

HSF4 Rabbit pAb

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Catalog # AP56314

Product Information

Application	WB, IHC-P, IHC-F, IF, E
Primary Accession	Q9ULV5
Predicted	Human, Mouse, Rat, Dog, Pig, Horse, Rabbit, Sheep
Host	Rabbit
Clonality	Polyclonal
Calculated MW	53011
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human HSF4
Epitope Specificity	21-120/492
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Nucleus.
SIMILARITY	Belongs to the HSF family.
Post-translational modifications	Phosphorylated mainly on serine residues. Phosphorylation on Ser-298 promotes sumoylation on Lys-293. Isoform HSF4B is constitutively sumoylated. Sumoylation represses the transcriptional activity and is promoted by phosphorylation on Ser-298. HSF4A is not sumoylated.
DISEASE	Defects in HSF4 are the cause of cataract zonular HSF4-related (CZ-HSF4) [MIM:116800]. A form of zonular cataract. Zonular or lamellar cataracts are opacities, broad or narrow, usually consisting of powdery white dots affecting only certain layers or zones between the cortex and nucleus of an otherwise clear lens. The opacity may be so dense as to render the entire central region of the lens completely opaque, or so translucent that vision is hardly if at all impeded. Zonular cataracts generally do not involve the embryonic nucleus, though sometimes they involve the fetal nucleus. Usually sharply separated from a clear cortex outside them, they may have projections from their outer edges known as riders or spokes. Defects in HSF4 are the cause of cataract Marner type (CAM) [MIM:116800]. A form of cataract with variable and progressive opacities. Affected individuals present with zonular cataract, although some have nuclear, anterior polar, or stellate cataract. Finger malformation is observed in some kindreds.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Heat-shock transcription factors (HSFs) activate heat-shock response genes under conditions of heat or other stresses. HSF4 lacks the carboxyl-terminal hydrophobic repeat which is shared among all vertebrate HSFs and has been suggested to be involved in the negative regulation of DNA binding activity. Two alternatively spliced transcripts encoding distinct isoforms and possessing different transcriptional activity have been described. [provided by RefSeq, Jul 2008]

Additional Information

Gene ID	3299
Other Names	Heat shock factor protein 4, HSF 4, hHSF4, Heat shock transcription factor 4, HSTF 4, HSF4
Target/Specificity	Expressed in heart, skeletal muscle, eye and brain, and at much lower levels in some other tissues.
Dilution	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,ICC/IF=1:100-500,IF=1:100-500,ELISA=1:5000-10000
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	HSF4
Function	Heat-shock transcription factor that specifically binds heat shock promoter elements (HSE) (PubMed: 22587838 , PubMed: 23507146). Required for denucleation and organelle rupture and degradation that occur during eye lens terminal differentiation, when fiber cells that compose the lens degrade all membrane-bound organelles in order to provide lens with transparency to allow the passage of light (By similarity). In this process, may regulate denucleation of lens fiber cells in part by activating DNASE2B transcription (By similarity). May be involved in DNA repair through the transcriptional regulation of RAD51 (PubMed: 22587838). May up-regulate p53/TP53 protein in eye lens fiber cells, possibly through protein stabilization (PubMed: 28981088). In the eye lens, controls the expression of alpha-crystallin B chain/CRYAB and consequently may be involved in the regulation of lysosomal acidification (By similarity).
Cellular Location	Nucleus.
Tissue Location	Expressed in heart, skeletal muscle, eye and brain, and at much lower levels in some other tissues

Background

Heat-shock transcription factors (HSFs) activate heat-shock response genes under conditions of heat or other stresses. HSF4 lacks the carboxyl-terminal hydrophobic repeat which is shared among all vertebrate HSFs and has been suggested to be involved in the negative regulation of DNA binding activity. Two alternatively spliced transcripts encoding distinct isoforms and possessing different transcriptional activity have been described. [provided by RefSeq, Jul 2008]

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