ABSTRACT

WRITING AND POSTER DESIGN
**KEY INFO FOR THE SPRING SEMESTER**

- **Abstracts** are due March 1. All abstracts must have titles. These are submitted online. We will send you the link.

- **Posters** are due April 11th (but people are encouraged to print elsewhere). Posters are submitted online. The Aresty Center has poster templates available for download at [https://aresty.rutgers.edu/research-symposium/poster-design-and-printing](https://aresty.rutgers.edu/research-symposium/poster-design-and-printing).

- **Symposium** is April 27th in the Livingston Student Center. There are two sessions (AM and PM) and students will select which one they prefer when they submit their abstracts.

- **Group projects** should submit one abstract/poster that lists the name of everyone in the group.

- **Faculty mentors** must be given time to review abstracts and posters. Students who submit posters without faculty approval will not be permitted to present.
Your symposium audience is diverse

Your audience will include:

Experts in your field
- Your professors
- Colleagues
- Students in your major

Intelligent non-experts
- Professors outside your field
- Graduate students outside your field
- Judges

Novices
- Friends
- Family
- Prospective students

Your abstract, poster and presentation itself should be able to balance the demands of each of these groups.
THE PARTS OF A POSTER

1. **Abstract or Introduction**: What is the research question and why is it important?
2. **Background or Overview**: How does it relate to previous work and how is your approach different?
3. **Materials and Methods**: What is the method for answering the question?
4. **Results**: What did you find?
5. **Conclusions**: What are the implications for your field, businesses, or individuals?
6. **Citations**: Who influenced this work or made it possible?
WHAT MAKES AN EFFECTIVE POSTER?

Consider the following posters and discuss these questions:

1. Where does your eye go first when you view a poster?
2. Compare images on the various posters. What purpose do the charts and graphs play? What makes them more or less clear?
3. What makes the flow (the arrangement of the sections) easier or harder to follow?
4. What do you notice about the amount of text and use of white space on various posters?
5. At what point does it become hard to keep reading a poster?
Obesity Propensity Differentially Alters Locus Coeruleus Norepinephrine Neural Activity

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Backgrounds

Obesity is associated with a variety of metabolic and lifestyle disruptions, including reduced mood, lower quality of life scores, and an elevated risk for cardiovascular diseases. It is a widespread health issue in the United States. According to the Center for Disease Control and Prevention, more than one-third (35.5%) of American adults are categorized as obese.

Food intake is regulated by several projections to forebrain areas. One of which is the locus coeruleus norepinephrine (LC-NE) system. The LC-NE is an important modulator of affect, stress response, and sympathetic activation. Despite this, little is known of obesity’s influences on the LC-NE system.

Single-unit electrophysiology is a reliable technique to directly characterize neuron firing patterns. When utilized in vivo, electrophysiology could be used to investigate sensory, motor, and regulatory neurons in their intact circuitry. Likewise, locus coeruleus neurons demonstrate spontaneous and biphasic responses to painful sensations that can be observed through electrophysiology.

Motivations & Approach

- Given the high number of afflicted Americans, obesity and its propensity are important research topics.
- Novel understandings of obesity’s influences on the LC-NE system could provide insights into future treatments.
- Locus coeruleus neurons exhibit reliable biphasic responses that are also sensitive to specific physiological manipulations.
- Previous electrophysiology experiments by the Bello Lab have demonstrated that dietary-induced binge-eating dampens locus coeruleus activation.

The aim of this experiment is to characterize the effects of obesity propensity on the LC-NE circuitry.

Materials & Methods

- The present study utilizes obesity animal models in Sprague-Dawley rats selectively bred to be obese-prone (OP) or -resistant (OR).
- These two strains are further split into groups fed with either high-fat (45%), low-fat (10%), or chow ad lib for 10 weeks.
- See table below for grouping summary.
- Non-invasive cardiovascular data was taken at the 10th week.
- The locus coeruleus neurons of these animals were subsequently recorded through single-unit in vivo electrophysiology under isoflurane anesthesia.
- During each electrophysiology recording, 3 minutes of spontaneous activity was recorded followed by 50 trials of contralateral sciatic nerve stimulations applied at 0.2 Hz.
- 2-10 cells were recorded per rat and the data was compiled into averaged peri-stimulus histograms for analysis.

<table>
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<tr>
<th>DIET</th>
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<th>Obese-Resistant</th>
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<td>High-Fat</td>
<td>OP-HF n=6</td>
<td>OR-HF n=7</td>
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<tr>
<td>Low-Fat</td>
<td>OP-CD n=7</td>
<td>OR-CD n=6</td>
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Results

Figure A: body weight progression of each group across the ten-week time span on their designated diets. By week 10, the OP animals demonstrated a roughly 25% higher body weight than the OR animals. HF diet also increased body weight as expected, to a lesser extent.

Figure B: the final recording body weight of each group. Bar indicates average body weight ± SEM. Each group exhibited significantly different body weight ranges.

Figure C: cardiovascular data taken on week 10. Bar indicates mean ± SEM. Systolic and diastolic values demonstrated no significant differences between groups. The heart rates of the OP group (273 ± 12.2 bpm) was significantly slower than that of the OR group (281.3 ± 12.9 bpm). At the same time, the high-fat group (275.8 ± 11.1 bpm) also had significantly slower heart rate than the low-fat group (312.6 ± 15.4 bpm) (**, p < 0.005). However, no strain-diet interactions were detected.

Figure D: the averaged peri-stimulus histogram of strain comparison. X-axis indicates time from stimulus. This histogram revealed significant strain effects in the evoked and spontaneous activity of the firing pattern. For rate analysis, see Figure F and G below. OP animals also exhibited a shorter inhibition period indicated by the earlier recovery phase.

Figure E: the averaged peri-stimulus histogram of diet comparison. X-axis indicates time from stimulus. This histogram exhibited no significant dietary effects.

Figure F: rate of spontaneous discharges. Bar indicates mean rate ± SEM. There was a higher spontaneous discharge rate for OP (0.05 cells, 2.18 ± 0.09 Hz) compared to OR (0.12 ± 0.09 Hz) (*, p < 0.005). Post-hoc analysis further revealed that the OP-CD spontaneous activity is significantly different lower than the three other groups. Similarly, OP demonstrated heightened tonic activity (300 ms before stimulus onset, 1.67 ± 0.09 Hz, bar graph not shown) than OR (1.33 ± 0.08 Hz) (p < 0.005).

Figure G: rate of evoked activation firing. The OP group (5.45 ± 0.35 Hz) exhibited higher evoked firing rate than the OR group (4.64 ± 0.34 Hz) (**, p < 0.005).

Figure H: signal-noise ratio analysis of signal-shock (evoked-tonic) firing rate. There was a significant strain interaction in this ratio analysis (**, p < 0.005). Post-hoc analysis also showed that OR-CD has the highest signal-to-noise ratio of all four groups.

Conclusions

- The obesity rat models sufficiently represented the phenotypes of human obesity by having reached the top 5% of the body weights with food ad lib.
- The OP group expressed significantly damped evoked activation of the LC-NE system versus the OR group.
- The OR animals also expressed an elevated level in the spontaneous discharge rate of locus coeruleus neurons compared with the OP animals.
- The signal-to-noise ratio analysis revealed that the groups of different obesity propensities respond differently to high- and low-fat diets.

Discussion

The present data are the first evidences for the involvement of the LC-NE in obesity susceptibility. Similarly, these results provide further insights into the chronic influences of obesity on the LC-NE system and, therefore, on mood and sympathetic activity. These findings indicate that the LC-NE targeting treatments of obesity and related emotional disorders.

To complement this experiment, an additional group of non-selected strain of Sprague-Dawley rats will be recorded after appropriate dietary conditioning to represent the theoretical basal level of obesity propensity. Furthermore, the relationship between obesity propensity and the fat-content level in diets is also a topic of interest.

Future research directions look to investigate mechanisms of modern weight loss drugs such as GLP-1 agonists, in relation with the LC-NE system. Having characterized the obesity propensity models at hand allows us to investigate how these drugs effectively mediate the LC-NE disruption caused by obesity.

Acknowledgements

We would like to thank the Arysta Research Center for providing the necessary funding for this experiment. Additional thanks goes to members of the Bello Lab who made this project possible.
Are Haspin and Bub1 kinases redundant for female meiotic chromosome segregation in Drosophila?

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The chromosomal passenger complex is required for accurate spindle assembly

- The chromosomal passenger complex (CPC) regulates spindle assembly and chromosome segregation. It is composed of four proteins and localizes in a ring around chromatin to organize the spindle in meiosis [2].
- When CPC member proteins INCENP and Aurora B (Aurora B) are knocked down in oocytes, CPC ring localization is not observed and no spindle assemblies [3].
- There is also evidence for CPC localization at centromeres in early metaphase during female meiotic spindle assembly (S. Radford, personal communication).

Haspin and Bub1 kinases recruit CPC subunits

- Through interaction with Pds5, Haspin kinase phosphorylates Histone H3 at Thr1 and recruits the CPC member protein Survivin, which positions Aurora B at centromeres in mitosis [4].
- The phosphorylation of histone H3 at Thr120 by Bub1 kinase recruits Shugoshin protein MEI-3332 to centromeres, which in turn contributes to the localization of CPC member Borealin and activation of Aurora B [5].

Haspin is not independently essential for chromosome segregation in oocytes

- In order to get a deletion of Haspin, we used a Minos element 418 bp upstream of the coding region to excise the gene. 225 excisions were obtained, but no deletions.
- These data collected using a 85% knockdown of Haspin suggest that Haspin kinase is not required for mitosis or female meiosis.
- This scoring for nondisjunction events in females expressing this same Haspin RNAi indicates that Haspin is not essential for accurate chromosome segregation in oocytes.

Bub1 is not essential for female meiotic chromosome segregation

- Expression of Bub1 RNAi in various tissues was performed using a 98% knockdown of Bub1 transcript. These results suggest Bub1 is not required for mitosis or female meiosis.
- Females expressing Bub1 RNAi in their germlines did not have increased nondisjunction events. This indicates that Bub1 is not essential for accurate chromosome segregation in oocytes.

Haspin and Bub1 kinases are not redundant for female meiotic spindle assembly or chromosome segregation

- Previous research in mitotic HeLa cells suggest these two CPC recruitment pathways are functionally redundant. Furthermore, cells lacking both Haspin and MEI-3332 are synthetic lethal [6].
- Females expressing, both, Haspin and Bub1 RNAi in their germlines are fertile and form bipolar oocyte spindles.
- There is no increase in nondisjunction in the Haspin and Bub1 double RNAi females when compared with wild type. This suggests there is no evidence that Haspin and Bub1 are functionally redundant for oocyte chromosome segregation.

Understanding CPC Localization

- To look at CPC localization in meiosis of Haspin and Bub1 double RNAi females, we will image their oocytes and stain for CPC member INCENP.
- Since these experiments rely on the use of a double knockdown that can have variable effects on protein expression, we will create a Pds5 and mei-3332 double mutant to further test the hypothesis of two redundant pathways in female meiotic CPC localization.

Acknowledgements

We would like to thank TRP at Harvard University for RNAi lines; the Rutgers University Division of Life Sciences and the Arsey Research Center for funding; and the members of the McKim lab for all of their assistance and continued support.
Mutations in the Ataxia Telangiectasia Mutated (ATM) gene cause a neurodegenerative disorder known as Ataxia Telangiectasia (A-T). When the ATM protein is altered, it leads to the production of DNA repair, a weakening of the immune system, and an impairment of movement and coordination, among other symptoms. Our aim is to use human embryonic kidney cells (HEK293) and ATM affected induced pluripotent stem cells (iPSCs) to better understand the mechanisms behind the disease. Through the use of various genome editing techniques, we planned to insert a known sequence surrounded by 2 "LoxP" sites into the HEK 293 cells to replace a specific exon associated with A-T so that it will be removable with the addition of an enzyme known as Cre recombinase at the LoxP sites. These cells have been chosen for their relative hardness and the ease with which they can be transfected in order to create a positive control for future experiments. We intend on working with the iPS cells in order to learn more about the effects of genome editing and study the causation/repair of the problems associated with A-T. Then, cellular function can be assessed by such methods as radiation treatment to see if genome correction can occur in the cells of affected individuals and how it will affect these cells.

The experiment began with 48 iPS (cardioblast) -sensitive HEK293 cells (pictured below) placed in medium without G418. They were then grown in a monolayer on a plate until they had become confluent (covered most of the plate).

These cells were then transfected using the CRISPR-Cas9 Genome Editor directed to Exon 6 of the ATM gene by guide RNA. The guide RNA binds the target sequence, which leads to the binding of Cas9 to the target site and a double strand break produced by it (pictured below). This promotes homologous repair of the genome.

The new donor DNA sequence from the designed plasmid (pictured below) is then inserted into this double strand break during the transfection through homologous recombination to replace the recently excised DNA. This leads to some cells carrying G418 resistance conferred on it by the neomycin resistance gene (neo) contained in the plasmid originally. The plasmid was cut at the KpnI and AccI (not shown) sites to linearize the plasmid to promote homologous recombination. These sites do not interrupt the inserted parts of the plasmid. Green Fluorescent Protein (GFP) was also transduced into the cells, and successfully transfected cells would mean fluorescence would be observable in them within 24-48 hours, as seen above.

Since the cells now have this new gene, they can be placed in medium that contains G418. Cells that survive and grow as a monolayer have been "selected" as they must carry G418 resistance. All cells beyond this point should contain the neo gene as well as the LoxP sites.

Once these cells are confluent, they are plated at a density of 1 cell per well in a 96 well plate to be able to track results in specific, clonal cell lines. If homologous directed repair is observed, these cells should not be mixed up with non-homologous recombinant cells, as non-homologous combination doesn’t insert the LoxP sites where we want them to be. These cells become confluent in the 96 well plate and are then collected and used for further DNA testing.

These collected cells are then lysed to recover DNA. Then, using a technique called "spike-in" using clonal sequences, the DNA is separated from the other parts of the lysed cells and collected.

Combining this DNA with primers of known length and sequence, a Polymerase Chain Reaction (PCR) is started. This allows us to amplify the quantity of relevant DNA sought out by the primers so that when they are later run on a gel, there will be more DNA and thus a stronger band.

After we run the gel electrophoresis with this amplified DNA, we expect to see a band in the region corresponding to the primer length, which would indicate the donor sequence had inserted into the HEK293 genome.

There are still many more samples left to test, but the positive results seen in Figure 3 show that it is possible to insert the desired neonycin resistance sequence in the genome at the correct location. We want to expand upon these results and garner further proof of the successful homologous recombination. There were a couple of experiments run with TALENs as well that did not progress as far as this CRISPR, so it’d be nice to see that work since it has its own benefits, such as greater specificity of targeting. We can use what we have learned and confirmed from this experiment to try to correct mutations in afflicted individuals to see if this can change the observed symptoms/phenotypes. Since these cells can serve as a positive control, this also allows us to work with the various genome editing techniques and see how effectively and efficiently they work for future studies, especially in iPSCs. Genome editing is the future of biotechnology and has the potential to help the lives of many people with debilitating genetic defects. Having full control of the mutations associated with this disease certainly opens up a world of possibilities in studying and potentially curing A-T.

References


Acknowledgements

Thank you to Dr. Ronald Hart and Dr. Jennifer Moore for providing me with guidance, assistance, training, and the knowledge necessary to complete this project. I’d also like to thank Michael D’Eclesiess, Alana Toro-Ramos, and Mavis Swordel for putting up with me and helping me through whatever problems I had even when I may have seemed like a lost cause.
Examining whether CED-3 cleaves RPM-1 to promote neuronal regeneration in C. elegans
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Abstract

Neurons of the human central nervous system do not regenerate well after injuries such as stroke or spinal cord injury. Currently, effective therapies to treat these injuries are lacking, but researchers are working towards the ultimate goal of promoting neuronal regeneration and completely restoring neuronal function. To achieve this goal, a more comprehensive understanding of the neuronal intrinsic and extrinsic factors regulating regeneration is needed. In recent years, a C. elegans model for studying regeneration was developed using a pulsed laser to sever axons of individual fluorescently labeled neurons. Regeneration of these neurons can then be analyzed over the next several hours and days by measuring the extent of regrowth. A laser anatomy setup and the laser anatomy protocol were established in the lab. In addition, the Driscoll lab determined that the apoptotic execution protein CED-3 promotes regeneration through the conserved DLK-1 MAPK kinetics pathway. CED-3 may modulate DLK-1 activity through the DLK-1 negative regulator RPM-1, an E3 ubiquitin ligase. RPM-1 has two putative CED-3 caspase cleavage sites. Furthermore, the f-box protein FSN-1, which acts in conjunction with RPM-1 to ubiquitinate DLK-1, is also being investigated. Our working model proposes that CED-3 cleaves RPM-1 in response to neuronal injury allowing DLK-1 to promote regeneration. The ced-3, rpm-1, and fsn-1 mutants were selectively crossed and preliminary data indicate that the ced-3; rpm-1 double mutant displays similar regenerative outgrowth to the rpm-1 strain, suggesting that CED-3 may cleave RPM-1 to promote regeneration. Similarly, the ced-3; fsn-1 double mutant displays similar regenerative outgrowth to the fsn-1 strain, suggesting FSN-1 operates downstream of CED-3.

Laser anatomy paradigm

Figure 1. C. elegans are mounted and immobilized on slides using agarose pads and 0.01% poly-L-lysine. GFP and CFP labeled neuron touch neurons are visualized. Laser pulses (485 nm) are then used to cut ALM axons. Worms are rescued from the slide, allowed to recover for 24 hours, and remounted to measure regenerative outgrowth.

Driscoll lab laser anatomy setup

Figure 2. The Driscoll lab’s laser anatomy setup (I). The fluorescent ALM neuron is visualized and crossed/denoting laser target is aligned with the axon (II). Laser pulses are used to sever the axon, confirmed by a gap in fluorescence (III).

24 hour regenerative outgrowth

Figure 3. GFP labeled wild type ALM touch neuron before laser anatomy (I), immediately after anatomy (II), and then 24 hours later (III). Wild type ALMs generate approximately 100 um of regenerative outgrowth during the first 24 h after anatomy.

Working model for CED-3 regeneration pathway

Figure 4. Neuronal injury leads to an influx of calcium into the cell that is amplified by the ER calcium chaperone CRT-1 (calreticulin). CED-4 binds to calcium through two putative EF hand domains which leads to oligomerization of CED-4 and CED-3 binding. CED-3 can then auto-activate (similar mechanism as apoptosis) and promote regeneration through the DLK-1 MAPK pathway, possibly by cleavage/activation of a complex formed by RPM-1 (an E3 ubiquitin ligase and negative regulator of DLK-1) and FSN-1.

Does CED-3 act through RPM-1/FSN-1 to promote regeneration?

Figure 5. Wild type ALM neurons expressing GFP prior to anatomy (I), immediately after anatomy (II), and 24 h later (III). Mutations in ced-3 decrease 24 h regrowth (IV), while rpm-1 (V), ced-3; rpm-1 (VI), fsn-1 (VII), and ced-3; fsn-1 (VIII) all regenerate more than wild type. The rpm-1 and fsn-1 mutations are epistatic to ced-3 suggesting that RPM-1/FSN-1 acts downstream of CED-3.

Regenerative outgrowth in ced-3, rpm-1, and fsn-1

Figure 6. Regenerative outgrowth measured 24 h after laser anatomy indicates ced-3 mutants regenerate approximately half that of wild type animals. rpm-1, ced-3; rpm-1, fsn-1, and ced-3; fsn-1 animals regenerate significantly more than wild type. (T-test **P < 0.05)

Putative RPM-1 cleavage sites

Figure 7. Two putative cleavage sites have been identified in RPM-1. Future studies include mutating either or both of these sites to determine if any mutations will impact regeneration and thereby suggesting CED-3 cleaves RPM-1 to promote regeneration.

Summary

C. elegans were used as an in vivo model to study neuronal regeneration where neurons visualized using GFP are axotomized using a pulsed laser and new regenerative outgrowth measured. The Driscoll lab identified a novel regeneration pathway involving the core apoptotic proteins CED-3 and executioner caspase CED-3. CED-3 acts to promote re-extension through the conserved dkl-1 pathway to promote regeneration but exactly how remains unclear. One possible mechanism for CED-3 modulation of the dkl-1 pathway being investigated is that CED-3 cleaves RPM-1, an E3 ubiquitin ligase and negative regulator of DLK-1, which contains two putative caspase cleavage sites. A ced-3 mutant was crossed with an rpm-1 and an fsn-1 mutant and were tested for regeneration along with each single mutant. Data indicates that ced-3 mutants regenerate less than wild type while rpm-1, fsn-1, ced-3; rpm-1, and ced-3; fsn-1 mutants regenerate more than wild type and ced-3. This suggests that CED-3 may promote regeneration through the DLK-1 pathway by cleaving the negative regulator RPM-1.

References


Investigations have indicated that aerobic fitness is strongly associated with reduced risk of mortality from both cardiovascular and non-cardiovascular diseases (Laakkanen et al., 2001). One proposed mechanism for the protective effect of cardiovascular fitness is attenuation of cardiovascular reactivity (CVR) to psychological stress and improved recovery from stressors (Spalding et al., 2004). In contrast, trait anxiety and hostility have been shown to be independent risk factors for coronary heart disease (CHD) (Miller et al., 1996). Therefore, the purpose of this study was to examine the relationships between aerobic fitness and trait anxiety on CVR and recovery from psychological stressors. A secondary purpose was to examine the moderating influence of aerobic fitness on the trait anxiety and stress-related CVR relationship.

**HYPOTHESES**
1. Greater aerobically fit individuals will exhibit lower levels of CVR and faster recovery in response to psychological stress.
2. Individuals with higher trait anxiety levels will exhibit greater cardiovascular responses to psychological stress.
3. Aerobic fitness will moderate the relationship between trait anxiety and cardiovascular stress responses.

**INTRODUCTION**
- Evidence exists for the cross-stressor adaption theory, which suggests that adaptations resulting from regular physical activity-aerobic fitness lead to similar adaptations in response to psychological stressors (Hull, Young & Ziegler, 1984).
- For example, regular physical activity has resulted in decreased heart rate, attenuated blood pressure, increased parasympathetic activation and decreased sympathetic activity in response to psychological stressors (Bord et al., 2000; O’Sullivan & Bell, 2003).
- Fit individuals show significantly attenuated HR and SBP reactivity to stressors, as well as a trend toward attenuated DBP reactivity (Forcier et al., 2006).
- Previous studies of the effects of exercise on blood pressure responses during psychological stress have generally failed to directly measure cardiorespiratory fitness (VO2peak) and control for behavioral or personality variables (e.g., anger, hostility, anxiety) that might result in greater CVR and slower recovery to stressors (Dishman et al., 2002).

**METHODS**
- **Maximal Aerobic Fitness**
  - VO2peak: peak oxygen consumption
- **Psychological Stressors**
  - Modified Stroop task: 2-digit number (17, 13, and 18) from a random 4-digit number
  - Serial Subtraction task: 2-digit number (17, 13, and 18) from a random 4-digit number

**RESULTS**
- A significant rise in DBP was observed in the low fit group during the Stroop task, p < .05.
- Greater high frequency HRV was observed for fit individuals at baseline and during the 5-min recovery period, p < .05.
- Higher anxiety group displayed higher SBP and DBP for the Stroop task, trending towards significance, p > .05.
- No significant differences between hostility and CVR or recovery from stress were found, p > .05.

**CONCLUSIONS**
- Greater anxiety is associated with greater CV reactivity and slower DBP recovery from psychological stressors. These results further suggest that aerobic fitness training may increase the ability of cardiovascular systems to control responses to acute stressors.
- Second, the stressors were effective as demonstrated by the CV variables as well as lower HF-HRV (parasympathetic cardiac control) during psychological stress exposure.
- Contrary to our expectations, trait hostility was not associated with CV stress responses.
- Physical fitness was associated with attenuated DBP responses to the Stroop task and although not statistically significant, also had a moderate effect on SBP responses.
- Fitness was related to lower HR and DBP values at rest, suggesting that lower absolute cardiovascular oscillations in fit individuals might explain the attenuated stress responses observed among fit individuals.
- Fitness was associated with high frequency HRV at baseline and during recovery, indicating higher vagally-mediated respiratory sinus arrhythmia.
- Associations between aerobic fitness and attenuated cardiovascular reactivity may provide an additional mechanism through which exercise may lead to improvements in cardiovascular health and decreased risk for adverse cardiovascular outcomes, including hypertension and coronary heart disease.

**ACKNOWLEDGEMENT**
We would like to thank Rutgers University, the Aresty Research Center for Undergraduates, and the School of Arts and Sciences for their funding and assistance with this project.
Walking Through Italian Literature and Film
Mihaela Sanderson, Aresty Center Research Assistant to Professor Andrea Baldi, Ph.D
Rutgers University - School of Arts and Sciences - Department of Italian

Introduction
Engaging in what at first seems like an ordinary fact of life, upon reflection we realize that walking becomes vial in establishing our perception of place. An essential kinesthetic behavior helps us alter our surroundings and use our environment to shape our philosophical thinking. In the moment we start walking, we essentially define the topography and the landscaping of the city from an individual, unique perspective. By that very act, as passersby, we become the sole architects in redefining and mapping the city in a new, different way. The common practice of walking not only permits us to geographically elaborate on the countless facets of the outside world, but often leads us to serious thought, consideration, reflection and soul searching.

Background
The Walking Through Italian Literature and Film research project originated from the larger concept of flânerie.
16
The objective of this research project is to supplement the base for a new course designed to examine different literary texts and films, all bound by a common theme i.e. the practice of walking. Through the practice of walking we are engaging in defining not only the geography of the space, but we are projecting new dimensions of cultural memory, the prioritization of individual freedom in relation to an ever-changing socio-economic framework, marked by the constant scientific and technological progress of the modernity. Among such literary texts, Manilfe Serio’s realist short story entitled Una Fiorita2 is perfectly detecting such dynamics. In this short story Serio not only reveals the practice of walking and the mapping of the city from that individual perspective, but also exposes the cultural practices and different characteristics pertaining to the social dimensions of that time.

Glossary
1 Kinesthetic- the use of the body and senses in learn about the world around you.
2 Flânerie- a 19th/20th century French term denoting strolling, idling. The term was further explored in 19th century in the writings of Charles Baudelaire accumulating the significant meaning of the casual wanderer, the observer and the reporter of the street life in the modern city.
3 Una Fiorita- The Flower girl.
4 Risanamento della Citta di Napoli 1899 - Rehabilitation of the City of Naples. This project initiated in 1895, a year after the cholera outbreak in 1884.

Methods
My research aimed to study the representation of walking in the Italian Literature and Film of the 19th and 20th century. I have solely worked on the short story Una Fiorita by Matilde Serao. The first step in my research was to break down and organize the protagonist’s journey into four main sections. Two documents found at the New York Public Library were essential. After carefully studying a map of the city of Naples dating 1899, the document entitled Risanamento della Citta di Napoli del 1899, which I translated from Italian into English, I reconstructed and captured in digital maps (Google Maps) the main character’s journey in order to reach a deeper understanding of the relationship between the protagonist and her urban environment. Such relationships shed light on historical intricacies, social and cultural dynamics of that period.

Urban Structure of the Journey in “Una Fiorita”

NYPL Topographic Map of the City of Naples 1899

NYPL Topographic Map of the City of Naples 1899

Digital Google Maps of the City of Naples

Discussion
The main character’s meandering experience on the streets of Naples is strictly delimited by specific geographical parameters and in conformity with the reality of her social dimension. Serio’s story sets the stage of a broken Naples, in which the cityscape is minutely observed through the visual, auditory and olfactory senses, thus revealing the spatial relationship within the city as bounded to two distinct spheres: the bright, dominant squares anchoring luxurious shops, and the dark slums of the city marked by the lack of sanitation, the accumulation of sewage, high rates of disease, crime and poverty.

While working on the short story I have come across two particular details which influenced my research and at the same time guided its outcome. Such findings are closely connected to Naples’ major urban intervention started in 1885 as a result of a serious cholera outbreak, and later on, the intense destruction of World War II. Both events radically changed the architecture of the most historic districts of the city. However, it was Naples’ urban rehabilitation that enabled extensively the replacement of pre-existing structures with new buildings, roads and squares. In reality, instead of solving the problems, it created a facade meant to conceal the poverty and the degradation of those areas. As a result, I have created a map that exposes the changes brought by the Rehabilitation, which helps us appreciate the city as it was then, as well as the struggles of the Neapolitan people.

Serio’s use of the practice of walking within the city space is an important tool that highlights much larger issues of social injustice and economic disparities, universal themes that calls for further research in order to better understand the pass in relation to today’s social and cultural practices.

Acknowledgements
I would like to express my gratitude to Professor Baldi for giving me this extraordinary opportunity to learn and grow; for his encouragement, continuous support and guidance throughout the year. Many thanks to Francesca Gianetti, Digital Humanities Librarian at Archibald S. Alexander Library for providing me with a crash course in digital mapping. I cannot express enough thanks to the Aresty Research Center, the School of Arts and Sciences, and to my friends Nicola Rosano and Carol Cofo for their invaluable advice. I would also like to acknowledge the New York Public Library staff, especially the map division for their patience and help provided during my week long stay while doing research.
ABSTRACTS ARE MICRO COSMS OF POSTERS

• **Title** – A succinct description of the study or its findings. This is also the title of the poster.

• **Introduction** – What is the research question and why is it important?

• **Background** – How is your specific approach unique?

• **Methods or mode of analysis** - What is the method for addressing your question?

• **Results** - What did you find?

• **Conclusions** – What are the implications? Why should we care?
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<th>Title</th>
<th>The Relationship between Undergraduate Research Participation and Subsequent Research Performance of Early Career STEM Graduate Students</th>
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<tr>
<td>Intro</td>
<td>Undergraduate research experiences have been adopted across higher education institutions.</td>
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<td>Back-ground</td>
<td>However, most studies examining benefits derived from undergraduate research rely on self-report of skill development.</td>
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<tr>
<td>Methods</td>
<td>This study used an empirical assessment of research skills to investigate associations between undergraduate research experiences and research skill performance in graduate school. Research experience characteristics including duration, autonomy, collaboration, and motivation were also examined.</td>
</tr>
<tr>
<td>Results</td>
<td>Undergraduate research experience was linked to heightened graduate school performance in all research skills assessed. While autonomy and collaboration were highlighted in student interviews, duration was most strongly correlated to significant increases in research skill performance.</td>
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<td>Conclusions</td>
<td>Based on these findings, we advocate for the inclusion of research experiences into the undergraduate science curriculum coupled with the creation of centralized offices of undergraduate research and faculty incentives for involving undergraduates in their research.</td>
</tr>
</tbody>
</table>

QUESTIONS/CONTACT US

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