

APP Human, HEK

Amyloid beta (A4) Precursor Protein Human Recombinant, HEK

RCP0085

Product Overview

Name	APP Human, HEK
Accession(Primary)	P05067
Description	Amyloid beta (A4) Precursor Protein Human Recombinant, HEK

Target information (P05067)

Gene Name

About Gene Symbol

APP (AID(50)) is a cell surface marker involved in cell surface signaling. It is an important research target in immunology, flow cytometry, and cancer immunotherapy and is widely studied to elucidate cellular mechanisms and identify therapeutic targets.

Background

Alzheimer's disease (AD) is a neurodegenerative disorder characterized by the accumulation of amyloid plaques and neurofibrillary tangles in the brain, leading to cognitive decline and memory loss. The amyloid beta (A β) peptide, derived from the amyloid beta (A4) precursor protein, has been identified as a key player in the pathogenesis of AD. This research aims to explore the significance of the amyloid beta precursor protein, its processing, and the implications it holds for understanding and treating Alzheimer's disease. The amyloid beta precursor protein (APP) is a transmembrane protein widely expressed in various tissues, with higher concentrations found in the brain. APP undergoes sequential proteolytic processing by enzymes known as secretases, leading to the generation of A β peptides of different lengths. Of particular importance is the production of the A β 42

peptide, which has a propensity to aggregate and form the characteristic amyloid plaques in AD. Understanding the processing and metabolism of APP is crucial for unraveling the mechanisms underlying AD pathology. Mutations in the APP gene and dysregulation of its processing have been associated with familial forms of AD, highlighting the pivotal role of APP in disease development. Investigating the function of APP and its proteolytic fragments can provide valuable insights into the molecular events leading to AD and potentially lead to the identification of therapeutic targets. This research will delve into the processing of the amyloid beta precursor protein, shedding light on the different cleavage pathways mediated by α -, β -, and γ -secretases. The paper will discuss the impact of these proteolytic events on the generation of A β peptides and how alterations in these pathways contribute to AD pathogenesis. Furthermore, it will explore the aggregation properties of A β peptides and their role in the formation of amyloid plaques, as well as their impact on neuronal function and viability. The study will also examine the potential of APP and A β as biomarkers for AD diagnosis and progression monitoring. Investigating the levels of APP and A β peptides in biological fluids and utilizing imaging techniques to detect amyloid plaques could enhance early diagnosis and facilitate the development of novel therapeutic interventions. By elucidating the molecular mechanisms involving APP and A β in AD, this research aims to contribute to the understanding of the disease pathogenesis and identify potential therapeutic targets. Additionally, it underscores the importance of ongoing research in this field to develop effective strategies for early diagnosis, disease modification, and improved patient outcomes.