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Does Stress Make You Fat?

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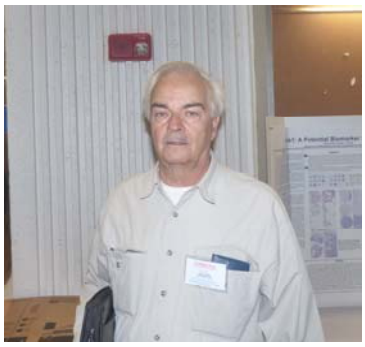






Instructions for Authors

IN VIVO is published three times yearly during the Fall, Winter, and Spring. Original research articles in the field of biology in addition to original articles of general interest to faculty and students may be submitted to the editor to be considered for publication. Manuscripts can be in the form of a) full length manuscripts, b) mini-reviews or c) short communications of particularly significant and timely information. Manuscripts will be evaluated by two reviewers.

Articles can be submitted electronically to *invivo@mec.cuny.edu* or mailed as a printed copy (preferably with a diskette that contains the file) to the Editorial Board at Medgar Evers College. All submissions should be formatted double spaced with 1 inch margins. The title of the article, the full names of each author, their academic affiliations and addresses, and the name of the person to whom correspondence should be sent must be given. As a rule, full length articles should include a brief abstract and be divided into the following sections: introduction, materials and methods, results, discussion, acknowledgments and references. Reviews and short communications can be arranged differently. References should be identified in the text by using numerical superscripts in consecutive order. In the reference section, references should be arranged in the order that they appeared in the text using the following format: last name, initials., year of publication. title of article, journal volume number: page numbers. (eg. - [1]Hassan, M. and V. Herbert, 2000. Colon Cancer. *In Vivo* **32:** 3 - 8). For books the order should be last name, initial, year of publication, title of book in italics, publisher and city, and page number referred to. (eg. - Prosser, C.L., 1973. *Comparative Animal Physiology*, Saunders Co., Philadelphia, p 59.). Abbreviations and technical jargon should be avoided. Tables and figures should be submitted on separate pages with the desired locations in the text indicated in the margins.

In This Issue:

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has anti-inflammatory activity (Nguyen A, personal communication) and we have data that suggest it is involved in cell signaling. MTE4a also produces a compound with antibiotic activity that has been partially purified. We describe here the various assays performed using MTE4a grown both in solid and liquid media to elucidate its potential to produce active secondary metabolites.

Does Stress Make You Fat? T. Rhodes, M. Pazienza, C. Nunez and J.F. Evans, Molloy College, Rockville Centre, New York, USA.

ACTH is a major hormone of the stress axis or hypothalamic-pituitary-adrenal (HPA) axis. It is derived from pro-opiomelanocortin (POMC) the precursor to the melanocortin family of peptides. POMC produces the biologically active melanocortin peptides via a series of enzymatic steps in a tissue-specific manner, yielding the melanocyte-stimulating hormones (MSHs), corticotrophin (ACTH) and β-endorphin. The melanocortin system plays an imperative role in energy expenditure, insulin release and insulin sensitivity. Bone marrow derived mesenchymal stem cells circulate in the blood stream and as progenitor cells have the potential to differentiate into many cell types such as osteoblasts, chondrocytes and adipocytes. Here we examine the effects of ACTH on the mouse D1 bone-marrow derived MSC. ACTH significantly increased lipid accumulation during the adipogenic differentiation of D1 cells in a concentration- dependent manner. ACTH also shifts the temporal pattern of D1 adipogenic differentiation to the left i.e. differentiation occurs earlier with ACTH treatment. No significant differences in protein expression of peroxisome proliferator-activated receptor gamma (PPAR-γ2), a regulating transcription factor of adipogenesis were found. Therefore the effects of ACTH are suggested to be mediated by an alternative pathway. Overall the results indicate a connection between increased adipose deposition and the elevated circulating ACTH associated with stress.

Preliminary Characterization of the *XRR1* Gene in the Yeast *S. cerevisiae.* Vanessa Rivera[1] and Marci J. Swede, Long Island University, Post, Brookville, NY.

We have characterized a novel gene, *XRR1* (eXhibits Rapamycin Resistance), whose null mutant exhibits temperature sensitive resistance to the anti-fungal drug rapamycin. The *XRR1* gene product was shown via a systematic yeast two-hybrid analysis (Uetz *et al*, 2000) to physically interact with FKBP12 (FPR1p). The FKBP12 protein is responsible for binding rapamycin and causing cell-cycle arrest via the *TOR* signaling pathway. The *XRR1* gene exhibits sequence homology to the A1pp (Macro) domain specific for binding ADP-ribose. This domain is found in the C -terminus of the mammalian macroH2A histone variant. Initial characterization of *XRR1* null mutants indicates that they have no overall growth defect across a temperature range of 25°C to 37° C. Further characterization of the *XRR1* gene suggests a role in the rapamycin resistance pathway. We have observed that the *XRR1* null mutation results in an increase in growth in the presence of 100 ng/ml rapamycin at 37°C compared to growth at 30°C when exposed to the drug. The degree of resistant growth observed in the null mutant is greater than that observed by wildtype isogenic yeast under the same growth conditions. This drug resistance is not observed when growth at 30°C. We propose a model in which the *XRR1* gene product is involved in stabilizing the FKBP12-rapamycin complex, which is responsible for cell cycle arrest in response to rapamycin treatment. The Swede lab gratefully acknowledges Dr. Hinnebusch at the NICHD for the gift of mutant strains. This research is supported by a grant from the Dextra Baldwin McGonagle Foundation in support of undergraduate research to MJ Swede and by a faculty research development grant from LIU, Post to MJ Swede.

The Effects of Resveratrol Compounds on the Motility and Proliferation of F10 Melanoma Cells. Christian Rivoira[1], Valery Morris[2] and Susan Rotenberg[2], [1]Queensborough Community College, Bayside, NY and [2]Queens College, Flushing, NY.

Resveratrol is a phytochemical found in grapes and wine and has been reported to possess anti-carcinogenic effects. The effects of several cis and trans isomers of resveratrol were tested on highly metastatic mouse B16 F10 cells to see the effect on cell motility. Cells were seeded onto 96-well plates, treated with each compound and then run through a proliferation assay. F10 cells were also plated on 6-well plates and allowed to grow until 100% confluent for a wound healing assay. These samples were then scratched and treated with the compounds.