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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, Condiace L. Floyd, Tracy L. D’Alessandro, William J. Hubbard, and Irshad H. Chaudry

*Journal of Neurotrauma. September 2013, Vol. 30 (17): 1531-1541*

Day NL, Floyd CL, D’Alessandro TL, Hubbard WJ, Chaudry IH.
17β-Estradiol Confers Protection after Traumatic Brain Injury in the Rat and Involves Activation of G Protein-Coupled Estrogen Receptor 1

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

Abstract

Traumatic brain injury (TBI) is a significant public health problem in the United States. Despite preclinical success of various drugs, to date all clinical trials investigating potential therapeutics have failed. Recently, sex steroid hormones have sparked interest as possible neuroprotective agents after traumatic injury. One of these is 17β-estradiol (E2), the most abundant and potent endogenous vertebrate estrogen. The goal of our study was to investigate the acute potential protective effects of E2 or the specific G protein-coupled estrogen receptor 1 (GPER) agonist G-1 when administered in an intravenous bolus 1 hour post-injury in the lateral fluid percussion (LFP) model of TBI. The results of this study show that, when assessed at 24 hours post-injury, E2 or G-1 confers protection in adult male rats subjected to LFP brain injury. Specifically, we found that an acute bolus dose of E2 or G-1 administered intravenously 1 hour post-TBI significantly increases neuronal survival in the ipsilateral CA 2/3 region of the hippocampus and decreases neuronal degeneration and apoptotic cell death in both the ipsilateral cortex and CA 2/5 region of the hippocampus. We also report a significant reduction in astrocytosis in the ipsilateral cortex, hilus, and CA 2/3 region of the hippocampus. Finally, these effects were observed to be dose-dependent for E2, with the 5mg/kg dose generating a more robust level of protection. Our findings further elucidate estrogens as a clinically relevant pharmacotherapeutic strategy for treatment of secondary injury following TBI, and intriguingly, reveal a novel potential therapeutic target in GPER.

Key words: apoptosis; estrogen; lateral fluid percussion; neuronal degeneration; neuroprotection

Introduction

Traumatic brain injury (TBI) is a significant public health problem in the United States. Annually, approximately 1.7 million TBIs are incurred, 53,000 people die, and 32–3.3 million others are living with long-term disabilities as a result. Despite preclinical successes, to date all clinical trials investigating potential therapeutics have failed. In addition, the financial burden associated with TBI is estimated at roughly $76 billion a year. 1,2 Traumatic brain injury (primary injury) and neurochemically-mediated damage (secondary injury) often lead to deficits in cognition, neuropsychiatry, and physical functioning. 3 Secondary injury mechanisms remain targets in the pathophysiology of TBI that could be manipulated by therapeutic interventions for prevention of further cell destruction and dysfunction. Thus, there is an urgent need for novel drug therapies that efficaciously target aspects of secondary injury.

Recently, sex steroid hormones have sparked interest as possible therapeutic agents following traumatic injury. One of these is 17β-estradiol (E2), the most abundant and potent endogenous vertebrate estrogen. Our research group has previously reported that E2 administration confers protection in models of spinal cord injury (SCI) and severe blood loss. 4,5 In prior TBI research, E2 has been shown to reduce cerebral contusion volume, apoptosis, blood-brain barrier permeability, edema, levels of pro-inflammatory cytokines, and intracranial pressure (ICP), as well as to upregulate expression of anti-apoptotic receptors Bid-2, increase cerebral perfusion pressure (CPP), and improve neurologic scores. 6-8 Taken together, these data suggest that E2 is protective and warrants further study as a potential therapeutic for treatment of TBI.

E2 signals through the classical estrogen receptors and β (ERβ) and the newly characterized G protein-coupled estrogen receptor 1 (GPER), which binds E2 and various estrogenic compounds, including the GPER-specific agonist, G-1, and initiates rapid intracellular signaling events. 9-10 However, GPER's role in the CNS has yet to be fully characterized, and its potential contributions to protection in TBI remains uninvestigated. Because GPER binds E2 as well as other more specific ligands, it could serve as a novel therapeutic target.
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Department of Obstetrics, University of Alabama at Birmingham, Birmingham, Alabama 35294, USA.

Abstract

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

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

RESULTS: Primary samples were composed of low densities of ALDH1A1, CD44, and CD133. Tumors collected immediately after primary therapy were more densely composed of each marker, whereas samples collected after initiating secondary therapy were composed of similar percentages of each marker. In tumors collected from recurrent platinum-resistant patients, only CD133 was significantly overexpressed in recurrent compared with matched primary samples, with knockdown of genes of interest, including endoglin/CD105 and the hedgehog mediators Gil1 and GI2, showing a novel contribution to cisplatin resistance.

CONCLUSIONS: These data indicate that ovarian tumors are enriched with CSCs and stem cell pathways, especially at the completion of primary therapy. This suggests that stem cell subpopulations contribute to chemoresistance and ultimately recurrent disease.

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Interleukin 17-producing CD4⁺ effector T cells develop via a lineage distinct from the T helper type 1 and 2 lineages

Harrington, L.E.¹, Hatton, R.D.², Mangan, P.R.⁵, Turner, H.⁴, Murphy, T.L.³, Murphy, K.M.⁴, Weaver, C.T.³

¹ Department of Pathology, University of Alabama at Birmingham, Birmingham, AL 35294, United States
² Department of Pathology and Immunology, Howard Hughes Medical Institute, Washington University School of Medicine, St. Louis, MO 63110, United States
³ Department of Microbiology and Immunology, Oregon Health and Science University, Portland, OR 97201, United States
⁴ Department of Pathology, The University of Texas M. D. Anderson Cancer Center, Houston, TX 77030, United States
⁵ Department of Immunobiology, Yale University School of Medicine, New Haven, CT 06520, United States

Abstract

CD4⁺ T cells producing interleukin 17 (IL-17) are associated with autoimmunity, although the precise mechanisms that control their development are undefined. Here we present data that challenge the idea of a shared developmental pathway with T helper type 1 (Th1) or Th2 lineages and instead favor the idea of a distinct effector lineage we call Th17. The development of Th17 cells from naive precursor cells was potently inhibited by interferon-γ (IFN-γ) and IL-4, whereas committed Th17 cells were resistant to suppression by IFN-γ or IL-4. IL-23 induced naive precursor cells to differentiate into Th17 cells independently of the transcription factors STAT1, T-bet, STAT4 and STAT6. These findings provide a basis for understanding how inhibition of IFN-γ signaling enhances development of pathogenic Th17 effector cells that can exacerbate autoimmunity. © 2005 Nature Publishing Group.

Indexed keywords

IL-17 family cytokines and the expanding diversity of effector T cell lineages
Weaver, C.T., Hatton, R.D., Mangan, P.R. (2007) Annual Review of Immunology

Expanding the effector CD4⁺ T-cell repertoire: the Th17 lineage
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Chapter 5 Emergence of the Th17 Pathway and Its Roles in Host Defense

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Review of the Literature

A review of the nutrition education literature attests to the widely-recognized value of incorporating research into dietetics curriculum. Several studies\(^{14-16}\) have found that students who lack research skills are at risk for not being able to apply research evidence into practice.\(^{17}\) Several authors have reported the results of training students to be evidence-based practitioners.\(^{18}\)

Smith describes a series of courses designed to teach students how to utilize evidence-based practice in their future work.\(^{19}\)

References


3. Hymel GM: Integrating Research Competencies in Massage Therapy Education.
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 ZHI†,‡ and Victor M. DARLEY-USMAR†,‡

*Mitochondrial Medicine Laboratory, University of Alabama at Birmingham, Birmingham, AL 35294, U.S.A.
†Department of Pathology, University of Alabama at Birmingham, Birmingham, AL 35294, U.S.A.
‡Seahorse Bioscience, North Billerica, MA 01862, U.S.A.
§Division of Gastroenterology and Hepatology, Department of Internal Medicine, University of Alabama at Birmingham, Birmingham, AL 35294, U.S.A.
¶Department of Veteran Affairs Medical Center, Birmingham, AL 35294, U.S.A.
**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 leukocytes. 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 test is available to assess for this. Bioenergetics, liver disease, cancer and environmental toxins can be dynamically assessed using a new parameter: the 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 leukocytes 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 provide evidence of importance of dysfunctional metabolism in disease progression.
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