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 (1-28)

中文名称：**β淀粉样肽1-28单克隆抗体**

别　名:beta Amyloid(1-28); beta Amyloid 1-28; beta-Amyloid 1-28; Amyloid 1-28; P3(28); 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; Beta-amyloid protein 28.

研究领域:细胞生物 免疫学 神经生物学 细胞凋亡 Alzheimer's

抗体来源:Mouse

克隆类型:Monoclonal

克 隆 号3C8

交叉反应:(predicted: Human, Mouse, Rat,)

产品应用:ELISA=1:5000-10000

not yet tested in other applications.

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

理论分子量:3kDa

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 derived from human beta-Amyloid: 1-28/42 <Cytoplasmic>

亚　型:IgG

纯化方法:affinity purified by Protein A

保存条件:Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.

β淀粉样肽1-28单克隆抗体注意事项:This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

Function:

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 (By similarity). 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 brain. 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).