

Preface

Since its discovery, p53 research has been the highlight of understanding the crucial role of oncogenes and tumor suppressors in regulated or deregulated cell growth. In the last decade, special attention was focused on gain of function mutations of p53 that can turn the tumor suppressor to an oncoprotein, as well as on abnormal expression of oncogenes such as MDM2 and MDMX that could inactivate the tumor suppressor function of p53 in the hope of devising cancer treatment. A wealth of information has emerged regarding what genes the gain-of-function mutants of p53 activate and how they induce oncogenesis, how these mutants are stabilized in cancer cells, how they respond to chemotherapy, and how interaction of p53 mutants with p53 family members may induce oncogenesis. Similarly there are exciting reports on how the oncoprotein MDM2, known to exist to control p53, can activate signaling pathways independent of p53 when overexpressed, and how MDMX is involved in the regulation of p53 by MDM2.

Mutant p53 and MDM2 in Cancer includes 19 chapters that discuss the activation of diverse oncogenic pathways consequent to p53 mutation and overexpression of MDM2 and MDMX and their splice variants. This book also includes chapters that discuss p53 mutation in hereditary cancer, response of cancers with p53 mutation to chemotherapy and radiation, structural aspects of mutant p53 that make it an oncoprotein and targeting of these structures for cancer therapy. The function of wild type p53 in response to stress and regulation of this function by MDM2 has also been included. Overall, this book provides an insight into the primary molecular events leading to oncogenesis consequent to p53 mutation and overexpression of MDM2. The information should be invaluable for beginning or experienced researchers, and even for future researchers opting to commit to cancer biology. To dissect the oncogenic functions of mutant p53 and MDM2, the book focuses primarily on human systems. Since a large volume of literature is available for the mouse models, perhaps it calls for a separate volume.

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