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EYA4 Rabbit pAb

Catalog # AP98911

Product Information

Application	IHC-P, IHC-F, IF
Primary Accession	O95677
Other Accession	O95677
Reactivity	Rat
Predicted	Pig, Human, Mouse, Rabbit, Chicken, Dog
Host	Rabbit
Clonality	Polyclonal
Calculated MW	69505
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human EYA4
Epitope Specificity	131-230/639
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the HAD-like hydrolase superfamily. EYA family.
DISEASE	Defects in EYA4 are the cause of deafness autosomal dominant type 10 (DFNA10) [MIM:601316]. DFNA10 is a form of sensorineural hearing loss. Sensorineural deafness results from damage to the neural receptors of the inner ear, the nerve pathways to the brain, or the area of the brain that receives sound information. Defects in EYA4 are the cause of cardiomyopathy dilated type 1J (CMD1J) [MIM:605362]. Dilated cardiomyopathy is a disorder characterized by ventricular dilation and impaired systolic function, resulting in congestive heart failure and arrhythmia. Patients are at risk of premature death.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	A gene of chromosome 6q23 encodes the 640 amino acid protein, EYA4 (eyes absent) (1). EYA is one of four members of the eyes absent family (1). A 271 amino acid domain at the carboxyl terminal is highly conserved amongst the members of the eyes absent family (1). EYA4 is expressed in the craniofacial mesenchyme, the dermamyotome, and the limb (1). The conserved region in other EYA proteins interacts with SIX, DACH, and G-proteins, which regulate transcription in early embryonic development (1,2,3,4). SIX translocates EYA1-3 to the nucleus, and G-proteins can stop this interaction (3,4). Premature stop codon mutations in EYA4 cause postlingual, progressive autosomal dominant hearing loss in humans (2). This shows that EYA4 is also vital to the mature organ of Corti (2). EYA4 may cause oculo-dento-digital syndrome, based on its expression pattern and map postion (1).

Additional Information

Gene ID	2070

Other Names	CMD1J; DFNA10; B130023L16Rik; EYA4_HUMAN; EYA4; Eyes absent homolog 4; 3.1.3.48; EYA4_MOUSE; EYA transcriptional coactivator and phosphatase 4; eyes absent (Drosophila) homolog 4; eyes absent homolog 4 (Drosophila)
Dilution	IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	EYA4
Function	Tyrosine phosphatase that specifically dephosphorylates 'Tyr- 142' of histone H2AX (H2AXY142ph). 'Tyr-142' phosphorylation of histone H2AX plays a central role in DNA repair and acts as a mark that distinguishes between apoptotic and repair responses to genotoxic stress. Promotes efficient DNA repair by dephosphorylating H2AX, promoting the recruitment of DNA repair complexes containing MDC1. Its function as histone phosphatase probably explains its role in transcription regulation during organogenesis. May be involved in development of the eye (By similarity).
Cellular Location	Cytoplasm {ECO:0000250|UniProtKB:Q99502}. Nucleus {ECO:0000250|UniProtKB:Q99502}
Tissue Location	Highly expressed in heart and skeletal muscle.

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