Effects of STAT3 on Pathological Retinal Angiogenesis

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Purpose: Retinopathy of prematurity (ROP), caused by abnormal retinal vessel growth, is a leading cause of worldwide blindness in children today. A treatment of ROP is to prevent pathological angiogenesis by using an antibody to bind to VEGF. Vascular endothelial growth factor (VEGF) is a signal protein that binds to its receptor VEGFR2 to help with angiogenesis. However, limiting VEGF causes problems in growing infants. For a better treatment, the Hartnett Lab tested the reducing STAT3, which initiates angiogenesis when VEGF and VEGFR2 bind together. This treatment used a rat oxygen induced retinopathy (OIR) model to mimic human ROP that included subretinal injections of gene therapies such as inhibitors to prevent STAT3 function. This STAT3 reduction resulted in reduction of intravitreal neovascularization, an abnormal growth of vessels. Thus, my hypothesis was that STAT3 reduction will reduce retinal vessel tortuosity. Procedure: The artery and overall lengths of the retinal images of rat OIR models were scored and measured. The tortuosity ratio of each artery was computed by dividing the vessel length by the overall length. Then those values were averaged by each eye group. Next each eye was sorted into the gene therapy groups where each of the averages and standard deviation was calculated along with a one-tailed t-test. To have a significant difference, the p-value of the t-test needed to be less than 0.05. Results: The p-value calculated to be 0.197. The average of the luciferase gene therapy group was about 1.048 and STAT3 group was about 1.042. Conclusion: The p-value was greater than 0.05, showing that there was no significant difference between the gene therapy groups which concludes that reduction of STAT3 does not reduce retinal vessel tortuosity.