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Produktinformation



Forschungsprodukte & Biochemikalien



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Lieferung & Zahlungsart

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Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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PRODUCT INFORMATION



sFRP1 (human, recombinant)

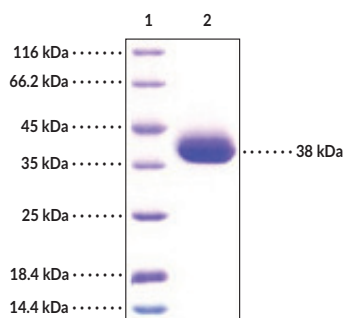
Item No. 38061

Overview and Properties

Synonyms:	SARP-2, Secreted Apoptosis-related Protein 2, Secreted Frizzled-related Protein 1
Source:	Active recombinant human C-terminal His-tagged sFRP1 expressed in HEK293 cells
Amino Acids:	32-314
Uniprot No.:	Q8N474
Molecular Weight:	34 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	≥97% estimated by SDS-PAGE
Supplied in:	Lyophilized from sterile PBS, pH 7.4
Endotoxin Testing:	<1.0 EU/μg, determined by the LAL endotoxin assay
Bioactivity:	See figures for details
Specific Activity:	5-30 μg/ml (ED ₅₀)

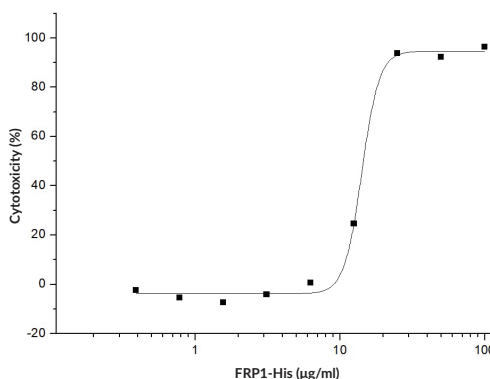
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Images



Lane 1: MW Markers
Lane 2: sFRP1 (human, recombinant)

SDS-PAGE Analysis of sFRP1 (human, recombinant). This protein has a calculated molecular weight of 34 kDa. It has an apparent molecular weight of approximately 38 kDa by SDS-PAGE under reducing conditions due to glycosylation.



Cytotoxicity of sFRP1. Measured by its ability to inhibit proliferation of HeLa human cervical epithelial carcinoma cells. The ED₅₀ value for this effect is typically 5-30 μg/ml.

WARNING
THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA
This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent via email to your institution.

WARRANTY AND LIMITATION OF REMEDY
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PRODUCT INFORMATION



Description

Secreted frizzled-related protein 1 (sFRP1) is a Wnt inhibitor and a member of the sFRP family.^{1,2} It is composed of a signal peptide, a cysteine-rich domain, and a netrin-like domain.¹ sFRP1 is ubiquitously expressed, with the highest levels in the heart, ovary, testis, and small intestine.³ It inhibits Wnt signaling by sequestering Wnt directly *via* the cysteine-rich or netrin-like domains or competing with Wnt for binding to the cysteine-rich domain of Frizzled (Fz) receptors.¹ sFRP1 is involved in apoptosis, adipocyte differentiation, angiogenesis, osteoblast proliferation and differentiation, development, and hematopoietic stem cell maintenance.^{1,2} Myocardial overexpression of *Sfrp1* protects against decreases in cardiac function and increases in myocardium apoptosis in a mouse model of heart failure induced by transverse aortic constriction.⁴ Tumor protein levels of sFRP1 are decreased in early-stage breast cancer, which is associated with poor prognosis.⁵ Cayman's sFRP1 (human, recombinant) protein can be used for cell-based assays. This protein consists of 294 amino acids, has a calculated molecular weight of 34 kDa, and a predicted N-terminus of Ser32 after signal peptide cleavage. By SDS-PAGE, under reducing conditions, the apparent molecular mass of the protein is 38 kDa due to glycosylation.

References

1. Guan, H., Zhang, J., Luan, J., *et al.* Secreted frizzled related proteins in cardiovascular and metabolic diseases. *Front. Endocrinol.* **12**, 712217 (2021).
2. Bodine, P.V.N. and Komm, B.S. Wnt signaling and osteoblastogenesis. *Rev. Endocr. Metab. Disord.* **7(1-2)**, 33-39 (2006).
3. Melkonyan, H.S., Chang, W.C., Shapiro, J.P., *et al.* SARPs: A family of secreted apoptosis-related proteins. *Proc. Natl. Acad. Sci. USA* **94(25)**, 13636-13641 (1997).
4. Pan, S., Zhao, X., Wang, X., *et al.* Sfrp1 attenuates TAC-induced cardiac dysfunction by inhibiting Wnt signaling pathway- mediated myocardial apoptosis in mice. *Lipids Health Dis.* **17(1)**, 202 (2018).
5. Klopocki, E., Kristiansen, G., Wild, P.J., *et al.* Loss of SFRP1 is associated with breast cancer progression and poor prognosis in early stage tumors. *Int. J. Oncol.* **25(3)**, 641-649 (2004).

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