9051 Mira Mesa Blvd, #910094
San Diego, CA 92191
Tel: 760-390-3989

APP Human, HEK

Amyloid beta (A4) Precursor Protein Human Recombinant, HEK
RCP0085

Product Overview

Name	APP Human, HEK

Description

Amyloid beta (A4) Precursor Protein Human Recombinant, HEK

Accession (Primary)	P05067

Synonyms

ABPP, APPI, Alzheimer disease amyloid A4 protein homolog, Alzheimer disease amyloid protein, Amyloid precursor protein, Amyloid-beta precursor protein, Amyloid-beta A4 protein, Cerebral vascular amyloid peptide, CVAP, PreA4, Protease nexin-II, PN-II, A4, AD1, beta-amyloid peptide, beta-amyloid precursor protein, testicular tissue protein Li 2, AAA, ABETA, alpha-sApp, CTF gamma, PN2.

Source

HEK293 Cells.

Physical Appearance

Sterile Filtered colorless solution.

Formulation

The APP solution (0.5mg/ml) contains 10% Glycerol and Phosphate-Buffered Saline (pH 7.4).

Stability

Store at 4°C if entire vial will be used within 2-4 weeks. Store, frozen at -20°C for longer periods of time. For long term storage it is recommended to add a carrier protein (0.1% HSA or BSA). Avoid multiple freeze-thaw cycles.

Purity

> 90% by SDS-PAGE.

Amino acid sequence

LEVPTDGNAG LLAEPQIAMF CGRLNMHMNV QNGKWDSDPS GTKTCIDTKE GILQYCQEVY PELQITNVVE ANQPVTIQNW CKRGRKQCKT HPHFVIPYRC LVGEFVSDAL LVPDKCKFLH QERMDVCETH LHWHTVAKET CSEKSTNLHD YGMLLPCGID KFRGVEFVCC PLAEESDNVD SADAEEDDSD VWWGGADTDY ADGSEDKVVE VAEEEEVAEV EEEEADDDED DEDGDEVEEE AEEPYEEATE RTTSIATTTT TTTESVEEVV REVCSEQAET GPCRAMISRW YFDVTEGKCA PFFYGGCGGN RNNFDTEEYC MAVCGSAMSQ SLLKTTQEPL ARDPVKLPTT AASTPDAVDK YLETPGDENE HAHFQKAKER LEAKHRERMS QVMREWEEAE RQAKNLPKAD KKAVIQHFQE KVESLEQEAA NERQQLVETH MARVEAMLND RRRLALENYI TALQAVPPRP RHVFNMLKKY VRAEQKDRQH

9051 Mira Mesa Blvd, #910094
San Diego, CA 92191
Tel: 760-390-3989

TLKHFEHVRM VDPKKAAQIR SQVMTHLRVI YERMNQSLSL LYNVPAVAEE IQDEVDELLQ KEQNYSDDVL ANMISEPRIS YGNDALMPSL TETKTTVELL PVNGEFSLDD LQPWHSFGAD SVPANTENEV EPVDARPAAD RGLTTRPGSG LTNIKTEEIS EVKMDAEFRH DSGYEVHHQK LVFFAEDVGS NKGA HHHHHH .

Biological Activity

The inhibitory function of APP on activity of trypsin was measured by a fluorometric assay using Mca-RPKPVE-Nval-WRK(Dnp)-NH2 at pH 7.5 at 37C. The IC50 ≤ 1 nM.

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 (Aβ) 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 Aβ peptides of different lengths. Of particular importance is the production of the Aβ42 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 Aβ peptides and how alterations in these pathways contribute to AD pathogenesis. Furthermore, it will explore the aggregation properties of Aβ 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 Aβ as biomarkers for AD diagnosis and progression monitoring. Investigating the levels of APP and Aβ 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 Aβ 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.

Precautions

APP Human, HEK is for research use only and not for use in diagnostic or therapeutic procedures.

Target Information: (P05067)

9051 Mira Mesa Blvd, #910094
San Diego, CA 92191
Tel: 760-390-3989

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 (Aβ) 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 Aβ peptides of different lengths. Of particular importance is the production of the Aβ42 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 Aβ peptides and how alterations in these pathways contribute to AD pathogenesis. Furthermore, it will explore the aggregation properties of Aβ 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 Aβ as biomarkers for AD diagnosis and progression monitoring. Investigating the levels of APP and Aβ 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 Aβ 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.