**Product datasheet**

**Anti-p53 (phospho S15) antibody ab1431**

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<th><strong>Applications</strong></th>
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<td>Our Abpromise guarantee covers the use of ab1431 in the following tested applications.</td>
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<td>The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.</td>
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Function
Acts as a tumor suppressor in many tumor types; induces growth arrest or apoptosis depending on the physiological circumstances and cell type. Involved in cell cycle regulation as a trans-activator that acts to negatively regulate cell division by controlling a set of genes required for this process. One of the activated genes is an inhibitor of cyclin-dependent kinases. Apoptosis induction seems to be mediated either by stimulation of BAX and FAS antigen expression, or by repression of Bcl-2 expression. Implicated in Notch signaling cross-over. Isoform 2 enhances the transactivation activity of isoform 1 from some but not all TP53-inducible promoters. Isoform 4 suppresses transactivation activity and impairs growth suppression mediated by isoform 1. Isoform 7 inhibits isoform 1-mediated apoptosis.

Tissue specificity
Ubiquitous. Isoforms are expressed in a wide range of normal tissues but in a tissue-dependent manner. Isoform 2 is expressed in most normal tissues but is not detected in brain, lung, prostate, muscle, fetal brain, spinal cord and fetal liver. Isoform 3 is expressed in most normal tissues but is not detected in lung, spleen, testis, fetal brain, spinal cord and fetal liver. Isoform 7 is expressed in most normal tissues but is not detected in prostate, uterus, skeletal muscle and breast. Isoform 8 is detected only in colon, bone marrow, testis, fetal brain and intestine. Isoform 9 is expressed in most normal tissues but is not detected in brain, heart, lung, fetal liver, salivary gland, breast or intestine.

Involvement in disease
Note=TP53 is found in increased amounts in a wide variety of transformed cells. TP53 is frequently mutated or inactivated in about 60% of cancers. TP53 defects are found in Barrett metaplasia a condition in which the normally stratified squamous epithelium of the lower esophagus is replaced by a metaplastic columnar epithelium. The condition develops as a complication in approximately 10% of patients with chronic gastroesophageal reflux disease and predisposes to the development of esophageal adenocarcinoma. Defects in TP53 are a cause of esophageal cancer (ESCR) [MIM:133239]. Defects in TP53 are a cause of Li-Fraumeni syndrome (LFS) [MIM:151623]. LFS is an autosomal dominant familial cancer syndrome that in its classic form is defined by the existence of a proband affected by a sarcoma before 45 years with a first degree relative affected by any tumor before 45 years and another first degree relative with any tumor before 45 years or a sarcoma at any age. Other clinical definitions for LFS have been proposed (PubMed:8118819 and PubMed:8718514) and called Li-Fraumeni like syndrome (LFL). In these families affected relatives develop a diverse set of malignancies at unusually early ages. Four types of cancers account for 80% of tumors occurring in TP53 germline mutation carriers: breast cancers, soft tissue and bone sarcomas, brain tumors (astrocytomas) and adrenocortical carcinomas. Less frequent tumors include choroid plexus carcinoma or papilloma before the age of 15, rhabdomyosarcoma before the age

### Application

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<th>Application</th>
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<tr>
<td>IHC-Fr</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use at an assay dependent concentration.</td>
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<td>ICC/IF</td>
<td>⭐⭐⭐⭐⭐</td>
<td>1/100 - 1/500.</td>
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<td>IHC-P</td>
<td>⭐⭐⭐⭐⭐</td>
<td>Use a concentration of 20 µg/ml. Perform heat mediated antigen retrieval before commencing with IHC staining protocol. PubMed: 23365256</td>
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<tr>
<td>IP</td>
<td>⭐⭐⭐⭐⭐</td>
<td>1/10 - 1/100.</td>
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### Target

**Function**
Acts as a tumor suppressor in many tumor types; induces growth arrest or apoptosis depending on the physiological circumstances and cell type. Involved in cell cycle regulation as a trans-activator that acts to negatively regulate cell division by controlling a set of genes required for this process. One of the activated genes is an inhibitor of cyclin-dependent kinases. Apoptosis induction seems to be mediated either by stimulation of BAX and FAS antigen expression, or by repression of Bcl-2 expression. Implicated in Notch signaling cross-over. Isoform 2 enhances the transactivation activity of isoform 1 from some but not all TP53-inducible promoters. Isoform 4 suppresses transactivation activity and impairs growth suppression mediated by isoform 1. Isoform 7 inhibits isoform 1-mediated apoptosis.

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of 5, leukemia, Wilms tumor, malignant phyllodes tumor, colorectal and gastric cancers. Defects in TP53 are involved in head and neck squamous cell carcinomas (HNSCC) [MIM:275355]; also known as squamous cell carcinoma of the head and neck. Defects in TP53 are a cause of lung cancer (LNCR) [MIM:211980]. Defects in TP53 are a cause of choroid plexus papilloma (CPLPA) [MIM:260500]. Choroid plexus papilloma is a slow-growing benign tumor of the choroid plexus that often invades the leptomeninges. In children it is usually in a lateral ventricle but in adults it is more often in the fourth ventricle. Hydrocephalus is common, either from obstruction or from tumor secretion of cerebrospinal fluid. If it undergoes malignant transformation it is called a choroid plexus carcinoma. Primary choroid plexus tumors are rare and usually occur in early childhood. Defects in TP53 are a cause of adrenocortical carcinoma (ADCC) [MIM:202300]. ADCC is a rare childhood tumor of the adrenal cortex. It occurs with increased frequency in patients with the Beckwith-Wiedemann syndrome and is a component tumor in Li-Fraumeni syndrome.

Sequence similarities
Belongs to the p53 family.

Domain
The nuclear export signal acts as a transcriptional repression domain. The TAD I and TAD II motifs (residues 17 to 25 and 48 to 56) correspond both to 9aaTAD motifs which are transactivation domains present in a large number of yeast and animal transcription factors.

Post-translational modifications
Acetylated. Acetylation of Lys-382 by CREBBP enhances transcriptional activity. Deacetylation of Lys-382 by SIRT1 impairs its ability to induce proapoptotic program and modulate cell senescence.
Phosphorylation on Ser residues mediates transcriptional activation. Phosphorylated by HIPK1 (By similarity). Phosphorylation at Ser-9 by HIPK4 increases repression activity on BIRC5 promoter. Phosphorylated on Thr-18 by VRK1. Phosphorylated on Ser-20 by CHEK2 in response to DNA damage, which prevents ubiquitination by MDM2. Phosphorylated on Thr-55 by TAF1, which promotes MDM2-mediated degradation. Phosphorylated on Ser-46 by HIPK2 upon UV irradiation. Phosphorylation on Ser-392 is required for acetylation by CREBBP. Phosphorylated on Ser-392 following UV but not gamma irradiation. Phosphorylated upon DNA damage, probably by ATM or ATR. Phosphorylated on Ser-15 upon ultraviolet irradiation; which is enhanced by interaction with BANP.
Dephosphorylated by PP2A-PPP2R5C holoenzyme at Thr-55. SV40 small T antigen inhibits the dephosphorylation by the AC form of PP2A.
May be O-glycosylated in the C-terminal basic region. Studied in EB-1 cell line.
Ubiquitinated by MDM2 and SYV1, which leads to proteasomal degradation. Ubiquitinated by RFWD3, which works in cooperation with MDM2 and may catalyze the formation of short polyubiquitin chains on p53/TP53 that are not targeted to the proteasome. Ubiquitinated by MKRN1 at Lys-291 and Lys-292, which leads to proteasomal degradation. Deubiquitinated by USP10, leading to its stabilization. Ubiquitinated by TRIM24, which leads to proteasomal degradation. Ubiquitination by TOPORS induces degradation. Deubiquitination by USP7, leading to stabilization. Isoform 4 is monoubiquitinated in an MDM2-independent manner.
Monomethylated at Lys-372 by SETD7, leading to stabilization and increased transcriptional activation. Monomethylated at Lys-370 by SMYD2, leading to decreased DNA-binding activity and subsequent transcriptional regulation activity. Lys-372 monomethylation prevents interaction with SMYD2 and subsequent monomethylation at Lys-370. Dimethylated at Lys-373 by EHMT1 and EHMT2. Monomethylated at Lys-382 by SETD8, promoting interaction with L3MBTL1 and leading to repress transcriptional activity. Demethylation of dimethylated Lys-370 by KDM1A prevents interaction with TP53BP1 and represses TP53-mediated transcriptional activation. Sumoylated by SUMO1.

Cellular localization
Cytoplasm; Cytoplasm. Nucleus. Nucleus > PML body. Endoplasmic reticulum. Interaction with BANP promotes nuclear localization. Recruited into PML bodies together with CHEK2; Nucleus. Cytoplasm. Localized in both nucleus and cytoplasm in most cells. In some cells, forms foci in the
nucleus that are different from nucleoli; Nucleus. Cytoplasm. Localized in the nucleus in most cells but found in the cytoplasm in some cells; Nucleus. Cytoplasm. Localized mainly in the nucleus with minor staining in the cytoplasm; Nucleus. Cytoplasm. Predominantly nuclear but localizes to the cytoplasm when expressed with isoform 4 and Nucleus. Cytoplasm. Predominantly nuclear but translocates to the cytoplasm following cell stress.

Images

All lanes: Anti-p53 (phospho S15) antibody (ab1431) at 1 µg/ml

Lane 1: HEK-293T (Human epithelial cell line from embryonic kidney transformed with large T antigen) cells at 20 µg
Lane 2: HEK-293T cells incubated with etoposide at 20 µg
Lane 3: MCF7 (Human breast adenocarcinoma cell line) cells at 20 µg
Lane 4: MCF7 cells incubated 6 hours with camptothecin at 20 µg
Lane 5: MCF7 cells incubated 16 hours with camptothecin at 20 µg
Lane 6: MCF7 cells incubated 24 hours with camptothecin at 20 µg
Lane 7: HEK-293T incubated with etoposide at 7.5 µg
Lane 8: Lambda phosphatase (400 times-diluted)-treated extract of HEK-293T incubated with etoposide at 7.5 µg
Lane 9: Lambda phosphatase (100 times-diluted)-treated extract of HEK-293T incubated with etoposide at 7.5 µg
Lane 10: Lambda phosphatase (25 times-diluted)-treated extract of HEK-293T incubated with etoposide at 7.5 µg

Secondary
All lanes: Goat anti rabbit IgG(H&L)-HRP at 1/10000 dilution

Developed using the ECL technique.

Performed under reducing conditions.

Predicted band size: 53 kDa
Observed band size: 53 kDa

Exposure time: 2 minutes

SDS PAGE performed under reducing conditions (100mM DTT, Sample heated at 50°C).
Blocking: 5% Milk + PBS for 3 hours at RT.
Primary antibody: 5% BSA + PBS overnight at 4 C.
Secondary antibody: 5% Milk + PBS for 2 hour at RT.

**ab1431 staining p53 in rat bone marrow cells by Immunocytochemistry/Immunofluorescence.**

The cells were fixed in paraformaldehyde, permeabilized in 0.1% Triton X-100 and then blocked using 5% BSA for 1 hour at 25°C. Samples were then incubated with primary antibody at 1:100 for 9 hours at 4°C. The secondary antibody used was a goat anti-rabbit Alexa Fluor® 488 (green) ab150077) used at a 1/250 dilution. DAPI was used to stain the cell nuclei (blue).

These pictures were taken in the different fields of the same bone marrow cell slide.

**Immunohistochemical analysis of mouse fetal lung tissue labeling p53 with ab1431 at 1/100 dilution, followed by Goat anti-Rabbit IgG at 1/200 dilution.**

**ab1431 staining p53 in mouse bone marrow WBCs cells by ICC/IF (Immunocytochemistry/immunofluorescence).**

Cells were fixed with formaldehyde, permeabilized with 0.1% Triton X-100 and blocked with 5% serum for 2 hours at 25°C. Samples were incubated with primary antibody (1/100 in PBS) for 12 hours at 4°C. An Alexa Fluor® 488-conjugated Goat anti-rabbit IgG polyclonal (1/500) was used as the secondary antibody.
Western blot - Anti-p53 (phospho S15) antibody (ab1431)

All lanes: Anti-p53 (phospho S15) antibody (ab1431)

Lane 1: HeLa whole cell lysate (ab150035)
Lane 2: HeLa DNA Damage Whole Cell Lysate Set: UV Treated and Untreated Control (ab157396)

Predicted band size: 53 kDa

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