Drug Resistance In Cancer Chemotherapy As An Optimal

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Overcoming Drug Resistance to Cancer Therapies Lung Cancer — Overcoming Drug Resistance

Targeting Cancer Pathways: Tumor Resistance

Overcoming drug resistance in ovarian cancer The challenges of drug resistance with targetted cancer therapies

Understanding Drug ResistanceOvercoming Drug Resistance in Ovarian Cancer | Dana-Farber Cancer Institute Charles Sawyers - Overcoming Cancer Drug Resistance The evolution of drug resistance and the orthodoxy of chemotherapy | Andrew Read **Drug-resistant tumor cell model generation**

Epigenetic Reversal of Chemotherapy Resistance in Cancers*Drug Resistance and the Treatment of Cancer* How to Remember Classification of Anticancer Drugs?? anti-NEO PLASTIC man - A Review of Common Toxicities with Traditional Chemotherapy Medications Oncogenetics - Mechanism of Cancer (tumor suppressor genes and oncogenes) *Pharmacology - CANCER DRUGS - HORMONAL*

THERAPY (MADE EASY) **6.6 - Cancer: Adaptive chemotherapy** ~~Update on Mechanisms of Resistance in Castration-Resistant Prostate Cancer~~ Mechanisms of Resistance to EGFR TKI and New Treatment Strategies *Lactate enhances lung cancer cells' resistance to chemotherapy drug etoposide*

Cancer cells plasticity and drug resistance Prof. Yehuda Assaraf - drug resistance leukemia breast cancer ~~Drug Resistance~~ Science Spotlight - Lineage Plasticity and Cancer Drug Resistance | Memorial Sloan Kettering Cancer Cells Send Signals Boosting Survival and Drug Resistance in Other Cancer Cells *\"What is drug resistance and how does it impact the effectiveness of cancer therapies?\"* ~~Antibiotic resistance puts surgery, chemo patients at risk~~ Drug Resistance In Cancer Chemotherapy
Resistance to cancer chemotherapy: failure in drug response from ADME to P-gp Abstract. Cancer chemotherapy resistance (MDR) is the innate and/or acquired ability of cancer cells to evade the... Background. In US only, the newly diagnosed cancer patient is 1,665,540 every year and the estimated ...

Resistance to cancer chemotherapy: failure in drug ...
Drug resistance in chemotherapy treatment. One obstacle to success with chemotherapy treatment is drug resistance. Patients receiving chemotherapy can develop resistance to previously effective drugs to the point that the drugs are no longer effective. Resistance – also called tachyphylaxis – occurs when a cancer cell develops the ability to keep the chemotherapy drug from entering it, or reduces the amount that can enter to a level that does not cause damage.

Drug resistance in chemotherapy treatment
Resistance to current chemo- and radiation therapy is the principal problem in anticancer treatment.

Although intensively investigated, the therapeutic outcome is still far from satisfactory. Among the multiple factors which contribute to the drug resistance in cancer cells, the involvement of autophagy is becoming more and more evident. Autophagy describes a cellular self-digestion process, in which cytoplasmic elements can be selectively engulfed and finally degraded in autophagolysosomes ...

Drug Resistance in Cancer - an overview | ScienceDirect Topics

Chemotherapy resistance occurs when cancers that have been responding to a therapy suddenly begin to grow. In other words, the cancer cells are resisting the effects of the chemotherapy. You may hear statements like the "cancer chemotherapy failed." When this occurs, the drugs will need to be changed.

Chemotherapy Resistance - What is Chemotherapy? - Chemocare

An international research team, led by scientists from Mater Research—The University of Queensland, have discovered they can overcome chemotherapy resistance in an ovarian cancer subtype by using...

Drug overcomes chemotherapy resistance in ovarian cancer

The drug imatinib mesylate (Gleevec, formerly STI571; Novartis, Basel) is a small-molecule inhibitor of the Bcr-Abl kinase and can achieve sustained hematologic and cytogenetic responses in chronic phase disease. Treatment of blast crisis, however, often fails because of drug resistance (19).

Drug resistance in cancer: Principles of emergence and ...

That is why combining treatments that have different mechanisms of action can kill more cancer cells and reduce the chance that drug resistance will emerge. Most of the research on drug resistance has

focused on identifying genetic mechanisms, such as mutations that alter a protein such that it impairs the binding of a drug. Research is revealing the importance of additional mechanisms of drug resistance, such as epigenetic factors that regulate the activity of genes and the dynamics between ...

Cancer Drug Resistance - National Cancer Institute

Chemotherapy is the standard internal medical treatment for cancer. However, the resistance of cancer cells to nearly all kinds of chemotherapeutic drugs and targeted drugs has become prevalent, and approximately 80-90% of deaths in cancer patients are directly or indirectly attributed to drug resistance. The progress of new drug research and development has also been impeded by the occurrence of drug resistance, which has emerged as a considerable challenge in cancer therapy.

Natural products to prevent drug resistance in cancer ...

Definition. Antineoplastic resistance, synonymous with chemotherapy resistance, is the ability of cancer cells to survive and grow despite different anti-cancer therapies, i.e. their multiple drug resistance. There are two general causes of antineoplastic therapy failure: Inherent resistance, such as genetic characteristics, giving cancer cells their resistance from the beginning, which is rooted in the concept of cancer cell heterogeneity and acquired resistance after drug exposure.

Antineoplastic resistance - Wikipedia

Though chemotherapy might kill most of the cancer, tiny populations of drug-resistant cancer cells manage to survive and propagate. Unlike the more familiar case of antibiotic-resistant bacteria,...

How Cancer Can Become Therapy-Resistant - Scientific American

Commonly known as drug resistance, this phenomenon is one of the most challenging problems facing cancer researchers and patients today. When cancer cells resist the effects of drugs used for treatment, they can grow and reform tumors, a process known as recurrence or relapse.

Drug Combinations to Overcome Treatment Resistance ...

Moreover, cancer cells cultured in a low-glucose condition reduced the proportion of chemoresistant cells. Conclusion: Starvation therapy can be used as a new method to reverse drug resistance in cancer. Keywords: cancer drug-resistance, P-glycoprotein, starvation therapy, nanoparticles, resistance reversal

Effect of Starvation in Reversing Cancer Chemoresistance ...

Drug resistance still impedes successful cancer chemotherapy. A major goal of early concepts in individualized therapy was to develop in vitro tests to predict tumors' drug responsiveness. We have developed an in vitro short-term test based on nucleic acid precursor incorporation to determine clinical drug resistance. This test detects inherent and acquired resistance in vitro and ...

Prediction of Cancer Drug Resistance and Implications for ...

Multidrug resistance (MDR) -- a process in which tumors become resistant to multiple medicines -- is the main cause of failure of cancer chemotherapy. Tumor cells often acquire MDR by boosting...

Calcium bursts kill drug-resistant tumor cells -- ScienceDaily

However, drug resistance in cancer cells significantly reduces the effectiveness and sensitivity of

chemotherapy, leading to recurrence and treatment failure. Cancer cells inherently have...

New drug that can prevent drug resistance and adverse effects

This was further confirmed by ectopic overexpression of sirt1, which induced expression of P-glycoprotein and rendered cells resistant to doxorubicin. Collectively, these findings uncovered a novel function for the longevity gene sirt1 as a potential target for diagnosis and/or treatment of cancer resistance to chemotherapy.

Control of Multidrug Resistance Gene mdr1 and Cancer ...

Combination chemotherapy uses drugs that target different cancer hallmarks, resulting in synergistic or additive toxicity. This strategy enhances therapeutic efficacy as well as minimizes drug resistance and side effects. In this study, we investigated whether silver nanoparticles act as a combinatorial partner to cisplatin. In so doing, we compared post-exposure biological endpoints ...

Elucidating the cellular response of silver nanoparticles ...

About 15 percent of lung cancer cases are small cell lung cancer. Chemotherapy has been the mainstay of this subtype's treatment for decades, but after an initial response, tumors quickly develop drug resistance and the disease progresses. When their tumors recur, patients find that their doctors have little to offer.

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.

Over the last several decades, the introduction of new chemotherapeutic drugs and drug combinations has resulted in increased long term remission rates in several important tumor types. These include childhood leukemia, adult leukemias and lymphomas, as well as testicular and trophoblastic tumors. The addition of high-dose chemotherapy with growth factor and hemopoietic stem cell support has increased clinical remission rates even further. For the majority of patients with some of the more common malignancies, however, palliation (rather than cure) is still the most realistic goal of chemotherapy for metastatic disease. The failure of chemotherapy to cure metastatic cancer is commonly referred to among clinicians as "drug resistance". This phenomenon can, however, often be viewed as the survival of malignant cells that resulted from a failure to deliver an effective drug dose to the (cellular) target because of anyone of or combination of a multitude of individual factors. Clinically, this treatment failure is often viewed as the rapid occurrence of resistance at the single cell level. However, in experimental systems, stable drug resistance is usually relatively slow to emerge.

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

An accessible and sophisticated account of the biological mechanisms of drug resistance in cancer and their mathematical modelling.

In order to avoid late-stage drug failure due to factors such as undesirable metabolic instability, toxic metabolites, drug-drug interactions, and polymorphic metabolism, an enormous amount of effort has been expended by both the pharmaceutical industry and academia towards developing more powerful

techniques and screening assays to identify the metabolic profiles and enzymes involved in drug metabolism. This book presents some in-depth reviews of selected topics in drug metabolism. Among the key topics covered are: the interplay between drug transport and metabolism in oral bioavailability; the influence of genetic and epigenetic factors on drug metabolism; impact of disease on transport and metabolism; and the use of novel microdosing techniques and novel LC/MS and genomic technologies to predict the metabolic parameters and profiles of potential new drug candidates.

Drug Resistance as a Biochemical Target in Cancer Chemotherapy covers the proceedings of the 13th Bristol-Myers Squibb Symposium on Cancer Research, entitled ""Drug Resistance as a Biochemical Target in Cancer Chemotherapy"", hosted by the Japanese Foundation for Cancer Research in Tokyo. This book is divided into four parts encompassing 18 chapters that summarize the results of both preclinical and clinical research on circumvention of drug resistance. The first two parts discuss the genetic aspects of multidrug resistance and the proteins involved in drug resistance. These parts also examine the structure, function, and expression of P-glycoproteins, with an emphasis on the role of these proteins as targets for cancer chemotherapy. The third part describes the methods for detection of P-glycoprotein and its antagonists to counter clinical drug resistance. This topic is followed by a discussion on the interactions among steroid hormones, steroid hormone receptors, antiandrogens, biological-response modifiers, and cytotoxic drugs in human breast cancer. The concluding part explores the clinical applications of chemosensitizers in cancer therapy. This part also considers the alternative clinical approaches against drug failure, including non-crosss-resistant therapies, autologous bone marrow transplantation, dose-intensive therapy, and high-dose chemotherapy. Biomedical scientists and researchers and clinicians will find this book invaluable.

Drug Efflux Pumps in Cancer Resistance Pathways: From Molecular Recognition and Characterization to Possible Inhibition Strategies in Chemotherapy, Volume Seven, describes the fundamental aspects of efflux pumps of the ATP-binding cassette superfamily in cancer resistance pathways, along with strategies to target and improve chemotherapy efficacy. Pumps of the ATP-binding cassette superfamily (ABCs) regulate the access of drugs to the intracellular space. In this context, the overexpression of ABCs is a well-known mechanism of multidrug resistance in cancer and is associated with therapeutic failure. Cancer types discussed include breast, endocrine, hematologic, gastrointestinal, musculoskeletal, lung, skin and central nervous system cancers. The book is a valuable source for researchers and advanced students in cancer, biology, pharmacology, pharmaceutical sciences, biomaterials and medical/clinical sciences that are interested in accessing a comprehensive compendium on efflux pumps in mechanisms of cancer resistance. Offers comprehensive and detailed descriptions of the basic aspects of efflux pumps in a very schematic and didactic manner Describes the involvement of efflux pumps in cancer resistance in different cancer types Encompasses an updated overview on state-of-the-art approaches that capitalize on their inhibition to improve chemotherapy and overcome resistance

?????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.

Chemotherapy is one of the major treatment options for cancer patients; however, the efficacy of chemotherapeutic management of cancer is severely limited by multidrug resistance, in that cancer cells become simultaneously resistant to many structurally and mechanistically unrelated drugs. In the past three decades, a number of mechanisms by which cancer cells acquire multidrug resistance have been discovered. In addition, the development of agents or strategies to overcome resistance has been the subject of intense study. This book contains comprehensive and up-to-date reviews of multidrug resistance mechanisms, from over-expression of ATP-binding cassette drug transporters such as P-glycoprotein, multidrug resistance-associated proteins, and breast cancer resistance p- tein to the drug ratio-dependent antagonism and the paradigm of cancer stem cells. The book also includes strategies to overcome multidrug resistance, from the development of compounds that inhibit drug transporter function to the modulation of transporter expression. In addition, this book contains techniques for the detection and imaging of drug transporters, methods for the investigation of drug resistance in animal models, and strategies to evaluate the efficacy of resistance reversal agents. The book intends to provide a state-of-the-art collection of reviews and methods for both basic and clinician investigators who are interested in cancer multidrug resistance mechanisms and reversal strategies. Tianjin, China Jun Zhou v Contents Preface. . . . . v Contributors. . . . . ix 1 Multidrug

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.