Perspective

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Alzheimer's Disease is a Neurodegenerative Condition

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Description

Alzheimer's disease is a neurodegenerative condition in which cognitive abilities gradually decline. Neuroimaging techniques like resting-state functional Magnetic Resonance Imaging (fMRI) have been widely used to investigate brain activity linked to neurodegenerative diseases. The binary classification of Alzheimer's disease and mild cognitive impairment has been the only focus of previous research. However, little research has been done on the use of computer-aided diagnosis in the various stages of Alzheimer's disease progression. Six stages of Alzheimer's disease are analyzed and methods for multi-label classification using rs-fMRI and deep learning are presented in this study. The k-fold cross-validation method was used to evaluate the results of the suggested models. The neurological condition known as Alzheimer's disease, which affects millions of people worldwide and is caused by brain proteins misfolding, has received the most research attention.

Exosomes Can Be Manipulated

The pathogenesis of AD primarily involves tau and amyloid. The only benefit of therapeutic interventions and advances in nanotechnology is the management of AD symptoms, and there is still no cure for the disease. Given their roles in AD progression and treatment, exosomes, which come from the majority of cell and tissue types, are regarded as a double-edged sword. Exosomes can be manipulated to act as drug delivery vehicles for a wide variety of therapeutic cargos, including small and large molecules. Thus, we survey the jobs of exosomes in the pathology, determination, and treatment of Promotion and feature their application as a medication transporter to the mind for Promotion treatment. The development of effective Alzheimer's disease treatments is now more important than ever because of the disease's severity and the growing number of patients. Two of the most characteristic histopathological and pathophysiological symptoms are the accumulation of Amyloid-Beta (A) plaques and tau protein tangles in the nerve tissue of the brain.

The brain's decrease in acetylcholine levels is another important factor in Alzheimer's disease's etiology. Cholinesterase inhibitors and an antagonist of the N-methyl-D-aspartate receptor, two medications currently in use to treat Alzheimer's disease, can temporarily reduce dementia symptoms but cannot stop or reverse the progression of the disease. In addition, it has been demonstrated that a number of medicinal plants, either in their whole form or as their individual chemicals, can lessen the degenerative features that are associated with Alzheimer's disease. A neurodegenerative disorder known as Alzheimer's disease is accompanied by changes in mood, such as apathy and depression. Alzheimer's disease is characterized by increased levels of glutamate, a neurotransmitter that is thought to be excitatory and causes the overproduction of glutamate, which may result in neural damage that affects memory and learning. Neural changes in Alzheimer's disease reveal the formation of amyloid plaques. Adipose tissue has an endocrine function, Neurodegenerative dementias have been shown to have decreased circulating levels of adipokines. Alzheimer's disease patients had the lower levels of resistin than the control group. Plasma levels of resistin were found to be correlated with liquoral amyloid levels of 1-42 in dementia patients. In Western societies, cognitive decline is frequently brought on by Alzheimer's Disease (AD) and Frontotemporal Dementia (FTD), both of which contribute significantly to the social cost. In recent years, significant scientific and financial efforts have been directed toward the identification of biomarkers of neurodegeneration that are more precise and predictive.

Amyloid-Beta Plaques

In rats with Alzheimer's disease caused by AlCl3, GA and hesperidin enhanced cognition. When AlCl3 was used to cause Alzheimer's disease in rats, both gallic acid and hesperidin improved neurotransmission. In alzheimer's rats, hesperidin and gallic acid reduced inflammation in the brain. In alzheimer's rats, the antioxidant status of the brain was improved by gallic acid and hesperidin. Necroptosis-induced inflammation can exacerbate Alzheimer's disease. Necroptosis, which is also a key characteristic in AD, causes insulin resistance. Necrosis is another type of cell death that can be found in almost any tissue. The cell membrane disruption that triggers an inflammatory immune response is its most distinctive feature. Through a literature review, the purpose of this study was to investigate the role that necroptosis plays in the onset of Alzheimer's disease. Necroptosis was found to occur in Alzheimer's disease and may also play a significant role due to a number of other factors. Alzheimer's disease is referred to as diabetes type 3 because hyperglycemia causes the switch from apoptosis to necroptosis. Second, excessive production of

reactive oxygen species during necroptosis influences the production of Alzheimer's disease amyloid beta. Necrosis-related inflammation is also a major cause of neurodegeneration and the overproduction of amyloid beta.

The starving brain theory of Alzheimer's disease is supported by these connections, and insulin resistance exacerbates the role of necroptosis in Alzheimer's disease progression. Due to a variety of factors, necroptosis may cause Alzheimer's disease to spiral downward, making it an important therapeutic target that requires further investigation. Alzheimer's disease frequently presents with mood and sleep disorders as comorbidities. Endoplasmic reticulum stress may cause prostate cancer and Alzheimer's disease. Memory loss is the primary characteristic of Alzheimer's disease. The medications that are currently available to treat AD only alleviate symptoms and do not stop the progression of the disease. As a result, it is crucial to look for effective therapeutic alternatives with multiple target actions. Thiazolidin-4-one, a substance with antioxidant, anticholinesterase, and amnesic properties, is one possible substitute. In the cerebral cortex, hippocampus, and cerebellum, memory, acetyl cholinesterase activity, phosphorylated tau protein levels, and oxidative stress were examined. In the blood and serum, biochemical and hematological parameters were examined and Memory disability.