

DESCRIPTION

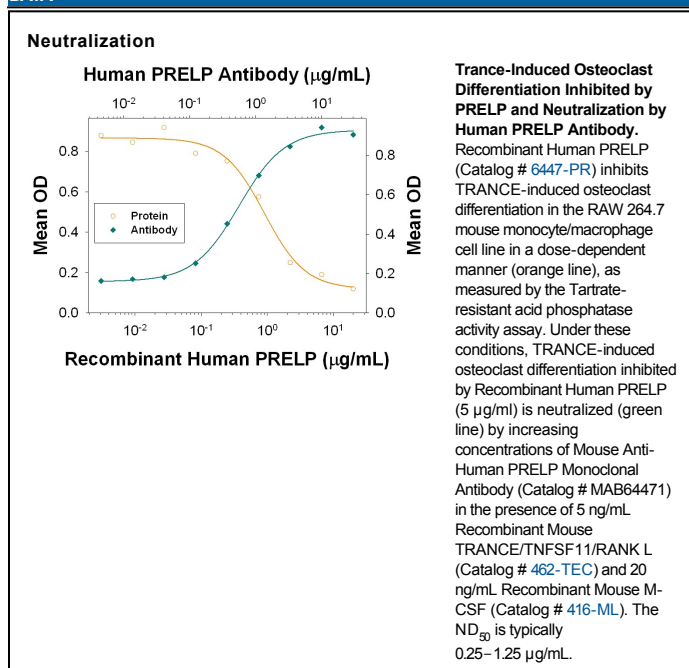
Species Reactivity	Human
Specificity	Detects human PRELP in direct ELISA and Western blot.
Source	Monoclonal Mouse IgG _{2A} Clone # 754636
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese hamster ovary cell line CHO-derived recombinant human PRELP Gln21-Ile382 Accession # P51888
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

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.

Neutralization	Measured by its ability to neutralize PRELP inhibition of TRANCE-induced osteoclast differentiation in the RAW 264.7 mouse monocyte/macrophage cell line. Rucci, N. <i>et al.</i> (2009) <i>Journal of Cell Biology</i> 187 : 669. The Neutralization Dose (ND ₅₀) is typically 0.25-1.25 µg/ml in the presence of 5 µg/mL Recombinant Human PRELP, 5 ng/mL Recombinant Mouse TRANCE/TNFSF11/RANK L, and 20 ng/mL Recombinant Mouse M-CSF.
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DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

PRELP (Proline Arginine-rich End Leucine-rich repeat Protein; also Prolargin) is a 55-62 kDa secreted glycoprotein that belongs to the small leucine-rich proteoglycan (SLRP) superfamily of extracellular matrix (ECM) molecules (1-4). Within this family, it is considered a class II member, implying that it is unlikely to form dimeric structures (3). PRELP is synthesized as a 382 amino acid (aa) precursor that contains a 20 aa signal sequence plus a 362 aa mature region (1, 5). Like other SLRPs, PRELP contains an N-terminal extension (aa 72-107) coupled to multiple Leu-rich repeats (LRRs) (aa 95-382) (6). Unlike other SLRPs, PRELP does not contain any proteoglycan chains, and its N-terminal extension is highly basic in charge. The N-terminus reportedly binds to negatively-charged heparin/heparin-sulfate, chondroitin sulfate, and Gram⁻ bacterial cell walls, while the LRR region participates in protein-protein interactions (7-9). Although PRELP is known to be synthesized by only a few cell types, including osteoblasts, skeletal muscle and chondrocytes, its expression is likely to be more widespread, given its presence in the basement membrane (BM) of Bowman's capsule, epididymal epithelium and the stratified squamous epithelium of the skin (1, 10, 11). The dual binding profile of PRELP is key to its function. In cartilage, PRELP likely links chondrocyte cell membrane heparin sulfate (HS) chains to endogenous type II collagen. Within the context of the BM, PRELP likely plays an anchoring role. The BM is composed of type IV collagen and laminin, linked together by nidogen. BM Perlecan reinforces this linkage by binding to all three components. PRELP, on the edge of the BM, can bind to free perlecan HS chains (via its N-terminus), and to underlying type I collagen (via its LRRs), thus forming an anchor for the BM (11). Notably, the N-terminus appears to do more than simply provide part of a linkage mechanism. In bone, osteoblast secreted PRELP is hypothesized to undergo proteolysis by enzymes such as LysC and glutamyl endopeptidase. This will generate 40-75 aa N-terminal fragments that can bind to chondroitin sulfate adducts that exist on the surface of pre-fusion osteoclast precursors. Following binding, PRELP is internalized, complexed to annexin-II, and translocated to the nucleus, where it interacts with NFκBp65 to block osteoclast maturation (8). In tissue, PRELP may also undergo proteolytic processing during inflammation to release an N-terminal fragment containing aa 21-42 of the precursor (7). This sequence has been shown to possess potent antimicrobial activity by creating pores in bacterial cell walls. Mature human PRELP shares 91% aa identity with mouse PRELP (10).

References:

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