GDF-15 Protein, Mouse (Biotinylated, HEK293, Fc)

Cat. No.:	HY-P77946
Synonyms:	GDF-15; MIC-1; NAG-1; PDF; PLAB; PTGFB; GDF15; MIC1; RG-1; Placental TGF-beta; PTGF-beta; PTGFBPTGF-beta
Species:	Mouse
Source:	HEK293
Accession:	Q9Z0J7 (S189-A303)
Gene ID:	23886
Molecular Weight:	40-50 kDa

PROPERTIES

AA Sequence	S A H A H P R D S C P L G P G R C C H L E T V Q A T L E D L G W S D W V L S P R Q L Q L S M C V G E C P H L Y R S A N T H A Q I K A R L H G L Q P D K V P A P C C V P S S Y T P V V L M H R T D S G V S L Q T Y D D L V A R G C H C A
Biological Activity	Immobilized Mouse GFRAL,His Tag at 2 μg/mL (100μl/Well) on the plate. Dose response curve for Bietinylated Mouse GDF15, hFc Tag with the EC_{50} ≤ 8 ng/mL dletermined by ELISA.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.22 μm filtered solution of PBS, pH 7.4. Normally 8% trehalose is added as protectant before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
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	Growth differentiation factor 15 (GDF-15) is a polypeptide hormone belonging to the transforming growth factor β (TGF-β) superfamily. GDF-15 was highly expressed in placenta, low in prostate and colon, and to some extent in kidney. So GDF-15 is also known as placental transforming growth factor PGF-β, placental bone morphogenetic protein PLAB, and prostatus-derived factor PDF. GDF-15 has a wide range of biological functions in physiology and pathology, especially in aging, cancer, and metabolic processes. GDF-15 is initially stored in the extracellular matrix (ECM), where it undergoes proteolytic hydrolysis upon external stimulation to form an active form that is quickly secreted into circulation. In mouse

cardiomyocytes, the cleavage process of GDF-15 may be catalyzed by the enzymes of the PCSK family, resulting in a mature dimer form. Upstream of the GDF15 promoter site, there are binding sites for various transcription factors, including specific protein 1 (Sp1), early growth response protein 1 (Egr-1), p53 and COUP transcription factor 1 (COUP-TF1). The receptor of GDF-15 is alpha-like protein (GFRAL), a receptor of the glial cell derived neurotrophic factor (GDNF) family. The GFRAL-GDF15 complex binds to the tyrosine kinase co-receptor RET, leading to RET phosphorylation. Subsequently, GFRAL-GDF15 continued to activate the intracellular signaling pathways of AKT, ERK1/2, and phospholipase C (PLCγ), but not the SMAD pathway. GDF-15 is overexpressed during and after many pathological states such as tissue injury and inflammation. The stimulating factors that contribute to this result include oxidized low-density lipoprotein (oxLDL), cytokines, and growth factors such as IL-1β, TNF-α, angiotensin II, macrophage colony-stimulating factor M-CSF, and TGFβ. GDF-15, also known as the NSAIDS drug activator gene NAG-1, may play an anti-inflammatory role by inhibiting macrophage activation. GDF-15 also inhibits the activity of NFκB or the expression of several cytokines, including interferon (IFN-γ), interleukin-6 (IL-6), monocyte chemotactic protein-1 (MCP-1), and tumor necrosis factor-α (TNF-α). GDF-15 has significant resistance to endotoxin-induced sepsis caused by acute kidney injury (AKI) and myocardial dysfunction. GDF-15 also appears to promote tumor growth in the later stages of malignancy. Elevated serum GDF15 levels have been reported as potential biomarkers for cancer progression, including breast, colon, pancreatic, and prostate tumors, among others. In human and cynomolgus monkeys, the amino acid sequence similarity of GDF-15 protein was high, and the similarity rate was 91.56%. Compared with the amino acid sequences of mice and rats, the similarity of human GDF-15 was low (59.73% and 59.39%, respectively) [1].

REFERENCES

[1]. Assadi A, et al. GDF15, an update of the physiological and pathological roles it plays: a review. Pflugers Arch. 2020 Nov;472(11):1535-1546.

[2]. Breen DM, et al. GDF-15 Neutralization Alleviates Platinum-Based Chemotherapy-Induced Emesis, Anorexia, and Weight Loss in Mice and Nonhuman Primates. Cell Metab. 2020 Dec 1;32(6):938-950.e6.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA