Efficacy of Anti-Annexin 2 Antibodies on Retinal Neoangiogenesis in a Model of Oxygen-Induced Retinopathy

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Retinopathy of prematurity (ROP) is a retinal angiogenic disorder that results from rapid microvascular proliferation after exposure to oxygen supplementation in preterm infants. It is currently the leading cause of childhood blindness worldwide. A mouse model of oxygen-induced retinopathy (OIR) is a standardized approach to investigating ROP by reproducing the two phases of the disorder: vaso-obliteration in a state of hyperoxia followed by neovascularization in a subsequent state of relative hypoxia. The proliferative phase of OIR requires transcriptional induction of the annexin A2 (A2) gene, which forms a heterotetramer with protein S100A10 (p11) to accelerate tissue plasminogen activator-dependent activation of plasmin, a fibrinolytic protease. This study targets A2 through the use of three anti-A2 antibody injections at varying dosages to block perivascular activation of plasmin and OIR-related angiogenesis. The number of retinal tuft formations, defined as any abnormal vascular bundles protruding from the outer bounds of the ganglion cell layer into the inner limiting membrane, was used as the metric for neovascularization. These data show that 5 micrograms per milliliter of the 2E6 antibody and 15 micrograms per milliliter of the 1A7 antibody resulted in a statistically significant reduction of the number of neovascular tufts formed in eyes treated under OIR. Such a reduction clearly implies that A2 is a new, viable therapeutic target for ROP and other proliferative retinopathies that has the potential to resolve complications associated with retinal angiogenic disorders. These promising results justify expanding the study of anti-A2 antibodies to larger mammalian species, and eventually humans.

Awards Won:
Third Award of $1,000