# Theoretical study to find a novel drug inhibitor of SFRP1 as a new treatment for Alzheimer's disease.

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## Abstract

Alzheimer's disease (AD) is a neurodegenerative disease of the central nervous system characterized by progressive neuronal death that mainly affects cortical regions of the brain and part of the limbic system. Alterations are produced in memory, language, perception, orientation and executive functions, being one of the major causes of dementia in the world. One of the main characteristics of AD is the appearance of senile or amyloid plaques. Senile plaques are extraneural deposits formed by the accumulation of the Aβ-42 peptide (β-amyloid peptide), produced by the proteolysis of amyloid precursor protein (APP) by β-secretase and γ- secretase. In contrast, non-pathological cleavage of APP occurs by α- secretase and γ-secretase. AD is expected to affect more than 150 million people by 2050, becoming a serious socioeconomic problem. Despite all efforts, no effective therapy has been achieved yet. Due to inefficiencies in therapies against currently known therapeutic targets, in the present work we have investigated the SFRP1 protein (Figure 1) as a new potential therapeutic target. SFRP1 is overexpressed in patients with AD producing inhibition of ADAM10 (α-secretase) (Figure 2), and hence promoting the formation of the β-amyloid peptide.

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