# Molecular Mechanisms Of Tumor Cell Resistance To Chemotherapy Targeted Therapies To Reverse Resistance Resistance To Targeted Anti Cancer Therapeutics

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*Molecular mechanisms of cellular stress responses in cancer and their therapeutic implications* - Megan Chircop 2015-03-06

In response to stress, cells can activate a myriad of signalling pathways to bring about a specific cellular outcome, including cell cycle arrest, DNA repair, senescence and apoptosis. This response is pivotal for tumour suppression as all of these outcomes result in restriction of the growth and/or elimination of damaged and pre-malignant cells. Thus, a large number of anti-cancer agents target specific components of stress response signalling pathways with the aim of causing tumour regression by stimulating cell death. However, the efficacy of these agents is often impaired due to mutations in genes that are involved in these stress-responsive signalling pathways and instead the oncogenic potential of a cell is increased leading to the initiation and/or progression of tumourigenesis. Moreover, these genetic defects can increase or contribute to resistance to chemotherapeutic agents and/or radiotherapy. Modulating the outcome of cellular stress responses towards cell death in tumour cells without affecting surrounding normal cells is thus one of the ultimate aims in the development of new cancer therapeutics. To achieve this aim, a detailed understanding of cellular stress response pathways and their aberrations in cancer is required. This Research topic aims to reflect the broadness and complexity of this important area of cancer research.

Drug and Hormonal Resistance in Breast Cancer - Robert Brent Dickson 1995

Featuring contributions from expert authors of international standing, this book explores emerging studies on the success and/or failure of chemotherapy in breast cancer. Covers the clinical resistance of breast cancer to treatment; chemo-hormonal reactions; anti-hormonal resistance; multidrug resistance; and fu/antimetabolites. For cancer and hormonal researchers and development scientists in pharmaceuticals and biomedicine. Previously announced in 7/93 PTR Catalog.

**Ferroptosis as New Therapeutic Targets in Cancer: from**

**Molecular Mechanisms to Therapeutic Opportunities** - Xu Chen 2022-11-08

*Drug Resistance as a Biochemical Target in Cancer Chemotherapy* - Takashi Tsuruo 1992

The development of drug resistance hinders the effectiveness of cancer chemotherapy. This book, drawing upon recent molecular biological and biochemical studies that are helping shed light on the molecular mechanisms of drug resistance, summarizes recent clinical and preclinical studies.

*Molecular Mechanisms in Cancer* - Metin Budak 2022-08-17

Cancer is a major public health problem and much research is being conducted to develop effective treatments for various types of malignancies. In doing so, researchers must have comprehensive knowledge about what causes cancer. This book explains the mechanisms of different types of cancers in twelve chapters organized into three sections on oncogenes, tumor suppressor genes, and viral oncogenes.

*Holland-Frei Cancer Medicine* - Robert C. Bast, Jr. 2017-03-10

Holland-Frei Cancer Medicine, Ninth Edition, offers a balanced view of the most current knowledge of cancer science and clinical oncology practice. This all-new edition is the consummate reference source for medical oncologists, radiation oncologists, internists, surgical oncologists, and others who treat cancer patients. A translational perspective throughout, integrating cancer biology with cancer management providing an in depth understanding of the disease An emphasis on multidisciplinary, research-driven patient care to improve outcomes and optimal use of all appropriate therapies Cutting-edge coverage of personalized cancer care, including molecular diagnostics and therapeutics Concise, readable, clinically relevant text with algorithms, guidelines and insight into the use of both conventional and novel drugs Includes free access to the Wiley Digital Edition providing search across the book, the full reference list with web links, illustrations and photographs, and post-publication updates

*Drug Resistance in Colorectal Cancer: Molecular Mechanisms and Therapeutic Strategies* - Chi Hin Cho 2020-05-24

Drug Resistance in Colorectal Cancer: Molecular Mechanisms and Therapeutic Strategies, Volume Eight, summarizes the molecular mechanisms of drug resistance in colorectal cancer, along with the most up-to-date therapeutic strategies available. The book discusses reasons why colorectal tumors become refractory during the progression of the disease, but also explains how drug resistance occurs during chemotherapy. In addition, users will find the current therapeutic strategies used by clinicians in their practice in treating colorectal cancer. The combination of conventional anticancer drugs with chemotherapy-sensitizing agents plays a pivotal role in improving the outcome of colorectal cancer patients, in particular those with drug-resistant cancer cells. From a clinical point-of-view, the content of this book provides clinicians with updated therapeutic strategies for a better choice of drugs for drug-resistant colorectal cancer patients. It will be a valuable source for cancer researchers, oncologists and several members of biomedical field who are dedicated to better treat patients with colorectal cancer. Presents a systemic summary of molecular mechanisms for a quick and in-depth understanding Updates current trends in the field with pioneering information on drug resistance Encompasses both basic and clinical approaches for a better understanding of unsolved problems from a holistic point-of-view

Dissecting the Molecular Mechanisms of Therapeutic Resistance in Cancer - Vibhuti Agrawal 2017

Therapeutic resistance continues to be a persistent challenge in medical oncology. In clinical settings, resistance can occur at the beginning of treatment, or may be acquired after an initial clinical response to the therapy. Several mechanisms of drug resistance have been described in cancer, including alterations in the drug transport and metabolism process, mutations in drug-target, activation of bypass signaling pathways, inhibition of cell-death pathways, and induction of an epithelial to mesenchymal transition (EMT) in response to cytotoxic or targeted therapies. In this study, I have investigated the molecular mechanisms underlying ZEB 1-induced EMT and established a new computational framework that uses inter-animal heterogeneity to identify drivers

responsible for variable phenotypic responses across different animals. EMT describes a cell-state switching process wherein epithelial cells lose their tight cell-cell junction contacts, and acquire the ability to migrate and invade the surrounding stroma to enter into blood circulation. Given the widespread role of EMT in drug resistance, it is imperative to identify therapeutic strategies to inhibit this transition. To identify druggable targets to block EMT progression, and therefore overcome EMT-mediated therapeutic resistance, I studied the effects of ZEB 1 expression on cellular signaling networks. By quantifying changes in tyrosine phosphorylation at different time points during ZEB 1-induced EMT, I found that Src family kinases (SFKs) were activated within 24 hours of ZEB 1 expression. Inhibition of SFKs blocked not only ZEB 1-induced EMT, but also EMT initiated by TGFp- and EGF signaling pathways in both breast and NSCLC cell-lines. SFK inhibition also prevented EGFR inhibitor-induced EMT and drug resistance in NSCLC cells both in vitro and in vivo. Mechanistically, SFK activation stabilized ZEBI by promoting ERK1/2-mediated phosphorylation on three serine residues, S583, S646, and S679. Consequently, MEK inhibition phenocopied the effects of blocking SFK activity with regards to decreasing stability of ZEB 1 and inhibiting ZEB 1-induced EMT. These results provide a new therapeutic application of SFK inhibitors as a potential anti-EMT therapy, to enhance the susceptibility of cancer cells to chemo- or targeted therapies. In the second part of this thesis, I have described a computational framework that leverages inter-animal heterogeneity to identify molecular mechanisms underlying variable phenotypic responses across different animals. Substantial inter-animal variability in phenotypes within the same treatment group, limits our ability to draw conclusions or gain meaningful insights about a biological process by simply averaging the data. To identify molecular drivers for heterogeneous phenotypic responses, I have established a method where each animal is considered as an individual entity whose phenotypic response is dependent on the state of its underlying signaling networks. As a proof of concept, I have used this method to successfully predict the resistance mechanisms of CDK4/6 inhibitor, palbocilib in two GBM PDX and one MPNST PDX models. The GBM6 model activated EGFR signaling upon treatment with palbociclib whereas the GBM22 and MPNST3 models activated SFKs and PDGFRa signaling in resistant tumors. Across all three PDX tumor models, treatment with combination therapies, consisting of palbociclib and an inhibitor targeting the activated bypass signaling pathway, substantially prolonged survival of mice. Thus, these results suggest that inter-animal variability can be used as a tool to predict drivers for a specific phenotypic response across different treatment conditions.

Cancer Drug Resistance - Beverly A. Teicher 2007-11-09

Leading experts summarize and synthesize the latest discoveries concerning the changes that occur in tumor cells as they develop resistance to anticancer drugs, and suggest new approaches to preventing and overcoming it. The authors review physiological resistance based upon tumor architecture, cellular resistance based on drug transport, epigenetic changes that neutralize or bypass drug cytotoxicity, and genetic changes that alter drug target molecules by decreasing or eliminating drug binding and efficacy. Highlights include new insights into resistance to antiangiogenic therapies, oncogenes and tumor suppressor genes in therapeutic resistance, cancer stem cells, and the development of more effective therapies. There are also new findings on tumor immune escape mechanisms, gene amplification in drug resistance, the molecular determinants of multidrug resistance, and resistance to taxanes and Herceptin.

**Targeted Therapies** - Daniel Gioeli 2011-06-02

This volume explores the mechanisms of resistance to targeted therapeutics. The focus is on the cancer cell signaling network, although other mechanisms of resistance including target mutation, and new areas of study such as cancer stem cells are included. Targeted Therapies: Mechanisms of Resistance highlights examples of changes in the signaling network in response to inhibition of a signaling event and underscores the importance in having a mechanistic understanding of the signaling network in cancer for developing effective targeted cancer therapies. Moreover, cutting edge tools to analyze the cell signaling network will be discussed. This includes the leading edge of techniques as well as

computational biology and systems theory. This volume provides the reader with both an overview as well as a detailed perspective on the mechanisms of resistance to targeted therapeutics and will be of great value to the oncologist, the physician-scientist treating patients and the translational scientist working on any aspect of targeted therapeutics.

**Platinum and Other Heavy Metal Compounds in Cancer Chemotherapy** - Andrea Bonetti 2009-01-09

Cisplatin, the first member of the family of platinum-containing chemotherapeutic agents, was discovered by Barnett Rosenberg in 1965 and approved by the FDA for marketing in 1978. After 30 years of use in the clinic, cisplatin remains a central element of many treatment regimens. Cisplatin is still an irreplaceable component of a regimen that produces high cure rates in even advanced nonseminomatous germ-cell cancers, and is widely used in the treatment of ovarian cancers and other gynecologic cancers, head and neck, and numerous other tumor types. The development of carboplatin has reduced some of the adverse events associated with cisplatin treatment, and the introduction of the DACH platinum compound oxaliplatin has broadened the spectrum of activity of the platinums to include gastro-intestinal cancers, especially colorectal cancer. The clinical importance of this family of drugs continues to drive investigation into how these drugs work and how to improve their efficacy and reduce their toxicity. The papers in this volume were presented in Verona, Italy, during the tenth International Symposium on Platinum Coordination Compounds in Cancer Chemotherapy. The symposium was jointly organized by the Department of Oncology of the Mater Salutis Hospital – Azienda Sanitaria Locale 21 of the Veneto Region – and by the Department of Medicine and Public Health, Section of Pharmacology, the University of Verona. They reflect the vitality of this field and the increasing use of new molecular and cell biologic, genetic, and biochemical tools to identify approaches to further improve their use.

**Mechanisms of Drug Resistance in Neoplastic Cells** - Paul V. Woolley 2017-01-31

Bristol-Myers Cancer Symposia, Volume 9: Mechanisms of Drug Resistance in Neoplastic Cells provides information on both basic scientific and clinical studies on the causes and implications of tumor cell resistance to common antineoplastic agents. The book describes the colon cancer as a model for resistance to antineoplastic drugs; mathematical modeling of drug resistance; and the mechanism of induced gene amplification in mammalian cells. The text also discusses the cellular concomitants of multidrug resistance; resistance to alkylating agents; and the phosphoprotein and protein kinase C changes in human multidrug-resistant cancer cells. Novel drugs that affect glutathione metabolism; the regulation of genes encoding drug-metabolizing enzymes in normal and preneoplastic tissues; and the relevance of glutathione S-transferases to anticancer drug resistance are also considered. The book further tackles the cellular resistance to cyclophosphamide; the preclinical and clinical experiences with drug combinations designed to inhibit DNA repair in resistant human tumor cells; and the modification of the cytotoxicity of DNA-directed chemotherapeutic agents by polyamine depletion. The text also demonstrates multidrug resistance and the circumvention of resistance. Oncologists, molecular biologists, biochemists, geneticists, and pharmacologists will find the book invaluable.

**Multiple Drug Resistance in Cancer 2** - Martin Clynes 2013-04-17

Resistance to chemotherapy, and especially multi-drug resistance, represents a significant barrier to the successful treatment of cancer. This multi-author volume brings together a wide range of up-to-date reviews on different aspects of our knowledge of drug-resistance mechanisms, written by experts in the different areas. Particular attention is paid to recently discovered mechanisms relating to oncogene expression and in particular to proteins involved in regulation and execution of apoptosis. Other important topics covered include DNA repair, topoisomerases, cell cycle control, oxygenation and vascularisation of tumours, LRP, intermediate filament proteins and low-level resistance. Recent developments in understanding the role of efflux pumps (P-170, MRP) in multi-drug resistance are also reviewed. This book will be useful to clinicians and scientists working in the areas of chemotherapy, drug resistance, DNA repair and apoptosis research.

**Molecular Mechanisms of Drug Resistance And Strategies of Sensitization in Breast Cancer** - Yan Cheng 2022-03-02

*Molecular Mechanisms of Tumor Cell Resistance to Chemotherapy* - Benjamin Bonavida 2013-07-04

This volume gives the latest developments in on the mechanisms of cancer cell resistance to apoptotic stimuli, which eventually result in cancer progression and metastasis. One of the main challenges in cancer research is to develop new therapies to combat resistant tumors. The development of new effective therapies will be dependent on delineating the biochemical, molecular, and genetic mechanisms that regulate tumor cell resistance to cytotoxic drug-induced apoptosis. These mechanisms should reveal gene products that directly regulate resistance in order to develop new drugs that target these resistance factors and such new drugs may either be selective or common to various cancers. If successful, new drugs may not be toxic and may be used effectively in combination with subtoxic conventional drugs to achieve synergy and to reverse tumor cell resistance. The research developments presented in this book can be translated to produce better clinical responses to resistant tumors.

**Resistance to Molecular Therapies for Hepatocellular Carcinoma** - Augusto Villanueva 2017-07-14

This volume evaluates the clinical patterns of resistance to sorafenib, the impact of trial design in the second-line setting and the current gold standard to define radiological resistance; describes the molecular mechanisms responsible for treatment resistance in HCC patients, including components of the immune system and tumor microenvironment; determines the role of the cancer stem cell phenotype in resistance; reviews the experimental models to study resistance; and addresses new approaches to overcome resistance to sorafenib, using successful examples from other malignancies.

*Characterization of Extracellular Matrix-induced Molecular Mechanisms of Cancer Cell Resistance in 2- and 3-dimensional Culture Systems* - Nilly Shimony 2010

*The Molecular Mechanisms of Tumor Cell Progression and Resistance to Polychemoterapy in Primary Hepatobiliary Cancers* - 2013

**Pancreatic Cancer** - Sanjay Srivastava 2012-03-23

This book provides the reader with an overall understanding of the biology of pancreatic cancer, hereditary, complex signaling pathways and alternative therapies. The book explains nutrigenomics and epigenetics mechanisms such as DNA methylation, which may explain the etiology or progression of pancreatic cancer. Book also summarizes the molecular control of oncogenic pathways such as K-Ras and KLF4. Since pancreatic cancer metastasizes to vital organs resulting in poor prognosis, special emphasis is given to the mechanism of tumor cell invasion and metastasis. Role of nitric oxide and Syk kinase in tumor metastasis is discussed in detail. Prevention strategies for pancreatic cancer are also described. The molecular mechanisms of the anti-cancer effects of curcumin, benzyl isothiocyante and vitamin D are discussed in detail. Furthermore, this book covers the basic mechanisms of resistance of pancreatic cancer to chemotherapy drugs such as gemcitabine and 5-flourouracil.

**Molecular and Cellular Biology of Multidrug Resistance in Tumor Cells** - I.B. Roninson 2012-12-06

The ability of neoplastic cells to survive exposure to various chemotherapeutic drugs represents the main obstacle to successful cancer chemotherapy. This book deals with a particular type of resistance in tumor cells that represents a single but especially important aspect of the multifaceted problem of cancer drug resistance. This type of resistance, known as multidrug or pleiotropic drug resistance, is characterized by cross-resistance of cells to several different classes of cytotoxic drugs, including some of the most commonly used anticancer agents. Over the last several years, there has been a veritable explosion of genetic, biochemical, and clinical information on multidrug resistance, which followed the identification and cloning of the genes responsible for this phenotype and the isolation of monoclonal antibodies against P-glycoproteins, the products of these genes. Elucida tion of the molecular

mechanism of multidrug resistance has led to the formulation of novel approaches to the prediction of tumor response to chemotherapeutic drugs and increasing the efficacy of cancer therapy. Analysis of the structure and function of P glycoproteins from multidrug-resistant mammalian cells has also established a prototype for a novel class of eukaryotic membrane proteins, which have now been associated with a variety of transport processes in different organisms. This book summarizes the results of molecular biological, pharmacological, bio chemical, cytogenetic, immunological, and pathological studies on multidrug resistance in mammalian cells. Most of the chapters deal at least to some extent with the structure and expression of P-glycoprotein and its role in multidrug resistance.

*Tumor Microenvironment: Molecular Mechanisms and Signaling Pathways Involved in Metastatic Progression* - Antonella Zannetti 2021-09-30

**Apoptosis, Cell Signaling, and Human Diseases** - Rakesh Srivastava 2007-11-05

These volumes present a concise synthesis of recent developments in the understanding of both cell survival and apoptotic pathways. Particular attention is given to apoptosis in human diseases, such as different forms of cancer and neurodegenerative diseases. These comprehensive volumes integrate the most innovative and current findings from several related disciplines of scientific research, including pathology, genetics, virology, cell biology, immunology, and molecular biology.

**YY1 in the Control of the Pathogenesis and Drug Resistance of Cancer** - Benjamin Bonavida 2020-10-20

YY1 Is Pivotal in the Control of the Pathogenesis and Drug Resistance of Cancer: A Critical Therapeutic Target describes the current state-of-the-art of the transcription factor YY1 that is overexpressed in the majority of cancers and a central factor that regulates all of the major features and characteristics of human cancers. This book emphasizes the biochemical, molecular and genetic underlying mechanisms by which YY1 regulates its pro-cancerous activities. In addition, it also describes the role of YY1 in the regulation of tumor cell resistance to conventional chemo and immunotherapies and the important role of inhibiting YY1 in cancer. This book is a valuable source for cancer researchers, oncologists and several members of medical and biomedical field who are interested in understanding further the role of YY1 in cancer. Provides a thorough understanding of the underlying mechanisms by which YY1 regulates cancer cell phenotype and unique characteristics Discusses the novel mechanisms of YY1 regulation of tumor cell resistance and means to overcome resistance Encompasses new examples of newly developed non-toxic and selective inhibitors targeting YY1

**Molecular and Clinical Advances in Anticancer Drug Resistance** - Robert F. Ozols 2012-12-06

The importance of drug resistance in cancer chemotherapy cannot be over stated. The 500,000 patients who die every year from cancer in the United States have, in most cases, been treated with chemotherapy. Many of these patients responded initially to chemotherapy, but death resulted from the development of drug-resistant tumors. In the first volume in the series. Drug Resistance in Chemotherapy the results of comprehensive laboratory studies aimed at understanding the mechanisms for resistance to individual agents and to the development of broad cross-resistance were described. In the past 2 years there has been substantial progress in understanding the molecular biology associated with these mechanisms of drug resistance. For the first time we are starting to understand which mechanisms are playing an im portant role in human tumors, and even more importantly, clinical trials have recently been initiated in an effort to reverse specific forms of drug resistance. The purpose of this volume is to describe the new advances, both at the molecular level and in the clinic regarding mechanisms of drug resistance and potential ways this resistance can be circumvented. This volume is focused upon mechanisms of resistance associated with two major classes of anticancer drugs: alkylating agents (including cisplatin) and the natural products (e. g. , adriamycin and vinblastine). The first section of the book describes new insights into the genetic mechanisms associated with drug resistance.

**Understanding Cancer** - David Tarin 2023-02-02

This book provides a unique, wide-ranging description of the phenomenon of cancer and its pathological effects in diverse species including humans, domesticated and wild animals, invertebrates, and plants. The broad scope of information presented is used to construct radical new insights into biological self-regulation and explain their relevance to its disruption by cancerous growth and spread within the human body. Mechanisms of action of carcinogenic agents, initiation, progression, metastasis, inappropriate gene expression, dormancy, latency, regression, and reasons for susceptibility and/or resistance to cancer are all considered. Also discussed are criteria for pathological diagnosis, advances in treatment, implications for public health, and pitfalls in diagnosis and interpretation of experimental results. The book describes operational mechanisms of cancer at the levels of whole individual, organ, tissue, cell, molecular, and even atomic (quantum) scales of structural and physiological order. Evidence is assembled from all these levels of organization to show that cancer is a dynamically changing disorder and that it is an inherent and perpetual risk of multicellular composition. This provides pragmatic new biological and clinical perspectives on malignant neoplasia. The biological insight is that it is a consequence of progressing miscommunication within a cellular society. The clinical perspective is realistic but optimistic in reasoning that, although cancer can never be completely eradicated from human life, because it is a disorder of our intrinsic biological constitution, it can be controlled and ameliorated and even cured in a proportion of individuals. The text is profusely illustrated with over 300 macroscopic and microscopic pictures. It will stimulate curiosity and interest specialists, as well as beginners, in many scientific disciplines and provides copious references to the medical and scientific literature supporting its conclusions. Readers from fields as diverse as medicine, pathology, veterinary sciences, cell biology, molecular biology, developmental biology, and epidemiology will find the information the book contains thought-provoking, interesting, and useful. Additionally, specialists in occupational and environmental health and legal experts focusing on exposure to carcinogenic materials and pollution will find the contents valuable and informative.

**Molecular Mechanisms and Their Clinical Application in Malignancies** - Daniel E. Bergsagel 2013-10-22

Molecular Mechanisms and their Clinical Application in Malignancies is a collection of manuscripts presented at the 12th Annual Bristol-Myers Squibb Symposium on Cancer Research, held in Toronto, Canada on September 26-27, 1989. This symposium reviews the current understanding of the mechanisms of malignant transformation and the application of several technologies to the diagnosis, evaluation, and treatment of malignancies. This book is divided into 14 chapters. The opening chapters deal with the genetic basis of neoplasia and the molecular biology of oncogenes, the regulation of transcription, and the rearrangement of T- and B-cell genes during development and in malignancies. The subsequent chapters focus on the genetic abnormalities detected in specific tumors, such as retinoblastoma, colorectal carcinoma, and lung cancer. These chapters also examine the retinoid and thyroid hormone receptors. Other chapters explore the genetic basis of the cellular response to therapy, drug resistance, cachectin-tumor necrosis factor in the biology of disease, acute myelogenous leukemia, and the stages in tumor progression. The final chapters look into the application of molecular biology to clinical treatment in the form of receptor specific intoxication on tumor cells, the molecular genetic analysis of the phakomycoses, and the structural design of antitumor compounds. This book will prove useful to oncologists, molecular biologists, clinicians, and researchers.

Chemoresistance and Metastasis in Breast Cancer: Molecular Mechanisms and Novel Clinical Strategies - Qifeng Yang 2022-03-18

**Insulin Resistance and Cancer** - I. George Fantus 2011-08-19

This book reviews the epidemiological associations between insulin resistance and cancer. This is followed by reviews of animal models which support this relationship and provide insight into potential mechanisms. Several chapters then provide detailed examination of the cellular and molecular changes characterizing the insulin resistant state, such as hyperinsulinemia, abnormal metabolism and hormone signaling, and how

these interact with various tumor characteristics. For example some tumors present increased quantities of the fetal form of the insulin receptor, unique regulation of oxidative (Krebs' cycle) metabolism (Warburg effect), as well as mutations in various relevant signaling pathways. Finally, the clinical implications of these data are integrated with considerations of insulin "sensitization" and potential metabolic interventions to prevent and treat cancer. It should be noted that while a number of cancers are associated with obesity the authors here have focused primarily on breast cancer as a key and significant model.

**Insights Into the Molecular Mechanisms of Phospholipase D-mediated Cancer Cell Survival** - Ronald Chase Bruntz 2014

**Biological Mechanisms and the Advancing Approaches to Overcoming Cancer Drug Resistance** - Andrew Freywald 2020-12-04

Biological Mechanisms and the Advancing Approaches to Overcoming Cancer Drug Resistance, Volume 12, discusses new approaches that are being undertaken to counteract tumor plasticity, understand and tackle the interactions with the microenvironment, and disrupt the rewiring of malignant cells or bypass biological mechanism of resistance by using targeted radionuclide therapies. This book provides a unique opportunity to the reader to understand the fundamental causes of drug resistance and how different approaches are applied. It is a one-stop-shop to understand why it is so difficult to treat cancer, and why only a very few patients respond to therapy and a significant portion develop resistance. Despite a rapid development of more effective anti-cancer drugs and combination therapies, cancer remains the leading cause of lethality in the developed world. The main reason for this is the ability of heterogeneous subpopulations of tumor cells interacting with constantly evolving tumor microenvironment to resist elimination and eventually, trigger cancer relapse. In this book, experts review current concepts explaining molecular and biological mechanisms of cancer drug resistance and discussing advancing approaches for overcoming these therapeutic challenges. Provides the most updated knowledge on the mechanisms of cancer drug resistance and the emerging therapeutic approaches reviewed by experts in the field Brings detailed analyses of most important recently reported developments related to drug resistance and their relevance to overcoming it in cancer patients Discusses in-depth molecular mechanisms and novel concepts of cancer resistance to conventional and advanced therapies

Cancer Metastasis - Richard G. Vile 1995-04-25

A concise description of the metastasis process and its therapeutic relevance in controlling the clinical outcome of the tumor. Explains why the clinical importance of metastasis could be the most life-threatening element in the tumorigenic procedure.

**Glioblastoma Resistance to Chemotherapy: Molecular Mechanisms and Innovative Reversal Strategies** - Ramasamy Paulmurugan 2021-06-25

Glioblastoma Resistance to Chemotherapy: Molecular Mechanisms and Innovative Reversal Strategies brings current knowledge from an international team of experts on the science and clinical management of glioblastoma chemoresistance. The book discusses topics such as molecular mechanisms of chemoresistance, experimental models to study chemoresistance, chemoresistance to drugs other than Temozolomide, and specific strategies to reverse chemoresistance. Additionally, it encompasses information on how to mitigate chemoresistance by targeted enhancement of p53 function. This book is a valuable resource for cancer researchers, oncologists, neuro-oncologists and other members of the biomedical field. Glioblastoma (GBM) is the most invasive and malignant primary brain tumor in humans with poor survival after diagnosis, therefore it is imperative that molecular and cellular mechanisms behind therapy resistant GBM cells, as well as the therapeutic strategies available to counter the resistance are comprehensively understood. Provides comprehensive, core knowledge related to the entire discipline of glioblastoma chemoresistance, from its many etiological mechanisms, to specific strategies to reverse resistance Presents current information from an international team of experts on the basic science, pre-clinical research, and clinical management of glioblastoma chemoresistance Discusses molecular and cellular

mechanisms behind therapy resistant glioblastoma cells, as well as the therapeutic strategies available to counter this resistance

**Inflammatory Tumor Immune Microenvironment: Molecular Mechanisms and Signaling Pathways in Cancer Progression and Metastasis** - Xu Wang 2022-03-25

Breast Cancer Chemosensitivity - Dihua Yu 2009-12-30

In Breast Cancer Chemosensitivity, a group of world leading experts review critical aspects of resistance to systemic therapy in breast cancer patients. Beginning with a clinical overview of the problem, the book then focuses on the latest findings of molecular mechanisms of drug resistance. Coverage provides an example of using novel approaches for chemosensitization of breast cancer cells that gives readers an idea about the future direction in breast cancer treatment. It allows those who are interested in breast cancer therapy to get a jump-start on critical issues in breast cancer therapeutic resistance.

**Molecular Biology of the Cell** - Bruce Alberts 2004

**Apoptosis, Cell Signaling, and Human Diseases** - Rakesh Srivastava 2007-11-05

Apoptosis, Cell Signaling, and Human Diseases: Molecular Mechanisms, Volumes 1 and 2, present a concise synthesis of recent developments in the understanding of both cell survival and apoptotic pathways. Particular attention is given to apoptosis in human diseases, such as different forms of cancer. These comprehensive volumes integrate the most innovative and current findings. The contributors are at the forefront of scientific discovery.

Resistance to Proteasome Inhibitors in Cancer - Q. Ping Dou 2014-09-16

The book explores cutting-edge strategies to overcome proteasome inhibitor resistance, including the second generation 20S proteasome inhibitors, novel combinational therapies, and new targets in the ubiquitin-proteasome pathway (e.g., ubiquitin E3 ligases, deubiquitinases, 19S proteasomal ATPases, histone deacetylases, oxidative stress and proteotoxic stress pathways and pharmacogenomic signature profiling) in resistant cancer cells. The mechanisms of action and resistance of proteasome inhibitors, such as bortezomib and carfilzomib in human cancers, including multiple myeloma, mantle cell lymphoma, acute leukemia, and solid tumors are explored in depth in this volume. This timely volume unveils the most current discoveries of the mechanisms behind proteasome inhibitor resistance, which will help illuminate the future of cancer therapies.

*Cytotoxic Drug Resistance Mechanisms* - Robert Brown 2008-02-01

There is now a range of cytotoxic drugs that have considerable clinical usefulness in producing responses in tumors and even, in a small proportion of cases, cure. However, the acquisition of drug resistance is a major clinical problem and is perhaps the main limiting factor in successful treatment of cancer. Thus, a tumor initially sensitive to chemotherapy will, in the majority of cases, eventually recur as a resistant tumor, which will then progress. Much of our understanding of drug resistance mechanisms comes from the study of tumor cell lines grown in tissue culture. We now understand many of the - lecular mechanisms that can lead to a cell acquiring resistance to antic- cer drugs; however, we still do not know which mechanism(s) are those most relevant to the problem of clinical drug resistance. Indeed, given that many of the cytotoxic anticancer drugs were discovered by random screening, it is - clear what features give a clinically useful anticancer drug a sufficient the- peutic index to be of value. The aim of Cytotoxic Drug Resistance Mechanisms is to provide pro- cols that are appropriate for examining the mechanisms of cellular resistance to anticancer cytotoxics in human tumor samples. Tumor cell lines have been enormously useful as experimental models of drug resistance mechanisms, however they have limitations and we need to address the relevance of such mechanisms in patients' tumors. Examining drug resistance in tumors is much more problematic than in cell lines.

Molecular Mechanisms of Programmed Cell Death - Yufang Shi 2013-06-29

The 2002 Nobel Prize in Physiology or Medicine was awarded to Sydney Brenner, H. Robert Horvitz, and John E. Sulston for their seminal discoveries concerning "genetic regulation of organ development and

programmed cell death." This clearly marked the prime importance of understanding the molecular mechanisms controlling cell death. The 1 st International Symposium on Programmed Cell Death was held in the Shanghai Science Center of the Chinese Academy of Sciences on September 8-12, 1996. A number of key issues in apoptosis were discussed at the meeting, and progress in major areas of apopto sis research was summarized by expert participants at the meeting and published by Plenum Publishing Corporation as a book entitled Programmed Cell Death. In the last six years, we have witnessed a real explosion in our knowledge on how cells undergo apoptosis, thereby participating in various developmental and pathophysiological processes. At this ever exciting time, we organized the 2nd International Symposium on Programmed Cell Death.

**MHC Class I Antigens In Malignant Cells** - Natalia Aptsiauri 2013-02-26

Abnormal expression of MHC class I molecules in malignant cells is a frequent occurrence that ranges from total loss of all class I antigens to partial loss of MHC specific haplotypes or alleles. Different mechanisms are described to be responsible for these alterations, requiring different therapeutic approaches. A complete characterization of these molecular defects is important for improvement of the strategies for the selection and follow-up of patients undergoing T-cell based cancer immunotherapy. Precise identification of the mechanism leading to MHC class I defects will help to develop new personalized patient-tailored treatment protocols. There is significant new research on the prevalence of various patterns of MHC class I defects and the underlying molecular mechanisms in different types of cancer. In contrast, few data is available on the changes in MHC class I expression during the course of cancer immunotherapy, but the authors have recently made discoveries that show the progression or regression of a tumor lesion in cancer patients undergoing immunotherapy depends on the molecular mechanism responsible for the MHC class I alteration and not on the type of immunotherapy used. According to this notion, the nature of the preexisting MHC class I lesion in the cancer cell has a crucial impact on determining the final outcome of cancer immunotherapy. This SpringerBrief will present how MHC class 1 is expressed, explain its role in tumor progression, and its role in resistance to immunotherapy.