



## Recombinant Protein Technical Manual

**Recombinant Human APP/Protease nexin-II Protein  
(Fc Tag)(Active)**  
RPES1288

### Product Data:

**Product SKU:** RPES1288

**Size:** 10µg

**Species:** Human

**Expression host:** HEK293 Cells

**Uniprot:** NP\_958816.1

### Protein Information:

**Molecular Mass:** 101 kDa

**AP Molecular Mass:** 15060 kDa

**Tag:** C-Fc

**Bio-activity:** Measured by its ability to inhibit trypsin cleavage of a fluorogenic peptide substrate, Mca-RPKPVE-Nval-WRK(Dnp)-NH<sub>2</sub>, (R&D Systems, Catalog # ES002). The IC<sub>50</sub> value is < 1.2 nM.

**Purity:** > 95 % as determined by reducing SDS-PAGE.

**Endotoxin:** < 1.0 EU per µg as determined by the LAL method.

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

**Shipping:** This product is provided as lyophilized powder which is shipped with ice packs.

**Formulation:** Lyophilized from sterile PBS, pH 7.4

**Reconstitution:** Please refer to the printed manual for detailed information.

**Application:**

**Synonyms:** AAA;ABETA;ABPP;AD1;APPI;CTFgamma;CVAP;PN-II;PN2

## Immunogen Information:

**Sequence:** Met 1-Leu 669

## 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  $\beta$ - and  $\gamma$ -secretase to produce the amyloid- $\beta$ -protein (A $\beta$ , 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,  $\alpha$ -secretase is involved in the cleavage of APP whose product exerts anti-amyloidogenic effect and prevention of the A $\beta$  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 Abeta 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 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.