Special Issue

Strategies for Anticancer in p53 Mutated Cancers

Message from the Guest Editors

The p53 gene is a key tumor suppressor but is frequently mutated in nearly all types of cancers, with an average mutation rate of ~50%. Most mutated p53 proteins are highly expressed and acquire oncogenic functions that promote cancer progression and confer high resistance to anticancer drugs. Various approaches have been explored to develop strategies against cancers with highly expressed mutant p53 genes. These strategies include the following: 1. The direct reactivation of mutated p53. 2. The activation of other p53 family members, such as p63 or p73. 3. The degradation of mutated p53. 4. Blocking oncogenic microRNAs or long non-coding RNAs downstream of mutated p53. New ideas, methods, and reviews regarding targeting mutant p53 genes in different cancer types are invited for inclusion in this special issue.

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Biomedicines (ISSN 2227-9059) is an open access journal devoted to all aspects of research on human health and disease, the discovery and characterization of new therapeutic targets, therapeutic strategies, and research of naturally driven biomedicines, pharmaceuticals, and biopharmaceutical products. Topics include pathogenesis mechanisms of diseases, translational medical research, biomaterial in biomedical research, natural bioactive molecules, biologics, vaccines, gene therapies, cell-based therapies, targeted specific antibodies, recombinant therapeutic proteins, nanobiotechnology driven products, targeted therapy, bioimaging, biosensors, biomarkers, and biosimilars. The journal is open for publication of studies conducted at the basic science and preclinical research levels. We invite you to consider submitting your work to Biomedicines, be it original research, review articles, or developing Special Issues of current key topics.

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