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Editorial on Alzheimer's Disease: Need for New Therapies

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Introduction

New targets and biomarkers are being discovered using a precision medicine strategy by the AMP Alzheimer's disease program. The objective of the first AMP Alzheimer's program (AD 1.0), which was launched in 2014, was to evaluate the efficacy of tau imaging as a biomarker for disease progression and treatment response. The second iteration of the AMP AD program-AMP AD 2.0-was launched in March 2021 . It expands on the initial efforts to discover new AMP AD targets and biomarkers and aims to enable a precision medicine approach. Alzheimer's disease is a progressive brain disorder in which memory and thinking abilities gradually decline. Alzheimer's disease is characterized by two distinct brain lesions: Neurofibrillary tangles (NFT), which are composed of aggregated tau proteins within the interior of cells, and plaque, which forms between nerve cells and is composed of fragments of the protein amyloid beta (A) [1]. It is the sixth leading cause of death in the United States and the most common diagnosis for dementia patients. Alzheimer's disease dementia affects as many as 5.8 million Americans aged 65 and older, and the prevalence is expected to rise to 13.8 million in the United States by 2050.

The cost of dementia to the economy is staggering; An estimate of $287,500 was spent on health care for a person with probable dementia during their final five years of life, according to an analysis supported by the NIH. Numerous biopharmaceutical companies have developed treatments as a result of the evidence linking A plaque accumulation to AD [2]. However, no one has yet demonstrated clinical efficacy in clinical trials with patients. It's possible that these failures are the result of issues with particular molecules or the design of the trial, rather than the hypothesis itself. Improved tools are urgently required to support target validation in patients prior to Phase III clinical trials and to identify new targets that offer alternative strategies for addressing the disease process. In addition, it is essential to locate trustworthy biomarkers that can predict a patient's response to a therapeutic intervention. Safe and efficient treatments for Alzheimer's disease and other dementias are still lacking, despite significant investments in research and development and advancements in our comprehension of the disease's pathogenesis. The fact that there are no treatments demonstrates the need to alter the ways in which the government, academic, and biopharmaceutical sectors involved in Alzheimer's disease research and therapy development generate, share, and utilize knowledge to propel therapeutic development [3]. The Alzheimer's research and drug development process must be transformed into one that is participatory, collaborative, well-integrated, and iterative if effective therapies are to be developed. This is what the AMP AD program aims to accomplish through a precompetitive partnership between government, business, and non-profit organizations that focuses on developing biomarkers to validate existing therapeutic targets and discovering novel, clinically relevant therapeutic targets.

The initial Target Discovery component's accomplishments serve as the foundation for the AMP AD 2.0 program. By improving the molecular characterization of Alzheimer's disease in brain, blood, and spinal fluid samples taken from a variety of populations, its goal is to make it possible to use a precision medicine approach to target and discover biomarkers [4]. The FAIR data infrastructure, the AD Knowledge Portal (link is external), and the portal-linked, open-source platform Agora are being developed in tandem with AMP AD 2.0's renewed commitment to open science practices for data, methods, and results sharing. Data from a variety of cell-based and animal models, as well as well-annotated, high-quality human multi-omic (genomic, proteomic, and metabolomic) samples, as well as the AD Knowledge Portal, can be quickly shared.

The use of statins to lower cholesterol may be beneficial in Alzheimer's. Antihypertensive and antidiabetic medications in individuals without overt cognitive impairment may decrease the risk of dementia by influencing cerebrovascular pathology. More research is needed to examine the relationship with Alzheimer's disease specifically. The use of statins to lower cholesterol may be beneficial in Alzheimer's. It is necessary to clarify the direct relationship between medications and other concurrent lifestyle changes (such as dieting, exercising, and quitting smoking). Depression is linked to an increased risk of Alzheimer's disease; management with antidepressants may serve as a preventative measure. Traditionally, it was believed that long-term use of non-steroidal anti-inflammatory drugs (NSAIDs) was linked to a lower risk of Alzheimer's disease because NSAIDs reduce inflammation. However, NSAIDs do not appear to be effective as a treatment. Additionally, it was once thought that estrogen deficiency during menopause was a risk factor since women have a higher incidence of Alzheimer's disease than men. However, there is insufficient evidence to suggest that menopausal hormone replacement therapy (HRT) lowers cognitive decline risk.

Conclusion

Acetylcholinesterase inhibitors are used to reduce the rate at which acetylcholine (ACh) is broken down, thereby increasing the concentration of ACh in the brain and combating the loss of ACh caused by the death of cholinergic neurons. There is evidence for the efficacy of these medications in mild to moderate Alzheimer's disease, and some evidence for their use in the advanced stage. The use of these drugs in mild cognitive impairment Muscle cramps, decreased heart rate (bradycardia), decreased appetite and weight, and increased gastric acid production are less common secondary effects of glutamate [5-7]. Although glutamate is an excitatory neurotransmitter of the nervous system, excessive amounts in the brain can lead to cell death through a process known as excitotoxicity, which consists of the overstimulation of glutamate receptors. These side effects occur in approximately

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10–20% of users and range in severity from mild to moderate. They Excitotoxicity is present in multiple neurological disorders, including multiple sclerosis and Alzheimer's disease. Memantine is a noncompetitive NMDA receptor antagonist that was initially utilized as an anti-influenza medication. Memantine has been shown to have a small benefit in the treatment of moderate to severe Alzheimer's disease Reported adverse events with memantine are infrequent and mild, including hallucinations, confusion, dizziness, headache, and fatigue. The combination of memantine and donepezil has been shown to be "of statistically significant but clinically marginal effectiveness."

References

1. Carney DN, Zweig MH, Ihde DC, Cohen MH, Makuch RW, et al. (1984) Elevated serum creatine kinase BB levels in patients with small cell lung cancer. Cancer Res 44: 5399-5403.
2. Niklinski J, Furman M, Laudanski J, Palynyczko Z, Welk M (1991) Evaluation of carcinoembryonic antigen (CEA) and brain-type creatine kinase (CK-BB) in serum from patients with carcinoma of the lung. Neoplasma 38: 129-135.
3. Niklinski J, Furman M, Palynyczko Z, Laudanski J, Bulatowicz J (1991) Carcinoembryonic antigen, neuron-specific enolase and creatine kinase-BB as tumor markers for carcinoma of the lung. Neoplasma 38: 645-651.
4. Zarghami N, Yu H, Diamandis EP, Sutherland DJ (1995) Quantification of creatine kinase BB isoenzyme in tumor cytosols and serum with an ultrasensitive time-resolved immunofluorometric technique. Clin Biochem 28: 243-253.
5. Zarghami N, Giai M, Yu H, Roagna R, Ponzone R, et al. (1996) Creatine kinase BB isoenzyme levels in tumour cytosols and survival of breast cancer patients. Br J Cancer73: 386-390.
6. Huddleston HG, Wong KK, Welch WR, Berkowitz RS, Mok SC (2005) Clinical applications of microarray technology: creatine kinase B is an up-regulated gene in epithelial ovarian cancer and shows promise as a serum marker. Gynecol Oncol 96: 77-83.
7. Graus F, Illa I, Agusti M, Ribalta T, Cruz-Sanchez F, et al. (1991) Effect of intraventricular injection of an anti-Purkinje cell antibody (anti-Yo) in a guinea pig model. J Neurol Sci 106: 82-87.