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High Glucose Enhances the Proliferation Effects of Stress Hormones in Mesenchymal Stem Cell Cultures

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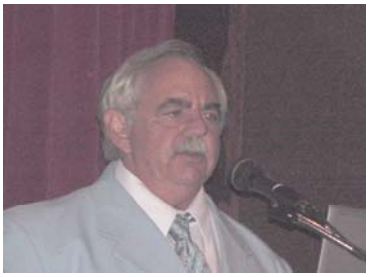



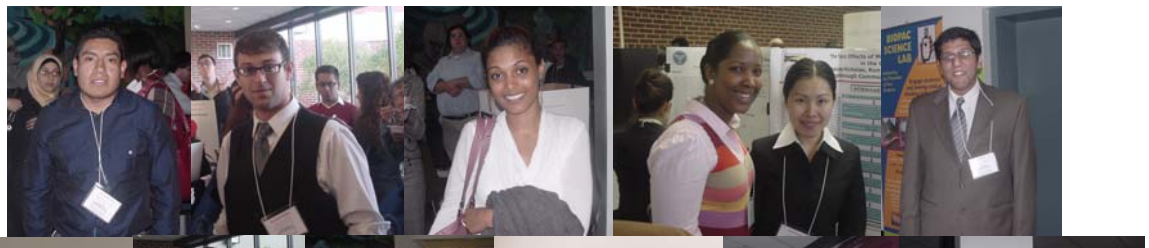


Instructions for Authors

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:

Election Results
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High Glucose Enhances the Proliferation Effects of Stress Hormones in Mesenchymal Stem Cell Cultures. Nancy Abrego[1,2], Jodi F. Evans[1,2] and Louis Ragolia[2], [1]Molloy College, Rockville Centre, NY and [2]Winthrop University Hospital, Mineola, NY.

Mesenchymal stem cells (MSC) are the progenitor cells to connective tissue cells, epithelial cells, and smooth muscle cells. These cells are currently being investigated as a cellular source for tissue regeneration and repair. The systemic and local tissue environment may have significant influence on the success of such efforts. We hypothesized that elevated glucose and exposure to stress hormones would influence the proliferation of MSC. MSC from the bone marrow of Wistar-Kyoto (WKY) rats were grown under both low glucose (5 mM) and high glucose (20 nM) conditions in the presence and absence of the synthetic glucocorticoid, dexamethasone, and adrenocorticotropin hormone (ACTH). Relative cell density was used to determine the rate of proliferation and was measured using methylene blue staining. Changes in cell density from initial plating were recorded at various stages of culture. Under low-glucose conditions, stress hormones reduced MSC proliferation and these changes were enhanced when cells were exposed to high glucose conditions. These data indicate that stress and elevated glucose can have a significant effect on the ability of MSC to proliferate and may prove to reduce their efficacy during tissue repair.

Are the Neurotoxic Effects of Manganese Due to Blockage of Post Synaptic Dopamine Receptors. Trevon Adams[1], Danilo Beaubrun[2], Michael Nelson[1], Margaret A. Carroll[1] and Edward J. Catapane[1], [1]Medgar Evers College and [2]Kingsborough Community College.

Manganese, a neurotoxin causing Manganism, a Parkinsons-like disease, disrupts dopaminergic neurotransmission. Lack of effective treatment for Manganism is major obstacle in its management. Recently, p-aminosalicylic acid (PAS) was reported an effective treatment. Lateral cilia of gill of *Crassostrea virginica* are controlled by serotonergic-dopaminergic innervations from their ganglia. We showed manganese blocks cilio-inhibitory effects of dopamine and this is prevented by PAS. We sought to determine if manganese exerts its effects by blocking dopamine post-synaptic receptor binding and if PAS prevents manganese from doing this. We observed membrane potentials of lateral ciliated cells with fluorescent dye while measuring cilia beating. Applying dopamine or 20 Hz electrical stimulation after exciting cilia repolarized the cell membrane and decreased beating. Manganese prevented this. PAS prevented the actions of manganese. Adding ATP to gill increased cilia beating without changing membrane potential. Applying MDL-12,330A, an adenylcyclase inhibitor, after manganese decreased cilia beating without affecting membrane potentials. The study shows the correlation between membrane potential of lateral ciliated cells and cilia beating rates. It helps elucidate the neurotoxic mechanism of action of manganese, showing the site of action is after the post-synaptic dopamine receptors. This information is helpful to understand causes and potential therapeutic treatments of Manganism.

The Synergistic Effect of Green Tea Polyphenols with Antiseptics and Antibiotics against the Growth of Potentially Pathogenic Bacteria. Sylvia Chinons Akuwudike, Bobby Haghjoo and Lee H. Lee, Montclair State University, Montclair, New Jersey, USA

Green tea leaves contain many polyphenolic compounds such as (-)-epicatechin, (-)-epicatechin-3-gallate, (-)-epigallocatechin, and (-)-epigallocatechin-3-gallate(EGCG). Green tea polyphenols (GTPs) have been implicated to have distinct properties that combat the harmful effects of cell proliferation. These compounds contain certain anti-viral and anti-microbial mechanisms that inhibit growth and perhaps reverse the process in which replication occurs. In this study, 2% GTP was used alone or with antiseptics and antibiotics to study its effect on different gram + and gram - bacteria. For the antiseptic study, disk diffusion test was carried out and for the antibiotics; Kirby-Bauer method was used. The zones of inhibition were measured in millimeter and bacterial resistant, intermediate, or susceptible was determined. The results suggested that GTP works best on the gram positive bacteria and had very little effect on the gram negative bacteria. The most powerful GTP effect can have zone of inhibitions reaching to more than 8mm.

Effect of Gap Junction Inhibitors on Breast Cancer Cell Migration. Vanessa Almonte[1] Maria L. Cotrina[2] and Regina Sullivan[1], [1]Queensborough Community College, Bayside, NY and [2]Columbia University, NY.

Connexins, a family of transmembrane proteins, form intercellular gap junctions in vertebrate cells. Gap junctions allow for cell-cell communication and the passage of small molecules between cells. Defective gap junctions have been identified in cancer cells however their role in cancer progression and the maintenance of a metastatic phenotype remains elusive. This study focused on the role of gap junction hemichannels in the migration of MDA 231, a highly metastatic breast cancer cell line. Experiments were done to assess the levels of functional gap functions in MD231 cells and compared to mouse astrocytes, a cell line that shows abundant gap junctions and to a human embryonic kidney cells which show low levels of gap junctions. The cells were grown to a confluent monolayer and treated with carboxy-dichloroflurescein. The monolayers were injured with a razor and the cells were imaged using an inverse phase fluorescent microscope fitted with a green filter. The wound healing assay was used to determine the effect of two gap junction inhibitors, carbenoxolone and meclofenamic acid, on the migration of 231 cells. The results of the dye transfer assay revealed that 231 cells have a low level of gap junctions. Interestingly, however ,carbenoxolone significantly inhibited cell migration while meclofenamic acid caused cellular morphological changes. Further studies will investigate the specificity of these effects.