Human MEPE/OF45 Alexa Fluor® 350-conjugated Antibody

Antigen Affinity-purified Polyclonal Goat IgG
Catalog Number: AF3140U
100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human MEPE/OF45 in direct ELISAs and Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human MEPE/OF45 Pro18-Asp525 Accession # Q9NQ76
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 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.*

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

MEPE (matrix extracellular phosphoglycoprotein), known as OF45 in mouse and rat, is a 55 kDa member of the SIBLING protein family. MEPE is primarily expressed in bone and dentin, where it regulates the mineralization of those tissues (1-3). The human MEPE cDNA encodes a 525 amino acid (aa) precursor that includes a 17 aa signal sequence. MEPE contains multiple consensus sites for post-translational modifications, including N-linked glycosylation, N-myristoylation, glycosaminoglycan attachment, and phosphorylation by a variety of kinases. MEPE also contains several putative proteolytic cleavage sites and one integrin-binding RGD motif (3, 4). There is therefore considerable potential for post-translational regulation of MEPE function and its degradation products. MEPE is secreted by osteoblasts and dental pulp stem cells during the mineralization process (5-7) and also by nonmineralizing tissues including epithelial cells in the renal proximal tubule and salivary duct (8, 9). MEPE has an inhibitory function in bone formation, (5) although a peptide corresponding to aa 242-264 stimulates new bone formation and the proliferation of osteoblasts and dental pulp stem cells (10, 11). MEPE contains one C-terminal ASARM motif common to SIBLING proteins. Similar to intact MEPE, the ASARM peptide inhibits bone mineralization and plays a central role in the phosphaturia and reduced mineralization of X-linked hypophosphatemic rickets (HYP) and tumor-induced osteomalacia (TIO) (12, 13). The zinc metalloprotease Phex binds directly to MEPE via the ASARM motif and prevents ASARM cleavage (13, 14). Multiple inactivating mutations in Phex are found in HYP and TIO and result in the increased liberation of ASARM peptide (15). Both MEPE and ASARM peptide are elevated in these disorders of mineralization and phosphate metabolism (12).

PRODUCT SPECIFIC NOTICES

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