BACE1 Inhibitors Reduce α-Amyloid Production, Provide Potential Treatment for Alzheimer’s Disease

OVERVIEW

Alzheimer’s disease is the most common form of dementia, affecting an estimated 27 million people worldwide. This disease has a large economic impact with estimated direct and indirect costs of $148 billion annually in the United States.

Plaques composed of aggregated β-amyloid peptide, which accumulate in the brains of patients with Alzheimer’s disease, play a key role in the pathogenesis of the disease. β-amyloid is formed when the enzyme BACE1 cleaves the β-amyloid precursor protein. Because BACE1 is the rate-limiting step in β-amyloid formation, it is considered a potential therapeutic target. A UW–Madison researcher previously showed that BACE1 is transiently acetylated by the acetyltransferases ATase1 and ATase2, a modification important to its role in β-amyloid production.

THE INVENTION

The UW–Madison researcher now has identified two compounds that can downregulate BACE1 levels and the rate of β-amyloid production and thus may be potential therapeutics for Alzheimer’s disease. These compounds inhibit the activity of ATase1 and ATase2.

APPLICATIONS

• Prevention or treatment of Alzheimer’s disease

KEY BENEFITS

• Successfully tested in cellular systems and an animal model of Alzheimer’s disease
• May delay the onset or progression of symptoms of Alzheimer’s disease
ADDITIONAL INFORMATION

Publications

Tech Fields
Pharmaceuticals & Vitamin D - Neurological & mental health

CONTACT INFORMATION

For current licensing status, please contact Rafael Diaz at rdiaz@warf.org or 608-960-9847.