

BMPR-II Protein, Human (HEK293, His-Fc)

Cat. No.:	HY-P72855
Synonyms:	Bone morphogenetic protein receptor type-2; BMPR-2; BMPR-II; PPH1
Species:	Human
Source:	HEK293
Accession:	Q13873 (S27-I151)
Gene ID:	659
Molecular Weight:	60-65 kDa

PROPERTIES

AA Sequence	<pre> MTSSLQRPWR VPWLPWTILL VSTAAASQNQ ERLCAFKDPY QQDLGIGESR ISHENGITILC SKGSTCYGLW EKSKGDINLV KQGCWSHIGD PQECHYEECV VTTTPPSIQN GTYRFCC CST DLCNVNFTEN FPPPDTTPLS PPHSFNRDET I </pre>
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 µm filtered solution of PBS, pH 7.4. Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/µg, determined by LAL method.
Reconstitution	It is not recommended to reconstitute to a concentration less than 100 µg/mL in ddH ₂ O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	<p>BMP Type II Receptor (BMPR2) is a member of the bone morphogenetic protein (BMP) receptor family of transmembrane serine/threonine kinases. BMPs are involved in endochondral bone formation and embryogenesis, transducing signals through the formation of heteromeric complexes of 2 types BMP receptors: type I receptors (50-55 kD) and type II receptors (70-80 kD). Type II receptors phosphorylate and activate type I receptors which autophosphorylate, then bind and activate SMAD transcriptional regulators^{[1][2]}. BMPR2 is highly expressed in the heart and liver, and involves in osteogenesis and cell differentiation, essential for embryogenesis, development, and adult tissue homeostasis. The BMPR2 pathway inhibits smooth muscle cell (SMC) proliferation within the pulmonary circulation, primarily within the small pulmonary arterioles. When mutated, BMPR2 is associated with an increased susceptibility to develop pulmonary arterial hypertension (PAH)^[1].</p>
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BMPR2 is essential for tubulin stability, as recent studies have shown that BMP2 regulates cell survival signaling events in cancer cells independent of the BMP type 1 receptor (BMPR1) or the Smad-1/5 transcription factor. Mutations in BMPR2 trafficking proteins leads to overactive BMP signaling, which leads to neurological diseases caused by BMPR2 stabilization of the microtubules. Inhibition of BMPR2 destabilizes the microtubules, thus leads to lysosomes activation in promoting cell death progress of cancer cells^[3]. Particularly, there is strong correlation between BMPR2 promoter DNA methylation and the severity of valvular heart disease (VHD), which makes BMPR2 serve as good biomarkers of VHD. Meanwhile, DNA methylation may cause PAH through deregulation of BMP signaling and increased apoptosis^[4]. The sequences of BMPR2 protein are conserved among different species, and the sequence of human shares a high similarity with rat (96.15%) and mouse (96.63%), respectively.

REFERENCES

- [1]. Xu B, et al. The role of TGF- β or BMPR2 signaling pathway-related miRNA in pulmonary arterial hypertension and systemic sclerosis. *Arthritis Res Ther*. 2021 Nov 25;23(1):288.
- [2]. ten Dijke P, et al. Signaling via hetero-oligomeric complexes of type I and type II serine/threonine kinase receptors. *Curr Opin Cell Biol*. 1996 Apr;8(2):139-45.
- [3]. Mondal A, et al. Bone morphogenetic protein receptor 2 inhibition destabilizes microtubules promoting the activation of lysosomes and cell death of lung cancer cells. *Cell Commun Signal*. 2021 Sep 25;19(1):97.
- [4]. Li N, et al. BMPR2 promoter methylation and its expression in valvular heart disease complicated with pulmonary artery hypertension. *Aging (Albany NY)*. 2021 Nov 18;13(22):24580-24604.
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