公司产品仅供科研研究实验，不得用于临床！

商品详情：

英文名称：beta-Amyloid(25-35)

中文名称：**β淀粉样肽（25-35）抗体**

别　名:beta Amyloid(25-35); beta Amyloid 25-35; beta-Amyloid 25-35; Amyloid 25-35; A4; AAA; ABETA; ABPP; AD1; Alzheimers Disease Amyloid Protein; Amyloid B; Amyloid Beta A4 Protein Precursor; Amyloid Beta; Amyloid of Aging and Alzheimer Disease; APP; APPI; B Amyloid; Beta APP; Cerebral Vascular Amyloid Peptide; CTFgamma; CVAP; PN II; PN2; PreA4; Protease nexin II; A beta; A4_HUMAN.

研究领域:免疫学 神经生物学

抗体来源:Rabbit

克隆类型:Polyclonal

交叉反应:Mouse, Rat, (predicted: Human, Chicken, Dog, Pig, Cow, Horse, Rabbit, Guinea Pig,)

产品应用:ELISA=1:5000-10000 IHC-P=1:100-500 IHC-F=1:100-500 IF=1:100-500 (石蜡切片需做抗原修复)

not yet tested in other applications.

optimal dilutions/concentrations should be determined by the end user.

理论分子量:4.4/72-83kDa

细胞定位:细胞核 细胞浆 细胞膜 细胞外基质

性　状:Liquid

浓　度:1mg/ml

免 疫 原:KLH conjugated synthetic peptide of human beta-Amyloid: 25-35/42

亚　型:IgG

纯化方法:affinity purified by Protein A

缓 冲 液:0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.

注意事项:This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

β淀粉样肽（25-35）抗体产品介绍:The cerebral and vascular plaques associated with Alzheimer's disease are mainly composed of Amyloid beta peptides. beta Amyloid is derived from cleavage of the Amyloid precursor protein and varies in length from 39 to 43 amino acids. beta Amyloid [1-40], beta Amyloid [1-42], and beta Amyloid [1-43] peptides result from cleavage of Amyloid precursor protein after residues 40, 42, and 43, respectively. The cleavage takes place by gamma-secretase during the last Amyloid precursor protein processing step. beta Amyloid [1-40], beta Amyloid [1-42], and beta Amyloid [1-43] peptides are major constituents of the plaques and tangles that occur in Alzheimer's disease. beta Amyloid antibodies and peptides have been developed as tools for elucidating the biology of Alzheimer's disease.

Functions as a cell surface receptor and performs physiological functions on the surface of neurons relevant to neurite growth,

neuronal adhesion and axonogenesis. Involved in cell mobility and transcription regulation through protein-protein interactions. Can promote transcription activation through binding to APBB1-KAT5 and inhibits Notch signaling through interaction with Numb. Couples to apoptosis-inducing pathways such as those mediated by G(O) and JIP. Inhibits G(o) alpha ATPase activity. Acts as a kinesin I membrane receptor, mediating the axonal transport of beta-secretase and presenilin 1. Involved in copper homeostasis/oxidative stress through copper ion reduction. In vitro, copper-metallated APP induces neuronal death directly or is potentiated through Cu(2+)-mediated low-density lipoprotein oxidation. Can regulate neurite outgrowth through binding to components of the extracellular matrix such as heparin and collagen I and IV. The splice isoforms that contain the BPTI domain possess protease inhibitor activity. Induces a AGER-dependent pathway that involves activation of p38 MAPK, resulting in internalization of amyloid-beta peptide and leading to mitochondrial dysfunction in cultured cortical neurons.

Beta-amyloid peptides are lipophilic metal chelators with metal-reducing activity. Bind transient metals such as copper, zinc and iron. In vitro, can reduce Cu(2+) and Fe(3+) to Cu(+) and Fe(2+), respectively. Beta-amyloid 42 is a more effective reductant than beta-amyloid 40. Beta-amyloid peptides bind to lipoproteins and apolipoproteins E and J in the CSF and to HDL particles in plasma, inhibiting metal-catalyzed oxidation of lipoproteins. Beta-APP42 may activate mononuclear phagocytes in the brain and elicit inflammatory responses. Promotes both tau aggregation and TPK II-mediated phosphorylation. Interaction with overexpressed HADH2 leads to oxidative stress and neurotoxicity.

Appicans elicit adhesion of neural cells to the extracellular matrix and may regulate neurite outgrowth in the brai.

The gamma-CTF peptides as well as the caspase-cleaved peptides, including C31, are potent enhancers of neuronal apoptosis.

N-APP binds TNFRSF21 triggering caspase activation and degeneration of both neuronal cell bodies (via caspase-3) and axons (via caspase-6).