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Short Communication

Core Characteristics of Alzheimer's Disease

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DESCRIPTION

There have been several unsuccessful attempts to use anti-amyloid methods to treat Alzheimer's Disease (AD). Both autopsy and imaging investigations have shown that the "amyloid cascade theory" does not adequately explain the neuronal damage in AD [1]. While there is continuous discussion over the specific function of neuro inflammation and whether it is beneficial or detrimental, it is clear. In this article, we concentrate on the putative glial activation mechanism and how regional and systemic variables affect disease development.

Neurological dysfunction may be caused by the ensuing hypo metabolic condition. In fact, compared to premenopausal women and age-matched males, premenopausal and postmenopausal women between the ages of 40 and 60 have an AD-end phenotype marked by lower metabolic activity and increased brain amyloid-beta deposition. In this article, the MT is discussed as a window of opportunity for therapeutic approaches to offset the brain bioenergetics crisis and counteract the ensuing elevated risk for AD in women.

A significant risk factor for developing late-onset Alzheimer's disease is female sex, which also includes old age and the Apo lipoprotein E (APOE)-4 genotype (AD). Given that AD pathology develops decades before clinical symptoms appear, the increased risk in women cannot only be explained by their longer life expectancy when compared to males [2]. Menopause Transition (MT), a female-specific midlife neuroendocrine transition state, has been linked to recent research into sex-specific pathophysiology pathways underlying AD risk.

Numerous symptoms of MT, which is frequently described as leading to reproductive senescence, are neurological in nature and include depression, impairment in several cognitive domains, and disturbance of oestrogen-regulated systems like thermoregulation, sleep, and circadian rhythms. Preclinical research has demonstrated that the oestrogen network separates from the brain's bioenergetics system during MT.

Despite decades of study, effective therapies for dementia-related NPS have not been discovered, and those that are now in use include significant hazards for individuals who take them. In the spring of 2010, an Alzheimer's Association Research Roundtable was held to examine the current understanding of NPS in Alzheimer's disease, to explore categorization, underlying neuro pathogenesis, and vulnerabilities, and to provide suggestions for novel treatment strategies. Since amyloid- was isolated and identified from post-mortem examinations of the brains of AD patients, the area of Alzheimer's Disease (AD) study has expanded dramatically over the past several decades [4]. The Journal of Alzheimer's Disease (JAD) recently published 300 research reports that were considered to be the most significant studies on AD since 2010.

We concentrate on neuro inflammation in AD, particularly in its early phases, which is characterized by a vicious cycle of pro-inflammatory factor production, glial priming, and neuronal injury. Reviewing imaging studies' findings regarding the timing of amyloid deposition and neuro inflammation, the impact of systemic inflammation on glial activation during both acute and chronic stimulation, and the utility of inflammation as a diagnostic and therapeutic the most significant developments in research on Alzheimer's Disease (AD). The most recent findings in genetics and epidemiology are included in this overview. Important insights into the causes of and variables that contribute to AD, as well as areas of focus for research into mechanisms and therapies, are emerging from epidemiological and genetic investigations [5]. The extensive use of genome-wide association studies has produced persuasive evidence of the genetic complexity of AD, with genes involved in the etiology of AD being linked to functions as varied as immunology and lipid metabolism.

Even though meditation is said to have been practiced for over 5,000 years, scientific study of it is still in its infancy. A topic that is very briefly mentioned when it comes to Alzheimer's Disease (AD) prevention is reducing the considerable harmful biochemical consequences of stress. The effects of meditation on cognition and wellbeing for reducing neuro degeneration and preventing AD are discussed, as well as lifestyle and stress as potential causes of AD [6]. This article focuses on Kirtan Kriya (KK), a simple, affordable meditation technique that only takes

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12 minutes a day and has been shown to improve memory in studies of people with subjective cognitive decline, mild cognitive impairment, and highly stressed caregivers-all of whom have a higher risk of developing AD in the future. It has also been shown to enhance genes that regulate insulin and glucose, upregulate immune system genes, increase telomerase by 43% that improves sleep, and decreases anxiety. Additionally, KK enhances psycho-spiritual health, which is crucial for maintaining cognitive function and preventing AD. Older people may easily learn and practice KK. The underlying assumption of this analysis is that KK, as well as other practices like dietary change, physical activity, mental stimulation, and socializing, may be helpful as a component of an AD preventive program.

CONCLUSION

Core characteristics of Alzheimer's disease and associated dementias include Neuropsychiatric Symptoms (NPS). These symptoms, which were once believed to appear more frequently in patients with late-stage illness, are now recognized to frequently appear in very early disease and in prodromal phases, such as moderate cognitive impairment. Neurological dysfunction may be caused by the ensuing hypo metabolic condition.

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