

MSX1 Mouse mAb

CatalogNo: YM0453

Key Features

Host Species

- Mouse

Reactivity

- Human

Applications

- WB,ELISA

MW

- 31kD (Calculated)

Recommended Dilution Ratios

WB 1:500-1:2000

ELISA 1:10000

Not yet tested in other applications.

Storage

Storage* -15°C to -25°C/1 year(Do not lower than -25°C)

Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Basic Information

Clonality Monoclonal

Immunogen Information

Immunogen Purified recombinant fragment of human Msx-1 expressed in E. Coli.

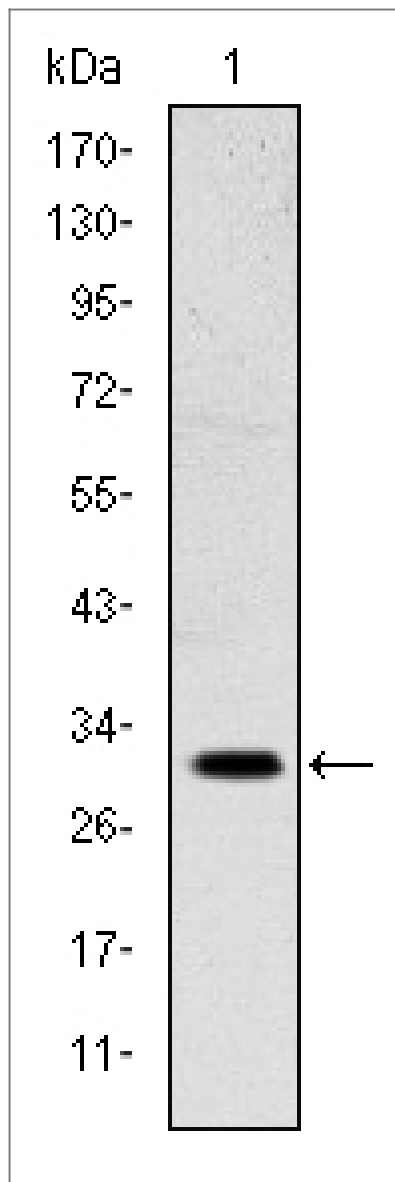
Specificity Msx-1 Monoclonal Antibody detects endogenous levels of Msx-1 protein.

Target Information

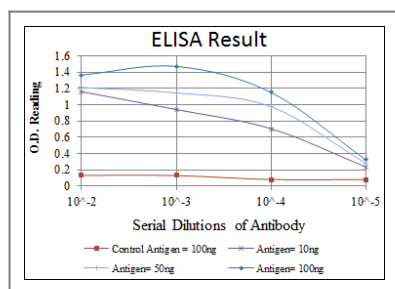
Gene name MSX1 HOX7

Protein Name	Homeobox protein MSX-1		
	Organism	Gene ID	UniProt ID
	Human	4487 ;	P28360 ;
	Mouse		P13297 ;
Cellular Localization	Nucleus.		
Tissue specificity	Expressed in the developing nail bed mesenchyme.		
Function	<p>Disease:A chromosomal aberration involving MSX1 is a cause of Wolf-Hirschhorn syndrome (WHS) [MIM:194190]. WHS is caused by sub-telomeric deletions in the short arm of chromosome 4. WHS is characterized by profound mental retardation, heart defects, and facial clefting.,Disease:Defects in MSX1 are a cause of autosomal dominant hypodontia (HYD1) [MIM:106600]; also known as familial or selective tooth agenesis. Absence of less than 6 teeth is referred to as hypodontia. Agenesis of one or more teeth constitutes one of the most common developmental anomalies in man. Reported incidences vary from 1.6% to 9.6%, excluding third molar (Wisdom tooth) agenesis, which occurs in 20% of the population.,Disease:Defects in MSX1 are the cause of non-syndromic orofacial cleft type 5 (OFC5) [MIM:608874]; also called non-syndromic cleft lip with or without cleft palate 5. Non-syndromic orofacial cleft is a common birth defect consisting of cleft lips with or without cleft palate. Cleft lips are associated with cleft palate in two-third of cases. A cleft lip can occur on one or both sides and range in severity from a simple notch in the upper lip to a complete opening in the lip extending into the floor of the nostril and involving the upper gum.,Disease:Defects in MSX1 are the cause of Witkop syndrome (WITS) [MIM:189500]. WITS is a form of ectodermal dysplasia also called tooth-and-nail syndrome or dysplasia of nails with hypodontia. Ectodermal dysplasias (EDs) constitute a heterogeneous group of developmental disorders affecting tissues of ectodermal origin. EDs are characterized by abnormal development of two or more ectodermal structures such as hair, teeth, nails and sweat glands, with or without any additional clinical sign. Each combination of clinical features represents a different type of ectodermal dysplasia. Witkop syndrome is characterized by abnormalities largely limited largely to teeth (some of which are missing) and nails (which are poorly formed early in life, especially toenails). This condition is distinguished from anhidrotic ectodermal dysplasia by autosomal dominant inheritance and little involvement of hair and sweat glands. The teeth are not as severely affected.,Function:Acts as a transcriptional repressor. May play a role in limb-pattern formation. Acts in cranofacial development and specifically in odontogenesis. Expression in the developing nail bed mesenchyme is important for nail plate thickness and integrity.,PTM:Sumoylated by PIAS1, desumoylated by SENP1.,similarity:Belongs to the Msh homeobox family.,similarity:Contains 1 homeobox DNA-binding domain.,tissue specificity:Expressed in the developing nail bed mesenchyme.,</p>		

| Validation Data



Western Blot analysis using Msx-1 Monoclonal Antibody against NTERA-2 cell lysate.



Contact information

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MSX1 Mouse mAb

