

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Neprilysin/CD10 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2B} Clone # 715823
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese hamster ovary cell line CHO-derived recombinant human Neprilysin/CD10 Tyr52-Trp750 Accession # P08473
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *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.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunohistochemistry	Optimal dilution of this antibody should be experimentally determined.

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

Neprilysin/CD10, also known as NEP and neutral endopeptidase 24.11, is a zinc metallopeptidase expressed at the cell surface of a variety of cells. The enzyme functions both as an endopeptidase with a thermolysin-like specificity and as a dipeptidylcarboxypeptidase. NEP has been shown to be involved in the degradation of enkephalins in the mammalian brain and the inactivation of circulating atrial natriuretic peptide (1, 2). NEP has also been identified as the common acute lymphocytic leukemia antigen (CALLA), and is expressed on the surface of lymphocytes in some disease states (3, 4). These and other observations have resulted in considerable interest in NEP as a target for analgesics and antihypertensive drugs. NEP is also a major degrading enzyme of amyloid β peptide (Aβ) in the brain, indicating that down-regulation of NEP activity, which could be caused by aging, can contribute to the development of Alzheimer's disease by promoting Aβ accumulation (5).

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