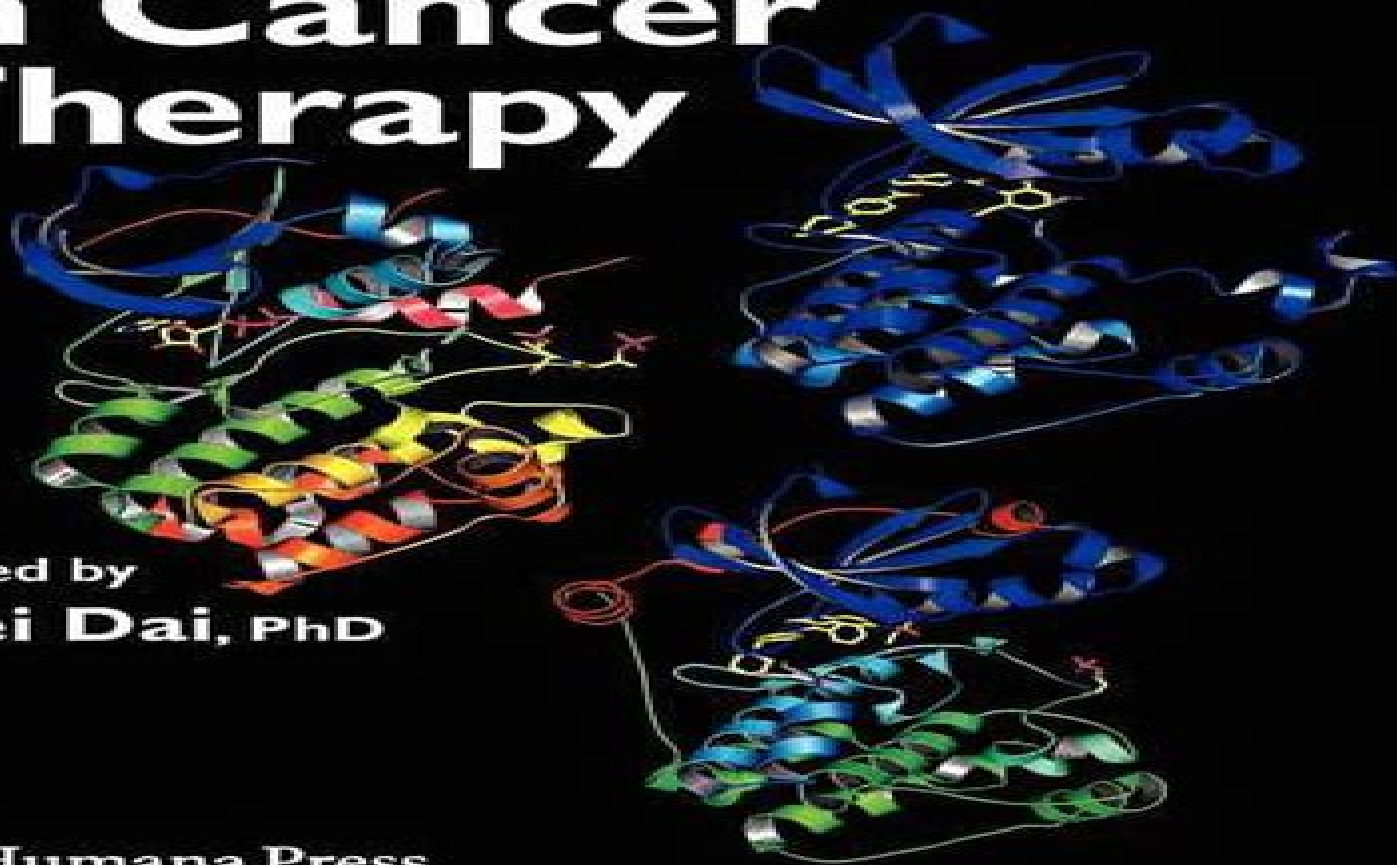


CANCER DRUG DISCOVERY AND DEVELOPMENT™

Checkpoint Responses in Cancer Therapy

Edited by
Wei Dai, PhD

 Humana Press



Checkpoint Responses In Cancer Therapy Cancer Drug Discovery And Development

Antonio Tito Fojo



Checkpoint Responses In Cancer Therapy Cancer Drug Discovery And Development:

Checkpoint Responses in Cancer Therapy Wei Dai, 2008-05-01 Extensive research has uncovered a set of molecular surveillance mechanisms commonly called checkpoints which tightly monitor cell cycle processes Today s anticancer drug development has identified many of these cell cycle checkpoint molecules as effective targets Research now promises to uncover a new generation of anticancer drugs with improved therapeutic indices based on their ability to target emerging checkpoint components Checkpoint Responses in Cancer Therapy summarizes the advances made over the past 20 years identifying components of cell cycle checkpoints and their molecular regulation during checkpoint activation and validating the use of checkpoint proteins as targets for the development of anticancer drugs This book s distinguished panel of authors takes a close look at topics ranging from the major molecular players affecting DNA synthesis and the response to DNA damage to advances made in the identification of chemical compounds capable of inhibiting individual mitotic kinases Illuminating and authoritative Checkpoint Responses in Cancer Therapy offers a critical summary of findings for researchers in the pharmaceutical and biotechnology industries and a valuable resource for academic scientists in cancer research and the study of cell cycle regulation signal transduction and apoptosis

Checkpoint Controls and Targets in Cancer Therapy Zahid H. Siddik, 2010-03-12 Much work over the last two decades has firmly established that loss of cell cycle checkpoint regulation and resultant unabated cellular proliferation is an inherent characteristic of cancer This loss may occur through aberration in any single component involved in signal transduction pathways that orchestrate checkpoint regulation which may manifest through either a failure to activate the checkpoint or a failure to respond to the activated checkpoint In normal cells checkpoint pathways are activated when genetic or cellular homeostasis is compromised and signals are then transduced to re stabilize homeostasis and failing this to activate the apoptotic machinery to induce a cellular suicidal response This implies that both survival and cell death pathways are induced following checkpoint activation and that the final decision is dependant on the net result of integrating the two sets of signals It is intriguing that checkpoint pathways are also critical in cancer therapy to provide an apoptotic stimulus when cellular damage induced by the therapeutic agent is detected by the sensor system Therefore it is not surprising that failure in pro survival checkpoint response will render tumor cells hypersensitive to cytotoxics and conversely failure in pro apoptotic checkpoint response will induce genetic instability and or therapeutic resistance Understanding the intricacies of checkpoint response is therefore central to the design of therapeutic regimen that will enhance antitumor effects Although early versions of this design entail combination of cytotoxic agents with cell cycle or checkpoint inhibitors a greater understanding of the concepts could make such combinations clinically more effective The contributions in this book will consolidate the current state of knowledge on checkpoint responses that may lay the foundation for hypothesis driven rational approaches in advancing the management of cancer The immediate attraction of the book to the scientific community is that it represents a timely opportunity to build upon existing

concepts of checkpoints to expand our understanding of the inner workings of the critical checkpoint machinery. The present understanding has provided ample appreciation that response to checkpoint activation is manifested through coordinated inhibition of cyclin dependent kinase CDK complexes in G1 S and or the G2 phase in order to arrest the cell cycle. Kinase inhibition can occur through several mechanisms including inhibitory phosphorylation of CDK, destruction of the cognate cyclins and recruitment of CDK inhibitors from the INK and WAF1/CIP1 families. However, the wealth of information from recent discoveries needs to be examined critically to consolidate our conceptual knowledge of checkpoints. At the same time, there is acute awareness in the diversity of checkpoint response between cytotoxic agents and this serves as a reminder of the magnitude of complexity that is inherent in checkpoint regulation. This volume is intended to bring the cancer research community closer toward an improved understanding of this regulation, how checkpoint abnormalities can impact negatively on cancer therapy and emerging strategies to target checkpoint response as a therapeutic end point.

Transforming Growth Factor-Beta in Cancer Therapy, Volume I Sonia B. Jakowlew, 2010-01-01. Transforming Growth Factor β in Cancer Therapy, Volume I. Basic and Clinical Biology. The present volume brings together a wealth of information that is fundamental to understanding the role of TGF in the pathogenesis, prevention and treatment of cancer. It is not even 25 years since TGF was first isolated and characterized as a dimeric peptide from both human and bovine sources [1-3] but the entire field of TGF research has grown and expanded so that it is now a central theme in all of cell biology. There is almost no tissue or organ in the mammalian body in which TGF does not play a central role in embryonic differentiation or in adult function and furthermore malfunction of the normal physiology of TGF can have disastrous consequences in almost all of these sites. Therefore the present comprehensive review of so many aspects of TGF function is a most welcome attempt to bring together a huge body of experimental data that is of the utmost importance in the field of oncology.

Targeting the DNA Damage Response for Anti-Cancer Therapy John Pollard, Nicola Curtin, 2018-05-26. Over the past decade a complex role for DNA damage response (DDR) in tumorigenesis has emerged. A proficient DDR has been shown to be a primary cause for cellular resistance to the very many DNA damaging drugs and IR that are widely used as standard of care across multiple cancer types. It has also been shown that defects in this network, predominantly within the ATM mediated signaling pathway, are commonly observed in cancers and may be a primary event during tumorigenesis. Such defects may promote a genomically unstable environment facilitating the persistence of mutations, any of which may provide a growth or survival advantage to the developing tumor. In addition, these somatic defects provide opportunities to exploit a reliance on remaining repair pathways for survival, a process which has been termed synthetic lethality. As a result of all these observations, there has been a great interest in targeting the DDR to provide anti cancer agents that may have benefit as monotherapy in cancers with high background DNA damage levels or as a means to increase the efficacy of DNA damaging drugs and IR. In this book we will review a series of important topics that are of great interest to a broad range of academic, industrial and clinical

researchers including the basic science of the DDR its role in tumorigenesis and in dictating response to DNA damaging drugs and IR Additionally we will focus on the several proteins that have been targeted in attempts to provide drug candidates each of which appear to have quite distinct profiles and could represent very different opportunities to provide patient benefit *Advances in DNA Repair in Cancer Therapy* Lawrence Panasci, Raquel Aloyz, Moulay

Alaoui-Jamali, 2012-12-09 A comprehensive review of the recent developments in DNA repair research that have potential for translational applications The book explains in detail the various biological mechanisms by which cancer cells can circumvent anticancer therapy and limits its usefulness in patients They also review the impact of such novel inhibitors of DNA repair mechanisms as methylguanine DNA methyltransferase Also examined are inhibitors of other DNA repair enzymes such as PARP and DNA PK The book captures for both cancer researchers and oncologists dealing with hallmark relapse or drug resistance phenomena on a daily basis the many exciting new uses of DNA repair inhibitors either alone or in combination with anticancer therapies *Transforming Growth Factor-Beta in Cancer Therapy, Volume II* Sonia B. Jakowlew, 2010-01-01

Transforming Growth Factor B in Cancer Therapy Volume II Cancer Treatment and Therapy The chapters in this volume confer an abundance of knowledge about the current state of our understanding of transforming growth factor B TGF B in cancer treatment and therapy Unlike several more traditional positive polypeptide growth factors that stimulate cellular proliferation the prototypical TGF B is now known to inhibit the growth of most normal cell types including those of epithelial and mesenchymal origin However there are examples of cell types that can be stimulated by TGF B under certain conditions TGF B also induces the accumulation of matrix molecules by stimulating their synthesis as well as inhibiting their degradation Moreover TGF B induces apoptosis of certain cell types thereby restricting their proliferation Overactivity of TGF has been linked to several diseases For instance the effect of TGF on matrix accumulation contributes to fibrotic conditions like glomerulonephritis lung fibrosis and liver cirrhosis 1 TGF has a very complicated role in cancer that is only beginning to be understood **The Role of Microtubules in Cell Biology, Neurobiology, and Oncology** Antonio Tito

Fojo, 2009-01-14 This book presents the first comprehensive exploration of the dynamic potential of microtubules anti cancer targets Written by leading anti cancer researchers this groundbreaking volume collects the most current microtubule research available and investigates the potential of microtubules in cancer therapy *Cancer Proteomics* Sayed S.

Daoud, 2008 This book covers current topics related to the use of proteomic strategies in cancer therapy as well as anticipated challenges that may arise from its application in daily practice It details current technologies used in proteomics examines the use of proteomics in cell signaling presents clinical applications of proteomics in cancer therapy and looks at the role of the FDA in regulating the use of proteomics **Drug-Diagnostics Co-Development in Oncology** Jan Trøst

Jørgensen, 2014-11-07 The idea of combining drugs and diagnostics in oncology is not new When the selective estrogen receptor modulator tamoxifen was developed in the 1970 s for the treatment of breast cancer a positive correlation between

receptor status and treatment outcome was found. As a result of this research it was suggested to use the estrogen receptor assay as a diagnostic test for selection of patients for tamoxifen treatment. Despite this suggestion was put forward nearly 40 years ago the adaptation of the drug diagnostic co development model has been relatively slow and it is only within the last decade that it has gained more widespread acceptance. The parallel development of the monoclonal antibody trastuzumab (Herceptin, Roche Genentech) and the immunohistochemistry assay for HER2 protein overexpression (HercepTest, Dako) seems to have served as an inspiration to a number of stakeholders such as pharma and diagnostic companies, regulatory agencies and academia. In recent years we have seen an increasing number of oncology drug development projects that have taken advantage of the drug diagnostic co development model as outlined below. Most of the new targeted anti cancer drugs that have been introduced in recent years such as BRAF, ALK, EGFR and HER2 inhibitors are more or less all a product of the drug diagnostic co development model. These drugs have shown remarkable high response rates in selected groups of patients within cancer diseases with great unmet medical needs.

Research Topic on Drug Diagnostic Co Development in Oncology

EGFR Signaling Networks in Cancer Therapy John D. Haley, William John Gullick, 2009-03-01

The epidermal growth factor (EGF) receptor and its downstream signal transduction networks have been implicated in the ontogeny and maintenance of tumor tissues which has motivated the discovery and development of molecularly targeted anti EGF receptor therapies. Over decades of study the EGF receptor structure, its ligand binding domains, the physical biochemistry underlying its intrinsic tyrosine kinase catalytic function and the modular interactions with SH2, PTB and SH3 domain containing signaling adaptor proteins required for signal transduction have been extensively dissected. Not only is the EGF receptor the nexus of many streams of information but it also forms one part of a calculating device by forming dimers and oligomers with the other three receptors in its family in response to at least eleven ligands, some of which are expressed in multiple forms with overlapping or quite distinct functions. This phenomenon while recruiting to the inner surface of the cell membrane and activating multiple second messenger proteins also allows the possibility of cross talk between these systems permitting a further layer of information to be exchanged. Less well described are the cross regulation of the EGF receptor and other anti apoptotic, mitogenic and metabolic signaling systems. The study of these systems has yielded new surprises. One hurdle in these efforts has been that signal transduction pathways have frequently been defined in the generic absence of their tissue specific or cell interaction specific context.

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