

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human DDR2 in direct ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant human DDR1 is observed.
Source	Monoclonal Mouse IgG _{2B} Clone # 290814
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human DDR2 Gln24-Arg399 Accession # Q16832
Conjugate	Alexa Fluor 532 Excitation Wavelength: 534 nm Emission Wavelength: 553 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

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).

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