

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse Dkk-2 in Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse Dkk-2 Ser26-Ile259 Accession # Q9QYZ8
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

Dickkopf related protein 2 (Dkk-2) is a member of the Dickkopf family of secreted Wnt modulators (1-3). Dkk proteins contain a signal peptide and two conserved cysteine-rich domains that are separated by a linker region. The second cysteine-rich domain, which shows a configuration of cysteines conserved in prokineticin and colipase families, mediates Dkk-2 binding activities (2-4). The 226 amino acid (aa), ~35 kDa mature mouse Dkk-2 shares 41% and 34% aa identity with mouse Dkk-1 and Dkk-4, respectively. It also shares 99%, 96%, 96%, 96% and 94% aa identity with rat, human, canine, equine and bovine Dkk-2, respectively, and can activate the canonical Wnt signaling pathway in *Xenopus* embryos (5). Dkk proteins modify Wnt engagement of a receptor complex composed of a Frizzled protein and a low-density lipoprotein receptor-related protein, either LRP5 or LRP6 (3). When LRP6 is overexpressed, direct high-affinity binding of Dkk-2 to LRP can enhance canonical Wnt signaling (6-8). However, when Dkk-2 and LRP6 form a ternary complex with Kremen2, Wnt signaling is inhibited due to internalization of Dkk-2/LRP6/Krm2 complexes (9, 10). Thus, depending on the cellular context, Dkk-2 can either activate or inhibit canonical Wnt signaling (3). In contrast, binding of Dkk-1 or Dkk-4 to LRP is consistently antagonistic (3). Dkk proteins are expressed in mesenchymal tissues and control epithelial transformations. Dkk-2 expression has been studied most in bone and eye. Mouse Dkk-1 or Dkk-2 deficiencies have opposite effects on bone homeostasis, despite downregulating Wnt antagonism in both cases (11, 12). Dkk-2 expression is induced by Wnts in bone, and is thought to enhance bone density by promoting terminal differentiation of osteoblasts and mineral deposition (11). In contrast, Dkk-1 negatively regulates late osteoblast proliferation, which limits bone density (12). Dkk-2-deficient mice are blind due to faulty differentiation of corneal epithelium (13).

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