

DESCRIPTION

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| Species Reactivity | Human/Mouse |
| Specificity | Detects human and mouse BACE-1 in direct ELISAs and human BACE-1 in Western blots. In Western blots, no cross-reactivity with recombinant human (rh) BACE-2, recombinant mouse (rm) BACE-2, rhADAM8, rmADAM9, rmADAM10, rhADAM15, or rhTACE is observed. |
| Source | Monoclonal Mouse IgG ₁ Clone # 137612 |
| Purification | Protein A or G purified from hybridoma culture supernatant |
| Immunogen | Mouse myeloma cell line NS0-derived recombinant human BACE-1 Thr22-Tyr460 Accession # P56817 |
| Conjugate | Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm |
| Formulation | Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions. |

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 |
|---|---------------------------------|---|
| Intracellular Staining by Flow Cytometry | 0.25-1 µg/10 ⁶ cells | Jurkat human acute T cell leukemia cell line fixed with paraformaldehyde and permeabilized with saponin |

PREPARATION AND STORAGE

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| Shipping | The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below. |
| Stability & Storage | Protect from light. Do not freeze. <ul style="list-style-type: none"> ● 12 months from date of receipt, 2 to 8 °C as supplied. |

BACKGROUND

BACE-1 (beta-site APP cleaving enzyme-1) is an aspartic protease and an integral membrane protein (1, 2). It is the major β secretase, and together with the γ secretase, is responsible for generating the amyloid β peptide (Aβ) from the amyloid precursor protein (APP) (3, 4). Because Aβ is a major component of amyloid plaques, BACE-1 has been implicated in the onset and/or progression of Alzheimer's disease. High levels of BACE-1 activity are sufficient to elicit neurodegeneration and neurological decline in vivo, indicating that inhibiting BACE-1 may block not only Aβ-dependent but also Aβ-independent pathogenic mechanisms (5). In addition to APP, BACE-1 also cleaves APP-like proteins 1 and 2, the cell adhesion protein P-selectin glycoprotein ligand-1 and β-galactoside α2,6-sialyltransferase, implying that BACE-1 may have additional functions involving the ectodomain shedding of membrane proteins (6-8).

References:

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