SOST Antibody – Monoclonal

PRODUCT OVERVIEW

Product Name	SOST, Monoclonal Antibody
Full Product Name	InVivoMAb Anti-Human SOST Antibody (Iv0185)
Product Gene Name	anti-SOST antibody
Product Synonym Names	SOST; Sclerostin
Clonality	Monoclonal
Isotype	IgG4, kappa
Clone Number	Iv0185
Species Reactivity	Human
Form/Format	Liquid; 0.01M PBS, pH7.4.
Purity/Purification	>95% by SDS-PAGE Protein A or G purified from cell culture supernatant.
Concentration	1mg/ml (lot specific)
Preparation and Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. Store at +4 degree C short term (1-2 weeks). Store at -20 degree C 12 months. Store at -80 degree C long term.
Molecular Weight	24,031 Da
Chromosome Location	Chromosome: 17; NC_000017.10 (41831099..41836156, complement). Location: 17q11.2
OMIM	122860
3D Structure	ModBase 3D Structure for Q9BQB4
Host Species	Human
Product Categories/Family for anti-SOST antibody	Invivo Antibodies
ISO Certification	Manufactured in an ISO 9001:2015 Certified Laboratory.
Applications Tested/Suitable for anti-SOST antibody	Blocking (BL), ELISA (EIA)
Related Product Information for anti-SOST antibody	MBS1579908 is an antibody targeting SOST. Also known as InVivoMAb Anti-Human SOST Antibody (Iv0185). Reactive in Human.
Other Notes	Small volumes of anti-SOST antibody vial(s) may occasionally become entrapped in the seal of the product vial during shipment and storage. If necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the container`s cap. Certain products may require to ship with dry ice and additional dry ice fee may apply.

ADDITIONAL DETAILS

NCBI/Uniprot data below describe general gene information for SOST. It may not necessarily be applicable to this product.

NCBI GI #	13376846
NCBI GeneID	50964
NCBI Accession #	NP_079513.1

UniProt Primary Accession #	Q9BQB4
UniProt Secondary Accession #	Q495N9
UniProt Related Accession #	Q9BQB4
GenBank Nucleotide #	NM_025237.2
NCBI Official Full Name	sclerostin
NCBI Official Synonym Full Names	sclerostin
NCBI Official Symbol	SOST
NCBI Official Synonym Symbols	CDD; VBCH
UniProt Protein Name	Sclerostin
Protein Family	Sclerostin
UniProt Gene Name	SOST
UniProt Entry Name	SOST_HUMAN
NCBI Protein Information	sclerostin
NCBI Summary for SOST	Sclerostin is a secreted glycoprotein with a C-terminal cysteine knot-like (CTCK) domain and sequence similarity to the DAN (differential screening-selected gene aberrative in neuroblastoma) family of bone morphogenetic protein (BMP) antagonists. Loss-of-function mutations in this gene are associated with an autosomal-recessive disorder, sclerosteosis, which causes progressive bone overgrowth. A deletion downstream of this gene, which causes reduced sclerostin expression, is associated with a milder form of the disorder called van Buchem disease. [provided by RefSeq, Jul 2008]

UniProt Comments for SOST	SOST: Negative regulator of bone growth that acts through inhibition of Wnt signaling and bone formation. Defects in SOST are the cause of sclerosteosis type 1 (SOST1). 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. Defects in SOST are a cause of van Buchem disease (VBCH). 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. A 52 kb deletion downstream of SOST results in SOST transcription suppression causing van Buchem disease. Defects in SOST are a cause of craniodiaphyseal dysplasia autosomal dominant (CDD). A severe bone dysplasia characterized by massive generalized hyperostosis and sclerosis, especially involving the skull and facial bones. The sclerosis is so severe that the resulting facial distortion is referred to as 'leontiasis ossea' (leonine faces) and the bone deposition results in progressive stenosis of craniofacial foramina. Respiratory obstruction due to choanal stenosis compromises the clinical outcomes of affected patients. Heterozygous mutations located in the secretion signal of the SOST gene prevent sclerostin secretion and can be responsible for craniodiaphyseal dysplasia. Belongs to the sclerostin family. 2 isoforms of the human protein are produced by alternative splicing. Protein type: Secreted; Secreted, signal peptide Chromosomal Location of Human Ortholog: 17q11.2 Cellular Component: extracellular matrix; Golgi apparatus; proteinaceous extracellular matrix; extracellular space; extracellular region Molecular Function: heparin binding; protein binding; transcription factor binding Biological Process: ossification; Wnt receptor signaling pathway; response to mechanical stimulus; positive regulation of transcription, DNA-dependent; negative regulation of ossification; negative regulation of protein complex assembly; negative regulation of BMP signaling pathway Disease: Sclerosteosis 1; Hyperostosis Corticalis Generalisata; Craniodiaphyseal Dysplasia, Autosomal Dominant
Research Articles on SOST	**1.** A novel frame shift mutation in the (SOST) gene causing loss of functional sclerostin was identified in a patient with sclerosteosis and her parents.
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