

Immunotag™ p53 (Acetyl Lys370) Polyclonal Antibody

| Antibody Specification | |
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| Catalog No. | ITK0035 |
| Product Description | Immunotag™ p53 (Acetyl Lys370) Polyclonal Antibody |
| Size | 50 µg, 100 µg |
| Conjugation | HRP, Biotin, FITC, Alexa Fluor® 350, Alexa Fluor® 405, Alexa Fluor® 488, Alexa Fluor® 555, Alexa Fluor® 647 |
| IMPORTANT NOTE | This product is custom manufactured with a lead time of 3-4 weeks. Once in production, this item cannot be returned for return. |
| Target Protein | p53 (Lys38000) |
| Clonality | Polyclonal |
| Storage/Stability | -20°C/1 year |
| Application | WB,IHC-p,ELISA |
| Recommended Dilution | Western Blot: 1/500 - 1/2000. IHC-p: 1:100-300 ELISA: 1/20000. Not yet tested in other applications. |
| Concentration | 1 mg/ml |
| Reactive Species | Human |
| Host Species | Rabbit |
| Immunogen | The antiserum was produced against synthesized Acetyl-peptide derived from human p53 around the Acetyl Lys370 |
| Specificity | Acetyl-p53 (K370) Polyclonal Antibody detects endogenous levels of p53 protein only when acetylated |
| Purification | The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific resin |
| Form | Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide. |
| Gene Name | TP53 |
| Accession No. | P04637 P02340 |
| Alternate Names | TP53; P53; Cellular tumor antigen p53; Antigen NY-CO-13; Phosphoprotein p53; Tumor suppressor p53 |

Antibody Specification

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| Description | tumor protein p53(TP53) Homo sapiens This gene encodes a tumor suppressor protein containing trans oligomerization domains. The encoded protein responds to diverse cellular stresses to regulate expression arrest, apoptosis, senescence, DNA repair, or changes in metabolism. Mutations in this gene are associated with hereditary cancers such as Li-Fraumeni syndrome. Alternative splicing of this gene and the use of alternative promoters generate multiple variants and isoforms. Additional isoforms have also been shown to result from the use of alternate transcripts (ENST00000209372, ENST00000209377). [provided by RefSeq, Feb 2013], |
| Cell Pathway/ Category | MAPK_ERK_Growth,MAPK_G_Protein,Cell_Cycle_G1S,Cell_Cycle_G2M_DNA,p53,Apoptosis_Inhibition,Apoptosis,Neurotrophin, T CELLNeurotrophin,Amyotrophic lateral sclerosis (ALS),Huntington's disease,Pathways in cancer,Colon cancer,Glioma,Prostate cancer,Thyroid cancer,Basal cell carcinoma,Melanoma,Bladder cancer,Chronic obstructive pulmonary disease, cell lung cancer, |
| Protein Expression | Blood,Brain,Classical Hodgkin Lymphoma,Esophageal squamous cell carcinoma,Glial cell,Glial |
| Subcellular Localization | nuclear chromatin,nucleus,nucleoplasm,replication fork,transcription factor TFIID complex,nucleolus,cytoplasm,mitochondrion, matrix,endoplasmic reticulum,cytosol,integral component of membrane,nuclear matrix, |

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Protein Function

cofactor: Binds 1 zinc ion per subunit., disease: Defects in TP53 are a cause of choroid plexus papilloma [MIM:102500], a growing benign tumor of the choroid plexus that often invades the leptomeninges. In children it is usually found in the fourth ventricle. Hydrocephalus is common, either from obstruction or from tumor secretion. After malignant transformation it is called a choroid plexus carcinoma. Primary choroid plexus tumors are rare and usually occur in children. TP53 are a cause of Li-Fraumeni syndrome (LFS) [MIM:151623]. LFS is an autosomal dominant familial cancer syndrome caused by the existence of a proband affected by a sarcoma before 45 years with a first degree relative affected by a sarcoma or a second degree relative with any tumor before 45 years or a sarcoma at any age. Other clinical definitions for LFS are given in PubMed:8718514 and called Li-Fraumeni like syndrome (LFL). In these families affected relatives develop tumors at very young ages. Four types of cancers account for 80% of tumors occurring in TP53 germline mutation carriers: sarcomas, brain tumors (astrocytomas) and adrenocortical carcinomas. Less frequent tumors include childhood leukemia before the age of 15, rhabdomyosarcoma before the age of 5, leukemia, Wilms tumor, malignant phyllodes tumor of the breast. TP53 are a cause of lung cancer [MIM:211980]., disease: Defects in TP53 are a cause of one form of hereditary retinoblastoma [MIM:202300]. ADCC is a rare childhood tumor, representing about 0.4% of childhood tumors, with a high incidence in patients with increased frequency in patients with the Beckwith-Wiedemann syndrome [MIM:130650] and is a cause of childhood cancer [MIM:151623]., disease: Defects in TP53 are found in Barrett metaplasia; also known as Barrett esophagus. In Barrett esophagus the squamous epithelium of the lower esophagus is replaced by a metaplastic columnar epithelium. The condition affects approximately 10% of patients with chronic gastroesophageal reflux disease and predisposes to the development of esophageal adenocarcinoma., disease: Defects in TP53 are involved in esophageal squamous cell carcinoma (ESCC) of the esophagus., disease: Defects in TP53 are involved in head and neck squamous cell carcinomas (HNSCC) of the head and neck., involved in oral squamous cell carcinoma (OSCC). Cigarette smoke is a prime mutagenic agent in cancer. TP53 may be associated with nasopharyngeal carcinoma [MIM:161550]; also known as nasopharyngeal carcinoma. TP53 is found in large amounts in a wide variety of transformed cells. TP53 is frequently mutated or inactivated in about 60% of human cancers. TP53 acts as a transcriptional repression domain., function: Acts as a tumor suppressor in many tumor types; induces growth arrest or apoptosis under physiological circumstances and cell type. Involved in cell cycle regulation as a trans-activator that acts to positively regulate a set of genes required for this process. One of the activated genes is an inhibitor of cyclin-dependent kinase activity. TP53 is mediated either by stimulation of BAX and FAS antigen expression, or by repression of Bcl-2 expression. TP53 is involved in many tumor types; induces growth arrest or apoptosis depending on the physiological circumstances and cell type. TP53 is a transcriptional activator that acts to negatively regulate cell division by controlling a set of genes required for this process. TP53 inhibits 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., online information: P53 entry, online information: The P53 database, online information: The Singapore human mutation and polymorphism database, PTM: Acetylation of Lys-382 increases transcriptional activity. Deacetylation of Lys-382 by SIRT1 impairs its ability to induce proapoptotic proteins and cell cycle arrest., PTM: Demethylation of di-methylated Lys-370 by KDM1/LSD1 prevents interaction with TP53 and inhibits transcriptional activation., PTM: Dephosphorylated by PP2A. SV40 small T antigen inhibits the dephosphorylation of Lys-382 and is glycosylated in the C-terminal basic region. Studied in EB-1 cell line., PTM: Monomethylated at Lys-372 by SMYD2, leading to decrease DNA-binding activity. Monomethylated at Lys-370 by SMYD2, leading to decrease DNA-binding activity. Lys-372 monomethylation prevents the interaction with SMYD2 and subsequent monomethylation of Lys-370. Lys-372 residues mediate transcriptional activation. Phosphorylated by HIPK1 (By similarity). Phosphorylation of Lys-382 inhibits the BIRC5 promoter. Phosphorylated on Thr-18 by VRK1, which may prevent the interaction with MDM2. Phosphorylation of Lys-382 prevents MDM2-mediated degradation. Phosphorylated on Ser-46 by HIPK2 upon UV irradiation. Phosphorylation of Lys-382 prevents MDM2-mediated degradation. Phosphorylated on Ser-392 following UV but not gamma irradiation. Phosphorylated upon DNA damage by HIPK2. Phosphorylated on Ser-15 upon ultraviolet irradiation; which is enhanced by interaction with BANP., PTM: Sumoylated by SUMO1, leading to proteasomal degradation., similarity: Belongs to the p53 family., subcellular location: Interaction with BANP. TP53 binds to DNA as a homotetramer., subunit: Interacts with AXIN1. Probably part of a complex consisting of TP53, HIF1, and AXIN1. TP53 is a homotetramer. Interacts with histone acetyltransferases EP300 and methyltransferases HRMT1L2 and DNMT3A. The interaction of TP53 with cancer-associated/HPV (E6) viral proteins leads to ubiquitination and degradation of TP53 and growth regulation. This complex formation requires an additional factor, E6-AP, which stably associates with TP53 (C-terminus) with TAF1; when TAF1 is part of the TFIID complex. Interacts with ING4; this interaction may be regulated by phosphorylation of TP53. Interacts with HIPK1, HIPK2, and P53DINP1. Interacts with WWOX. May interact with HCV core protein. Interacts with HSP90AB1. Interacts with CHD8; leading to recruit histone H1 and prevent transactivation activity. Interacts with CDKN2AIP and E4F1. Interacts with YWHAZ; the interaction enhances P53 transcriptional activity. Phosphorylation of Lys-382 prevents interaction. Interacts (via DNA-binding domain) with MAML1 (via N-terminus).,

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Usage

For Research Use Only! Not for diagnostic or therapeutic procedures.