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p53 Regulation Mechanisms

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Message from the Guest Editors

Dear Colleagues,

The tumor suppressor p53 is frequently mutated in various human cancers as it can prevent tumorigenesis by inducing cellular senescence, regulating energy metabolism, blocking metastasis, stopping cell proliferation, and inducing apoptosis, mainly via its transcriptional activity. In response to various stressors, p53 is activated to induce or repress the transcription of numerous target genes important for multiple biological functions. For example, the p53 target gene p21 is involved in p53-dependent cell cycle arrest, while the BH3-only-encoding target genes BBC3 (Puma) and PMAIP1 (Noxa) play key roles in p53-mediated apoptosis. Since activated p53 is generally cytotoxic, it is subjected to tight regulation by MDM2, which is encoded by a transcriptional target gene of p53. Via its N-terminal domain, MDM2 directly binds to the N and C termini of p53 and mediates its ubiquitin-dependent proteolysis, as it possesses intrinsic E3 ubiquitin ligase activity, thus creating a negative feedback loop. The main focus of this Special Issue will be on the broad spectrum of functions of p53 in cancer cells.

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Message from the Editorial Board

Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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