibitory balance critical for memory processing. These molecular and synaptic alterations synergistically hinder cognitive resilience (Lövdén et al., 2020).

Hippocampal neurogenesis, essential for memory formation, declines with age due to chronic stress (Schoenfeld & Cameron, 2015), inflammation (Kempermann et al., 2018), and sedentary lifestyles (Voss et al., 2019). Elevated glucocorticoids (Sapolsky, 2015) and pro-inflammatory cytokines (e.g., TNF-α, IL-6) (Di Benedetto et al., 2017) suppress neurogenesis and synaptic integrity, creating a vicious cycle of neuronal damage. Conversely, physical exercise and enriched environments enhance neurogenesis (Erickson et al., 2019), underscoring lifestyle modifications as actionable interventions.

Neuroinflammation exacerbates age-related cognitive decline, with chronic low-grade inflammation disrupting synaptic function and neuronal survival (Heneka et al., 2018). “Mitochondrial dysfunction and oxidative stress further amplify inflammatory cascades, accelerating neurodegeneration” (López-Otín et al., 2016, p. 89). This interplay between structural atrophy, synaptic dysfunction, and inflammation highlights the multifactorial nature of memory decline (Fjell et al., 2017).

In conclusion, age-related memory deficits arise from interconnected structural, molecular, and inflammatory processes. Targeting these mechanisms–via neurogenesis-promoting lifestyles (Erickson et al., 2019), anti-inflammatory therapies (Cunningham et al., 2019), “mitochondrial antioxidants” (Stefanatos & Sanz, 2018), or synaptic plasticity enhancers (Lynch et al., 2017)–could preserve cognitive resilience. Future research must prioritize translational strategies to bridge mechanistic insights from animal models into clinical practice, ultimately improving quality of life for aging populations (Hodes et al., 2016).