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Editorial

Individuals with Down's Syndrome and Alzheimer's Disease: The Opportunities for and the Difficulties of Creating Preventive Therapies

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ABSTRACT

At a relatively early age, individuals with Down's syndrome (DS) are at high risk of contracting Alzheimer's disease (AD). This elevated risk is not observed for causes other than DS in individuals with intellectual disabilities, and for this reason it is unlikely to be attributed to non-specific symptoms of neurodevelopmental illness, but instead a direct product of DS genetics (trisomy 21).

KEYWORDS: Down Syndrome, Trisomy 21, Chromosome Abnormality

INTRODUCTION

The amyloid cascade hypothesis is the dominant theory that accounts for this risk, considering the location of the amyloid precursor protein (APP) gene on chromosome 21, with other genetic and environmental influences modifying the age of onset and the path of the disease. Several new therapies are currently being studied that address the amyloid receptor and attempt to alter the progression of AD, which could also be beneficial for the treatment of AD in DS. However, given that AD-related neuropathology occurs several years before dementia occurs, all preventive therapy must begin long before symptoms begin. Plasma, CSF, brain, and retinal biomarkers are being tested as proxies for early diagnostic and outcome assessments for AD to facilitate studies of such therapies.

Given the situation of the amyloid precursor supermolecule (APP) factor on body 21, the amyloid cascade hypothesis is that the dominant theory accounting for this risk, with different genetic and environmental factors modifying the age of onset and also the course of the disease. Many potential therapies targeting the amyloid pathway and progressing to modify the course of AD are presently being investigated, which can even be helpful for treating AD in DS. However, only if the neuropathology related to AD begins a few years before dementedness manifests, any preventative treatment should start well before the onset of symptoms. To change trials of such interventions, plasma, CSF, brain, and retinal biomarkers are being studied as proxy early diagnostic and outcome measures for AD. During this systematic review, we have a tendency to think about the prospects for the event of potential preventative treatments of AD within the DS population and their evaluation.

In people with DS, pharmacological approaches to the treatment of AD have been primarily focused on the cholinergic theory of AD and on the use of anticholinesterase inhibitors. These early pharmacological therapies have been shown to relieve the effects of AD to a small degree in DS, but have not changed its subsequent course.

People with DS are at high risk, relatively early in life, of having Alzheimer's dementia. The "amyloid cascade hypothesis" is the most plausible reason for this risk. Thus, persons with DS are an excellent population to research AD and are therefore expected to benefit from novel therapies designed for this disease. Much of the available drugs at the moment, however, are symptomatic. In mouse models, the BACE antagonists, 2-aminooxazoline and 3-azaxanthene, and γ-secretase modulators, DAPT, have been shown to minimize Aβ but are still being studied in AD humans. Successful Aβ42 immunotherapy studied in DS mouse models has demonstrated increases in cognitive performance as well as reductions in neuronal atrophy without obvious side effects.

Since the pathological changes of AD ar gift before the onset of symptoms in individuals with DS, analysis on treatments for this malady have to be compelled to target triggers and steps within the amyloid pathway, reducing the assembly or enhancing the clearance of Aβ macromolecule within the brain and, consequently, stunning or delaying the event of the malady, assumptive that it's correct that it's excess amyloid and also the later cascade of neuropathological events that ar driving this method. additionally it's crucial to spot biomarkers for AD during this population therefore on be able to confirm the effectiveness of any new treatments early within the course of the underlying malady method and well before the AD-related pathology and cerebral atrophy became established.

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