abcam

Product datasheet

Anti-p63 (phospho S395) antibody ab92581

1 Image

Overview

Product name Anti-p63 (phospho S395) antibody

Description Rabbit polyclonal to p63 (phospho S395)

Host species Rabbit

Tested applications Suitable for: WB

Species reactivity Reacts with: Human

Predicted to work with: Mouse, Rat

Immunogen Synthetic peptide corresponding to Human p63 (phospho S395).

Positive control HepG2, A549 and LOVO cell extracts treated with nocodazole (1 µg/ml, 18 hours)

General notes

The Life Science industry has been in the grips of a reproducibility crisis for a number of years.

Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets

your needs before purchasing.

If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be

found below, along with publications, customer reviews and Q&As

Properties

Form Liquid

Storage instructions Shipped at 4°C. Store at -20°C. Stable for 12 months at -20°C.

Storage buffer pH: 7.40

Preservative: 0.02% Sodium azide

Constituents: PBS, 50% Glycerol, 0.87% Sodium chloride

Without Mg2+ and Ca2+

Purity Immunogen affinity purified

Purification notes Affinity chromatography using epitope specific phosphopeptide. The antibody against non

phosphopeptide was removed by chromatography using non phosphopeptide corresponding to

the phosphorylation site.

Clonality Polyclonal

1

Isotype IgG

Applications

The Abpromise guarantee

Our **Abpromise guarantee** covers the use of ab92581 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 - 1/1000. Predicted molecular weight: 77 kDa.

Target

Function

Acts as a sequence specific DNA binding transcriptional activator or repressor. The isoforms contain a varying set of transactivation and auto-regulating transactivation inhibiting domains thus showing an isoform specific activity. May be required in conjunction with TP73/p73 for initiation of p53/TP53 dependent apoptosis in response to genotoxic insults and the presence of activated oncogenes. Involved in Notch signaling by probably inducing JAG1 and JAG2. Plays a role in the regulation of epithelial morphogenesis. The ratio of DeltaN-type and TA*-type isoforms may govern the maintenance of epithelial stem cell compartments and regulate the initiation of epithelial stratification from the undifferentiated embryonal ectoderm. Required for limb formation from the apical ectodermal ridge.

Tissue specificity

Widely expressed, notably in heart, kidney, placenta, prostate, skeletal muscle, testis and thymus, although the precise isoform varies according to tissue type. Progenitor cell layers of skin, breast, eye and prostate express high levels of DeltaN-type isoforms. Isoform 10 is predominantly expressed in skin squamous cell carcinomas, but not in normal skin tissues.

Involvement in disease

Defects in TP63 are the cause of acro-dermato-ungual-lacrimal-tooth syndrome (ADULT syndrome) [MIM:103285]; a form of ectodermal dysplasia. 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. ADULT syndrome involves ectrodactyly, syndactyly, finger- and toenail dysplasia, hypoplastic breasts and nipples, intensive freckling, lacrimal duct atresia, frontal alopecia, primary hypodontia, and loss of permanent teeth. ADULT differs significantly from EEC3 syndrome by the absence of facial clefting.

Defects in TP63 are the cause of ankyloblepharon-ectodermal defects-cleft lip/palate (AEC) [MIM:106260]. AEC is an autosomal dominant condition characterized by congenital ectodermal dysplasia with coarse, wiry, sparse hair, dystrophic nails, slight hypohidrosis, scalp infections, ankyloblepharon filiform adnatum, maxillary hypoplasia, hypodontia and cleft lip/palate.

Defects in TP63 are the cause of ectrodactyly-ectodermal dysplasia-cleft lip/palate syndrome type 3 (EEC3) [MIM:604292]. EEC3 is an autosomal dominant syndrome characterized by ectrodactyly of hands and feet, ectodermal dysplasia and facial clefting.

Defects in TP63 are the cause of split-hand/foot malformation type 4 (SHFM4) [MIM:605289]. Split-hand/split-foot malformation is a limb malformation involving the central rays of the autopod and presenting with syndactyly, median clefts of the hands and feet, and aplasia and/or hypoplasia of the phalanges, metacarpals, and metatarsals. There is restricted overlap between

the mutational spectra of EEC3 and SHFM4.

Defects in TP63 are the cause of limb-mammary syndrome (LMS) [MIM:603543]. LMS is characterized by ectrodactyly, cleft palate and mammary-gland abnormalities.

Note=Defects in TP63 are a cause of cervical, colon, head and neck, lung and ovarian cancers. Defects in TP63 are a cause of ectodermal dysplasia Rapp-Hodgkin type (EDRH) [MIM:129400]; also called Rapp-Hodgkin syndrome or anhidrotic ectodermal dysplasia with cleft lip/palate. Ectodermal dysplasia defines a heterogeneous group of disorders due to abnormal development of two or more ectodermal structures. EDRH is characterized by the combination of anhidrotic ectodermal dysplasia, cleft lip, and cleft palate. The clinical syndrome is comprised of a characteristic facies (narrow nose and small mouth), wiry, slow-growing, and uncombable hair, sparse eyelashes and eyebrows, obstructed lacrimal puncta/epiphora, bilateral stenosis of external auditory canals, microsomia, hypodontia, cone-shaped incisors, enamel hypoplasia, dystrophic nails, and cleft lip/cleft palate.

Defects in TP63 are the cause of non-syndromic orofacial cleft type 8 (OFC8) [MIM:129400]. 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 p53 family.

Contains 1 SAM (sterile alpha motif) domain.

Domain

The transactivation inhibitory domain (TID) can interact with, and inhibit the activity of the N-

terminal transcriptional activation domain of TA*-type isoforms.

Post-translational modifications

May be sumoylated.

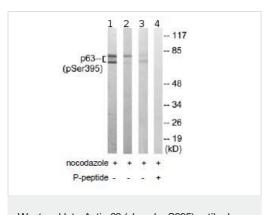
Ubiquitinated. Polyubiquitination involves WWP1 and leads to proteasomal degradation of this

protein.

Cellular localization

Nucleus.

Images



Western blot - Anti-p63 (phospho S395) antibody (ab92581)

All lanes : Anti-p63 (phospho S395) antibody (ab92581) at 1/500 dilution

Lane 1 : Extracts from HepG2 cells treated with nocodazole (1 µg/ml,

18 hours)

Lane 2: Extracts from A549

cells treated with nocodazole (1 µg/ml,

(8 hours

Lane 3 : Extracts from LOVO cells treated with nocodazole (1 μ g/ml,

18 hours)

Lane 4: Extracts from HepG2 cells treated with nocodazole (1 µg/ml,

18 hours) with immunizing peptide at 10 µg

Lysates/proteins at 30 µg per lane.

Predicted band size: 77 kDa

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