Human DDR2 Biotinylated Antibody

Antigen Affinity-purified Polyclonal Goat IgG
Catalog Number: BAF2538

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human DDR2 in Western blots. In Western blots, less than 5% cross-reactivity with recombinant human DDR1 is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human DDR2 Gln24-Arg399 Accession # Q16832
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

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.*

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Human DDR2 (Catalog # 2538-DR)

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 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

DDR2, also known as TYR010 and TKT, is a widely expressed 130 kDa type I transmembrane glycoprotein belonging to the discoidin-like domain containing subfamily of receptor tyrosine kinases (1). Mature human DDR2 consists of a 378 amino acid (aa) extracellular domain (ECD) that includes the discoidin-like domain, a 22 aa transmembrane segment, and a 434 aa cytoplasmic domain that includes the kinase domain (2). Within the ECD, human DDR2 shares 53% aa sequence identity with DDR1 and 97% aa sequence identity with mouse DDR2. The discoidin-like domain mediates DDR2 interactions with collagens I, III, and X (3-5). Collagens II and V are less efficacious ligands (3). DDR2 selectively recognizes the triple helical structure of collagen compared to monomeric or denatured collagen (3, 5, 6). Within collagen II, the D2 period is required for DDR2 binding, and the D1 period is additionally required to trigger DDR2 autophosphorylation (6). The ECD of DDR2 exists as a non-covalent dimer in solution, and dimerization of the receptor greatly enhances collagen binding (4, 7). DDR2 interaction with collagen I inhibits collagen fibrillogenesis and alters collagen fiber morphology (7). Ligand binding induces DDR2 autophosphorylation in the cytoplasmic domain (3, 5, 8), which promotes associations with Shc and Src (9). In addition to the above mechanism, DDR2 exhibits a distinct interaction with collagen X. A region other than the discoidin-like domain of DDR2 recognizes the non-helical NC1 domain of collagen X, and this interaction does not lead to receptor autophosphorylation (5). Activation of DDR2 by collagen induces upregulation of MMP-1, -2, and -13 as well as DDR2 itself (3, 8, 10). DDR2 is implicated in collagenous matrix destruction and cell invasiveness (8, 10). DDR2 is also upregulated in several pathological conditions, including hepatic fibrosis following injury, rheumatoid and osteoarthritis, and smooth muscle cell hyperplasia (8, 10-12).

References:

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