

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Cathepsin C/DPPI in direct ELISAs and Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human pro Cathepsin C/DPPI Asp25-Leu463 Accession # P53634
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 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

Cathepsin C, also known as dipeptidyl-peptidase I (DPPI), is a cysteine protease of the papain family (1). Cathepsin C sequentially removes dipeptides from the free N-termini of proteins and peptides. It has broad specificity except that it does not cleave a basic amino acid (Arg or Lys) in the N-terminal position or Pro on either side of the scissile bond. It requires halide ions for activity. The pro form contains a pro peptide and a catalytic region, which can be further processed into heavy/α and light/β chains that are linked by a disulfide bond. It is broadly distributed. Cathepsin C plays a role in the lysosomal degradation. It also functions as a key enzyme in the activation of granule serine proteases in cytotoxic T lymphocytes and natural killer cells (granzymes A and B), mast cells (tryptase and chymase), and neutrophils (Cathepsin G and elastase) by removing their N-terminal activation dipeptides (2). Loss of function mutations in the Cathepsin C gene result in periodontal disease and palmoplantar keratosis (3).

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