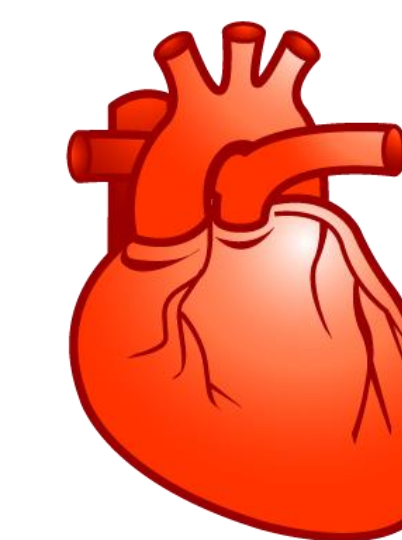
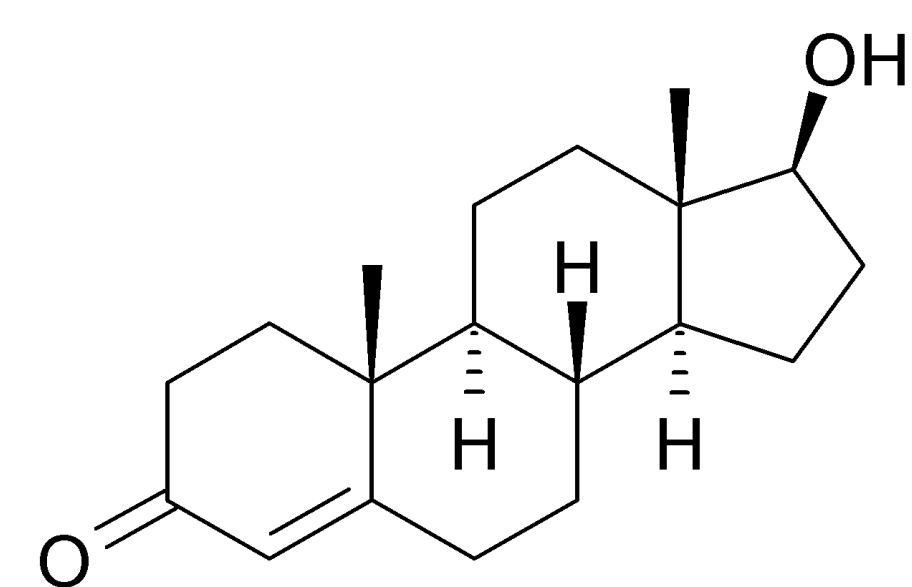




# The Effects of Exogenous Testosterone on Cardiovascular Stress

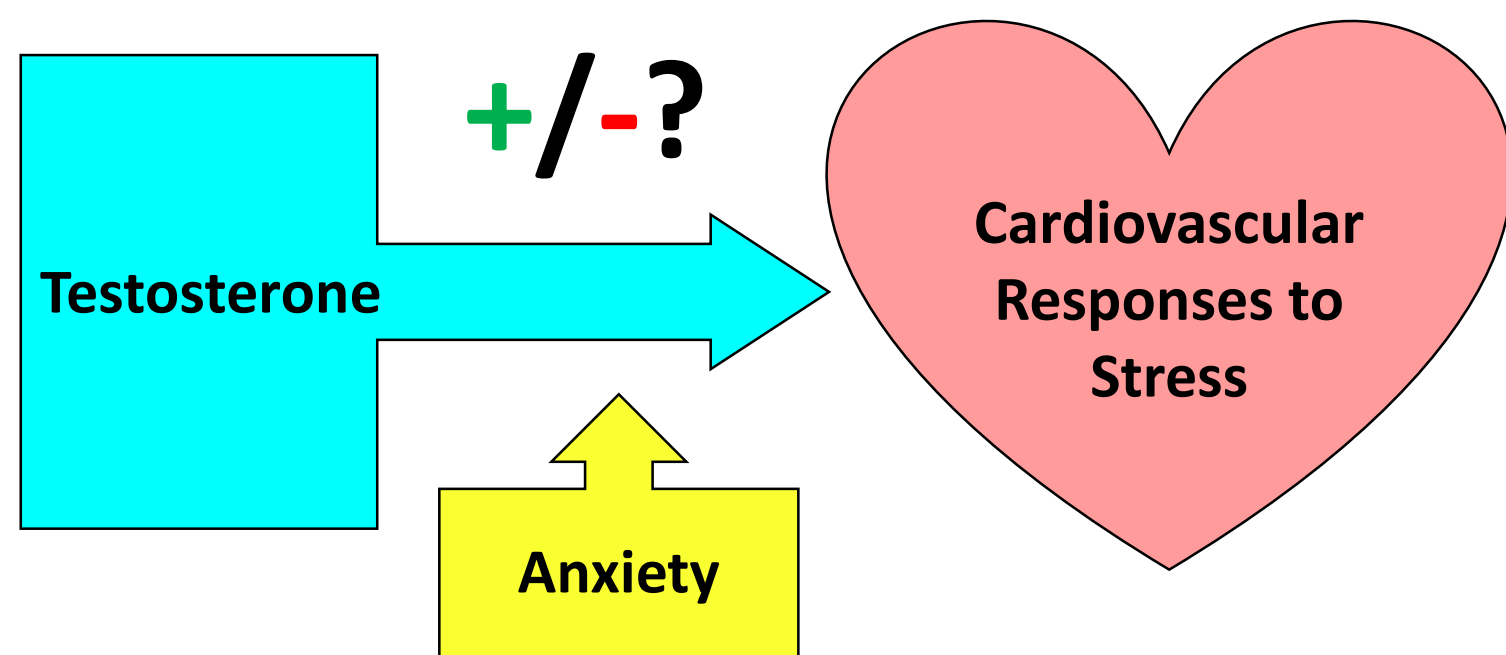
Kevin Soon-Sian Lai



A Thesis  
Presented to the Department of Psychology  
and the Robert D. Clark Honors College

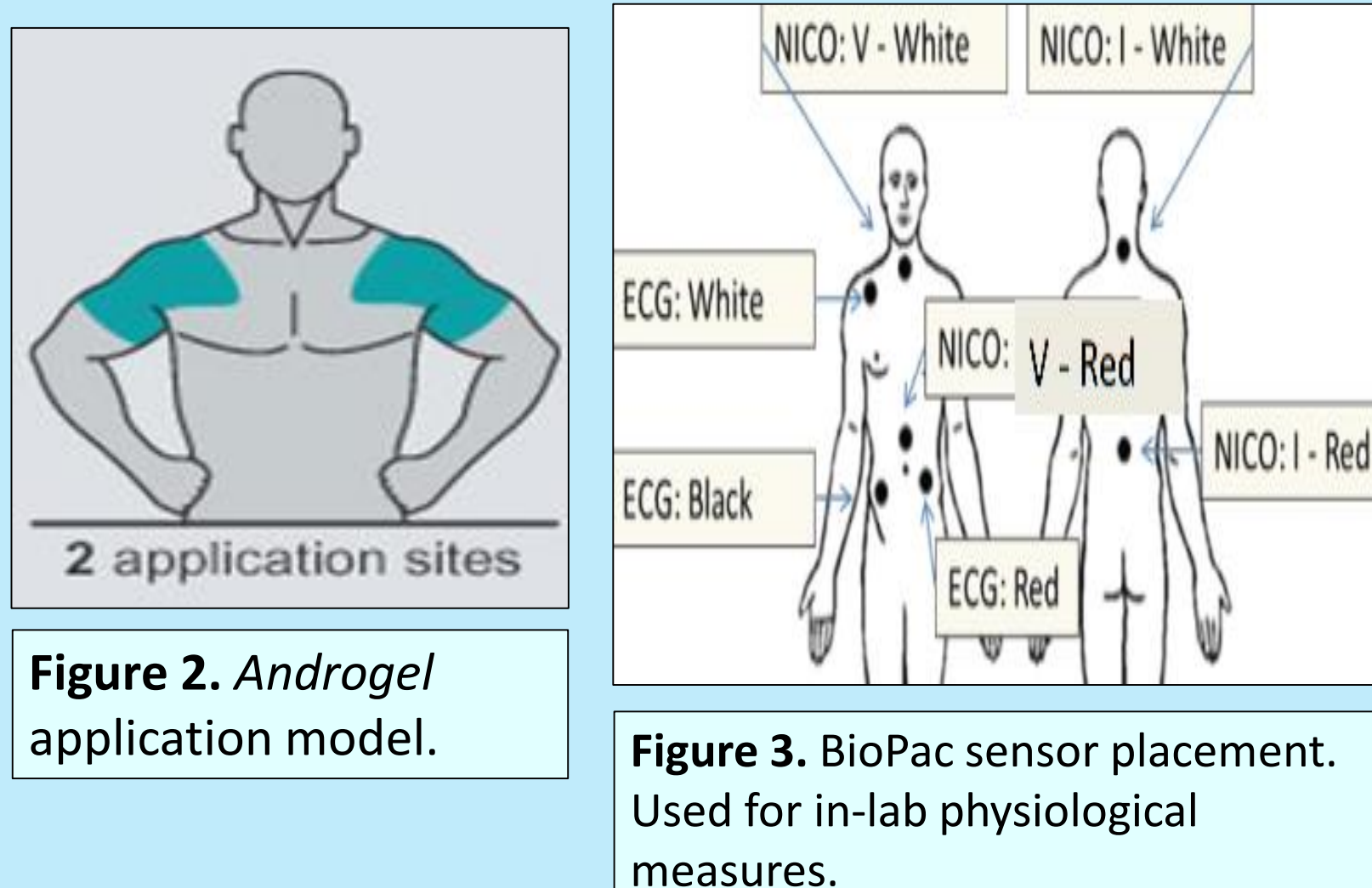
## INTRODUCTION

- Stress is a risk factor for cardiovascular disease, so it is important to understand the mechanisms that underlie cardiovascular responses to acute stress
- The steroid hormone testosterone is thought to play a key role in stress management
- Whether or not steroid hormone testosterone acts almost exclusively as enhancer<sup>1</sup> or buffer<sup>2</sup> of cardiovascular stress response is unknown



## Research Questions

- Does testosterone indeed have an effect on cardiovascular responses to stress, and is this effect enhancing or acting as a buffer?
- Does anxiety act as a psychological moderator on this proposed relationship?



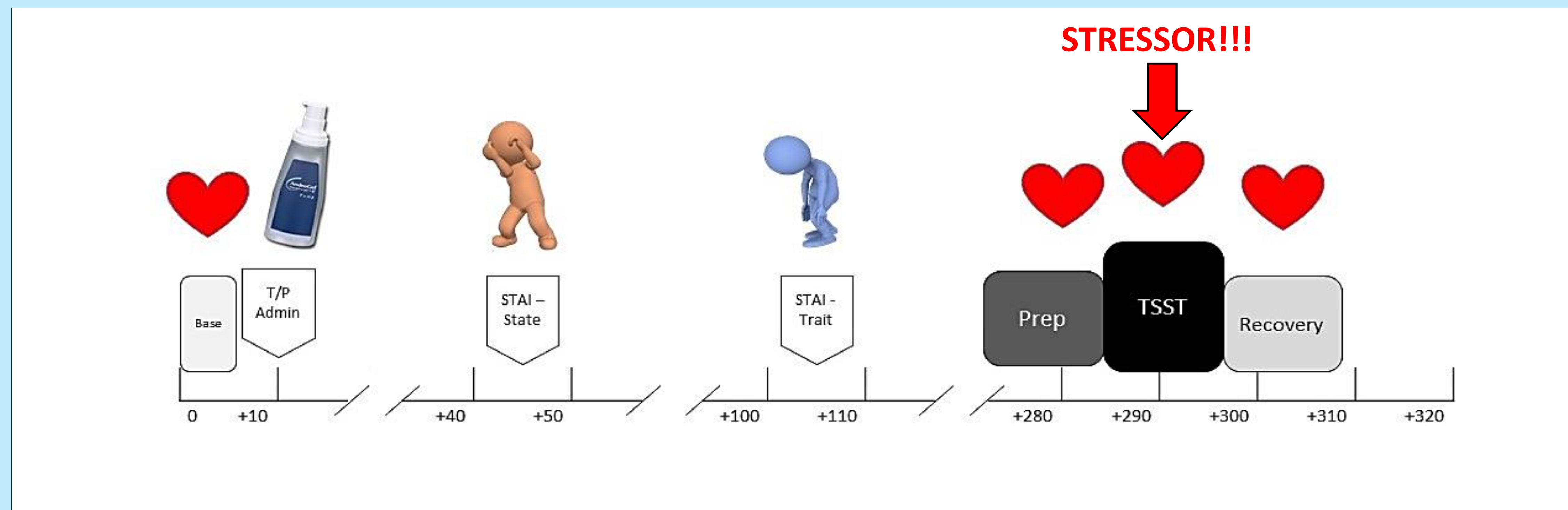
## METHODS

**Participants**  
N = 120 males (Mean age = 21.55 ± 3.49 years, Range = 18-39 years)

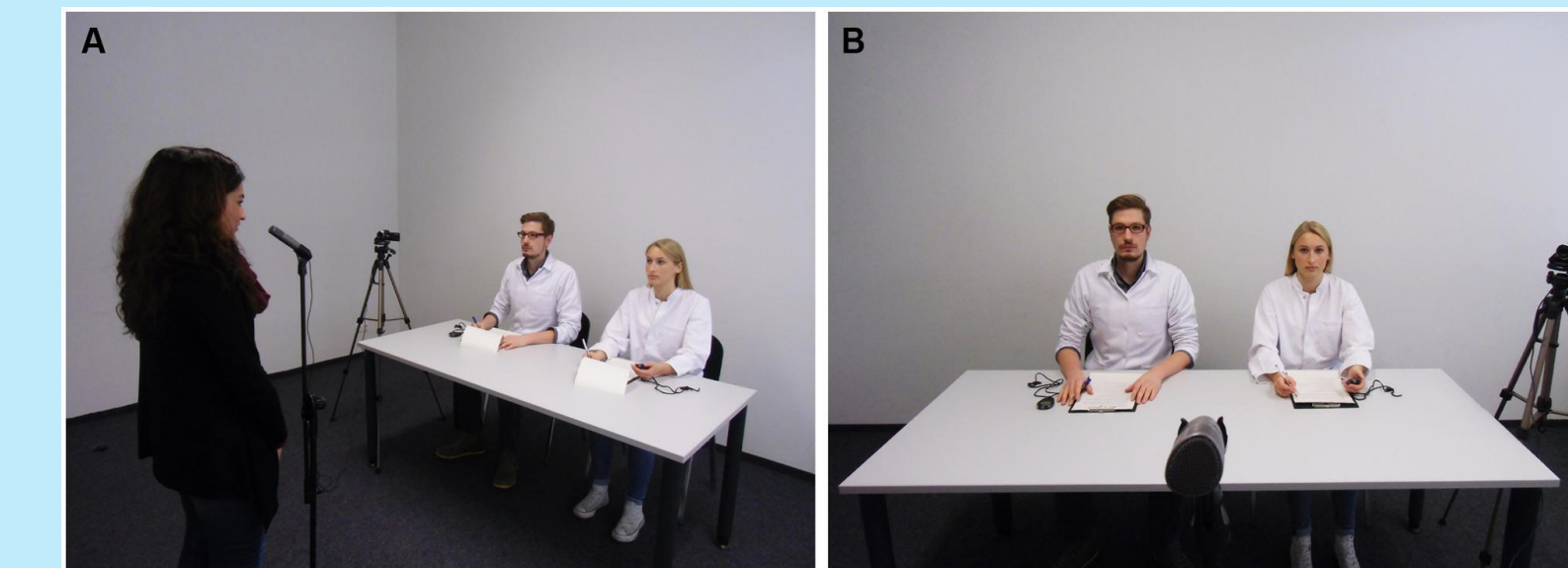
- Protocol**
- Testosterone.** Topical administration of *AndroGel* (containing testosterone/placebo), self-administered in morning
  - Stress.** Trier Social Stress Test<sup>3</sup> – Deliverance of speech & serial subtraction math task in front of 2 Judges (neutral affect) + video camera

- Measures**
- Cardiovascular Activity.** BioPac applied to participant to measure Heart Rate (HR) & High-frequency component of Heart Rate Variability (HRV) across 4 epochs: Baseline, TSST-Prep, TSST-Speech + Math, TSST-Recovery
  - Anxiety.** State-Trait Anxiety Inventory (STAI)<sup>4</sup> – Self-reported measures of anxiety

- Analyses**
- HR & HRV subjected to 4 (Epoch) × 2 (T/P) Repeated Measures General Linear Model (GLM)
  - State & Trait Anxiety Measures entered as continuous variable in 4 × 2 × 2 GLMs



**Figure 1.** Experimental procedure timeline. Duration denotes ECG recording time. Bottom units are in minutes, with + denoting minutes after recording of baseline HR and HRV. Hearts denote periods where cardiovascular activity was recorded.



**Figure 4.** Sample picture of a TSST panel<sup>9</sup> used for stress induction.

## RESULTS

- HR increased & HRV dropped during exposure to the stressor
- Testosterone did not influence cardiovascular stress responses
- When looking separately at individuals with high vs. low levels of state/trait anxiety, testosterone still do not impact cardiovascular stress responses

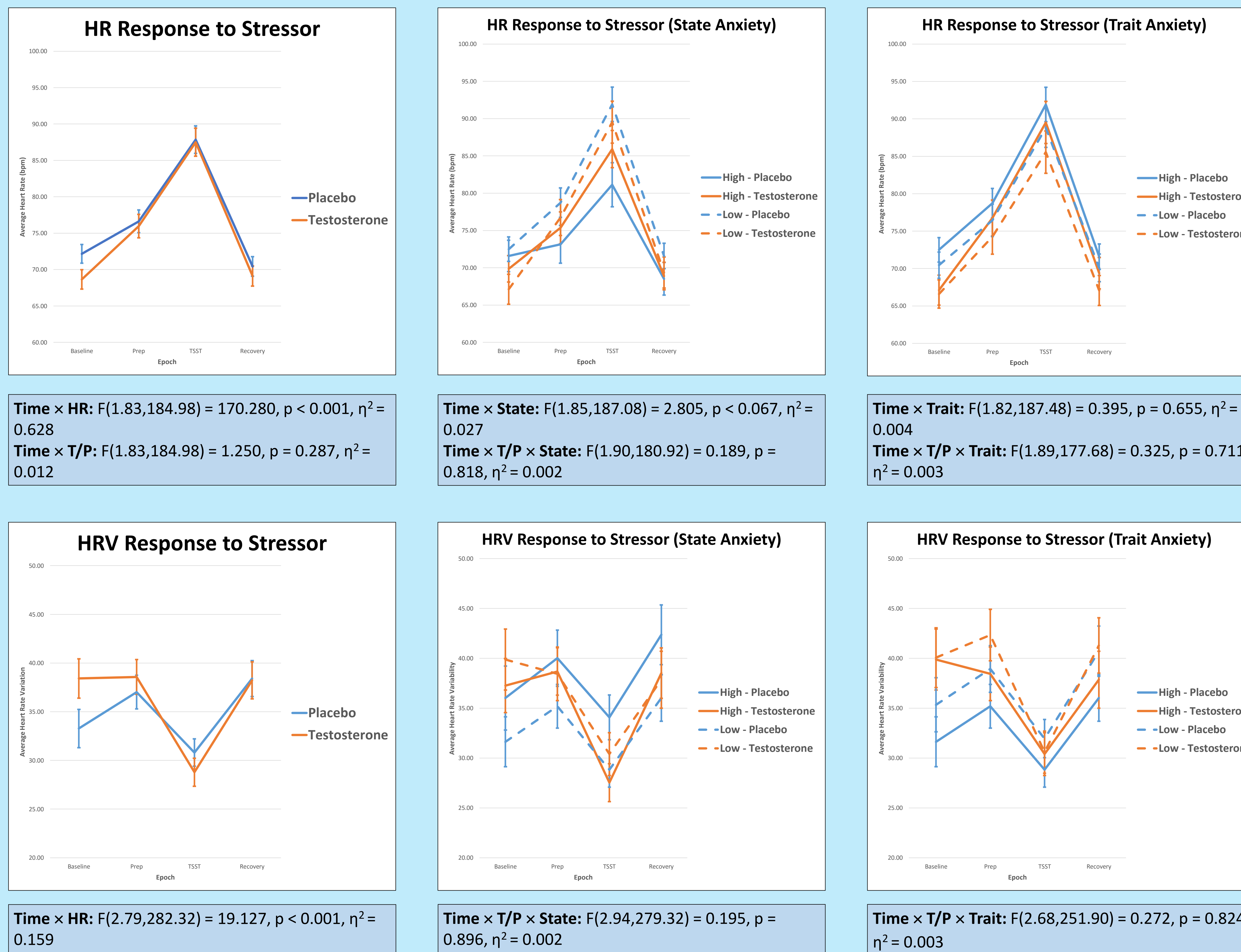
## CONCLUSIONS

- Testosterone does not play a significant role in moderating HR or HRV changes during exposure to a stressor
- Effects of testosterone on HRV were not moderated by individual levels of state or trait anxiety
- Although testosterone does indeed play a role in the human stress response by influencing other factors (e.g. cortisol), there is no direct relation to the cardiovascular component of the stress reactivity
- Despite null results, obtained outcomes will help encourage future important questions surrounding steroid hormones and their interplay with stress and cardiovascular health

## Future Directions

- Gender<sup>5</sup> & age<sup>6</sup> differences to T/P response
- Investigation of other possible moderators, such as negative/positive affect<sup>7</sup> & social status<sup>8</sup>

## EXPERIMENTAL DATA



**Figure 5.** Experimental results. Heart data recorded with BioPac MP150 and processed with Kubios HRV. Low state anxiety determined by STAI state score < 31. Low trait anxiety determined by STAI trait score < 38. Error bars represent standard error with 95% CI.



## ACKNOWLEDGEMENTS

**Primary Thesis Advisor:** Dr. Pranjal Mehta  
**Second Reader:** Erik L. Knight  
**CHC Representative:** Professor Mai-Lin Cheng  
**CHC Academic & Thesis Coordinator:** Miriam Alexis Jordan

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