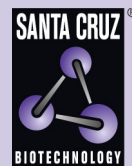


apoE (M-20): sc-6384



The Power to Question

BACKGROUND

Apolipoprotein-E (apoE) is a protein component of plasma lipoproteins that mediates the binding, internalization and catabolism of lipoprotein particles. It can serve as a ligand for several lipoprotein receptors, including the LDL (apoB/E) receptor and the hepatic apoE (chylomicron remnant) receptor. apoE is produced in most organs and occurs in all plasma lipoprotein fractions, constituting 10-20% of VLDL (very low density lipoprotein) and 1-2% of HDL (high density lipoprotein). Three major isoforms of apoE have been described in human (E2, E3 and E4) which differ by one to two amino acids. Estrogen receptor has been shown to upregulate apoE gene expression via the ER α -mediated pathway, indicating a potential role for apoE in atherosclerosis. This is consistent with studies in mice in which plasma apoE levels were raised, thereby protecting the mice from diet-induced atherosclerosis. apoE has also been shown to be a potent inhibitor of proliferation and thus may play a role in angiogenesis, tumor cell growth and metastasis.

REFERENCES

1. Mahley, R.W. 1988. Apolipoprotein E: cholesterol transport protein with expanding role in cell biology. *Science* 240: 622-630.
2. Shimano, H., et al. 1992. Overexpression of apolipoprotein E in transgenic mice: marked reduction in plasma lipoproteins except high density lipoprotein and resistance against diet-induced hypercholesterolemia. *Proc. Natl. Acad. Sci. USA* 89: 1750-1754.

CHROMOSOMAL LOCATION

Genetic locus: Apoe (mouse) mapping to 7 A3.

SOURCE

apoE (M-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of apoE of mouse origin.

APPLICATIONS

apoE (M-20) is recommended for detection of apoE of mouse and rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for apoE siRNA (m): sc-29709, apoE shRNA Plasmid (m): sc-29709-SH and apoE shRNA (m) Lentiviral Particles: sc-29709-V.

Molecular Weight of apoE: 36 kDa.

Positive Controls: rat liver extract: sc-2395, mouse liver extract: sc-2256 or mouse PBL whole cell lysate.

STORAGE

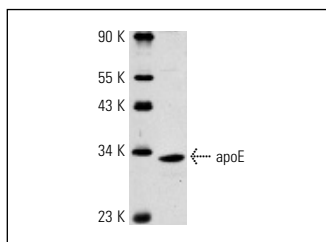
Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

PRODUCT

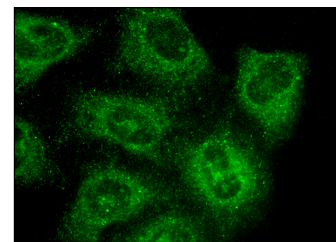
Each vial contains 200 μ g IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-6384 P, (100 μ g peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

DATA



apoE (M-20): sc-6384. Western blot analysis of apoE expression in rat liver extract.



apoE (M-20): sc-6384. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Naidu, A., et al. 2001. Conversion of brain apolipoprotein E to an insoluble form in a mouse model of Alzheimer disease. *Neuroreport*. 12: 1265-1270.
2. Wahrle, S.E., et al. 2008. Overexpression of ABCA1 reduces amyloid deposition in the PDAPP mouse model of Alzheimer disease. *J. Clin. Invest.* 118: 671-682.
3. Burgess, B.L., et al. 2008. ABCG1 influences the brain cholesterol biosynthetic pathway but does not affect amyloid precursor protein or apolipoprotein E metabolism *in vivo*. *J. Lipid Res.* 49: 1254-1267.
4. Nishida, Y., et al. 2009. Depletion of vitamin E increases amyloid β accumulation by decreasing its clearances from brain and blood in a mouse model of Alzheimer disease. *J. Biol. Chem.* 284: 33400-33408.
5. Wu, K., et al. 2009. Altered expression of genes functioning in lipid homeostasis is associated with lipid deposition in NOD mouse lacrimal gland. *Exp. Eye Res.* 89: 319-332.
6. Chua, C.C., et al. 2010. Altered apolipoprotein E glycosylation is associated with A β (42) accumulation in an animal model of Niemann-Pick Type C disease. *J. Neurochem.* 112: 1619-1626.
7. Fitz, N.F., et al. 2010. Liver X receptor agonist treatment ameliorates amyloid pathology and memory deficits caused by high-fat diet in APP23 mice. *J. Neurosci.* 30: 6862-6872.
8. Terwel, D., et al. 2011. Critical role of astroglial apolipoprotein E and liver X receptor- α expression for microglial A β phagocytosis. *J. Neurosci.* 31: 7049-7059.
9. Rolyan, H., et al. 2011. Telomere shortening reduces Alzheimer's disease amyloid pathology in mice. *Brain* 134: 2044-2056.

RESEARCH USE

For research use only, not for use in diagnostic procedures.