p53 Rabbit pAb

CatalogNo: YT3528 KD/KO Validated

Orthogonal Validated Comparable Abs

Key Features

Host Species
- Rabbit

Reactivity
- Human,Mouse,Rat,Monkey

Applications
- WB,IHC,IF,ELISA

MW
- 53kD (Observed)

Isotype
- IgG

Storage

Storage*	-15°C to -25°C/1 year (Do not lower than -25°C)
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Recommended Dilution Ratios

WB 1:500-1:2000
IHC 1:100-1:300
IF 1:200-1:1000
ELISA 1:10000
Not yet tested in other applications.

Basic Information

Clonality	Polyclonal

Immunogen Information

Immunogen	The antiserum was produced against synthesized peptide derived from human p53. AA range:10-59
Specificity	p53 Polyclonal Antibody detects endogenous levels of p53 protein.

Target Information

Gene name TP53 P53

Protein Name Antigen NY-CO-13;BCC7;Cellular tumor antigen p53;FLJ92943;LFS1;Mutant tumor protein 53;p53;p53 tumor suppressor;P53_HUMAN;Phosphoprotein p53;Tp53;Transformation related protein 53;TRP53;tumor antigen p55;Tumor protein 53;Tumor protein p53;Tumor suppressor p53

Organism	Gene ID	UniProt ID
Human	7157;	P04637;
Mouse	22059;	P02340;
Rat	24842;	P10361;

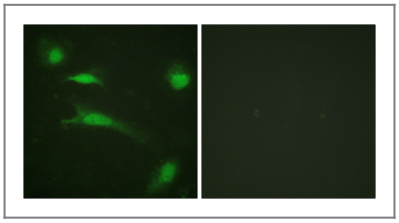
Cellular Localization Nuclear

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.

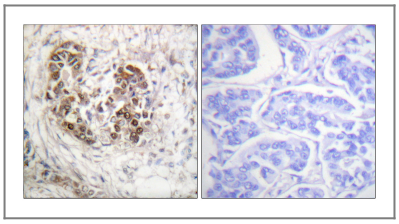
Function

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 activation. ,PTM:Dephosphorylated by PP2A. SV40 small T antigen inhibits the dephosphorylation by the AC form of PP2A. ,PTM:May be O-glycosylated in the C-terminal basic region. Studied in EB-1 cell line. ,PTM:Monomethylated at Lys-372 by SETD7 , leading to stabilize it and increase transcriptional activation. Monomethylated at Lys-370 by SMYD2 , leading to decrease DNA-binding activity and subsequent transcriptional regulation activity. Lys-372 monomethylation prevents the interaction with SMYD2 and subsequenct monomethylation at Lys-370. ,PTM: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 , which may prevent the interaction with MDM2. Phosphorylated on Thr-55 by TAF1 , which promotes MDM2-mediated degradation. Phosphorylated on Ser-46 by HIPK2 upon UV irradiation. Phosphorylation on Ser-46 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. ,PTM:Sumoylated by SUMO1. ,PTM:Ubiquitinated by SYVN1 , which leads to proteasomal degradation. ,similarity:Belongs to the p53 family. ,subcellular location:Interaction with BANP promotes nuclear localization. ,subunit:Binds DNA as a homotetramer. ,subunit:Interacts with AXIN1. Probably part of a complex consisting of TP53 , HIPK2 and AXIN1 (By similarity) . Binds DNA as a homotetramer. Interacts with histone acetyltransferases EP300 and methyltransferases HRMT1L2 and CARM1 , and recruits them to promoters. In vitro , the interaction of TP53 with cancer-associated/HPV (E6) viral proteins leads to ubiquitination and degradation of TP53 giving a possible model for cell growth regulation. This complex formation requires an additional factor , E6-AP , which stably associates with TP53 in the presence of E6. Interacts (via C-terminus) with TAF1; when TAF1 is part of the TFIID complex. Interacts with ING4; this interaction may be indirect. Found in a complex with CABLES1 and TP73. Interacts with HIPK1 , HIPK2 , and P53DINP1. Interacts with WWOX. May interact with HCV core protein. Interacts with USP7 and SYVN1. Interacts with HSP90AB1. Interacts with CHD8; leading to recruit histone H1 and prevent transactivation activity (By similarity) . Interacts with ARMC10 , BANP , CDKN2AIP and E4F1. Interacts with YWHAZ; the interaction enhances P53 transcriptional activity. Phosphorylation of YWHAZ on 'Ser-58' inhibits this interaction. Interacts (via DNA-binding domain) with MAML1 (via N-terminus) . ,

Validation Data



Immunofluorescence analysis of HeLa cells, using p53 Antibody. The picture on the right is blocked with the synthesized peptide.



Immunohistochemistry analysis of paraffin-embedded human breast carcinoma tissue, using p53 Antibody. The picture on the right is blocked with the synthesized peptide.

Contact information

Orders:	order@immunoway.com
Support:	tech@immunoway.com
Telephone:	877-594-3616 (Toll Free), 408-747-0185
Website:	http://www.immunoway.com
Address:	2200 Ringwood Ave San Jose, CA 95131 USA

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p53 Rabbit pAb

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