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The Tumor Suppressor TP53 in Colorectal Carcinoma

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Message from the Guest Editors

Dear Colleagues,

Despite the tremendous gain of knowledge in colorectal cancer (CRC) biology during the past years, this tumor type is still the second most common cancer worldwide, characterized by complex genetic, epigenetic, and biochemical defects. A few crucial intracellular signaling pathways, including Wnt/β-catenin, Ras, and p53 signaling, are often deregulated in CRC. The tumor suppressor TP53 is mutated in more than 60% of CRC, and there is no doubt that TP53 is one of the most crucial players in this deadly disease. As a guardian of genome stability, upon genotoxic stress, this multifaceted transcription factor is involved in the regulation of cell cycle arrest, senescence, apoptosis, non-apoptotic cell death, and DNA repair. Critical mutations (hot spots) in the *TP53* gene are key drivers of the transition from adenoma to adenocarcinoma. Thus, exploring the functional roles of *TP53* gain-of-function (GOF) mutations in tumor development is of utmost importance, since patients with mutant p53 are often resistant to current chemotherapies, experiencing a short lifespan upon diagnosis.

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Message from the Editor-in-Chief

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