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Role of Endomucin in Hypoxia-Induced Retinopathy of Prematurity

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ABSTRACT

Retinopathy of prematurity (ROP) is a major cause of blindness among premature, low birth weight infants as a result of pathological angiogenesis. Angiogenesis, the growth of new blood vessels from preexisting vessels, occurs in the veins and capillaries of the body. The process is highly regulated during early development and maturation. However, under abnormal conditions such as a decrease in oxygen levels or hypoxia, angiogenesis can become dysregulated and pathogenic. Currently, the best treatment for ROP is laser therapy, which does not significantly improve vision. Alternatively, glycoproteins are believed to play an important role in angiogenesis. Endomucin (EMCN), a glycoprotein, has been shown to be expressed by the venous and capillary endothelium. EMCN is believed to be associated with angiogenesis and could be a potential target for treatment of ROP. Thus, we hypothesize that EMCN is regulated by hypoxia and plays an important role in pathological angiogenesis.

Human retinal endothelial cells (HRECs), representative of endothelial cells involved in retinal angiogenesis, were deprived of oxygen using a hypoxia chamber. We established the optimal oxygen dosage, determined the optimal cell density, and monitored EMCN expression at different time points after exposure to hypoxia. Changes in gene expression in response to hypoxia were compared to control cells. Our preliminary data indicates that EMCN is regulated by hypoxia. Currently, we are investigating whether EMCN has similar effects in regulating revascularization in vivo. Taken together, our study indicates a novel role for EMCN during hypoxia-induced angiogenesis which may serve as a therapeutic target.

Keywords: angiogenesis, endomucin, hypoxia

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