

Immunotag™ p53 (phospho Ser315) Polyclonal Antibody

Antibody Specification	
Catalog No.	ITP0207
Product Description	Immunotag™ p53 (phospho Ser315) 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® 594, 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 cancelled from an order and is not eligible for return.
Target Protein	p53 (Ser315)
Clonality	Polyclonal
Storage/Stability	-20°C/1 year
Application	WB,IHC-p,IP,ELISA
Recommended Dilution	Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunoprecipitation: 2-5 ug/mg lysate. ELISA: 1/10000. Not yet tested in other applications.
Concentration	1 mg/ml
Reactive Species	Human
Host Species	Rabbit
Immunogen	The antiserum was produced against synthesized peptide derived from human p53 around the phosphorylation site of Ser315. AA range:281-330
Specificity	Phospho-p53 (S315) Polyclonal Antibody detects endogenous levels of p53 protein only when phosphorylated at S315.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen
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

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Description	tumor protein p53(TP53) Homo sapiens This gene encodes a tumor suppressor protein containing transcriptional activation, DNA binding, and oligomerization domains. The encoded protein responds to diverse cellular stresses to regulate expression of target genes, thereby inducing cell cycle arrest, apoptosis, senescence, DNA repair, or changes in metabolism. Mutations in this gene are associated with a variety of human cancers, including hereditary cancers such as Li-Fraumeni syndrome. Alternative splicing of this gene and the use of alternate promoters result in multiple transcript variants and isoforms. Additional isoforms have also been shown to result from the use of alternate translation initiation codons (PMIDs: 12032546, 20937277). [provided by RefSeq, Feb 2013],
Cell Pathway/ Category	Stem cell pathway, WNT,WNT-T CELL,β-Catenin, SAPK_JNK, AMPK, Cell_Cycle_G1S,Cell_Cycle_G2M_DNA, MAPK_ERK_Growth,MAPK_G_Protein, PI3K/Akt, Protein_Acetylation
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,mitochondrial 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: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., disease: 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 of 5, leukemia, Wilms tumor, malignant phyllodes tumor, colorectal and gastric cancers., disease: Defects in TP53 are a cause of lung cancer [MIM:211980]., disease: Defects in TP53 are a cause of one form of hereditary adrenocortical carcinoma (ADCC) [MIM:202300]. ADCC is a rare childhood tumor, representing about 0.4% of childhood tumors, with a high incidence of associated tumors. ADCC occurs with increased frequency in patients with the Beckwith-Wiedemann syndrome [MIM:130650] and is a component tumor in Li-Fraumeni syndrome [MIM:151623]., disease: Defects in TP53 are found in Barrett metaplasia; also known as Barrett esophagus. It is 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., disease: Defects in TP53 are involved in esophageal squamous cell carcinoma (ESCC) [MIM:133239]. ESCC is a tumor of the esophagus., disease: Defects in TP53 are involved in head and neck squamous cell carcinomas (HNSCC) [MIM:275355]., disease: Defects in TP53 are involved in oral squamous cell carcinoma (OSCC). Cigarette smoke is a prime mutagenic agent in cancer of the aerodigestive tract., disease: Defects in TP53 may be associated with nasopharyngeal carcinoma [MIM:161550]; also known as nasopharyngeal cancer., disease: TP53 is found in increased amounts in a wide variety of transformed cells. TP53 is frequently mutated or inactivated in about 60% of cancers., domain: The nuclear export signal acts as a transcriptional repression domain., 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., 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., online information: P53 entry, online information: Somatic and germline TP53 mutations in human cancers, online information: The Singapore human mutation and polymorphism database, PTM: 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., PTM: Demethylation of di-methylated Lys-370 by KDM1/LSD1 prevents interaction with TP53BP1 and represses TP53-mediated transcriptional

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Usage

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