

# Inhibiting the tumor cell growth by expressing the p53 protein and its interplay with other disease

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## ABSTRACT

**p53 is immunological factor that performed by apoptotic mechanism. In most of cancer the mutation happens into p53 protein i.e. 'mutant p53' and p53 consider as one of the tumor suppresser protein. It plays critical role in prevent DNA fidelity and DNA repair mechanism, through the regulation of cell cycle. The p53 perform apoptotic mechanism to inhibit the tumor cells growth and also performed phagocytosis after engulfing the tumor cell and pinocytosis at the time of releasing the granuals outside the cells. So in this article we will focus on how the p53 inhibits the tumor cells by degradation and its regulation.**

**Key words: p53, tumor suppresser protein, disease interplay, degradation, regulation.**

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## INTRODUCTION

Tumor suppressor protein p53, additionally referred to as p53, cell tumor antigen p53, the Guardian of the Genome, (Toufekhtan, E.; Toledo, F. (2018). phosphoprotein p53, tumor suppressor p53, antigen NY-CO-13, or transformation-associated protein 53 (TRP53), is any isoform of a protein encoded via way of means of homologous genes in diverse organisms, along with P53 (humans) and Trp53 (mice). This homolog (at the beginning concept to be, and frequently spoken of as, a unmarried protein) is vital in multicellular vertebrates, in which it prevents most cancers formation, and hence features as a tumor suppressor. ( Surget S, Khoury MP, Bourdon JC 2013). As such, p53 has been defined as "the mum or dad of the genome" due to its position in keeping balance via way of means of stopping genome mutation (AP, Strachan T (1999) Hence P53 is classed as a tumor suppressor gene ( McBride OW, Merry D, Givol D 1986). The call p53 changed into given in 1979 describing the obvious molecular mass; SDS-PAGE evaluation shows that it's far a 53-kilodalton (kDa) protein. However, the real mass of the full-duration p53 protein (p53 $\alpha$ ) primarily based totally at the sum of loads of the amino acid residues is simplest 43.7 kDa. This distinction is because of the excessive range of proline residues withinside the protein, which sluggish its migration on SDS-PAGE, hence making it seem heavier than it without a doubt is.

In addition to the full-duration protein, the human P53 gene encodes as a minimum 15 protein isoform, ranging in length from 3.5 to 43.7 kDa. All those p53 proteins are referred to as the p53 isoform ( Surget S, Khoury MP, Bourdon JC 2013). The P53 gene is the maximum often mutated gene (>50%) in human most cancers, indicating that the P53 gene performs a vital position in stopping most cancers formation. (Surget S, Khoury MP, Bourdon JC (December 2013) P53 gene encodes proteins that bind to DNA and alter gene expression to save you mutations of the genome ( Levine AJ, Lane DP, eds. 2010). In humans, the P53 gene is positioned on the quick arm of chromosome 17 (17p13.1) ( Kern SE, Kinzler KW, Bruskin A, Jarosz D, Friedman P, Prives C, Vogelstein B 1991). The gene spans 20 kb, with a non-coding exon 1 and a completely lengthy first intron of 10 kb. The coding collection includes 5 areas displaying a excessive diploma of conservation in vertebrates, predominantly in exons 2, five, 6, 7 and 8, however the sequences located in invertebrates display simplest remote resemblance to mammalian P53 ( May P, May E 1999) P53 orthologs were recognized in maximum mammals for which whole genome records are available.

In humans, as nicely not unusual place polymorphism includes the substitution of an arginine for a proline at codon role seventy two. Many researches have investigated a genetic hyperlink among this alteration and most cancers susceptibility; however, the outcomes were controversial. For instance, a meta-evaluation from 2009 failed to expose a hyperlink for cervical most cancers (Klug SJ, Rensing M, Koenig J, Abba MC, Agorastos T, Brenna SM, et al. 2009). A

2011 have a look at located that the P53 proline mutation did have a profound impact on pancreatic most cancers hazard amongst males ( Sonoyama T, Sakai A, Mita Y, Yasuda Y, Kawamoto H, Yagi T, Yoshioka M, Mimura T, Nakachi K, Ouchida M, Yamamoto K, Shimizu K 2011). A have a look at of Arab ladies located that proline homozygosity at P53 codon seventy two is related to a reduced hazard for breast most cancers( Alawadi S, Ghabreau L, Alsaleh M, Abdulaziz Z, Rafeek M, Akil N, Alkhalaf M 2011). One greater have a look at cautioned that P53 codon seventy two polymorphisms, MDM2 SNP309, and A2164G can also additionally together be related to non-oropharyngeal most cancers susceptibility and that MDM2 SNP309 in aggregate with P53 codon seventy two can also additionally boost up the improvement of non-oropharyngeal most cancers in ladies ( Yu H, Huang YJ, Liu Z, Wang LE, Li G, Sturgis EM, Johnson DG, Wei Q 2011) A 2011 have a look at located that P53 codon seventy two polymorphism changed into related to an accelerated hazard of lung most cancers( Piao JM, Kim HN, Song HR, Kweon SS, Choi JS, Yun WJ, Kim YC, Oh IJ, Kim KS, Shin MH 2011).

**Assembly of P53:** An acidic N-terminus transcription-activation area (TAD), additionally referred to as activation area 1 (AD1), which turns on transcription factors. The N-terminus includes complementary transcriptional activation domains, with a first-rate one at residues 1–forty two and a minor one at residues 55–75, mainly worried withinside the law of numerous pro-apoptotic genes ( Venot C, Maratrat M, Dureuil C, Conseiller E, Bracco L, Debussche L 1998).Activation area 2 (AD2) vital for apoptotic interest: residues 43–63. proline wealthy area vital for the apoptotic interest of p53 via way of means of nuclear exportation thru MAPK residues- 64–92. critical DNA-binding center area (DBD). Contains one zinc atom and numerous arginine amino acids-residues 102–292. This area is answerable for binding the p53 co-repressor LMO3 ( Larsen S, Yokochi T, Isogai E, Nakamura Y, Ozaki T, Nakagawara A 2010). Nuclear Localization Signaling (NLS) area, residues 316–325. C-terminal worried in downregulation of DNA binding of the critical area- residues 356–393 ( Harms KL, Chen X 2005). Mutations that deactivate p53 in most cancers commonly arise withinside the DBD. Most of those mutations ruin the capability of the protein to bind to its goal DNA sequences, and hence prevents transcriptional activation of those genes. As such, mutations withinside the DBD are recessive loss-of-characteristic mutations. Molecules of p53 with mutations withinside the OD dimerise with wild-kind p53, and save you them from activating transcription. Therefore, OD mutations have a dominant poor impact at the characteristic of p53.

**Role in different disease:** If the P53 gene is damaged, tumor suppression is significantly compromised. People who inherit best one purposeful replica of theP53 gene will maximum probable broaden tumors in early adulthood, a sickness called Li-Fraumeni syndrome. The P53 gene also can be changed through mutagens (chemicals, radiation, or viruses), growing the chance for out of control mobileular department. More than 50 percentage of human tumors comprise a mutation or deletion of the P53 gene (Hollstein M, Sidransky D, Vogelstein B, Harris CC 1991) Loss of p53 creates genomic instability that most customarily outcomes in an aneuploidy phenotype (Schmitt CA, Fridman JS, Yang M, Baranov E, Hoffman 2002). Increasing the quantity of p53 might also additionally appear an answer for remedy of tumors or prevention in their spreading. This, however, isn't always a usable technique of remedy, considering the fact that it is able to purpose untimely aging (Bradley A, Donehower LA. 2002). Restoring endogenous everyday p53 characteristic holds a few processes. Research has proven that this recovery can result in regression of sure most cancers cells without adverse different cells withinside the process. The approach through which tumor regression happens relies upon especially at the tumor kind. For example, recovery of endogenous p53 characteristic in lymphomas might also additionally set off apoptosis, whilst mobileular boom can be decreased to everyday Thus, pharmacological reactivation of p53 provides itself as a possible most cancers remedy option ( Herce HD, Deng W, Helma J, Leonhardt H, Cardoso MC) The first industrial gene therapy, Gencicine, become accredited in China in 2003 for the remedy of head and neck squamous It promises a purposeful replica of the p53 gene the use of an engineered adenovirus ( Pearson S, Jia H, Kandachi K. 2004).

**Pathogens incineration:** It also can have an effect on the p53 protein that the P53 gene expresses. One such example, human papillomavirus (HPV), encodes a protein, E6, which binds to the p53 protein and inactivates it. This mechanism, in synergy with the inactivation of the mobileular cycle regulator pRb through the HPV protein E7, permits for repeated mobileular department manifested clinically as warts. Certain HPV kinds, particularly kinds sixteen and 18, also can result in development from a benign wart to low or excessive-grade cervical dysplasia, which might be reversible sorts of Persistent contamination of the cervix through the years can purpose irreversible modifications main to carcinoma in situ and finally invasive cervical most cancers. This outcomes from the consequences of HPV genes, especially the ones encoding E6 and E7, which might be the 2 viral oncoproteins which can be preferentially retained and expressed in cervical cancers through integration of the viral DNA into the host genome (Angeletti PC, Zhang L, Wood 2000). The p53 protein is usually produced and degraded in cells of wholesome people, ensuing in damped oscillation. The degradation of the p53 protein is related to binding of MDM2. In a bad remarks loop, MDM2 itself is triggered through the p53 protein. Mutant p53 proteins regularly fail to set off MDM2, inflicting p53 to build up at very excessive levels.

Moreover, the mutant p53 protein itself can inhibit everyday p53 protein levels. In a few instances, unmarried missense mutations in p53 were proven to disrupt p53 balance and characteristic (Fersht AR 1997).

### Suppression of p53:

suppression of p53 in human breast most cancers cells is proven to result in accelerated CXCR5 chemokine receptor gene expression and activated mobileular migration in reaction to (Mitkin NA, Hook CD, Schwartz AM, Biswas S, Kochetkov DV, Muratova AM, Afanasyeva MA, Kravchenko JE, Bhattacharyya A, Kuprash DV 2015).

**Mutation inactivation of p53 protein:** mutation takes place in genes it become a not unusual place nuclear mechanism in the back of the disfunction of p53 protein. In maximum of most cancers instances there may be considerable mutation of p53 protein. Among them 95% of mutation have been determined inside genomic vicinity encoding the DNA binding area (Vousden, K.H, Lu X. 2002). In this mutation maximum of applicable amino acids which reasons to most cancers consisting of Arg-175, Gly-245, Arg-249, Arg-273, and Arg-282 (Joerger, A.C.; Fersht, A.R. 2007). This mutation discovered in DNA binding area of p53 protein and disrupt the conformation consequently it powerful in collection precise transcriptional activation dependant of untamed kind p53 binding element.

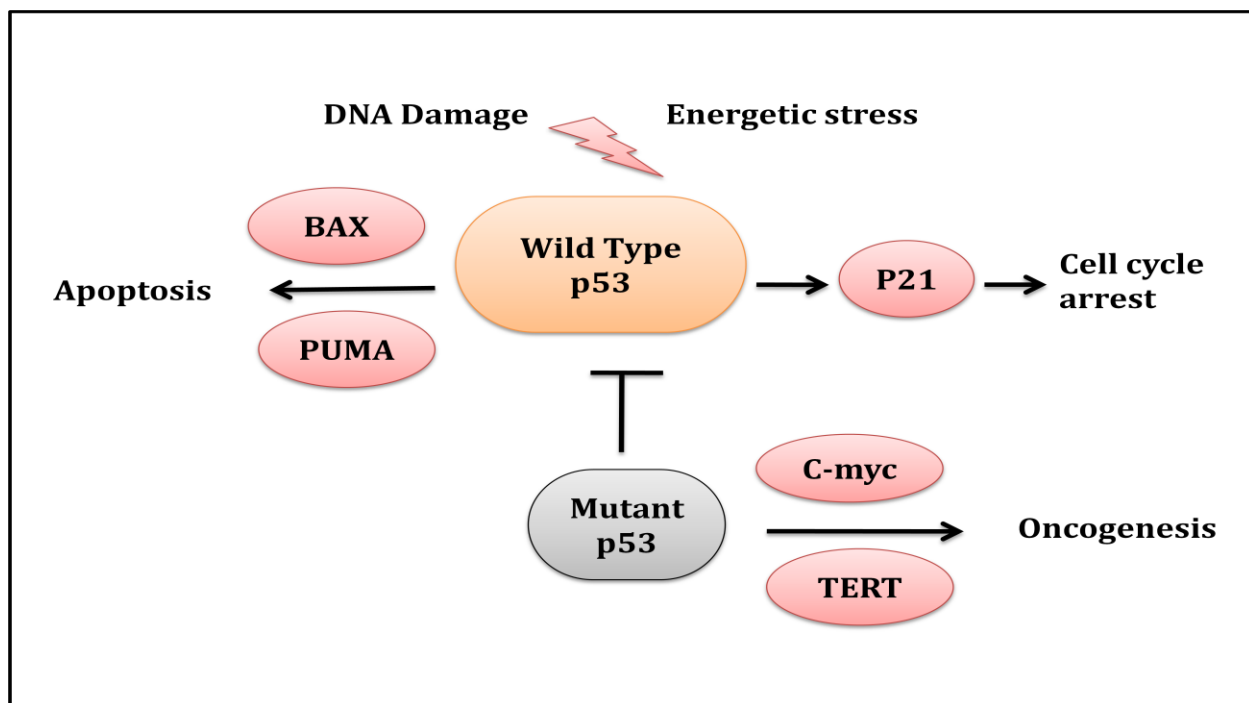


Fig.1. showing how the mutant p53 will dominant over the wild-type p53 and express the apoptosis and oncogenesis condition (Toshinori Ozaki 1 and Akira Nakagawara 2011)

The gathering proof proven that sure most cancers-derived mutant sorts of p53Transactivate diverse goal genes consisting of the more than one drug resistance gene 1 (MDR1), c-myc, proliferating mobileular nuclear antigen (PCNA), interleukin-6 (IL-6), insulin-like boom component 1 (IGF-1), fibroblast boom component (FGF) and epidermal boom component receptor (EGFR). ) discovered that most cancers-derived mutant p53 transactivate asparagine synthetase (ASNS) and telomerase opposite transcriptase (TERT) (Scian, M.J.; Stagliano, K.E.; Deb, D. 2004). Therefore, it's miles probable that sure most cancers-derived p53 mutants transactivate boom-selling and oncogenic genes, thereby main to the development of the competitive cancers Since the mutation look for p53 targeted at the genomic vicinity encoding the DNA-binding area, there ought to nonetheless be unidentified lack of characteristic mutations outdoor the DNA-binding area (Vousden, K.H.; Lu, X. 2002). Lomax et al. discovered factor mutations (L344P and R337C) withinside the COOH-terminal oligomerization area of p53. According to their outcomes, p53 bearing L344P mutation existed because the monomeric shape and lacked the transactivation capacity. On the opposite hand, p53 wearing R377C mutation shaped the tetramer, while this mutant displayed the notably decreased transcriptional and pro-apoptotic activities. Di-Giammarino et al. stated the presence of a factor mutation (R337H) withinside the COOH-terminal oligomerization area . p53 bearing R337H mutation shaped the native-like tetramer, however, its balance become notably decrease than that of untamed-kind p53. missing part of the oligomerization area

and nuclear localization indicators in human neuroblastoma-derived mobileular lines (Nakamura, Y.; Ozaki. 2000). We have discovered p53 based on our outcomes, p53 in large part expressed in cytoplasm and had notably decrease pro-apoptotic capacity in comparison with wild-kind p53.

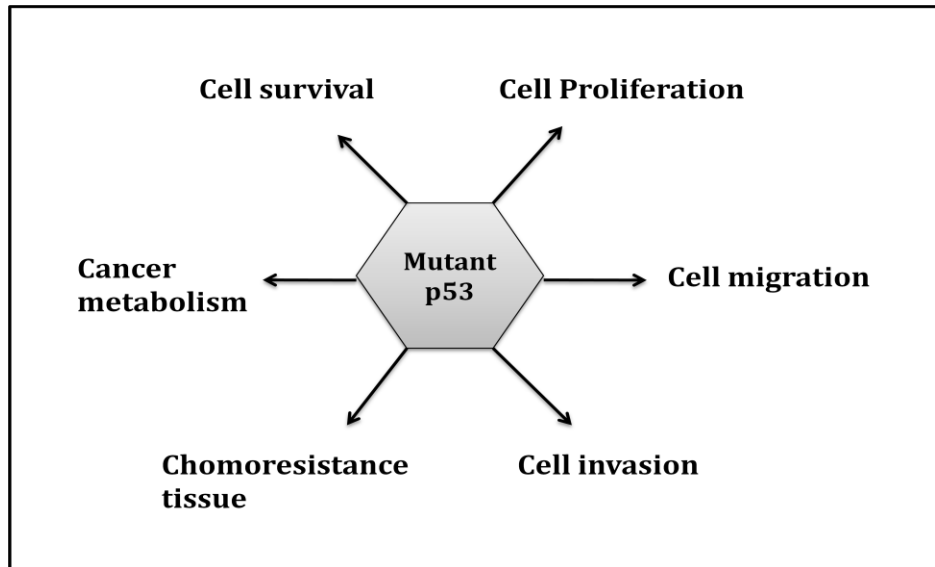


Fig.2. its showing the mutant p53 will regulating and expressing on these factor.

**Degradation of tumor cell by expressing p53 protein:**

P53 work as DNA repair and in apoptosis mechanism to eliminate the tumor cells. It prevent the replication of damage DNA and invansion it through the apoptosis mechanism. During the growth of tumor cell the p53 arrest that protein which activates transcription of that protein that involve in DNA repair mechanism.

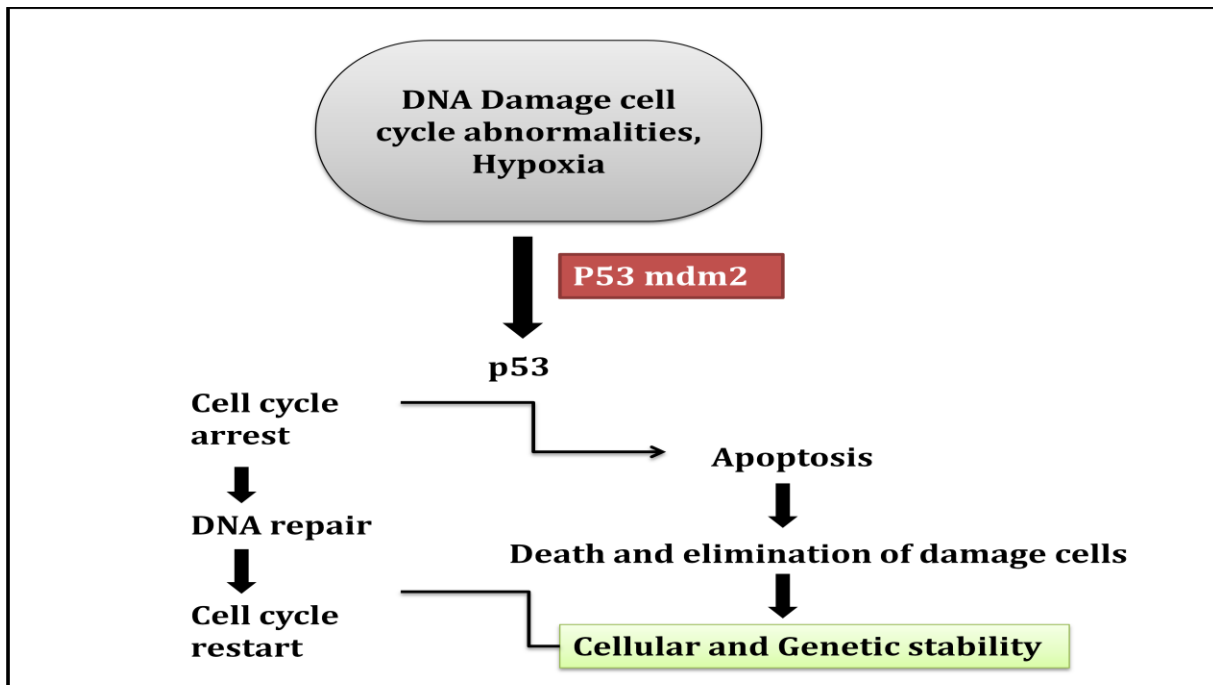


Fig.3. p53 pathway: In a normal cell, p53 is inactivated by its negative regulator, mdm2. Upon DNA damage or other stresses, various pathways will lead to the dissociation of the p53 and mdm2 complex. Once activated, p53 will induce a cell cycle arrest to allow either repair or survival of the cell or apoptosis to discard the damaged cell. How p53 makes this choice is currently unknown (Thierry Soussi 2007).

**Invasion of cancer cells:** Invasion of most cancers cells via the basement membrane right into a blood or lymphatic vessel. (Tsuji, Takanori; Soichiro Ibaragi; Guo-Fu Hu 2009). Intravasation is certainly considered one among numerous carcinogenic activities that provoke the break out of cancerous cells from their number one webweb sites. Other mechanisms consist of invasion via basement membranes, extravasation, and colonization of remote metastatic webweb sites. Cancer mobile chemotaxis additionally is predicated in this migratory conduct to reach at a secondary vacation spot targeted for most cancers mobile colonization. One of the genes that contributes to intravasation codes for urokinase (uPA), a serine protease this is capable of proteolytically degrade diverse extracellular matrix (ECM) additives and the basement membrane round number one izumi, Megumi; Wen Liu; Sudha K Pai; Eiji Furuta; Kounosuke Watabe 2008 ) uPA additionally turns on a couple of increase elements and matrix metalloproteinases (MMPs) that similarly make a contribution to ECM degradation, as a consequence permitting tumor mobile invasion and A newly recognized metastasis suppressor, p75 neurotrophin receptor (p75NTR), is capable of suppress metastasis in element with the aid of using inflicting unique proteases, inclusive (izumi, Megumi; Wen Liu; Sudha K Pai; Eiji Furuta; Kounosuke Watabe) Tumor-related macrophages (TAMs) were proven to be (izumi, Megumi; Wen Liu; Sudha K Pai; Eiji Furuta; Kounosuke Watabe 2008).

Studies have discovered that macrophages beautify tumor mobile migration and intravasation with the aid of using secreting chemotactic and chemokinetic elements, selling angiogenesis, transforming the ECM, and regulating the formation of collagen fibers. (Pollard, Jeffrey W. Groups of 3 mobile types (a macrophage, an endothelial mobile, and a tumor mobile) together called tumor microenvironment of metastasis (TMEM) can permit tumor cells to go into blood vessels. Tumors can use each energetic and passive strategies to go into vasculature (Bockhorn, Maximilian; Rakesh K Jain; Lance L. Munn 2007). Some research recommend that most cancers cells actively flow closer to blood or lymphatic vessels in reaction to nutrient or chemokine gradients (van Zijil, Franziska; Georg Krupitza; Wolfgang 2010) at the same time as others offer proof for the speculation that metastasis withinside the early degrees is greater accidental. In energetic intravasation, cancerous cells actively migrate towards after which into close by blood vessels.( Bockhorn, Maximilian; Rakesh K Jain; Lance L. Munn (May 2007). The first step on this system is unique adhesion to venular endothelial cells, accompanied with the aid of using adherence to proteins of the subendothelial basement membrane, inclusive of laminin and brands IV and V collagen.

The very last step is the adhesion of the metastatic tumor mobile to connective tissue factors inclusive of fibronectin, kind I collagen, and hyaluronan, that's required for the motion of the tumor mobile into the subendothelial stroma and next increase on the secondary webweb page of Passive intravasation refers to a system wherein tumors metastasize via passive shedding. Evidence for that is visible whilst the variety of tumor cells launched into the blood circulation will increase whilst the number one tumor stories trauma ( Liotta, L A; Sidel, M G; 1976). Additionally, cells developing in constrained areas were proven to push in opposition to every different inflicting blood and lymphatic vessels to collapse, probably forcing cells into the Apart from the cell and molecular outcomes above, p53 has a tissue-degree anticancer impact that works with the aid of using inhibiting angiogenesis. As tumors develop they want to recruit new blood vessels to deliver them, and p53 inhibits that with the aid of using;

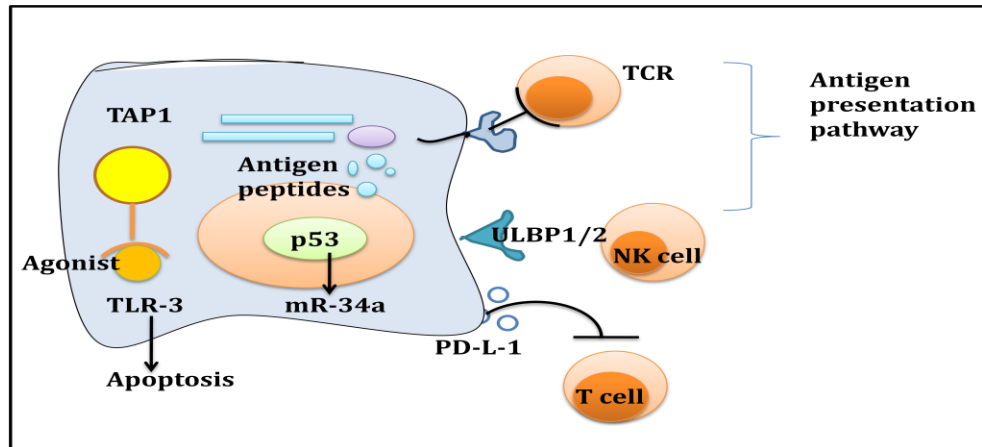
(i) Interfering with regulators of humor hypoxia that still have an effect on angiogenesis, inclusive of HIF1 and HIF2,

(ii) Inhibiting the manufacturing of angiogenic selling elements, and

(iii) Directly growing the manufacturing of angiogenesis p53 with the aid of using regulating Leukemia Inhibitory Factor has been proven to facilitate implantation withinside the mouse and probably human beings reproduction.

**Regulation of p53 protein into tumor degradation with the aid of using immune reaction:**

p53 will become activated in reaction to myriad stressors, together with however now no longer restricted to DNA harm (prompted with the aid of using both UV, IR, or chemical dealers inclusive of hydrogen peroxide),oxidative strain (Van Remmen H, Richardson A. osmotic shock, ribonucleotide depletion, and deregulated oncogene expression. This activation is marked with the aid of using most important activities. First, the half-lifestyles of the p53 protein is elevated drastically, main to a short accumulation of p53 in burdened cells. Second, a conformational alternate forces p53 to be activated as a transcription regulator in those cells. The crucial occasion main to the activation of p53 is the phosphorylation of its N-terminal area.



**Fig.4. Functions of p53 in tumour cells that regulate interactions with the immune system. p53 regulates endogenous antigen presentation through transcriptional control of ERAP1 and TAP1. In addition, p53 regulates the expression of the NKG2D ligands ULBP1 and ULBP2, either positively as a transcriptional target or negatively through the upregulation of miR-34a. The miR34 family also represses PD-L1 expression, an inhibitor of T cell activity. TLR3 and TLR9 are transcriptional targets of p53 that promote agonist-induced cell death ( Wang S, Yang D, Lin J. 2009).**

The N-terminal transcriptional activation area incorporates a huge variety of phosphorylation web sites and may be taken into consideration because the number one goal for protein kinases transducing strain The protein kinases which can be recognised to goal this transcriptional activation area of p53 may be kind of divided into groups. A first institution of protein kinases belongs to theMAPK family (JNK1-3, ERK1-2, p38 MAPK), which is understood to reply to numerous varieties of strain, inclusive of membrane harm, oxidative strain, osmotic shock, warmth shock, etc. A 2nd institution of protein kinases (ATR, ATM, CHK1 and CHK2, DNA-PK, CAK, P53RK) is implicated withinside the genome integrity checkpoint, a molecular cascade that detects and responds to numerous varieties of DNA harm as a result of genotoxic strain. Oncogenes additionally stimulate p53 activation, mediated with the aid of using the protein p14ARF.

**Stress/unstressed cells activation:** p53 ranges are stored low via a non-stop degradation of p53. A protein known as Mdm2 (additionally known as HDM2 in humans), binds to p53, stopping its motion and transports it from the nucleus to the cytosol. Mdm2 additionally acts as an ubiquitin ligase and covalently attaches ubiquitin to p53 and therefore marks p53 for degradation with the aid of using the proteasome. However, ubiquitylation of p53 is reversible. On activation of p53, Mdm2 is likewise activated, putting in a remarks loop. P53 ranges can display oscillations (or repeated pulses) in reaction to positive stresses, and those pulses may be vital in figuring out whether or not the cells live on the stress, or die (Alexander; Lahav, Galit MI-sixty three binds to MDM2, reactivating p53 in conditions in which p53's feature has emerge as inhibited( Wang S, Yang D, Lin J. 2009) . A ubiquitin particular protease, USP7 (or HAUSP), can cleave ubiquitin off p53, thereby protective it from proteasome-structured degradation thru the ubiquitin ligase This is one approach with the aid of using which p53 is stabilized in reaction to oncogenic insults. USP42 has additionally been proven to deubiquitinate p53 and can be required for the capacity of p53 to reply to stress( Hock AK, Vigneron AM, Carter S, Ludwig RL, Vousden KH Recent studies has proven that HAUSP is in particular localized withinside the nucleus, aleven though a fragment of it is able to be discovered withinside the cytoplasm and mitochondria.

**Over expression of HAUSP effects in p53 stabilization;** However, depletion of HAUSP does now no longer end result to a lower in p53 ranges however instead will increase p53 ranges because of the truth that HAUSP binds and deubiquitinates It has been proven that HAUSP is a higher binding companion to Mdm2 than p53 in unstressed cells. USP10 but has been proven to be positioned withinside the cytoplasm in unstressed cells and ubiquitinates cytoplasmic p53, reversing Mdm2 ubiquitination. Following DNA damage, USP10 translocates to the nucleus and contributes to p53 balance. Also USP10 does now no longer have interaction with Mdm2 ( Yuan J, Luo K, Zhang L, Chevillie JC, Lou Z. 2010). Phosphorylation of the N-terminal stop of p53 with the aid of using the above-referred to protein kinases disrupts Mdm2-binding. Other proteins, which include Pin1, are then recruited to p53 and set off a conformational alternate in p53, which prevents Mdm2-binding even more. Phosphorylation additionally permits for binding of transcriptional coactivators, like p300 and PCAF, which then acetylate the carboxy-terminal stop of p53, exposing the DNA binding area of p53, permitting it to spark off or repress particular Deacetylase enzymes, which include Sirt1

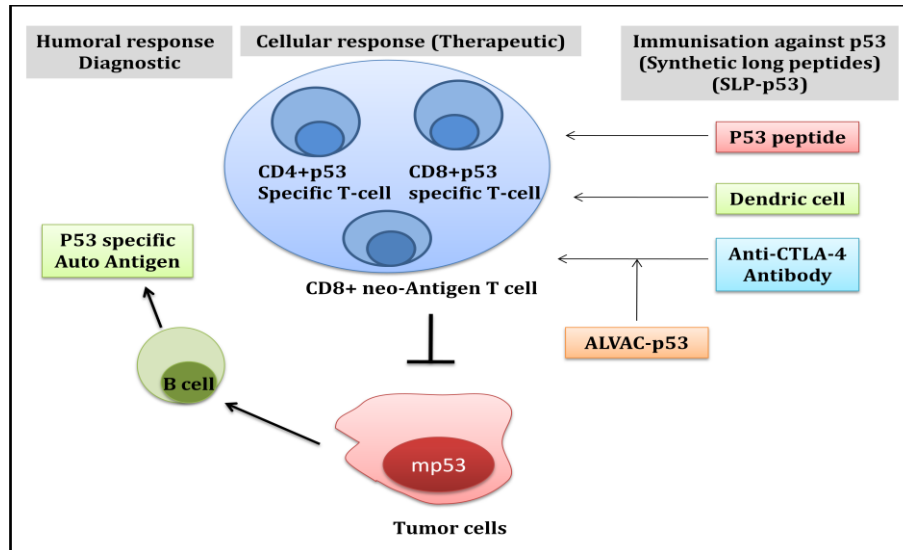
and Sirt7, can deacetylate p53, main to an inhibition of apoptosis. Some oncogenes also can stimulate the transcription of proteins that bind to MDM2 and inhibit its activity.

**P53 in stomatal populace throughout tumor formation and development:**

The tumour–stromal community is a heterogeneous populace of cells, originating from mesenchymal or lymphoid origins, that without delay or in a roundabout way have interaction with tumour cells (Mahadevan and Von Hoff, 2007; Pietras and Östman, 2010). While there was a focal point on how changes of p53 withinside the tumour cells make a contribution to most cancers progression, tumour cells expressing wild-kind p53 display improved boom while transplanted into p53-null hosts (Guo et al. , 2013), demonstrating a position for p53 withinside the non-most cancers-related stromal cells in modulating tumorigenesis.

**Immune recognition of p53 in most cancers cells:**

While wild-kind p53 ranges are very low in everyday cells, mutant p53 proteins have a tendency to build up at excessive ranges in most cancers cells. These observations enhance the opportunity that the tumour-particular expression of p53 ought to stimulate a B mobile (humoral) reaction, imparting diagnostic value, in addition to activating T cells that can be harnessed for vaccination.



**Fig.5. Immune responses to p53 expression in tumour cells (Wang S, Yang D, Lin J. 2009)**

p53 expression in most cancers cells can initiate immune recognition, maximum generally in reaction to the buildup of excessive ranges of mutant p53 protein. The presence of p53-particular autoantibodies may be detected in sufferers with quite a few cancers, which include colorectal, liver, ovarian, lung and breast cancers, and might have diagnostic value. p53 also can elicit T-mobile-particular responses each in CD4+ and CD8+ T cells, which include the ones recognising neo-antigens derived from point-mutated p53 proteins. Therapeutic processes the use of p53 vaccines or artificial lengthy peptides of p53 power T mobile responses, and feature proven a few efficacy in early medical trials. Vaccine-primarily based totally cures encompass use of ALVAC-p53 and MVAp53. Combination remedy with both CPG-ODN or anti-CLTA-four antibodies and MVAp53 elicits T mobile responses that could lessen tumour boom. SLP-p53 on my own and dendritic cells (DCs) pulsed with mutant p53 peptides also can power p53-particular T cell responses.

**CONCLUSION**

The feature of p53 throughout tumor formation and its depletion is studied on this article. We simply starts off evolved with the characters of p53 and it conduct via the tumor formation and its interaction with different sickness in brief and additionally if the mutation takes place at p53 protein then how it's going to suppress with the aid of using mobile cycle and apoptosis mechanism with the aid of using arresting oncogenic mobile When the mutation takes place it's going to modify and expressing those element Viz. mobile survival mobile proliferation, mobile migration, mobile invention Chomoresistance tissue and most cancers metabolism. P53 additionally enables into DNA-restore mechanism and set up cell and genetic balance with the aid of using expressing p53 pathway. While in law of immunological elements is

likewise invasion that p53 in tumor mobile that modify interplay with immune system, reaction to immune signaling and expression in tumor cells. The degradation of tumor cells is takes place with the aid of using phagocytosis mechanism and launched the degraded granuals with the aid of using pinocytosis for the duration.

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