# Recombinant Human APP Protein (Trx Tag)

**RPES8057**

## Product Information

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| **Product SKU**: | RPES8057 | **Expression Host**: | E.coli | **Size**: | 20µg |
| **Tag**: | N-Trx | **Reactivity**: | Human | **Accession**: | P05067-1 |

## Additional Information

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| **Calculated MW:** | 30.8 kDa | **Observed MW:** | 35 kDa |
| **Sequence**: | Asp672-Asn770 | | |

## Protein Information

**Background**: Amyloid precursor protein (APP) is a type I transmembrane protein expressed in many tissues and concentrated in the synapses of neurons, and is suggested as a regulator of synapse formation and neural plasticity. APP can be processed by two different proteolytic pathways. In one pathway, APP is cleaved by β-and γ-secretase to produce the amyloid-β-protein (Aβ, Abeta, beta-amyloid) which is the principal component of the amyloid plaques, the major pathological hallmark of Alzheimer's disease (AD), while in the other pathway, α-secretase is involved in the cleavage of APP whose product exerts antiamyloidogenic effect and prevention of the Aβ peptide formation. The aberrant accumulation of aggregated beta-amyloid peptides (Abeta) as plaques is a hallmark of AD neuropathology and reduction of Abeta has become a leading direction of emerging experimental therapies for the disease. Besides this pathological function of Abeta, recently published data reveal that Abeta also has an essential physiological role in lipid homeostasis. Cholesterol increases Abeta production, and conversely A beta production causes a decrease in cholesterol synthesis. Abeta may be part of a mechanism controlling synaptic activity, acting as a positive regulator presynaptically and a negative regulator postsynaptically. The pathological accumulation of oligomeric Abeta assemblies depresses excitatory transmission at the synaptic level, but also triggers aberrant patterns of neuronal circuit activity and epileptiform discharges at the network level. Abeta-induced

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dysfunction of inhibitory interneurons likely increases synchrony among excitatory principal cells and contributes to the destabilization of neuronal networks. There is evidence that beta-amyloid can impair blood vessel function. Vascular beta-amyloid deposition, also known as cerebral amyloid angiopathy, is associated with vascular dysfunction in animal and human studies. Alzheimer disease is associated with morphological changes in capillary networks, and soluble beta-amyloid produces abnormal vascular responses to physiological and pharmacological stimuli.

**Synonyms**: Amyloid Beta Precursor Protein, Amyloid Beta (A4) Precursor Protein, Alzheimer Disease Amyloid Protein, Cerebral Vascular Amyloid Peptide, Amyloid Precursor Protein, Peptidase Nexin-II, Protease Nexin-II, PreA4, PN-II, ABPP, APPI, CVAP, AD1, Beta-Amyloid Precursor Protein.

**Endotoxin**: < 10 EU/mg of the protein as determined by the LAL method

**Formulation**: Lyophilized from a 0.2 μm filtered solution in PBS with 5% Trehalose and 5% Mannitol.

**Purity**: > 90% as determined by reducing SDS-PAGE.

**Bio-Activity**: Not validated for activity

**Storage**: Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.

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