Winter 2015

Vascular Tissue-resident Mesenchymal Stem Cells; Friend or Foe?

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MACUB

IN VIVO

The Publication of the Metropolitan Association of College and University Biologists

Winter 2015 Volume 36, Issue 2

47th Annual MACUB Conference
Molloy College
Rockville Centre, New York
November 1, 2014







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.

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The 2015 MACUB Conference will be at

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Nov. 7, 2015

were titrated with various concentration of Zinc. Our results indicate that at lower ratio of citrate to glycine, both molecules are interacting with the metal. According to the FTIR spectra, there are two complexes formed in solution. Complex 1 is citrate interacting with Zn (II) and complex 2 is glycine and Zn (II). However, the FTIR data do not show that there is formation of a new glycine-Zn^{2+}-citrate complex at the ratio of 1:1 and 1:3. At the ratio of 1:5 and 1:10 citrate to glycine, the FTIR spectra depicts a new interaction between citrate, glycine and Zn (II). Understanding the nature of this interaction could be the key to possible implementation of such an amino acid-zinc delivery system. Washington Rammirez is a participant in the NIH Bridges to the Baccalaureate Program at Queensborough Community College.

Nuclear Tip60 Overexpression Exacerbates Chemotherapeutic Drug Ttreatment in Breast, Lung and Pancreatic Cancer Cell Lines. Priyadarshini Ravichandran and Daniel S. Ginsburg, LIU Post, Brookville, NY.

The Tip60 lysine acetyltransferase acetylates histones, the p53 tumor suppressor, the ATM kinase DNA repair enzyme, and the androgen receptor transcription factor. Tip60 plays a vital role in transcription, DNA repair, and apoptosis. There is conflicting evidence that Tip60 may function as both a tumor suppressor and an oncogene. We are interested in analyzing Tip60's role in breast, pancreatic, and lung carcinomas. We would also like to gauge Tip60's potential as a therapeutic agent in these cancers. We hypothesize that due to Tip60's role in DNA repair and apoptosis, it serves as a tumor suppressor in breast, pancreatic and lung cancers. Therefore, overexpression of Tip60 should decrease proliferation in tumor cells and enhance the effects of chemotherapeutic agents. We have shown that Tip60 levels in six different breast, pancreatic and lung cancer cell lines were significantly lower as compared to non-tumorigenic cells. While Tip60 overexpression itself was found to reduce cancer cell proliferation in only one cell line, Tip60 overexpression when accompanied with paclitaxel treatment, decreased proliferation 30-60% more than administration of paclitaxel alone amongst the cancer cell lines. In addition, we discovered that the subcellular localization of Tip60 varied amongst the cell lines, with cytoplasmic localization in cancer cells and nuclear localization in non-cancer cells. Overexpression of Tip60 containing an N-terminal nuclear localization signal (NLS) decreased cancer cell proliferation more than Tip60 lacking the extra NLS. These results suggest that Tip60 serves as a tumor suppressor in breast, lung, and pancreatic cancers, consistent with previous data, and that the proper localization of Tip60 may be more important for its tumor suppressor activity than overall Tip60 levels. Our work provides evidence that Tip60 may be useful as part of a cancer therapy in combination with currently used drugs. This work was supported by the LIU Post Research Committee.

Vascular Tissue-resident Mesenchymal Stem Cells; Friend or Foe? Heather Renna, Lauren McHugh, Eddie Bochynski, Victoria Flemm and Jodi F. Evans, Molloy College, Rockville Centre, NY

Mesenchymal stem cells (MSC) are ideal candidates for stem cell-based therapies of vascular inflammation. They are progenitor cells that can replace damaged cells and they can modulate immune response cells. MSC from bone marrow and adipose tissue regulate inflammation by promoting the switch of macrophage cell from an inflammatory to an anti-inflammatory phenotype. Much less is known about the tissue resident MSC's and their interaction with macrophage cells. We hypothesized that aorta-derived mesenchymal stem cells (mAo MSC) would also promote the expression of the anti-inflammatory phenotype among macrophage cells. The interaction of mAo MSC with the macrophage was examined by co-culturing the cells and exposing them to the inflammatory mediator, lipopolysaccharide (LPS). A bone marrow derived MSC cell line was used as a control. Nitric oxide (NO) and the tumor necrosis factor-α (TNFα) and interleukin-12 (IL-12) cytokines were measured using Griess reaction and ELISA assay respectively. The impact of the interaction on phagocytosis was measured using zymosan-A. We found that bone marrow derived MSC, when in co-culture with macrophage cells, performed as expected and suppressed NO, TNFα, and IL-12 production. Unexpectedly, the mAo MSC enhanced NO and TNFα production by the macrophage cells, which is indicative of the inflammatory phenotype; IL-12 production by macrophage remained unchanged. Macrophage cell phagocytic activity was significantly increased by contact with mAo MSC, which represents enhancement of the anti-inflammatory phenotype.