

Product datasheet

Recombinant human Sclerostin protein (Active) ab233662

Description

Product name	Recombinant human Sclerostin protein (Active)
Biological activity	Determined by its ability to downregulate alkaline phosphatase activity in differentiating MC3T3-E1 cells in the presence of 20ng/ml murine Wnt-3a.
Purity	>= 95 % SDS-PAGE. >= 95% by HPLC analysis.
Expression system	CHO cells
Accession	<u>Q9BQB4</u>
Protein length	Full length protein
Animal free	No
Nature	Recombinant
Species	Human
Sequence	QGWQAFKNDATEIPELGEYPEPPPELENNKTMNRAENG GRPPHHPFETK DVSEYSCRELHFTRYVTDGPCRSAPVTELVCSGQCGPA RLLPNAIGRGK WWRPSGPDFRCIPDRYRAQRVQLLCPGGEAPRARKVRL VASCKCKRLTRF HNQSELKDFGTEAARPQKGRKPRPRARSAKANQAELEN AY
Predicted molecular weight	22 kDa
Amino acids	24 to 213
Additional sequence information	This product is the mature full length protein from aa 24 to 213. The signal peptide is not included.

Specifications

Our **Abpromise guarantee** covers the use of **ab233662** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications	HPLC
	Functional Studies
	SDS-PAGE

Form	Lyophilized
Additional notes	Migrates with an apparent molecular mass of approximately 28-35 kDa by SDS-PAGE gel, under non-reducing conditions.

Preparation and Storage

Stability and Storage	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle. This product is an active protein and may elicit a biological response in vivo, handle with caution.
Reconstitution	For lot specific reconstitution information please contact our Scientific Support Team.

General Info

Function	Negative regulator of bone growth.
Tissue specificity	Widely expressed at low levels with highest levels in bone, cartilage, kidney, liver, bone marrow and primary osteoblasts differentiated for 21 days.
Involvement in disease	Defects in SOST are the cause of sclerosteosis (SOST) [MIM:269500]; also known as cortical hyperostosis with syndactyly. SOST is an autosomal recessive sclerosing bone dysplasia characterized by a generalized hyperostosis and sclerosis leading to a markedly thickened skull, with mandible, ribs, clavicles and all long bones also being affected. Due to narrowing of the foramina of the cranial nerves, facial nerve palsy, hearing loss and atrophy of the optic nerves can occur. Sclerosteosis is clinically and radiologically very similar to van Buchem disease, mainly differentiated by hand malformations and a large stature in sclerosteosis patients. Note=A 52 kb deletion downstream of SOST results in SOST transcription suppression and is a cause of van Buchem disease (VBCH) [MIM:239100]; also known as hyperostosis corticalis generalisata. VBCH is an autosomal recessive sclerosing bone dysplasia characterized by endosteal hyperostosis of the mandible, skull, ribs, clavicles, and diaphyses of the long bones. Affected patients present a symmetrically increased thickness of bones, most frequently found as an enlarged jawbone, but also an enlargement of the skull, ribs, diaphysis of long bones, as well as tubular bones of hands and feet. The clinical consequence of increased thickness of the skull include facial nerve palsy causing hearing loss, visual problems, neurological pain, and, very rarely, blindness as a consequence of optic atrophy. Serum alkaline phosphatase levels are elevated.
Sequence similarities	Belongs to the sclerostin family. Contains 1 CTCK (C-terminal cystine knot-like) domain.
Cellular localization	Secreted.

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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- Response to your inquiry within 24 hours

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