abcam

Product datasheet

Anti-MSX1 antibody ab86870

Overview

Product name	Anti-MSX1 antibody
Description	Rabbit polyclonal to MSX1
Host species	Rabbit
Tested applications	**Suitable for:** WB, ELISA, Dot blot
Species reactivity	**Reacts with:** Mouse, Rat, Human
Immunogen	Synthetic peptide (DEEGAKPKVSPSLLPFSVE) corresponding to amino acids 46-64 of mouse Msx1 protein. The peptide was post synthetically modified and conjugated to KLH using hetero bifunctional cross linker for immunization of rabbits. Run BLAST with Run BLAST with

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Storage buffer	Preservative: 0.02% Sodium Azide Constituents: PBS; Stabilizing proteins
Purity	Immunogen affinity purified
Purification notes	Msx1 antibody was affinity purified on immobilized antigen based affinity chromatography.
Clonality	Polyclonal
Isotype	IgG

Applications

Our Abpromise guarantee covers the use of **ab86870** in the following tested applications.

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

Application	Abreviews	Notes
WB		1/500 - 1/750. Detects a band of approximately 34-35 kDa (predicted molecular weight: 31 kDa).

Application	Abreviews	Notes
ELISA		1/20000 - 1/50000.
Dot blot		1/20000 - 1/50000.

Target

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.
Tissue specificity	Expressed in the developing nail bed mesenchyme.
Involvement in disease	Defects in MSX1 are the cause of tooth agenesis selective type 1 (STHAG1) [MIM:106600]. A form of selective tooth agenesis, a common anomaly characterized by the congenital absence of one or more teeth. Selective tooth agenesis without associated systemic disorders has sometimes been divided into 2 types: oligodontia, defined as agenesis of 6 or more permanent teeth, and hypodontia, defined as agenesis of less than 6 teeth. The number in both cases does not include absence of third molars (wisdom teeth). Tooth agenesis selective type 1 can be associated with orofacial cleft in some patients. Note=MSX1 is deleted in some patients with Wolf-Hirschhorn syndrome (WHS). WHS results from sub-telomeric deletions in the short arm of chromosome 4. Defects in MSX1 are the cause of Witkop syndrome (WITS) [MIM:189500]. WITS is a form of ectodermal dyslasia 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. 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.
Sequence similarities	Belongs to the Msh homeobox family. Contains 1 homeobox DNA-binding domain.
Post-translational modifications	Sumoylated by PIAS1, desumoylated by SENP1.
Cellular localization	Nucleus.

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