SANTA CRUZ BIOTECHNOLOGY, INC.

AChE (E-19): sc-6432

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Acetylcholinesterase (AChE) hydrolyzes acetylcholine at synaptic junctions. Alternative mRNA splicing gives rise to three forms of AChE. The T form, also known as the asymmetric form, is soluble and is present in synapses. The H form is also known as the globular form and is present on the outer surfaces of cell membranes. The R form is not known to be a functional species. AChE globular form subunits are GPI-anchored to cell membranes and asymmetric subunits are anchored to basal lamina components by a collagen tail. The catalytic sununits of AChE are oligomers composed of disulfide-linked homodimers. The loss of AChE from cholinergic and non-cholinergic neurons in the brain is seen in patients with Alzheimer's disease. However, AChE activity is increased around amyloid plaques, which may be due to a disturbance in calcium homeostasis involving the opening of L-type voltage-dependent calcium channels.

CHROMOSOMAL LOCATION

Genetic locus: Ache (mouse) mapping to 5 G2.

SOURCE

AChE (E-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of AChE of mouse origin.

PRODUCT

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

Blocking peptide available for competition studies, sc-6432 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

AChE (E-19) is recommended for detection of AChE of mouse and rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg 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 AChE siRNA (m): sc-29629, AChE shRNA Plasmid (m): sc-29629-SH and AChE shRNA (m) Lentiviral Particles: sc-29629-V.

Molecular Weight (predicted) of AChE: 68 kDa.

Molecular Weight (average of observed) of AChE: 71 kDa.

Positive Controls: C6 whole cell lysate: sc-364373, PC-12 cell lysate: sc-2250 or PC-12 + NGF cell lysate: sc-3808.

RESEARCH USE

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

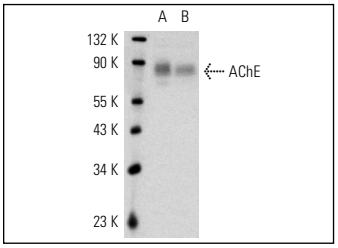
PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.

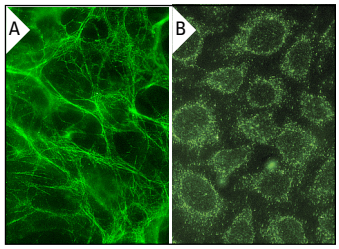
STORAGE

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

DATA



AChE (E-19): sc-6432. Western blot analysis of AChE expression in untreated (**A**) and NGF-treated PC-12 (**B**) whole cell lysates.



AChE (E-19): sc-6432. Immunofluorescence staining of methanol-fixed NIH/3T3 cells showing membrane localization (**A**). Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization (**B**).

SELECT PRODUCT CITATIONS

1. Sternfeld, M., et al. 2000. Excess "read-through" acetylcholinesterase attenuates but the "synaptic" variant intensifies neurodeterioration correlates. Proc. Natl. Acad. Sci. USA 97: 8647-8652.
2. Santos, S.C., et al. 2007. Expression and subcellular localization of a novel nuclear acetylcholinesterase protein. J. Biol. Chem. 282: 25597-25603.
3. Silveyra, M.X., et al. 2008. Presenilin 1 interacts with acetylcholinesterase and alters its enzymatic activity and glycosylation. Mol. Cell. Biol. 28: 2908-2919.
4. Noureddine, H., et al. 2008. Acetylcholinesterase associates differently with its anchoring proteins ColQ and PRiMA. J. Biol. Chem. 283: 20722-20732.
5. Sinis, N., et al. 2009. Electrical stimulation of paralyzed vibrissal muscles reduces endplate reinnervation and does not promote motor recovery after facial nerve repair in rats. Ann. Anat. 191: 356-370.
6 Halliday, A.C., et al. 2010. Evaluation of a technique to identify acetyl-cholinesterase C-terminal peptides in human serum samples. Chem. Biol. Interact. 187: 110-114.
7. Tsim, K.W., et al. 2010. Expression and localization of PRiMA-linked globular form acetylcholinesterase in vertebrate neuromuscular junctions. J. Mol. Neurosci. 40: 40-46.
8. Figueiró, M., et al. 2010. Acetylcholinesterase inhibition in cognition-relevant brain areas of mice treated with a nootropic Amazonian herbal (Marapuama). Phytomedicine 17: 956-962.
9. Petrov, K.A., et al. 2011. Different sensitivities of rat skeletal muscles and brain to novel anti-cholinesterase agents, alkylammonium derivatives of 6-methyluracil (ADEMS). Br. J. Pharmacol. 163: 732-744.
10. Xie, J., et al. 2011. Induction of a 55 kDa acetylcholinesterase protein during apoptosis and its negative regulation by the Akt pathway. J. Mol. Cell. Biol. 3: 250-259.