SEARCHING THE LITERATURE

UAB Resources and Services Supporting Translational Research

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UAB Lister Hill Library
lvucovi@uab.edu
4/13/2016
Objectives

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• Find research articles to support your work.
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<th>Title</th>
<th>Editors/Authors</th>
<th>Call Number</th>
<th>ISBN</th>
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<tr>
<td><strong>Autophagy: Cancer, Other Pathologies, Inflammation, Immunity, Infection, and Aging by M. A. Hayat (Editor)</strong></td>
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<td><strong>B Is for Balance by Sharon Weinstein</strong></td>
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<td><strong>Capstone Coach for Nursing Excellence by Linda Campbell; Marcia A. Gilbert; Gary Robert Laustsen</strong></td>
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<td>9781622021819</td>
<td>2014/2015 ed.</td>
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<td><strong>Modern Nutrition in Health and Disease by Shils; Katherine L. Tucker; Benjamin Caballer; Robert J. Cousins; A. Catharine Rosa; Thomas R. Ziegler</strong></td>
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<td>9781605474618</td>
<td>2014, 11th ed.</td>
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<td><strong>Molecularly Imprinted Catalysts by Songjun Li (Editor); Shunsheng Cao (Editor); Sergey A. Piletsky (Editor); Anthony P. F. Turner (Editor)</strong></td>
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<td>9780128014448</td>
<td>2016</td>
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<tr>
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17β-Estradiol Confers Protection after Traumatic Brain Injury in the Rat and Involves Activation of G Protein-Coupled Estrogen Receptor 1

Nicole L. Day, Condace L. Floyd, Tracy L. D'Alessandro, William J. Hubbard, and Irshad H. Chaudry


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PAY PER VIEW Journal of Neurotrauma - 30 (17):1531-1541; 17β-Estradiol Confers Protection after Traumatic Brain Injury in the Rat and Involves Activation of G Protein-Coupled Estrogen Receptor 1 (access for 24 hours for US $59.00)

ADD TO CART

Day NL¹, Floyd CL, D’Alessandro TL, Hubbard WJ, Chaudry IH.
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Poster child: Ready for a close-up
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Cancer metastases: Are one and all the same?
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Androgen receptor antagonists compromise T cell response against prostate cancer leading to early tumor relapse
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Cultured networks of excitatory projection neurons and inhibitory interneurons for studying human cortical neurotoxicity
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Individualizing liver transplant immunosuppression using a phenotypic personalized medicine platform
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Cyclosporin promotes atherosclerosis regression via macrophage reprogramming
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Letters

RESEARCH LETTER

Use of Open Access Platforms for Clinical Trial Data
Concerns over bias in clinical trial reporting have stimulated calls for more open data sharing. In response, multiple pharmaceutical companies have created mechanisms for investigators to access patient-level clinical trial data. However, if and how these shared clinical trial data are being used is unknown.

Methods | We evaluated how many clinical trials were publicly available to investigators through 3 open access platforms: ClinicalTrials.gov, the Yale University Open Data Access Project (YODA), and the Supporting Open Access for Researchers (SOAR) initiative. 2+ Sponsors depositing data in these platforms include GlaxoSmithKline, AstraZeneca, Boehringer Ingelheim, Eisai, Eli Lilly, Novartis, Roche, Sanofi, Takeda, Union Chimique Belge, ViVi Healthcare, Johnson & Johnson, Medtronic, and Bristol-Myers Squibb. Company policies on what trials are shared vary and are available online, but most include all trials within certain date ranges after regulatory review and publication of results.

Investigators submit a research proposal to the platforms, which is first reviewed for the availability of the trials requested and completeness of the application. Next, the proposal is reviewed by a panel comprising independent experts or members of the platform. The panel then rejects or approves the proposal based on scientific merit and adequacy of the research design to achieve scientific objectives. A data sharing agreement is then created. Each platform requires investigators to report any resulting publications.

We reviewed all proposals with data use agreements since inception of each platform (first in 2013 and December 31, 2015 from ClinicalTrials.gov and the YODA website or directly from SOAR), the characteristics of accepted proposals, and reported publications. We classified the main objective of the analysis based on review of the analytic plan and study design.
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Stem cell pathways contribute to clinical chemoresistance in ovarian cancer

Department of Obstetrics, University of Alabama at Birmingham, Birmingham, Alabama 35294, USA.

Abstract

PURPOSE: Within heterogeneous tumors, subpopulations often labeled cancer stem cells (CSCs) have enhanced tumorigenicity and chemoresistance in ex vivo models. However, whether these populations capable of surviving chemotherapy in de novo tumors is unknown.

EXPERIMENTAL DESIGN: We examined 45 matched primary/recurrent tumor pairs of high-grade ovarian adenocarcinomas for expression of CSC markers ALDH1A1, CD44, and CD133 using immunohistochemistry. Tumor samples collected immediately after completion of primary therapy were then laser capture microdissected and subjected to quantitative PCR array examining stem cell biology pathways (Hedgehog, Notch, TGF-β, and Wnt). Results were validated as important targets using siRNA-mediated downregulation.

RESULTS: Primary samples were composed of low densities of ALDH1A1, CD44, and CD133. Tumors immediately after primary therapy were more densely composed of each marker, whereas samples collected 1 year later, before initiating secondary therapy, were composed of similar percentages of each marker. In ovarian cancer cell lines, ALDH1A1 knockdown significantly decreased proliferation, while CD133 knockdown significantly decreased clonogenicity. CD133 knockdown reversed the effects of CD133 overexpression, tumor growth was significantly increased in transgenic mice overexpressing CD133 compared to control mice.

CONCLUSIONS: These data indicate that ovarian tumors are enriched with CSCs and stem cells, and that these populations contribute to clinical chemoresistance and ultimately recurrent disease.

MeSH Terms

Adenocarcinoma/metabolism
Adenocarcinoma/pathology
Aldehyde Dehydrogenase/analysis
Aldehyde Dehydrogenase/biosynthesis
Antigens, CD/analysis
Antigens, CD/biosynthesis
Antigens, CD/genetics
Antigens, CD44/analysis
Antigens, CD44/biosynthesis
Antigens, CD44/physiology
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Gene Expression Profiling
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Neoplasm Recurrence, Local/metabolism
Neoplastic Stem Cells/metabolism
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Nuclear Proteins/physiology
Ovarian Neoplasms/analysis
Ovarian Neoplasms/physiology
Ovarian Neoplasms/metabolism
Ovarian Neoplasms/pathology
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Receptors, Cell Surface/physiology
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Tumor necrosis factor α inhibitor therapy and cancer risk in chronic immune-mediated diseases


University of Pennsylvania, Department of Epidemiology and Biostatistics, 713 Blockley Hall, 423 Guardian Drive, Philadelphia, PA 19104, United States

University of Alabama at Birmingham, United States

Kaiser Permanente Northern California, Oakland, United States

Objective To compare the incidence of cancer following tumor necrosis factor α (TNFα) inhibitor therapy to that with commonly used alternative therapies across multiple immune-mediated diseases. Methods The Safety Assessment of Biological Therapeutics study used data from 4 sources: national Medicaid and Medicare databases, Tennessee Medicaid, pharmacy benefits plans for Medicare beneficiaries in New Jersey and Pennsylvania, and Kaiser Permanente Northern California. Propensity score-adjusted hazard ratios (HRs) and 95% confidence intervals (95% CIs) were computed to estimate the relative rates of cancer, comparing those treated with TNFα inhibitors to those treated with alternative disease-modifying therapies. The cancer-finding algorithm had a positive predictive value ranging from 31% for any leukemia to 89% for female breast cancer. Results We included 29,555 patients with rheumatoid arthritis (RA) (13,102 person-years), 6,367 patients with inflammatory bowel disease (HR 1.58, 95% CI 1.49-1.67), 1,298 patients with psoriasis (371 person-years), and 2,498 patients with psoriatic arthritis (618 person-years). The incidence of any solid cancer was not elevated in RA (HR 1.08, 95% CI 0.95-1.21), inflammatory bowel disease (HR 1.27, 95% CI 1.06-1.51), psoriasis (HR 0.95, 95% CI 0.82-1.11), or psoriatic arthritis (HR 0.94, 95% CI 0.72-1.23) within TNFα inhibitor therapy compared to disease-specific alternative therapy. Among RA patients, the incidence of any of the 10 most common cancers in the US and of nonmelanoma skin cancer was not increased with TNFα inhibitor therapy compared to treatment with comparator drugs. Conclusion Short-term cancer risk was not elevated among patients treated with TNFα inhibitor therapy relative to comparator drugs.

Indexed keywords
EMTREE drug terms: adalimumab; azathioprine; etanercept; hydroxychloroquine; infliximab; leflunomide; mercaptopurine; methotrexate; retinoid derivative; salazosulfapyridine; steroid; tumor necrosis factor alpha inhibitor
EMTREE medical terms: adult; aged; algorithm; ankylosing spondylitis; article; breast cancer; cancer incidence; cancer risk; enteritis; female; human; immunopathology; leukemia; major clinical study; male; phototherapy; priority journal; psoriasis; psoriatic arthritis; rheumatoid arthritis; side effect; skin cancer; solid tumor; steroid therapy; United States
MeSH: Adult; Aged; Antibodies, Monoclonal; Chronic Disease; Cohort Studies; Female; Follow-Up Studies; Humans; Immune System Diseases; Immunologic Factors; Incidence; Male; Middle Aged; Neoplasms; Retrospective Studies; Risk Factors; Tumor Necrosis Factor-alpha

Medline is the source for the MeSH terms of this document.

Cited by 26 documents

Related documents
Do inflammatory bowel disease causes cancer?
Mason, M., & Siegel, C.A. (2013) Inflammatory Bowel Diseases
Cancer risk in immune-mediated inflammatory diseases (IMID)
Infectious and malignant complications of TNF inhibitor therapy in IBD

Metrics
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References (36)
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Review of the Literature

A review of the nutrition education literature attests to the widely-recognized importance of incorporating research into dietetics curriculum. Several studies have found that nutrition professionals often lack research skills, which has proven to be an obstacle preventing them from effectively translating research evidence into practice. Several authors have reported the results of this skill deficiency among future nutrition professionals.

Smith describes a series of courses meant to progressively involve students in research activities.

References


3. Hymel GM: Integrating Research Competencies in Massage Therapy Education. *J Bodyw Mov*
The Bioenergetic Health Index: a new concept in mitochondrial translational research

Balu K. CHACKO†, Philip A. KRAMER†, Saranya RAVI†, Gloria A. BENAVIDES†, Tanecia MITCHELL†, Brian P. DRANKA†, David FERRICK†, Ashwani K. SINGHAL, Scott W. BALLINGER†, Shannon M. BAILEY†, Robert W. HARDY†, Jianhua ZHANG†, Degui ZHENG‡ and Victor M. DARLEY-USMAR††

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"Department of Biostatistics, University of Alabama at Birmingham, Birmingham, AL 35294, U.S.A.

Abstract
Bioenergetics has become central to our understanding of pathological mechanisms, the development of new therapeutic strategies and as a biomarker for disease progression in neurodegeneration, diabetes, cancer and cardiovascular disease. A key concept is that the mitochondrion can act as the ‘canary in the coal mine’ by serving as an early warning of bioenergetic crisis in patient populations. We propose that new clinical tests to monitor changes in bioenergetics in patient populations are needed to take advantage of the early and sensitive ability of bioenergetics to determine severity and progression in complex and multifactorial diseases. With the recent development of high-throughput assays to measure cellular energetic function in the small number of cells that can be isolated from human blood these clinical tests are now feasible. We have shown that the sequential addition of well-characterized inhibitors of oxidative phosphorylation allows a bioenergetic profile to be measured in cells isolated from normal or pathological samples. From these data we propose that a single value – the Bioenergetic Health Index (BHI) – can be calculated to represent the patient’s composite mitochondrial profile for a selected cell type. In the present Hypothesis paper, we discuss how BHI could serve as a dynamic index of bioenergetic health and how it can be measured in platelets and leucocytes. We propose that, ultimately, BHI has the potential to be a new biomarker for assessing patient health with both prognostic and diagnostic value.

Key words: aging, cardiovascular disease, haplotype, hepatotoxicity, neurodegenerative disease, oxidative reserve capacity

INTRODUCTION
Complex and chronic diseases with underlying mechanisms involving dysfunctional metabolism are a growing healthcare problem in the developed world [1–3]. The availability of low-cost high-calorie foods in combination with a contemporary sedentary lifestyle presents a unique combination of risk factors with multiple evolving co-morbidities, which increasingly challenges our healthcare system especially in terms of prediction and management. Defining energetic health has become a necessity for healthcare in the 21st Century, and at the present time no clinical practice guidelines exist for the assessment of energetic health in the 21st Century. Bioenergetic Health Index (BHI) in patient populations: Bioenergetic Health Index (BHI) in patient populations. This approach has the potential to be used as the basis of personalized cell-based measurements to quantify bioenergetic health.

Our recent findings support an emerging concept that circulating leucocytes and platelets can serve as ‘the canary in the coal mine’ by acting as early sensors or predictive biomarkers of mitochondrial function under conditions of metabolic stress [4–8]. These studies provided evidence that circulating leucocytes and platelets can serve as ‘the canary in the coal mine’ by acting as early sensors or predictive biomarkers of mitochondrial function under conditions of metabolic stress.
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