



Directed Expression of Dominant-negative P73 in Mouse Hearts Ying Zhang,2012 Mammalian cardiomyocytes lose their ability to proliferate shortly after birth. Previous studies have demonstrated that p53 plays an important role in maintaining cell cycle arrest of cardiomyocytes, which might account for the inability of human hearts to regenerate after injury, e.g. myocardial infarction. Thus, inhibition of p53 might be a promising strategy to reactivate cell cycle progression in cardiomyocytes although such a concept is limited by the fact that p53 is a well-known tumor suppressor. N-terminal truncated isoforms of the p53-related protein p73 function as antagonists against p53 and p73 during normal development without induction of tumor. Recombinant adenovirus encoding dominant-interfering p73 (Ad-p73DD) were generated to inhibit p53/p73 signaling in mouse hearts at different stages. The expression of p73DD led to the increase of the relative heart weights after 3 days and 14 days which was accompanied by a significant increase of proliferating cardiomyocytes (BrdU-incorporation, phosphorylation of histone3, expression of AuroraB) without induction of apoptosis. Progression of cardiomyocyte cell cycle went along with a significant downregulation of the p53-dependent CDK-inhibitor p21WAF both on mRNA and protein level. Furthermore, mRNA and protein expression of D-type cyclins and cyclin A, B2 and E were increased after Ad-p73DD injection. The cell cycle reentry of cardiomyocytes was not restricted to neonatal hearts, but was also found in adult hearts 5 days after intramyocardial injection of Ad-p73DD. Thus, expression of dominant-negative p73 might be utilized to stimulate proliferation of cardiomyocytes to improve cardiac regeneration.

**The P53 Homolog P73 Takes Hold of the Male Germ Line - a Novel Function of TAp73 in Protecting Sperm Cell Adhesion, Migration and Maturation Within the Seminiferous Epithelium of the Testis** Lena Holembowski,2012 The p53 family of transcription factors possesses diverge functions in tumorigenesis and development. The evolutionarily conserved role of the family member p63 to protect the genetic stability of germ cells and influence germ cell development is thought to be the ancestral protein function. Using p53, p63 and p73 knockout (KO) mice, the impact of these transcription factors on the female and male germ line was previously investigated. While mice deficient for p53 and p63 are fertile, loss of either all p73 isoforms or of the transcriptionally active TAp73 isoform alone was shown to cause i ...

*Regulation of P53 and P73 Function by PCAF and Adenovirus E1B 55-kDa Oncoprotein [microform]* Yue Liu,2002

*The Yes-Associated Protein Promotes P73 Stability Downstream of C-Jun Signalling* ,2007

The Related P53 and P73 Proteins Have Opposing Roles in Neuronal Survival and Apoptosis Christine D. Pozniak,2001 Together these studies have identified novel and opposing roles for the p53 family members in developing and mature neurons of the central and peripheral nervous systems. --

The Role and Regulation of P73 in Cisplatin-induced Apoptosis in Human Ovarian Cancer Cells in Vitro Shadia Mohamed Al Bahlani,2010

**Role and Regulation of the P53-homolog P73 in the Transformation of Normal Human Fibroblasts** Lars Hofmann,2007

Exploring the Role of P73-isoforms During Tumor Development Habib Abdullah Mohammad Sakil,2017

The Regulation of Neuronal Survival by the P53 Family Member P73, and by the Tyrosine Phosphatase SHP-2 Stevo Radinovic,2001 In the developing nervous system, neuron number and innervation are determined by the balance between the activity of pro-survival and pro-apoptotic proteins. We have examined the role of two such proteins, p73 and SHP-2, in cultured post-natal sympathetic neurons, a model system for developing peripheral neurons. p73 is a newly identified member of the p53 tumor suppressor family that exists as both truncated and full-length isoforms. We found that p73 is expressed in the developing nervous system as a truncated isoform (DeltaNp73) whose protein levels dramatically decreased when sympathetic neurons apoptose following NGF withdrawal. Ectopic expression of DeltaNp73 prevented this death. GST-pull down experiments suggested a physical interaction between DeltaNp73 and p53 in vitro, and expressing DeltaNp73 in sympathetic neurons attenuated p53-induced death. p73 <sup>-/-</sup> mice showed an increased amount of developmental sympathetic neuron death. These data indicate that DeltaNp73 is an important pro-survival protein that may function by directly inhibiting p53's pro-apoptotic activity. A second protein that was examined was SHP-2, a phosphotyrosine phosphatase. We observed that SHP-2 modulates the survival of sympathetic neurons through two independent pathways downstream of TrkA. Inhibiting SHP-2 enhanced GAB-1 phosphorylation and blocked ERK phosphorylation, suggesting that a normal role for SHP-2 in neurons is to enhance ERK activity and ERK-mediated pro-survival signals. --

Pfifty-three and P73 in Neurogenesis of the Adult Zebrafish Danio Rerio Julia Eich,2014

Mechanisms Used by P73, P63 and XIAP to Regulate Survival and Apoptosis of Sympathetic Neurons Daniel Kuan-te Ho,2004 Developmental neuron death is determined by the activities of pro-survival and pro-apoptotic proteins. Here, we investigate the functions of three such proteins---p73, p63 and XIAP---in cultured neonatal sympathetic neurons. p73 and p63 are members of the p53 family that exist in both full-length (TA) and truncated (DeltaN) isoforms. First, we ask whether DeltaNp73 mediates sympathetic neuron survival in p53-dependent and/or -independent fashions. Overexpression of DeltaNp73 in sympathetic neurons withdrawn from NGF prevents upregulation of the p53 targets p21waf1 and Apaf-1, induction of the BH3-only protein BimEL, and cleavage of the effector caspase-3. DeltaNp73 also promotes survival in p53-independent manner by acting upstream of p53 to decrease JNK phosphorylation. Second, we ask whether p63 functions as a pro-apoptotic molecule in a manner similar to that of p53. TAp63 levels markedly increase in sympathetic neurons withdrawn from NGF. Similar to p53, TAp63 overexpression in neurons maintained in NGF upregulates p21waf1 and Apaf-1 levels, induces caspase-3 cleavage, and does not induce BimEL levels and JNK phosphorylation. Third, we examine the role of the caspase inhibitor XIAP and ask whether XIAP is a substrate of the serine/threonine kinase Akt. XIAP rescues NGF withdrawal- and LY294002-induced apoptosis and promotes sympathetic neuron survival cooperatively with Ras. In addition, Akt associates with XIAP and phosphorylates XIAP at serine 87. --

**TNF-[alpha] Mediated Apoptosis in Vascular Smooth Muscle Cells Requires P73** Vincent Tang,2004

*Biophysical and Functional Studies of SAM Domain of Human P73[alpha].* Wooi Koon Wang,2001

*Activation of Expression of P15, P73 and E-cadherin in Myeloid Leukemia Cells by Different Concentrations of 5-aza-2'-deoxycytidine* Nuno Jorge dos Reis Farinha,2004

*Structural Investigations of the Tetramerization Domains of P53/p73-like Transcription Factors from the Tunicate Species Ciona Intestinalis* Jan Peter Heering,2014

Structural Diversity of P63 and P73 Isoforms Christian Osterburg,Volker Dötsch,2022

*Journal of the National Cancer Institute* ,1999

**AACR 2022 Proceedings: Part A Online-Only and April 10** American Association for Cancer Research,2022-05-09 The AACR Annual Meeting is the focal point of the cancer research community, where scientists, clinicians, other health care professionals, survivors, patients, and advocates gather to share the latest advances in cancer science and medicine. From population science and prevention; to cancer biology, translational, and clinical studies; to survivorship and advocacy; the AACR Annual Meeting highlights the work of the best minds in cancer research from institutions all over the world.

**Induction of C-Jun N-terminal Kinase Activity in the Absence of NFkB Activation Induces TRAF3, IgFBP6, P53 Associated Protein, P73 and is Responsible for Programming of Cell Death of Oral Tumor Cells** David Lam Le,2000

**Autoantibodies** Yehuda Shoenfeld,M. Eric Gershwin,M.E. Gershwin,Pier Luigi Meroni,2011-10-13 Autoantibodies was published and presented in November 2006 at the International Congress of Autoimmunity in Sorrento, a small town in Campania, Italy. The Congress also celebrated the 100th anniversary of the first routine test for autoantibodies. An autoantibody is a type of antibody that is produced by the immune system and that fights one or more of a person's own proteins. These autoantibodies cause autoimmune diseases such as lupus erythematosus. The authors and editors of this book provide a critical review of autoantibodies and their primary functions. They cite a number of major developments in the field of autoantibodies, including the detection of autoantibodies in which a healthy person is a carrier; the discovery that autoantibodies can be both pathogenic and protective in some cases; and the development of a device that will help monitor and detect a specific autoantibody using a small

amount of serum and proteomic arrays. Aside from the pathogenic and protective autoantibodies, the book also discusses irrelevant autoantibodies, as these may be relevant for future research. It also addresses the importance of the autoantibodies in a person’s body. Clinical physicians, as well as scientists interested in the significance of autoantibodies in the human body, will find this book relevant. It will also be of interest to those who suffer from an autoimmune disease. \* Incudes and exhaustive list of autoantibodies not covered by other publications \* Short reviews can easily be checked for quick reference information \* Both basic and clinical aspects are covered

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