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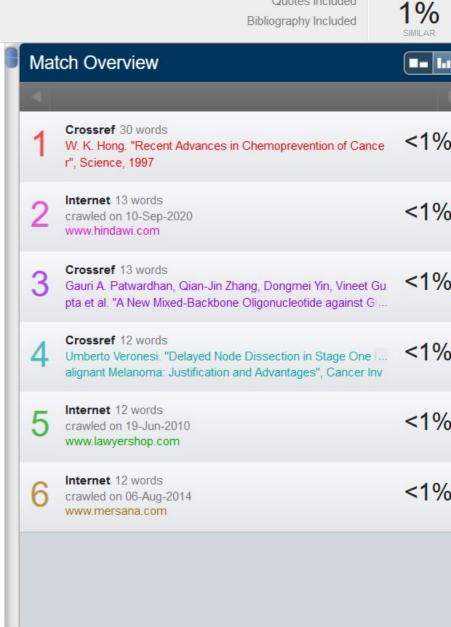
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Tumor-specific lytic path 'hyperploid progression mediated death': Resolving side effects through targeting retinoblastoma or p53 mutant

Frank-Un Hong, Miguel Castro, Klaus Linse

Abstract

A major advance was made to reduce the side effects of cancer therapy via the elucidation of the tumor-specific lytic path 'hyperploid progression-mediated death' targeting retinoblastoma (RB) or p53-mutants defective in G1 DNA damage checkpoint. The genetic basis of human cancers was uncovered through the cloning of the tumor suppressor RB gene. It encodes a nuclear DNA-binding protein whose self-





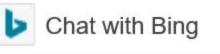
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Introduction. Lung cancer is the leading cause of cancer **death** worldwide. 1 Standard-of-care therapies for these patients include a combination of surgery, chemotherapy, and/or radiation therapy. The treatment of choice for patients with early stage disease is surgical resection in combination with adjuvant chemotherapy. 2 Cisplatin-based adjuvant chemotherapy, in particular, has been shown to ...

Cited by: 24 Author: Sofia R. Gameiro, Jorge A. Caballero, Ja...

Publish Year: 2012

Oncolytic virotherapy: Challenges and solutions ...

https://www.sciencedirect.com/science/article/pii/S0147027220301343

Aug 15, 2020 · The second mechanism of action of OVs is the enhancement of immune responses (). Following infection of tumor cells with OVs, cell **death**, and releasing tumor-associated antigens, **tumor-specific** immune responses increase which leads to the elimination of distant and uninfected tumor cells. 26 Tumor cell lysis also leads to release of cytokines (such as type I interferons [IFNs], ...

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May 22, 2015 · The key transcriptional target following **p53** stabilization is p21, which can impede G1/S transition **through** inhibition of the cyclin E/Cdk2 complex. The **retinoblastoma** protein (Rb) also has a key role in cell cycle **progression** by binding the E2F transcription factors and inhibiting **progression** ...

Cited by: 82 Author: Robert Hollingworth, Roger J Grand

Publish Year: 2015

Frontiers | Protein Glycosylation and Tumor ...

https://www.frontiersin.org/articles/10.3389/fonc.2019.00380/full •

Moreover, tumor hypoxia selects clones expressing **mutant p53**, facilitating the clonal expansion of cells that have a dominant-negative effect on the wild-type cells, thus evading growth suppression . Interestingly, the two canonical suppressors of cell proliferation, **p53** and RB, are regulated by O-GlcNAcylation (149, 150) .

Cited by: 23 Author: Andreia Peixoto, Marta Relvas-Santos, Ri...

Publish Year: 2019

7157 - Gene ResultTP53 tumor protein p53 [(human)]

https://www.ncbi.nlm.nih.gov/gene/7157

Oct 04, 2020 · 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 ...

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Cited by: 25 Author: Andreia Peixoto, Marta Relvas-Santos, Ri...

Publish Year: 2019

Oncolytic adenoviruses: A thorny path to glioma cure ...

https://www.sciencedirect.com/science/article/pii/S2352304214000336

Dec 01, 2014 · The glioma-specific oncolytic vector, referred to as ONYX105 or dl1520, was designed to replicate in the p53 deficient tumor cells with functional defects in p53 tumor suppressor signaling 21 and induce non-apoptotic cell death during viral infection. 22 However, over the past several decades, multiple scientific reports have evidenced that ...

Cited by: 8 Author: I.V. Ulasov, A.V. Borovjagin, B.A. Schroed...

Publish Year: 2014

Oncolytic virotherapy: Challenges and solutions ...

https://www.sciencedirect.com/science/article/pii/S0147027220301343

Aug 15, 2020 · Various strategies have been used to avoid off-target side effects and re-direct MeV specifically to cancer cells, including insertion of tumor-specific ligands, insertion of integrin-binding peptides, insertion of single-chain T-cell receptors, and modifications on the envelope fusion properties. 83 Moreover, defects in type 1 interferon