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商品详情:

英文名称: beta Amyloid 1-16

中文名称: **β淀粉样肽1-16/Aβ1-16 抗体**

别名: β-Amyloid 1-16; beta Amyloid(1-16); beta-Amyloid 1-16; A4; B Amyloid 1-16; A4_HUMAN; 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; Amyloid 1-16.

研究领域: 细胞生物 免疫学 神经生物学 信号转导 细胞凋亡

抗体来源: Rabbit

克隆类型: Polyclonal

交叉反应: (predicted: 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: 4.3kDa

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 rat beta-Amyloid: 1-16/42 <Extracellular>

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1-16/Aβ1-16 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.

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