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p53 Signaling and Cancer Cell Response to Genotoxic Stress: Beyond Cell Cycle Checkpoints and Apoptosis

Guest Editor:

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Deadline for manuscript submissions:
closed (31 December 2020)

Message from the Guest Editor

Dear Colleagues,

The landscape of the DNA damage response has changed. Notably, the biological outputs orchestrated by the p53 tumor suppressor extends far beyond conventional cell cycle arrest and/or apoptosis. Under physiological conditions (e.g., the absence of ectopic p53 gene expression), the activation of the p53 signaling pathway following exposure to ionizing radiation and chemotherapeutic agents serves to prevent death through apoptosis and other modes of cell death, and to induce a senescence-like proliferation arrest.

The purpose of this Special Issue is to bring together research/review articles on the growing complexity surrounding p53 in general, and the long-term biological outputs controlled by p53 and its key downstream effectors (e.g., CDKN1A) in particular. Articles on tumor heterogeneity, advances in single-cell detection methodologies to study cancer cell responses to genotoxic stress, and novel therapeutic approaches by targeting proliferation-arrested (dormant) cancer cells are particularly welcomed.

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Message from the Editor-in-Chief

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