

Perspective

Memory Loss Issues and Concerns in Old Age People

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DESCRIPTION

As patients get older, memory issues worry many of them. Gary small offers confidence and outlines a plan for evaluating agerelated memory decline and safeguarding brain health. Patients are expressing more anxiety about their common, aging-related memory problems as doctors and researchers have increased their attention on Alzheimer's disease and associated dementias. Our patients frequently ask us how they can maintain their memory as they age when the results of fresh research on early identification and prevention are made more widely available. Many of the doctors practicing now received their medical training at a time when little knowledge about these subjects was taught.

The question of whether or to what extent it is valid to attribute these differences to ageing person to some variable or class of variables that intervene between age and remembering arises laboratory tests showing that older adults typically perform worse than do younger adults on many types of memory tasks.

The majority of the study strongly suggests that slowing downs in information processing are a major factor in typical age-related memory decline. Other mechanisms, including as working memory, executive function, and sensory processes, are nevertheless sometimes crucial. Even while age-related memory loss is a phenomenological and factual reality and has been the subject of numerous explanations, there is still more research to be done to determine why it happens. Future prospects for memory ageing research are likely to be shaped by current discussions on the nature and methods of detecting shared and distinctive effects. Hippocampus-related topographic amnesia is the most common symptom of memory disorders in Alzheimer's

Disease (AD) patients. Latest findings have revealed that understanding-mediated DNA methylation, which is regulated by enzymes with DNA MethylTransferase (DNMT) activity, is essential for the development of recent remembrance as well as the maintenance of outlying memory. Notably, overexpression of DNMT in the hippocampus can reverse longitudinal memory deficits in aged mice. However, a decline in global DNA methylation was found in the autopsied hippocampi of patients with AD. Precisely, what endogenous factors that affect DNA methylation still persist to be elucidated.

Here, we report a marked increase in endogenic formaldehyde levels is associated with a decline in global DNA methylation in the autopsied hippocampus from AD patients. *In vitro* and *in vivo* results show that formaldehyde in excess of normal biological levels reduced global DNA methylation by interfering DNMTs. Interestingly, intra hippocampal injection of excess formaldehyde before spatial learning in healthy adult rats can mimic the learning difficulty of early stage of AD. Moreover, injection of excess formaldehyde after spatial culture can mimic the loss of remote longitudinal memory observed in late stage of AD. These conclusions imply that aging-associated formaldehyde impacts to topographical amnesia in AD patients.

Memory shortages associated with regular aging and alzheimer's disease have been associated to a decline in the volume of brain structures, such as the hippocampus and to genetic signs, such as apolipoprotein E. In this concern, examination of CSF for biomarkers of disorder can help in differentiating normal aged from Alzheimer's disease. Measures of oxidative stress and cholesterol in plasma relate with memory deficits; investigation recommends that treatments that reduce oxidative stress or cholesterol through physical activity, diet or the use of antioxidant vitamins may delay intellectual decline.

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Received: 02-Jan-2023, Manuscript No. JASC-23-19851; Editor assigned: 05-Jan-2023, Pre QC No. JASC-23-19851 (PQ); Reviewed: 20-Jan-2023, QC No JASC-23-19851; Revised: 26-Jan-2023, Manuscript No. JASC-23-19851 (R); Published: 03-Feb-2023, DOI: 10.35248/2329-8847.23.11.298.

Citation: Zan Z (2023) Memory Loss Issues and Concerns in Old Age People. J Aging Sci. 11:298.

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J Aging Sci, Vol.11 Iss.1 No:1000298

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