Online Proceedings

Poster Session 1

Balcony, Easel 98

11:00 AM to 1:00 PM

Mitochondrially-Targeted Enzyme Catalase Prevents DNA Damage in Lung Fibroblast Cells in Old Mice

Kateryna Tonyuk, Senior, Biochemistry
Mentor: Warren Ladiges, Comparative Medicine
Mentor: Soroosh Fatemie, Comparative Medicine

With increasing age and as a result of mitochondrial-generated reactive oxygen species such as hydrogen peroxide, lung fibroblasts cells undergo DNA damage which is leading to a loss of function and premature ageing (senescence). The objective of this project was to assess whether the presence of the mitochondrial-specific antioxidant enzyme catalase (mCAT) could affect the levels of DNA damage and senescence in fibroblasts collected from the lungs of 24 month old mCAT mice versus control. Immunostaining of lung fibroblast cultures, using specific antibodies against markers of DNA damage/premature ageing showed, a significant decrease in DNA damage and a decrease in cell division in mCAT lung fibroblasts against control. Aging is the most significant risk factor for developing cancer, and majority of malignant cancers that are treated in clinics, occur in older patients that are suffering from cellular senescence/aging and uncontrollable cell division, leading to cancer. Therefore, understanding the consequences of senescence may help with designing new strategies, such as anti-oxidant therapies that lead to prevention of DNA damage and age-related diseases, in the future.

Poster Session 1

Balcony, Easel 96

11:00 AM to 1:00 PM

Effect of GHK Peptide on Lung Fibroblasts *in vitro*

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Mentor: Warren Ladiges, Comparative Medicine

Lung diseases, including chronic obstructive pulmonary disease and idiopathic pulmonary fibrosis, are major health concerns in older people. There is currently a paucity of drugs available that are effective in treating these conditions. A potential drug target in the lungs is a cell type known as fibroblasts. These cells are very abundant and are essential in providing support and structure for alveolar cells, which are responsible for the vital gaseous exchange of oxygen and carbon dioxide. A drug that has been shown to target fibroblasts in the skin, and currently used in skin creams, is a tripeptide designated as GHK. The objective of this project was to see if GHK could target lung fibroblasts collected from old mice by restoring functional capacity. Lung fibroblasts were grown in culture to a monolayer and a scratch wound assay was performed to measure the rate of closure. Immediately proceeding the scratch, cells were treated with different concentrations of GHK while one received a placebo treatment. The closure rate was then measured every 24 hours for a total of 72 hours. The highest concentration treatment of GHK had a significantly faster migration rate than that of the placebo treated monolayers, suggesting that GHK enhances fibroblast migration and restores functionality to more youthful levels. These results provide the rationale for preclinical studies in mice to help determine if GHK might be a potential drug to consider for treating chronic lung diseases in the elderly.

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Decreased Serum Protein Factors are Associated with Increased Functional Decline in Old Mice

Anna Campbell (Anna) Schorr, Senior, Biology (General), Integrated Sciences
Mentor: Warren Ladiges, Comparative Medicine

Aging leads to the loss of physiological function such as cognition, mobility, and cardiovascular performance. One possibility for this loss is an age-dependent decrease in the generation of proteins necessary to support these functions at a youthful level. Many of these protein factors are secreted into the general circulation and can be detected in the serum of blood samples, this provides the opportunity to monitor their levels at various ages. The objective of this project was to determine if serum proteins could be identified that decreased with increasing age using mice as a preclinical model of aging. Mice with a mixed genetic background, designated as CB6F1, were used because they represent a heterogeneous gene pool and a co-morbid disease pattern similar to humans. Serum was collected from 8-month and 32-month old mice (comparable to 22 and 80 human years, respectively), and

tested for 200 serum proteins using a slide capture array (RayBiotech). Levels for 23 proteins, involved in supporting blood vessels, the immune response, brain cells, skin, muscle and bones, were significantly decreased in old mice compared to young mice, correlating with an increase in frailty, fatigue, heart failure, and cognitive impairment. This preliminary data suggest specific serum proteins, alone or in combination, may be useful markers for aging. This provides the rationale to investigate the ability of anti-aging drugs to restore specific protein factors to their youthful levels and monitor for any delay or reversal of age-related functional decline.