Human Acetylcholinesterase/ACHE Antibody

Monoclonal Mouse IgG_1 Clone # 872044

Catalog Number: MAB7574

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human ACHE in direct ELISAs.
Source	Monoclonal Mouse IgG_1 Clone # 872044
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese hamster ovary cell line CHO-derived recombinant human ACHE Met1-Leu614 Accession # P22303
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 μm filtered solution in PBS.

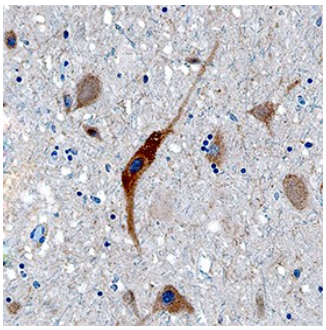
APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

	Recommended Concentration	Sample
Immunohistochemistry	8-25 µg/mL	See Below

DATA

Immunohistochemistry



Acetylcholinesterase/ACHE in Human Brain. Acetylcholinesterase/ACHE was detected in immersion fixed paraffin-embedded sections of human brain (substantia nigra) using Mouse Anti-Human Acetylcholinesterase/ACHE Monoclonal Antibody (Catalog # MAB7574) at 15 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Mouse HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS002) and counterstained with hematoxylin (blue). Specific staining was localized to neurons. View our protocol for Chromogenic IHC Staining of Paraffin-embedded Tissue Sections.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

The classical role of ACHE is to terminate cholinergic neurotransmission by hydrolysis of acetylcholine (ACH) (1). ACHE is thought to be involved in the pathology of Alzheimer's disease (AD) by accelerating the assembly of Aβ peptides into fibrillar species through forming complexes with Aβ via the peripheral anionic site on ACHE. ACHE inhibitors have been used to delay symptoms of AD patients by virtue of their ability to enhance ACH availability, as well as reduce amyloidogenesis and subsequent neurotoxicity (2). Its involvement in the cholinergic anti-inflammatory pathway connects ACHE with a possible marker of low-grade systemic inflammation in obesity, hypertension, coronary heart disease, and AD (3). Alternative splicing produces three isoforms: an amphipathic form that exists as both monomeric and multimeric forms, a soluble monomeric form lacking the cysteine residue near the C-terminus, and a GPI-anchored dimeric form found in the membranes of erythrocytes (1). The recombinant human ACHE (rhACHE) was expressed as the amphipathic form that consists of soluble monomer and multimeric forms.

References:

1. Grisaru, D. *et al*. (1999) Eur. J. Biochem, **264**:672.
2. Campbell, V. A. and Gowran, A. (2007) Br. J. Pharm. **152**:655.
3. Das, U. N. (2007) Med. Sci. Monit. **13**:RA214.

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