

DESCRIPTION

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 700 Excitation Wavelength: 675-700 nm Emission Wavelength: 723 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

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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