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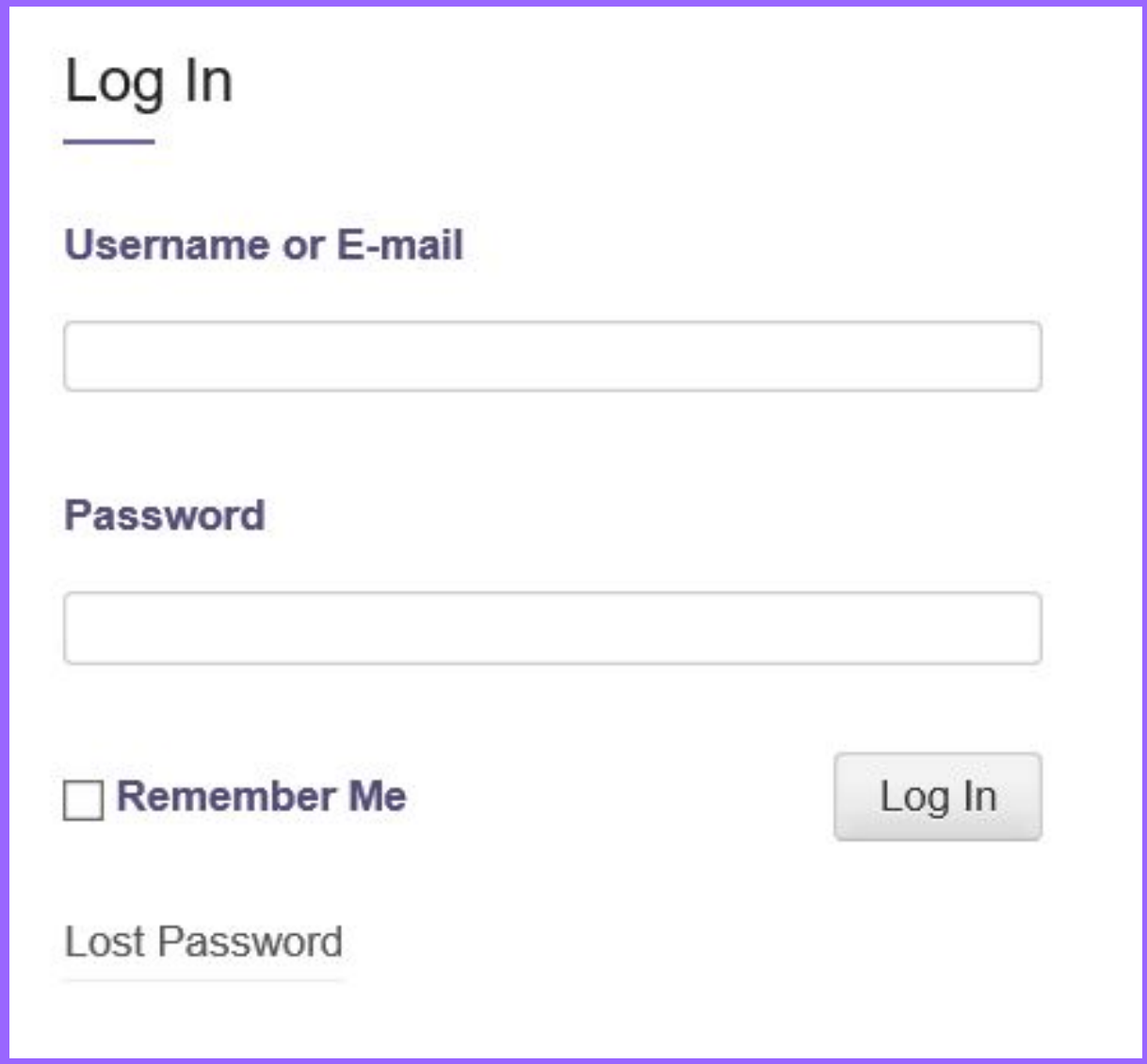
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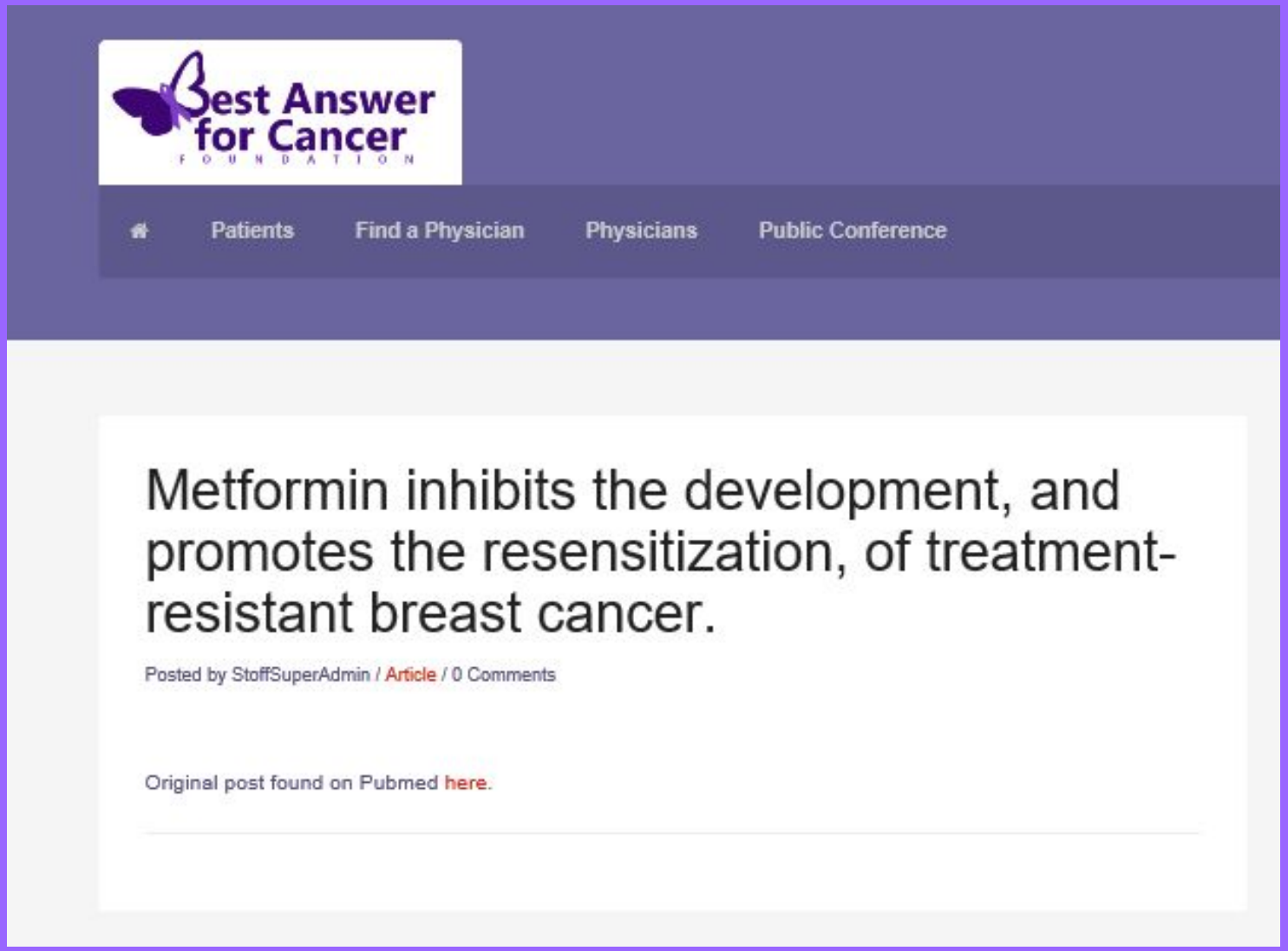
Breast Cancer X Metformin



PLoS One. 2017 Dec 6;12(12):e0187191. doi: 10.1371/journal.pone.0187191. eCollection 2017.

Metformin inhibits the development, and promotes the resensitization, of treatment-resistant breast cancer.

Davies G[1], Lobanova L[1], Dawicki W[2], Groot G[3], Gordon JR[2], Bowen M[2], Harkness T[1], Arnason T[1,2].

Author information

Abstract

Multiple drug resistant (MDR) malignancy remains a predictable and often terminal event in cancer therapy, and affects individuals with many cancer types, regardless of the stage at which they were originally diagnosed or the interval from last treatment. Protein biomarkers of MDR are not globally used for clinical decision-making, but include the overexpression of drug-efflux pumps (ABC transporter family) such as MDR-1 and BCRP, as well as HIF1α, a stress responsive transcription factor found elevated within many MDR tumors. Here, we present the important in vitro discovery that the development of MDR (in breast cancer cells) can be prevented, and that established MDR could be resensitized to therapy, by adjunct treatment with metformin. Metformin is prescribed globally to improve insulin sensitivity, including in those individuals with Type 2 Diabetes Mellitus (DM2). We demonstrate the effectiveness of metformin in resensitizing MDR breast cancer cell lines to their original treatment, and provide evidence that metformin may function through a mechanism involving post-translational histone modifications via an indirect histone deacetylase inhibitor (HDACi) activity. We find that metformin, at low physiological concentrations, reduces the expression of multiple classic protein markers of MDR in vitro and in preliminary in vivo models. Our demonstration that metformin can prevent MDR development and resensitize MDR cells to chemotherapy in vitro, provides important medical relevance towards metformin's potential clinical use against MDR cancers.

PMID: 29211738 PMCID: PMC5718420 DOI: 10.1371/journal.pone.0187191

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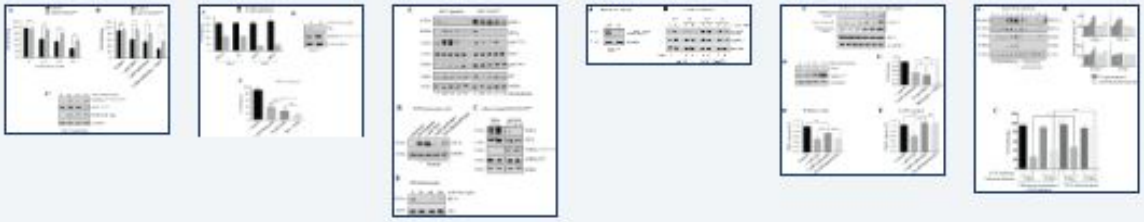

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October 19, 2016

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ERCC1, PD-L1, RRM1, TOP2A, TRKA/B/C, TS, TUBB3

Original post found here

Cell cycle arrest and autoschizis in a human bladder carcinoma cell line following Vitamin C and Vitamin K3

Immunotherapy for Bladder Cancer

How is Immunotherapy Changing the Outlook for Patients with Bladder Cancer?

Reviewed By: Padmanee Sharma, M.D., Ph.D. DETAILS +

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Bladder cancer was the first indication for which an immunotherapy was granted approval by the Food and Drug Administration (FDA) in 1990. Since 2016, two more immunotherapies have been approved, and there are currently a number of additional immune-based bladder cancer treatments in development. This page

FEATURED SCIENTIST

Clinical Trials for Bladder Cancer

Immunotherapy with BCG has reduced the risk of bladder cancer recurrence and increased the percentage of patients who experience a complete response after surgery, while atezolizumab and nivolumab have benefitted patients with advanced cases of bladder cancer. Investigational immunotherapies that train the immune system to recognize bladder cancer cells may further improve outcomes for bladder cancer patients. Below are descriptions of ongoing immunotherapy clinical trials for patients with bladder cancer.

A promising avenue of clinical research in bladder cancer is the use of immune checkpoint inhibitors. These treatments work by targeting molecules that serve as checks and balances in the regulation of immune responses. By blocking inhibitory molecules or, alternatively, activating stimulatory molecules, these treatments are designed to unleash or enhance pre-existing anti-cancer immune responses.

Atezolizumab (TECENTRIQ®): A PD-L1 Antibody

- A phase III study of atezolizumab as adjuvant therapy in patients with PD-L1-positive, high-risk muscle invasive bladder cancer after surgery to remove all or part of the bladder (NCT02450331).
- A phase II preoperative study of atezolizumab in patients with transitional cell cancer of the bladder (NCT02662309).
- A phase I/II study of atezolizumab in patients with advanced cancer, including bladder cancer, in combination with varlilumab (CDX-1127), an anti-CD27 antibody (NCT02543645).
- A phase I study of CPI-444, which targets the adenosine-A2A receptor that suppresses the anti-tumor activity of immune cells, +/- atezolizumab for patients with advanced cancer, including bladder cancer (NCT02655822).

Durvalumab (MEDI4736): A PD-L1 Antibody +/- Tremelimumab: A CTLA-4 Antibody

- A phase III trial of durvalumab +/- tremelimumab for patients with bladder cancer (NCT02516241).
- A phase I/II trial of durvalumab, tremelimumab, and Poly-ICLC, a Toll-like receptor 3 agonist, in patients with advanced, measurable cancers, including bladder cancer (NCT02643303).

Nivolumab (Opdivo®): A PD-1 Antibody +/- Ipilimumab (Yervoy®): A CTLA-4 Antibody

- A phase II trial of nivolumab +/- ipilimumab in patients with bladder cancer (NCT02553642).

Ott et al. Journal for ImmunoTherapy of Cancer (2017) 5:16
DOI 10.1186/s40425-017-0218-5

Journal for ImmunoTherapy of Cancer

REVIEW Open Access

Combination immunotherapy: a road map

Patrick A. Ott[1*], F. Stephen Hodi[1], Howard L. Kaufman[2], Jon M. Wigginton[3] and Jedd D. Wolchok[4]

Abstract

Cancer immunotherapy and in particular monoclonal antibodies blocking the inhibitory programed cell death 1 pathway (PD-1/PD-L1) have made a significant impact on the treatment of cancer patients in recent years. However, despite the remarkable clinical efficacy of these agents in a number of malignancies, it has become clear that they are not sufficiently active for many patients. Initial evidence, for example with combined inhibition of PD-1 and CTLA-4 in melanoma and non-small cell lung cancer (NSCLC), has highlighted the potential to further enhance the clinical benefits of monotherapies by combining agents with synergistic mechanisms of action. In order to address the current progress and consider challenges associated with these novel approaches, the Society for Immunotherapy of Cancer (SITC) convened a Combination Immunotherapy Task Force. This Task Force was charged with identifying and prioritizing the most promising prospects for combinatorial approaches as well as addressing the challenges associated with developing these strategies. As a result of the extensive clinical benefit and tolerable side effects demonstrated with agents inhibiting the PD-1 pathway, an overview of current evidence to support its promising potential for use as a backbone in combination strategies is presented. In addition, key issues in the development of these strategies including preclinical modeling, patient safety and toxicity considerations, clinical trial design, and endpoints are also discussed. Overall, the goal of this manuscript is to provide a summary of the current status and potential challenges associated with the development and clinical implementation of these strategies.

J Immunol. 2018 Jan 15;200(2):385-391. doi: 10.4049/jimmunol.1701302.

A Believer's Overview of Cancer Immunosurveillance and Immunotherapy.

Finn OJ[1].

Author information

Abstract

The field of tumor immunology has grown around the idea that one of the important roles of the immune system is to eliminate cancer. This idea was difficult to reconcile with the accepted notion that the immune system evolved to distinguish self from nonself and therefore tumors derived from self-tissues would not be recognized. Lack of appropriate animal models prevented experimental testing of cancer immunosurveillance. This changed with the realization that the immune system evolved to recognize danger and with the advent of mouse models deficient in one or more immune function, which showed predicted increases in susceptibility to cancer. Simultaneously, technical advances that enabled the study of the human immune system provided data for the existence of tumor-specific T cells and Abs and led to molecular identification of tumor Ags, fully validating the cancer immunosurveillance hypothesis. Immunotherapy designed to strengthen cancer immunosurveillance has achieved unprecedented clinical successes.

PMID: 29311379 PMCID: PMC5763509 [Available on 2019-01-15] DOI: 10.4049/jimmunol.1701302

Cell Cycle. 2002 Nov-Dec;1(6):369-74.

Cell cycle phase-specific chemotherapy: computational methods for guiding treatment.

Gardner SN[1].

Author information

Abstract

Computational models of cancer chemotherapy enhance the understanding of in vitro, in vivo, and clinical trial data and have the potential to contribute to the design of rational treatment regimens. In particular, mechanistic, predictive models are superior to statistical, phenomenological descriptions of data. Mechanistic models based on functional data from tumor biopsies will enable the response to treatment to be predicted for a specific patient, in contrast to statistical models in which the probability of response for a given patient may differ substantially from the population average. This review summarizes mathematical models developed to improve the design of treatment regimens using cell-cycle phase-specific chemotherapy. It starts with simple models of dose response, then moves to more complex models of scheduling cell-cycle phase-specific drugs, and finally discusses mechanistic models that incorporate both genetic drug resistance and cell cycle-mediated drug resistance. This last class of models will be most useful in designing treatment regimens tailored for individual patients.

PMID: 12548007 DOI: 10.4161/cc.1.6.258

Classification of Chemotherapy:

Drug Classification	Mechanism of Action
Alkylating agents	• Cell-cycle-non-specific • Break DNA helix strand, interfering with DNA replication • Most active in G0 phase
Antitumour Antibiotics	• Cell-cycle-non-specific • Bind to DNA and interfere with further replication of DNA and transcription of RNA
Antimetabolites	• Cell-cycle-specific to S phase • Resemble essential metabolic elements needed for cell growth • Inhibits RNA and DNA synthesis
Plant Alkaloids	• Cell-cycle-specific to M phase • Cause a cell cycle phase arrest
Hormones	• Cell-cycle-non-specific • Act in a variety of ways • Mechanism of action not fully understood • May have "direct lytic action" on cells in certain diseases (eg corticosteroids in leukemia, lymphoma) • Interfere with protein synthesis

- Anticancer strategies
  - Differentiation
  - Oncolytic virotherapy
  - Cytotoxic
  - Onco-immunology
  - Biochemical
  - Targeted therapies
  - Anti-angioneogenesis
  - Cancer Stem Cell Inhibitors
  - Oxidative
  - Anti-Inflammatory
  - Detox
  - Nutritional
  - Psychoneuroimmunology
  - Cancer Milieu Epigenetics
  - Cancer Genetics
  - Interventional, Radiation

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Thank You !