Do myoblasts enhance collateral arteriogenesis by normalizing endothelial dysfunction?

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Introduction

•Peripheral Artery Disease (PAD) is one of the largest contributors to cardiovascular morbidity with over 200 million cases worldwide

•PAD involves chronic arterial blockages and endothelial dysfunction

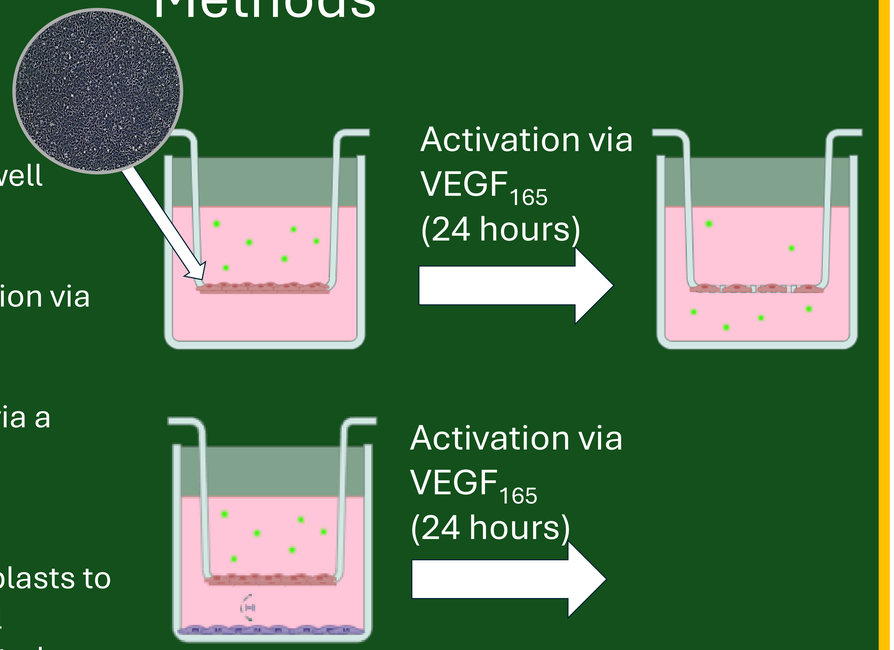
•Increased permeability correlates with endothelial cell (EC) dysfunction/activation

•Collateral arteriogenesis is a natural physiologic response to ischemic conditions driven by PAD, allowing blocked blood to bypass and provide oxygen

•Myoblasts may be a therapeutic candidate for PAD by inducing arteriogenesis

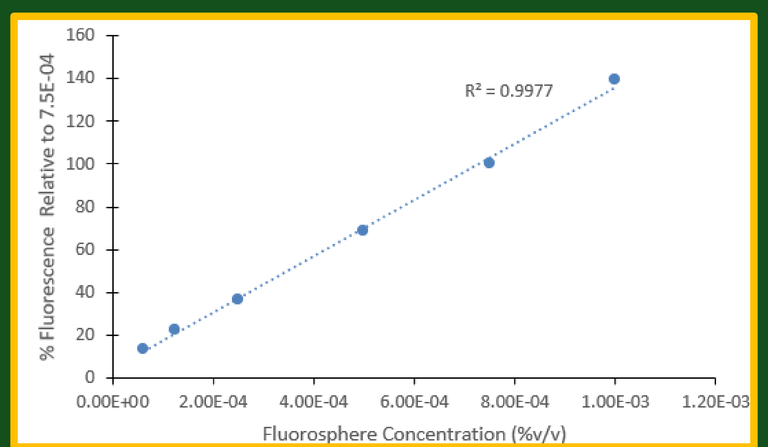
Methods

- Establish HUVEC monolayer on transwell inserts
- Stimulate EC activation via $VEGF_{165}$ (100 ng/ml)
- Quantify activation via a fluorescence-based permeability assay
- Coculture with myoblasts to assess if endothelial activation is modulated



Results

- A standard curve has been made to relate future fluorescence to a specific fluorosphere concentration
- A positive correlation was established with $R^2 = 0.9977$
- EC monolayer is steadily being established, all myoblasts that will be used have been collected and cryopreserved



Future Steps

- Completing coculture and analyzing for significant effects using one-way ANOVA & post hoc Tukey's test
- Exploring different modes of inducing activation such as hyperglycemic media

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