

Recombinant Protein Technical Manual

Recombinant Human BMPR2 Protein (His & Fc Tag)(Active) RPES2075

Product Data:

Species: Human

Size: 100µg

Expression host: HEK293 Cells

Uniprot: NP_001195.2

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Molecular Mass:	42 kDa
AP Molecular Mass:	60-65 kDa
Tag:	C-His & Fc
Bio-activity:	Measured by its binding ability in a functional ELISA.2. Immobilized human BMPR-II-Fc at 10 μ g/mL (100 μ l/well) can bind biotinylated human BMP2-Fc, The EC50 of biotinylated human BMP2-Fc (Cat:PKSH031985) is 8010 ng/mL.
Purity:	> 90 % as determined by reducing SDS-PAGE.
Endotoxin:	< 1.0 EU per μg as determined by the LAL method.
Storage:	Lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping:	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation:	Lyophilized from sterile PBS, pH 7.4
Reconstitution:	Please refer to the printed manual for detailed information.
Application:	Functional ELISA
Synonyms:	BMPR-II;BMPR3;BMR2;BRK-3;POVD1;PPH1;T-ALK

Immunogen Information:

Sequence: Met 1-Ile 151

Background:

The bone morphogenetic protein type II receptor (BMPR-II, or BMPR2), a receptor for the transforming growth factor (TGF)-beta/bone morphogenetic protein (BMP) superfamily. Reduced expression or function of BMPR2 signaling leads to exaggerated TGF-beta signaling and altered cellular responses to TGF-beta. In endothelial cells, BMPR2 mutation increases the susceptibility of cells to apoptosis. BMPR2 transduces BMP signals by forming heteromeric complexes with and phosphorylating BMP type I receptors. The intracellular domain of BMPR2 is both necessary and sufficient for receptor complex interaction. It had been identified that BMPR2 plays a key role in cell growth. Its mutations lead to hereditary pulmonary hypertension, and knockout of Bmpr-II results in early embryonic lethality. The C-terminal tail of BMPR2 provides binding sites for a number of regulatory proteins that may initiate Smad-independent signalling. BMPR2 mutations were predicted to alter the BMP and TGF-b1/SMAD signalling pathways, resulting in proliferation rather than apoptosis of vascular cells, and greatly increase the risk of developing severe pulmonary arterial hypertension. BMPR2 gene result in familial Primary pulmonary hypertension (PPH) transmitted as an autosomal dominant trait, albeit with low penetrance. Heterozygous germline mutations of BMPR2 gene have been identified in patients with familial and sporadic PPH, indicating that BMPR2 may contribute to the maintenance of normal pulmonary vascular structure and function. Tctex, a light chain of the motor complex dynein, interacts with the cytoplasmic domain of BMPR2 and demonstrate that Tctex is phosphorylated by BMPR-II, a function disrupted by PPH disease causing mutations within exon 12. BMPR2 and Tctex co-localize to endothelium and smooth muscle within the media of pulmonary arterioles, key sites of vascular remodelling in PPH.