BACKGROUND: Caffeine is a cyclic Adenosine Monophosphate phosphodiesterase inhibitor. Through this activity it has been shown to inhibit cell proliferation and angiogenesis in vitro and in vivo. It is unknown whether administration of caffeine exacerbates the toxic effects of oxygen on blood vessel growth and contributes to retinopathy of prematurity.

OBJECTIVE: To determine if caffeine affects vasculature growth by affecting 1.) differentiation of endothelial (EC) and smooth muscle cells (SMC) from their progenitors and 2.) ability of these differentiated cells to form normal blood vessels

DESIGN/METHODS: Mouse embryonic bodies (EB), a model of angiogenesis, were treated with increasing doses of caffeine (0-2mM). EC differentiation was determined by expression of specific EC markers. Tube formation and vascular density were assessed by immunofluorescence staining and mRNA analysis.

RESULTS: Caffeine did not effect EC differentiation shown by expression of EC markers. EB treated with caffeine failed to form vascular tubes. Instead PECAM-1and SMC staining revealed the presence of endothelial and smooth muscle cell clusters. Abnormal vascular anatomy with poor vessel branching and lack of angiogenic sprouting was noted with caffeine exposure (fig.2) as compared to the control (fig.1).
CONCLUSIONS: Caffeine does not inhibit differentiation of mesenchymal cells into endothelial and smooth muscle cells in an embryonic stem cell model. However, cells differentiated under caffeine exposure engaged poorly in vascular network formation which may play a role in the development of retinopathy of prematurity.
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