Genetic Differences in Neointimal Hyperplasia in Response to Vascular Injury

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Introduction:

Cardio-Vascular disease is the number killer in the state of Ohio accounting for approximately 42% of all deaths. One treatment option for the cardiovascular disease is angioplasty which has a very good initial success rate of 90-95% chance of opening up the clogged artery. This is an intial success rate though, and within 3-6 months 30-50% of the arteries become restenosed. The purpose of my research was to broaden the field of knowledge about restenosis and hopefully someday be able to use this information to try to erase this 30-50% restenosis rate.

Hypothesis:

It is the hypothesis that the restenosis resistant mouse (C57 Bl/6) has higher levels of MIPP (Multiple Inositol Polyphosphate Phosphatase) in it's smooth muscle cells compared to the susceptible mouse (C57 L/J), and this could be one reason why the C57 Bl/6 mouse is resistant to restenosis.

Methods:

A quantitative RT-PCR using GAPDH as the housekeeper gene was performed on the two strains of mice to check for their expression of the MIPP gene. An In-Situ hybridization was then run to check where the MIPP gene was being expressed.

Results:

The quantitative RT-PCR showed a greater than 2 fold difference in expression of the MIPP gene with localization of expression in the smooth muscle cells of the carotid arteries.

Conclusions:

With the increase in expression of MIPP in the resistant strain of mouse (C57 Bl/6) and with the localization of the gene of interest in the smooth muscle cells, it can be concluded that this gene is having an anti-proliferative effect on these SMC's. It could thus mean that this might be one of the protective mechanisms for this mouse that prevents it from having restenosis take place.