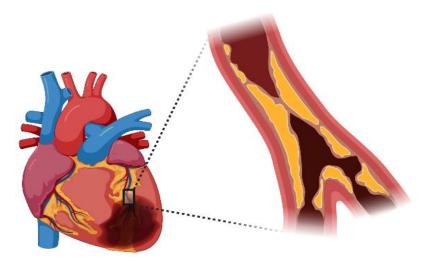


The Burden of Myocardial Infarction

Myocardial Infarction(MI), colloquially known as a 'heart attack' is estimated to cause 15% of all mortality and shortens the lifespans of survivors by up to 10%. Its destruction of cardiomyocytes and induction of scarring on the heart, even with the multitude of current treatments, is largely irreversible, and even among those who survive and are treated, approximately 20% of those will die within one year after the event.



Left: Myocardial Infarction caused by coronary artery blockage, leading to loss of blood flow and O2 deprivation to the heart and surrounding blood

vessels

Ephrin A1: Cardioprotective?

- Protein that has been linked cardio-protective processes
- In prior studies: Ephrin A1 Injection \rightarrow Decreased Infarct
- size • If proven to be safe, EphrinA1 could *reverse the* damage caused by myocardial infarction.

The Role of Sex Hormones in Myocardial Infarction

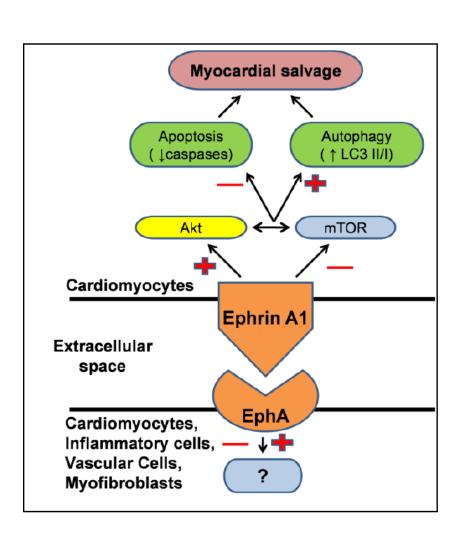
- Hormones may explain differing rates of MI: • Males > Females
 - Elderly > Young

Estradiol

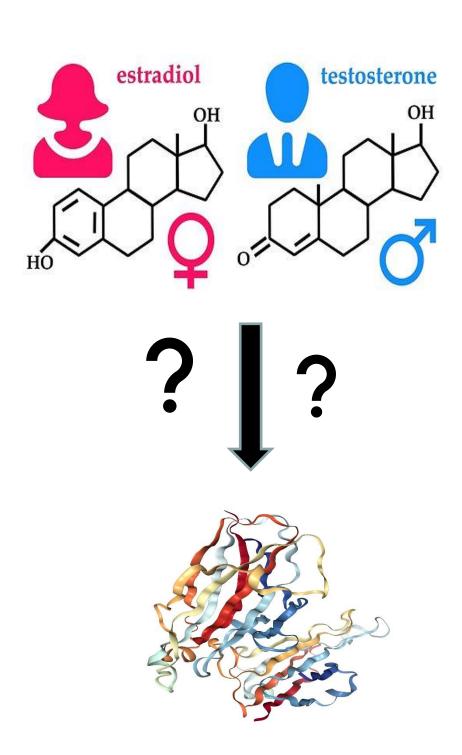
- Correlation to cardio-protective factors, such as vasodilation and anti-atherosclerotic effects.
- Decreased Estradiol \rightarrow \square MI Rates post-menopause?

Testosterone

- Studies have linked Testosterone to:
- Cardio-protective factors such as reducing infarct size and improving lipid profile.
- Cardio-harmful factors, such as increased vasoconstriction and inflammation



Top: Potential cardio-protective mechanism of EphrinA1

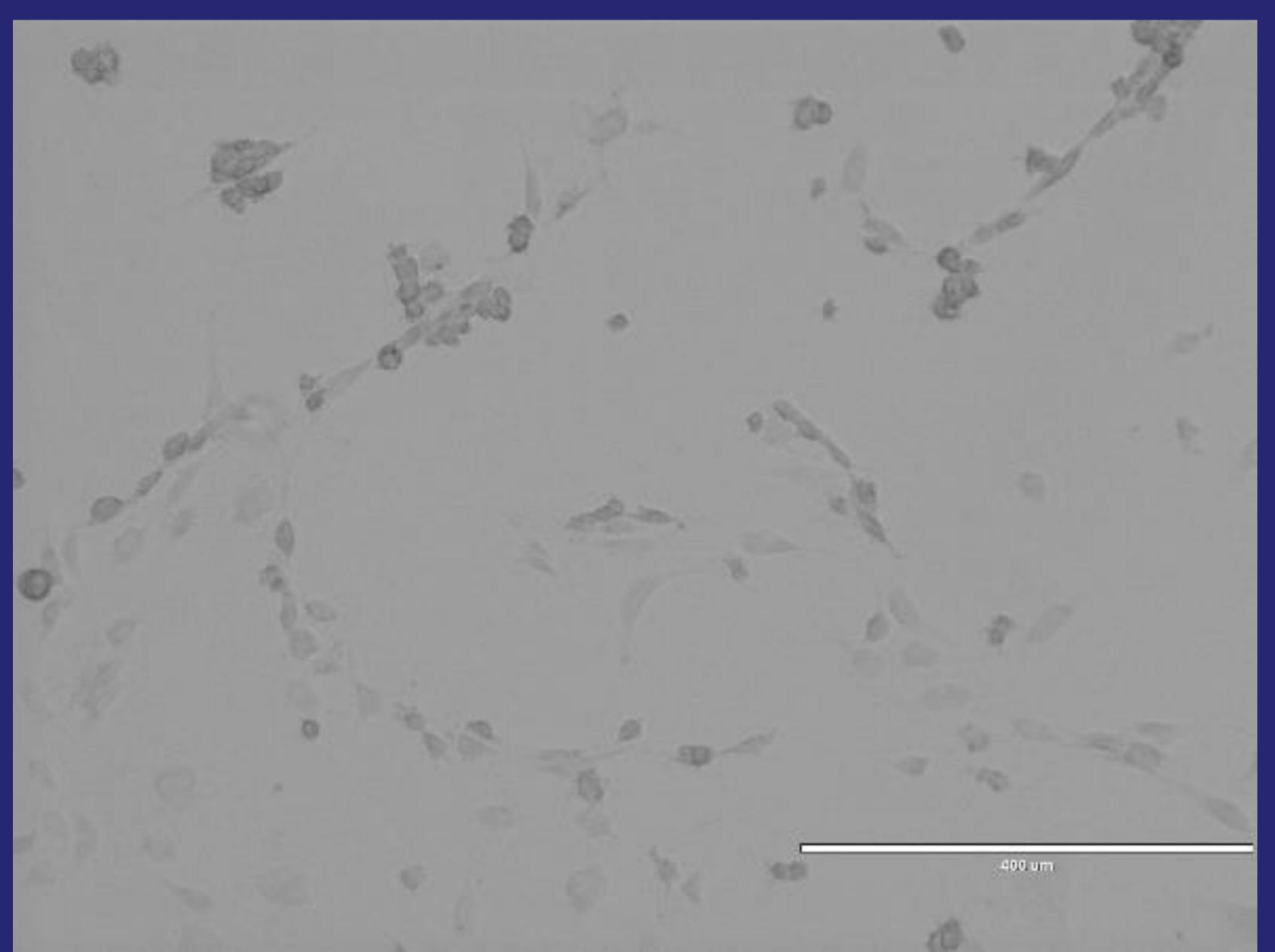


EphrinA1

Interplay between EphrinA1 and Estradiol/Testosterone?

The goal of this project was to observe whether application of Estradiol and Testosterone at various concentrations on Endothelial cells resulted in any change in EphrinA1 expression. Further understanding of the mechanisms affecting EphrinA1 are necessary in order to explore its use as a potential MI treatment.

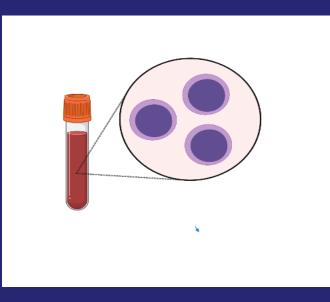
EphrinA1 levels in HUVEC's to be quantified and compared between different hormone levels



Above: HUVEC cells under microscope







Balloon Angioplasty Insertion of inflatable stent into atherosclerotic blood vessel May cause artery rupture, cardiac arrythmias, and induce M

Stem Cell Therapy Replacement of damaged cardiomyocytes May require long-term suppression of immune system





Take a picture of the QR code to view the abstract and references!

Investigating the effects of sex hormone exposure on Ephrin A1 expression in Human **Umbilical Vein Endothelial Cells(HUVEC) and implications for future treatment of Myocardial Infarction**

Ahmad Tejan-Sie



Nitroglycerin Potent Vasodilator Contraindicated in pregnant women and may induce rapid tolerance

Methods HUVEC's were obtained were maintained following protocol in 75cm² and grown in until they reached the 6th passage. At 6th passage, HUVEC cells were plated in a 24-well plate at a density of **50,000 cells per well**. After reaching 80% confluence, cells were exposed to Estradiol(E2), Testosterone(Test), or left unexposed to serve as controls. Concentrations of exposure hormones were derived based on physiological conditions within the human body.

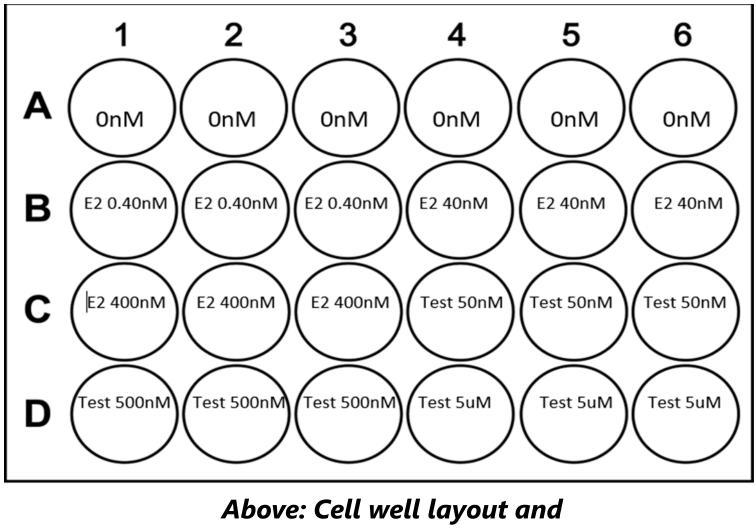
Future Research The EphrinA1 content of the HUVEC cells will be obtained via Western Blotting procedures. The levels of EphrinA1 between the different concentrations of Estradiol and Testosterone will inform as to whether either hormone may cause change in Ephrin levels. Due to its linkage with cardio-protection, we expect Estradiol levels to be directly correlated with an increase in EphrinA1. Testosterone's effects on heart function has been shown to be mixed, so we are unable to predict its effects on EphrinA1 expression.

Future research will utilize methods of this experiment on Human Induced Pluripotent-derived stem cell Cardiomyocytes(hipsc-CM) under hypoxic conditions, to mimic the conditions seen after Myocardial infarction. Estradiol in previous studies has been shown to promote cardiac regeneration and cardiac function, while Testosterone's effect on cardiomyocytes has been more mixed, with some studies suggesting it is cardioprotective while others indicate that it may promote hypertrophy of the myocardium, or inducing cardiomyocytes to be more reliant on glucose as fuel via the AMPK pathway instead of fatty acids. Their effect on EphrinA1 expression in Cardiomyocytes is unknown, and exploring the interaction between them will be beneficial in further understanding mechanism of the EphrinA1 protein.

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hormone concentrations