

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Insulysin/IDE in direct ELISAs and Western blots.
Source	Monoclonal Mouse IgG ₁ Clone # 334501
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>S. frugiperda</i> insect ovarian cell line Sf 21-derived recombinant human Insulysin/IDE Met42-Leu1019 Accession # P14735
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	HeLa cells 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. ● 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

Insulysin, or insulin-degrading enzyme (IDE), is a zinc metallopeptidase of the inverzincin family. IDE is primarily located in the cytosol, but has been detected as a secreted enzyme and associated with the plasma membrane as well (1). The enzyme is expressed in many tissues, with the highest levels in liver, kidney, brain, and testis (2). IDE hydrolyzes a variety of regulatory peptides, including insulin, glucagon, atrial natriuretic factor, and transforming growth factor- α *in vitro* (1). In addition, IDE has been shown to degrade the amyloid β (A β) peptide, which polymerizes into the plaques associated with Alzheimer's disease (3). Deficiencies in IDE activity may contribute to the pathogenesis of type 2 diabetes mellitus (DM2) and Alzheimer's disease. The IDE region of human chromosome 10q has been genetically linked to DM2 (4). When the IDE gene was specifically disrupted in mice, IDE -/- animals developed hyperinsulinemia and glucose intolerance, characteristics of DM2 (5). The IDE -/- mice were also shown to have a significant decrease in A β degradation in the brain, resulting in increased cerebral accumulation of A β peptide. This *in vivo* evidence is consistent with the hypotheses that IDE is important for the degradation of insulin in cells and for the clearance of A β peptide in the brain.

References:

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