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Mini Review

New Cells Vs Old Cells in Ageing Brain

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Our health is incomplete without mental wellbeing, and our nerve cells supremely own the cognitive and psychosocial wellness. Our cognitive abilities have domains like processing speed, attention, memory, language, visuospatial abilities, and executive functioning. Age-related decline in organ health is inevitable and this holds true even for the nerve cells' functional capabilities. Our cognitive skills gradually decline with age, manifesting as decremented memory; lack in attention; difficulties in learning and motor coordination; slow-paced decision-making; and detrimentally altered sensory perceptions (vision, hearing, smell, taste and touch) [1].

Waste clearance mechanisms in brain

- Brain cells are amongst the most highly active cells in our body with a high rate of metabolism. A substantial amount of toxic by-products/interstitial waste products are released into the brain due to the high metabolic activity of neurons. Such products like lactate, carbon dioxide and proteins (amyloid-β/Aβ and tau), require rapid exit through waste clearance (WC) mechanisms
- Waste clearance (WC) is essential for brain homeostasis. Regulation of metabolic by-products, and recycling of neurotransmitters, are essential for proper neuronal functioning and healthy brain cell aging [2].

Does the rate of old cell clearance slow down during ageing?

- Cellular senescence is a process characterized by an irreversible cell cycle arrest which gets triggered by varied stressors like mitochondrial dysfunction, glycation, oxidative stress, and telomere dysfunction, notably. Senescent cells get accumulated in ageing tissues.
- Senescent cells may detrimentally effect aging and relate with age-related diseases partially via the senescence-associated secretory phenotype (SASP). A chronic exposure to SASP may contribute to the spreading of senescence to otherwise healthy tissues [3,4].

Surprisingly, Neurogenesis or new brain cell formation continues to happen well in old age!

Science suggests it's possible to make new brain cells and improve your memory. The key is to get moving. Scientifically, it is proven that, even as we age, our brain is capable of continuing its new cell (neuron) formation or neurogenesis [5,6].

- The efficacy of neurogenesis in our brain, and how effectively we enhance it are crucial to solve the puzzle on age-related memory loss. And perhaps, this would pave way for preventing dementia, and curb the progression of neurodegenerative diseases like Alzheimer's disease [7].
- Considering our brain's ability to continue new cell development even during ageing, scientific advancements aim to enhance our brain's neurogenesis through best practices. This aligns with the ideology of a renowned neuroscientist,

Dr. Rudolph Tanzi, who is the co-director of McCance Center for Brain Health at Harvard-affiliated Massachusetts General Hospital.

- Our brain, on an average has 100 billion cells. A majority of these are formed prior to birth, and during early childhood the neurogenesis is rapid-paced. It continues through the rest of the life with a gradual, age-related decline. Interestingly, the process of neurogenesis doesn't stop as we age.
- Research states that an area of our brain called hippocampus produces nearly 700-1,500 new neurons every day. This region is responsible for learning and storing memories. It also vitally supports the health of existing neurons.

SHIELD yourself against neuronal degeneration

Our lifestyle has a huge impact on neuronal degeneration, being both causative when it's unhealthy and protective when it's healthy. Such key lifestyle components include food choices, eating pattern, meal timings, exercise schedule, sleep adequacy, stress response, substance abuse and environmental triggers. To address these key lifestyle aspects, simple and doable points have been compiled as a guideline 'SHIELD' (inspired by the advice from a famous neuroscientist, Dr. Rudolph Tanzi)-

- **S**: Sleeping six to eight hours, every night. Sleep quality and adequacy are essential to remove amyloid plaque and flush out other toxins from the brain cells. Their build-up eventually leads to the risk of Alzheimer's disease.
- **H**: Handling stress. Stress induces increased cortisol production and release from the adrenal glands. Increased cortisol adversely affects memory and cognition.
- **I**: Interaction with friends and family. Constant loneliness can double the risk of Alzheimer's disease.
- **E**: Exercise. Regular aerobic activity helps induce neurogenesis. Aerobic exercise enhances the production of a vital protein, brain-derived neurotrophic factor/BDNF in the brain which maintains prime nerve health (by helping neuronal growth and survival). The BDNF for our brain health is as crucial as a fertilizer for a plant 'seed' growth.
- **L**: Learning new things. Learning increases synapses (communication channels between neurons) and improves the brain's resilience.
- **D**: Diet. Our gut is called the second brain. And the link between how the gut and brain communicate with each other via the gut-brain axis is crucial is understanding nerve health. Fresh fruit and vegetable intake equating to at least five servings daily is an elixir for preserving nerve health. The reason is, their dietary fiber gets fermented to the short chain fatty acids/SCFAs such as acetate, propionate, butyrate, isobutyric acid and formic acid, which prevent gut inflammation and downstream neurodegeneration. In Parkinsonism, gut dysbiosis is evidenced as the SCFA-producing bacteria (Bacteroides, Prevotella, and Ruminoccoccus) are reduced in count, while the endotoxin-producing bacteria (Actinobacteria and Proteobacteria) show a rise [8].

Gene-nutrient interactions impact nerve health through gut-brain axis

The G-protein coupled receptor (GPR109A) lodged on the intestinal epithelial surface vitally controls intestinal permeability and inflammatory cascade. During an inflammatory state, butyrate functions through GPR109A to increase tight junction protein' concentration and favorably controls intestinal permeability. Genetic polymorphisms (such as rs676823, rs2454726, rs7314976, rs12372280, rs601339, rs2454722, rs7972971) that alter GPR109A receptor-binding might decrease the concentration of tight junction proteins, resulting in increased intestinal permeability and inflammation susceptibility. GPR109A being a high-affinity niacin receptor, dietary niacin enhances its nerve-health benefits. Niacin shifts the macrophage polarization in favor of anti-inflammatory profile, mitigating the risk of pro-inflammation. Niacin is the precursor for converting nicotinamide adenine dinucleotide/NAD to its reduced form, NADH; NADH supply is much-needed for dopamine production in the striatum. Moreover, the dopaminergic medications of Parkinson's disease can cause niacin deficiency. An increased NAD/NADH ratio also boosts mitochondrial functions, favoring cell survival [8-10].

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