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Distinct Pathways to the Same Goal for P53 Activation in Genetic Diseases

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Introduction

The current era of therapeutic research has been transformed by the concept of genome modification. From gene modifications in primary cells to genetic modifications in animals, studies of genome modification have advanced considerably. The predetermined gene expression may be modulated (either upregulated or downregulated) by the targeted genetic modification. Cas9. The CRISPR-Cas9 genome editing technology was able to participate in cancer clinical trials as a result of this. The CRISPR-Cas9 tool can be used to create genetically inhibited animal models for drug discovery and development in addition to having therapeutic potential. The origins of CRISPR-Cas9 systems and their therapeutic potential against various genetic disorders, such as cancer, allergy, immunological disorders, Duchenne muscular dystrophy, cardiovascular disorders, neurological disorders, liver-related disorders, cystic fibrosis, blood-related disorders, eye-related disorders and viral infection, are the subject of this comprehensive review. Last but not least, we go over the various difficulties, safety concerns and methods for overcoming them in CRISPR-Cas9-mediated therapeutic approaches [1].

Description

Human cancers frequently alter the p53 gene, indicating its biological and clinical significance. Around one hundred target genes, most notably those involved in apoptosis, DNA repair and cell cycle regulation, are activated by p53. Through the actions of its downstream targets, such as p21, p53 also represses gene expression. Ablation of Trp53 induces tumors in mice at an early age, given that p53 is involved in tumor suppression. In addition, Trp53-deficient mice exhibit developmental defects that are distinct between strains of mice with distinct genetic backgrounds, indicating that p53 plays a role in cell differentiation and development. Additionally, p53 coordinates metabolic homeostasis and participates in various metabolic pathways; metabolic disorders and possibly tumorigenesis may result from dysregulated p53 function. Moreover, multiple signaling pathways are used by p53 to control cell senescence and aging. Depending on the type or intensity of the cellular stress, p53 may either elicit senescence or apoptosis, which respectively stop the spread of damage and eliminate damaged cells, or transient cell cycle arrest, which enables damage repair. Tissue degeneration is accelerated when p53 activation is abnormal or persistent. For instance, neurodegenerative conditions like Alzheimer's disease are associated with excessive p53-induced neuronal death. Additionally, a variety of congenital disorders have been linked to excessive p53 activation during embryonic development. Several sets of genetic mutations that contribute to excessive p53 activation and cause phenotypic abnormalities in congenital disorders are discussed in this overview [2-4].

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A large number of rare diseases caused by mutations in nuclear genes encoding proteins or enzymes involved in the import or catabolism of fatty acids in the mitochondria are known as inborn mitochondrial FAO disorders. The enzymology of mitochondrial FAO has been the subject of numerous reviews and is well-described. The long chain acyl coenzyme A (CoA)s enter the ß-oxidation pathway for a brief time after being imported into the mitochondria, where they are shortened into acetyl CoA, which is then fully oxidized to CO2 during the tricarboxylic acid cycle (TCA) cycle. Although genetic publications on Lebanese subjects began as early as 1950, we only see a significant amount of genetic literature after the end of the Lebanese civil war in the late 1990s. A previous study that looked at the biomedical bibliometric output from Lebanon up until 2007 showed that the number of publications was going up. Our analysis of the year-by-year distribution of the selected articles reveals a similar trend in publications over time, indicating an increase in genetic research. We were also interested in keeping an eye on how medical genetic studies were developing, particularly in light of the development of more recent molecular technologies. The percentage of research articles that contain molecular data is clearly on the rise, especially in the past ten years, when clinicians in Lebanon started using NGS techniques to diagnose genetic diseases. It is interesting to note that the number of such publications has decreased over the past few years, most likely as a result of Lebanon's current economic crisis [5].

Conclusion

A systematic effort to identify and counsel as many people as possible in a population who are at genetic risk, regardless of whether they have a family history of a genetic disorder, is known as a screening program for genetic carriers. Premarital screening for haemoglobinopathies has been implemented in several Arab nations.

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Conflict of Interest

There are no conflicts of interest by author.

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