SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com

[Marker Primary Antibody]

Rabbit Anti-SIX1/FITC Conjugated antibody

Catalog Number: SL17504R-FITC

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	Anti-SIX1/FITC
Immunogen:	KLH conjugated synthetic peptide derived from human SIX1
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Applications:	ICC/IF=1:50-200 IF=1:100-500 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	(predicted:Human,Mouse,Rat,Cow,Rabbit,Sheep)
Background:	background: The protein encoded by this gene is a homeobox protein that is similar to the Drosophila 'sine oculis' gene product. This gene is found in a cluster of related genes on chromosome 14 and is thought to be involved in limb development. Defects in this gene are a cause of autosomal dominant deafness type 23 (DFNA23) and branchiootic syndrome type 3 (BOS3). [provided by RefSeq, Jul 2008] **Function:** May be involved in limb tendon and ligament development. **Subcellular Location:** Nucleus. **Tissue Specificity:** Specifically expressed in skeletal muscle. DISEASE: Defects in SIX1 are the cause of deafness autosomal dominant type 23 (DFNA23) [MIM:605192]. A form of non-syndromic deafness characterized by prelingual, bilateral, symmetric hearing loss with a conductive component present in some but not all patients. Defects in SIX1 are the cause of branchiootic syndrome type 3 (BOS3)

Important Note: This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com

	[MIM:608389]. BOS3 is a syndrome characterized by usually bilateral branchial cleft fistulas or cysts, sensorineural and/or conductive hearing loss, pre-auricular pits, and structural defects of the outer, middle or inner ear. Otic defects include malformed and hypoplastic pinnae, a narrowed external ear canal, bulbous internal auditory canal, stapes fixation, malformed and hypoplastic cochlea. Branchial and otic anomalies are as those seen in individuals with the branchiootorenal syndrome. However, renal anomalies are absent in branchiootic syndrome patients. Note=Defects in SIX1 could be a cause of branchiootorenal syndrome (BOR). BOR is an autosomal dominant disorder manifested by various combinations of preauricular pits, branchial fistulae or cysts, lacrimal duct stenosis, hearing loss, structural defects of the outer, middle, or inner ear, and renal dysplasia. Associated defects include asthenic habitus, long narrow facies, constricted palate, deep overbite, and myopia. Hearing loss may be due to mondini type cochlear defect and stapes fixation. Penetrance of BOR syndrome is high, although expressivity can be extremely variable. **Similarity:** Belongs to the SIX/Sine oculis homeobox family. Contains 1 homeobox DNA-binding domain. **Database links:** **Entrez Gene:** 6495 Human **Entrez Gene:** 20471 Mouse **Entrez Gene:** 114634 Rat Omim: 601205 Human **SwissProt:** Q15475 Human **SwissProt:** Q62231 Mouse **SwissProt:** Q8BSP4 Mouse **Unigene:** 54416 Human **Unigene:** 713114 Human **Unigene:** 4645 Mouse **Unigene:** 23396 Rat Important **Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.