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Editorial

Can we Stretch Our Memory? An Anatomical Update

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Human beings have been blessed (by the process of evolution) with the gift of its cognitive ability. Among all the cognitive abilities, memory has always remained an important domain whose role in our daily lives is immense. As a natural (could be physiological or pathological or combination of both) process, with aging our memory gets a halt first, followed by variable rates of fading which, in its extreme forms can clinically present to us as a neurobehavioral symptom complex named as dementia. Massive research has been conducted globally to 'pause' this decline and recent evidence of 'possible memory enhancement' correlating its anatomical basis has gained much attention as well.

Classically, memory has been divided into short term or working memory and long term memory and following retrieval the long term memory goes for reconsolidation [1]. The areas of brain attributed to different types of memory resides mostly around medial temporal lobe, or at hippocampus being more specific. Other brain areas, like roles of various cortical e.g. frontal for working/episodic memory, temporal, parietal for semantic memory and subcortical areas e.g. cerebellum and basal ganglia(putamen)for procedural memory has also been established [2].

In the previous days, it was mostly considered that post puberty human brain or its capacity remains fixed with gradual decline in the adulthood and so on. However, the 'ceiling effect' of memory has been proved as a myth, and different individual's capacity of different cognitive function, memory reserve and retention has been argued against it.

Over the years, various evidences have been accumulated supporting the hope or possibility of enhancement of human memory even after attaining the 'so called' adult age. Neurogenesis of adult hippocampus has been proved by various investigators, the practical utility of which is well supported through various treatment options. Existence of memory suppressor genes in various animal or insect models (like Aplysia, Drosophila, house mouse) and effect of their deletion on the behaviour has established the genetic base of cognition and possible role of mutagenesis or gene deletion as a targeted treatment option [3]. The molecular basis and protein translation for the short term and long term memory has been explained by CamKII based auto phosphorylation and CREB dependent protein synthesis (along with modulatory roles of GPRCs) respectively [4-6] The hippocampal neurogenesis in adult brain has been observed to generate new excitatory granule cells (in dentate gyrus), the axons of which then makes mossy fibre tracts further connecting to CA3. These granule cells are derivatives of type 1 radial glia-like precursor cells, which over several morphological changes (like type 2a glial, type 2b neuronal, type 3 neuroblast like stage) and cell cycles, turns same as older granule cells and increases the synaptic plasticity [7].

Cognitive Remediation Therapy (CRT), defined as a 'behavioural-training based intervention that aims to improve cognitive processes (attention, memory, executive function, social cognition, or metacognition) with the goal of durability and generalization [8] is a recent advance among the non-pharmacological treatment options in mental health disorders, which initially though has been tried mostly on people suffering from Schizophrenia, in current days it's application has gained much popularity in other mental illnesses too, like anxiety disorder, depression, eating disorder or early stage dementia [9-12].

Both clinicians administered or computerised CRT (like Cogpack, Cogrehab) has shown promising result in cognitive improvement, and has remained a wonderful evidence of brain neurogenesis in chronic and severe mental illnesses [13,14]. The role of neurogenesis has been postulated in favour of CRT induced neuro cognitive improvement, and effect on hippocampus has been mostly attributed for this. The anatomical brain regions or clusters having effects of CRT are, left middle frontal gyrus (MFG), left inferior frontal gyrus (IFG), left superior frontal gyrus, pre- and post-central gyrus, bilateral insula, parietal lobe, and medial frontal gyrus etc, as evidenced by neuroimaging studies [15]. Cognitive remediation training (CRT) also supports restorative functioning in prefrontal and thalamic areas [15].

To conclude, some has feared that the attempt of enhancing memory or cognition might make our brain 'a wasteland of non-interpretable junk memory traces' which will listen, see and remember every stimulus irrespective of its importance or actual need, though this has been countered by the evidence of bi directionality of brain plasticity, hence the 'adjustability' of memory and other cognitive functions [16-18].

The steady and progressive decline of various cognitive functions in Alzheimer's Disease and similar neurodegenerative disorders has remained an area of interest for researchers since long, and needless to say, anatomical correlates of all the pharmacological or behavioral attempt of memory enhancements will guide the future researchers even better, both for understanding and implementation of the same.

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