Poster Design
Parts

1. Poster (use PowerPoint or Publisher)
2. Oral Presentation

- They should complement each other.
- A poster is visual: **show** what was done.
- Everything should address a central question.

A good poster…
- organizes your ideas and results.
- facilitates short presentations.
- can stand alone.
- Is visually appealing.
Common Poster Construction

1. Abstract
2. Background/Overview
3. Materials and Methods
4. Results
5. Conclusions
6. Citations
1. Abstract

- You have already constructed this piece!
- It may be updated from what you submitted on March 1.

A sulfamate ester is a suitable linker for a variety of carrier-linker prodrugs. We have chosen to use a sulfamate ester because it seems to resist human enzyme cleavage. We will show that a sulfamate ester linker will undergo hydrolysis in a carrier-linker prodrug and will properly facilitate the sustained release of steroid drugs with short metabolic half-lives. Most steroids have very short half-lives in vivo. Thus, the prodrug structure we are developing would extend the half-life of steroid-type drugs with phenol groups. We are starting with ethinyl estradiol.
2. Background/Overview

- Introduce and motivate the problem you solved
- Definitions and/or technical framework (**but not too much**)
- Place your problem in context
3. Materials & Methods

- How did you do the experiment?
- Techniques, measures
- Why do these techniques work?

Remark: You need not elaborate on procedures that are very common in your field.
4. Results

What did you find?
These may be slightly technical.
It is common to use graphs/illustrations here.
5. Conclusions

- Why do your results matter?
- How does this fit into the big picture?
- What further work may be done in the future?
- May also add future direction of research here
6. Citations

• Use smaller font (14 – 18 pt)
• Keep the length reasonable
• Use only the most important references to conserve space
Formatting Basics

• Use column format
• Poster must be 3 ft. tall x 4 ft. wide
• Leave 40% of poster blank to separate sections
• Font should be one style and easily readable
• Use graphics saved in .gif, .jpeg, .eps, or .png format
• Use a plain background that doesn't cause distraction
• Please include Aresty logo
• Be concise - less is more!
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 (35.7%) 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 reactivity, 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 to future treatments.
- Locus coeruleus neurons exhibit reliable biphasic responses that are also sensitive to specific physiological manipulations.
- Previously 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%) or low-fat (10%) diet ad lib for 10 weeks.
- See table below for groupings.
- Non-invasive cardiovascular data was taken 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>
<thead>
<tr>
<th>DIET STRAIN</th>
<th>Obese-Prone</th>
<th>Obese-Resistant</th>
</tr>
</thead>
<tbody>
<tr>
<td>High-Fat</td>
<td>OP-HF n=6</td>
<td>OR-HF n=7</td>
</tr>
<tr>
<td>Low-Fat</td>
<td>OP-CD n=7</td>
<td>OR-CD n=6</td>
</tr>
</tbody>
</table>

Results

**A** Body weights progression of each group across the ten-week time span. Graphs show data for week 8-10. The OR animals demonstrated a roughly 25% higher body weight than the OR animals. HF diet also increased body weight as expected. No statistical differences between groups were observed.

**B** Record body weight of each group. Bars indicate average body weight ± SEM. Each group exhibited significantly different body weight ranges.

**C** Cardiovascular data taken on week 10. Bars indicate mean ± SEM. Analysis of difference between groups. The heart rates of the OP group (273 ± 12 bmp) was significantly lower than that of the OR group (316 ± 12 bmp) at 0.05. The same time, the high-fat group (273 ± 11 bpm) had significantly lower heart rate than the low-fat group (212 ± 15.4 bpm). However, no strain-diet interactions were detected.

**D** Mean latency to first spontaneous activity (in seconds) for each group. The OR group had a significantly lower latency than the OP group. The high-fat diet significantly delayed the onset of spontaneous activity.

**E** Mean number of spontaneous spikes for each group. The OP group had a significantly higher number of spontaneous spikes than the OR group. The high-fat diet significantly increased the number of spontaneous spikes.

**F** Rate of spontaneous discharge. Bar indicates mean ± SEM. There was a higher spontaneous discharge rate for OP (0.95 spikes, 2.1 spike per stimulus) compared to OR (0.72 spikes, 1.07 ± 0.09 Hz) (p < 0.001). Post-hoc analysis further revealed that the OP-CD spontaneous activity is significantly different from the OR group. Similarly, OP demonstrated heightened tonic activity (500 ms before stimulus onset, 1.87 ± 0.09 Hz, bar graph not shown) than OR (1.53 ± 0.09 Hz) (p < 0.001).

**G** Rate of evoked activity firing. The OR group (0.83 ± 0.35 Hz) exhibited lower evoked firing rate than the OR group (0.84 ± 0.35 Hz) (p < 0.005).

Conclusions

- The obesity rat models sufficiently represented the phenotypes of human obesity by having reached the top 25% 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 propensity responds differently to high- and low-fat diets.

Discussion

The present data are the first evidence 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 provide grounds for our LC-NE targeting treatments of obesity and related emotional disruptions.

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 baseline of obesity propensity. Furthermore, the relationship between obesity propensity and the fat-content level in diets is also a topic of interest.

Future research direction looks 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 Artery Research Center for providing the necessary funding for this experiment. Additional thanks go to the members of the Bello Lab who made this project possible.
Spindle assembly in female meiosis

During oocyte meiosis, microtubules nucleate around the chromosomes which condense to form the karyosome and extend outward to build the meiotic spindle. This process occurs in the absence of microtubule organizing centers called centrosomes which guide mitotic spindle assembly [1].

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 Thr13 and recruits the CPC-member protein Survivin, which positions Aurora B at centromeres in mitosis [4].
- The phosphorylation of histone H3 at Thr126 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.

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

- Previous research in mitotic HeLa cells suggests that 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 germline are fertile and form bipolar oocyte spindles.

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.

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.

References


Acknowledgements

We would like to thank TRP at Harvard University for RNAi lines; the Rutgers University Division of Life Sciences and the Artery Research Center for funding; and the members of the McKinn lab for all of their assistance and continued support.
Walking Through Italian Literature and Film
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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 vital in establishing our perception of place. An essential kinesthetic behavior helps us to discover our surroundings and use our environment to shape our philosophical thinking. In our daily activities, we essentially define the topography and the landscape of the city from an individual perspective. By that very act, we become the landscape 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 Flâneur. 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, namely, the practice of walking. Through the practice of walking we are engaging in defining not only the geography of the space, but the most important aspect of cultural memory, to prioritize 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, Marilde Serio’s realist short story entitled Una Fioreria 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.

**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 Fioreria written by Matilde Serio. The first step in my research was to break down and organize the protagonist’s journey into four major 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 The Risanamento della Città di Napoli del 1899, which I translated from Italian into English, I re-constructed 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.

**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, domineering 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’s urban rehabilitation that enabled extensive 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, continued support, and guidance throughout the year. Many thanks to Francesca Gianetti, Digital Humanities Librarian Research 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 Nicolita Romano and Carol Cofone 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.

**Glossary**

- *Kinesthetic*: the use of the body and senses to learn about the world around you.
- *Flâneur*: 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 reportor of the social life in the modern city.
- *Una Fioreria*: The Flower girl.
- *Risanamento della Città di Napoli 1899*: Rehabilitation of the City of Naples 1899 document, but the project initiated in 1885, a year after the cholera outbreak in 1884.
Modeling, Understanding, and Correcting Ataxia Telangiectasia
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Department of Cell Biology and Neuroscience, Rutgers University Piscataway, NJ

Abstract

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 prevention of DNA repair, a weakening of the immune system, and an impairment of movement and coordination, among other symptoms. Our goal is to use human embryonic kidney cells (HEK293) and A-T 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, and with A-T 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 ease with which they can be manipulated to provide a platform for future experiments. We intend on working with the iPSCs 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.

Methods

The experiment began with G418 (antibiotic-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).

The experiment was then transferred using the CRISPR/Cas9 Genome Editor directed to Exon 6 of the ATM gene by guide RNA. The guide RNA finds the target sequence, which leads to the binding of Cas9 to the target site and a double-strand break precipitating the cell death (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 because it contains the neomycin resistance gene (neo) contained in the plasmid originally. The plasmid was cut at the Kpnl and AccI (not shown) sites, which is necessary to insert 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 to specific cloning cell lines. If homologous directed repair is observed, these cells shouldn’t be mixed up with non-homologous corrected cells, as non-homologous contamination doesn’t insert the loxP sites where we want them to be. These cells become confluent in the 96 well plate and are then picked/cultivated for further DNA analysis.

These collected cells are then lysed to recover DNA. Then, using a technique called “sequencing by synthesis and amplification,” 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 the 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 run the gel electrophoresis with this amplified DNA, we expect to see a band in the region corresponding to the expected size, which would indicate that the donor sequence had inserted into the HEK293 genome.

Results

Fig. 2. An example of a gel showing no insertion of donor, with 2 ladders on the left as a scale, 6 numbered samples, and a donor plasmid along with an A-T carrier patient’s DNA as controls. The donor control has the correct donor band (765 bp) but no genomic band. The chart control has the correct genomic band (679 bp) but not the donor. None of the samples had both bands, which is what we are looking for to prove insertion of the donor. See orange arrows in Fig. 4 for primer location

Fig. 3. A gel with ladder on the left and 6 different lettered samples than the ones used before. Note the faint band for Sample A next to the white arrow in the ladder. This is located where a fragment for the genome + loxP should be (1664 bp), proving that the donor DNA inserted into the correct location. The same goes for Samples F, which also has the same band. See blue arrows in Fig. 4 for primer location

Fig. 4. Diagram of the region in the genome where the plasmid should have recombined. Primers were started (for Fig. 3) in the left adjacent region (present in HEK293 cells, not in plasmid) and ended in the loxP site (in plasmid, not in non-transfected HEK293 cells), while in Fig. 2 it starts in the left homology region (present in plasmid and cells) and ends after the loxP (in plasmid, not non-transfected cells.) The fragment obtained from Fig. 3 would thus only be present in correctly recombined HEK293 cells, while Fig. 2 simply tries to show that it is present, not that it is in the correct location.

Conclusions

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 neomycin 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 one, so it’s 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 symptomatic 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 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’Ecellissa, Aluna 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
Isaac Song, Christopher Reina, and Monica Driscoll
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Rutgers University, Piscataway NJ

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 complete 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 axotomy setup and the laser axotomy protocol were established in the lab. In addition, the Driscoll lab determined that the apoptotic executioner protein CED-3 promotes regeneration through the conserved DLK-1 MAPKKK regeneration pathway. CED-3 may be modulating 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 axotomy paradigm

Figure 1. C. elegans are mounted and immobilized on slides using agarose pads and 0.01% polylysine microbeads and GFP labeled ALM touch neurons are visualized. Laser pulses (435 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.

Driscol lab laser axotomy setup

Figure 2. The Driscoll lab’s laser axotomy setup (i). The fluorescent ALM neuron is visualized and crosshair denoting laser target is aligned with the axon (ii). Laser pulses are used to sever the axon, confirmed by a gap in fluorescence (iii).

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

Figure 5. Wild type ALM neurons expressing GFP prior to axotomy (i), immediately after axotomy (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.

24 hour regenerative outgrowth

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

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-3 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 in apoptosis) and promote regeneration through the DLK-1 MAPKK 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.

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

Figure 6. Regenerative outgrowth measured 24 h after laser axotomy indicates ced-3 mutants regenerate approximately half 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 the point 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-4 and executioner caspase CED-3. CED-3 appears to act through the conserved DLK-1 regeneration pathway to promote regeneration but exactly how remains unclear. One possible mechanism for CED-3 modulation of the dlk-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

Katherine King
Professor Dr. Geoffrey Wallace, Political Science Department, Rutgers University

ABSTRACT

Free media is fundamental to good governance. In recognition of its political influence and ability to serve as a check on governments and businesses, the free press is often referred to as "The Fourth Estate." Yet, because of this power, journalists often become targets of violence internationally. While many organizations track the killing of journalists, research analyzing the data has been scant. To facilitate such evaluation, I have collated information from six independent sources to create a dataset tracking journalistic killings over an eleven-year period. I proceed by examining trends in variables such as medium, main coverage, and regime-type in order to establish which factors increase the likelihood of violence toward journalists. Preliminary evaluations suggest that the form of government in a country correlates with media-targeted violence, with the lowest journalists being killed under authoritarian regimes. Thorough investigation of the data will allow journalist to better prepare for and respond to threats while in the field. Also, further research can guide international institutions to selectivity pressure regions and countries where journalists are most endangered.

BACKGROUND

Because of journalists power to monitor the activities of governments, business, and criminal organizations, they are often subject to violence and murder. Many organizations monitor violence against journalists, however, there are discrepancies in their definitions of journalistic fatalities. This leads to organizational biases and incomplete coverage of the threats that journalists face. The purpose of this research is to create an accurate dataset of all media killings, in order to more accurately assess the threats that journalists endure. With this information, detailed analysis can be performed to understand which factors most increase the likelihood of media fatalities. A thorough understanding of these factors and how they interact can prepare journalist to respond to threats in the field and know which assignments are especially dangerous.

MATERIALS AND METHODS

Data was collected from six international journalist organizations, each of which creates databases tracking the killing and deaths of journalists across the globe. These organizations include The Committee to Protect Journalists (CPJ), Freedom Forum (FFP), The International Federation of Journalists (IFJ), The International Press Institute (IPI), Reporters Without Borders (RSF), and the World Association of Newspapers and News Publishers (WAN, IFRA). By cross-referencing these databases, I ensured that the information for each observation is as accurate and complete as possible. Observations were collated into Microsoft Excel spreadsheets, disaggregated by year. Further, specific information about each case was coded based on a rubric for each variable. Variables included information about the journalists such as gender, nationality, job, organization, and medium. Additional variables tracked information regarding the attack such as attack type, attacker nature, the method by which the journalist was killed, and whether the journalist was taken captive, threatened, or tortured prior to death. This information was then combined and analyzed over a period from 1992 - 2002.

RESULTS

Number of Journalists Killed Over Time

Observations By Data Source

Journalists Killed Over Time, By Medium

Journalist Killed, by Main Area of Coverage

The Influence of On the Targeting of Journalists

CITATIONS


ACKNOWLEDGEMENTS

I would like to thank Dr. Geoffrey Wallace for inviting me to work on this research with him. This has been an invaluable learning experience, and has increased my interest in more in-depth research endeavors. Also, I would like to thank the Rutgers Research Center for Undergraduates for providing me the opportunity to present this research.

RUTGERS
Aresty Research Center
for Undergraduates
Relations Between Aerobic Fitness, Trait Anxiety, and Cardiovascular Responses to Stress

Colleen D. Schreier, Ryan L. Olson, Brandon L. Alderman
Department of Exercise Science, Rutgers University

ABSTRACT

Investigations have indicated that aerobic fitness is strongly associated with reduced risk of mortality from both cardiovascular and non-cardiovascular diseases (Laukkanen 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 physically fit individuals will exhibit lower levels of CVR and faster recovery in response to psychological stress.
2. Individuals with higher trait anxiety levels will evidence 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 (Borbély et al., 2000; O’Sullivan & Bell, 2001).
- Fit individuals show significantly attenuated HR and SBP reactivity to stressors, as well as a trend toward attenuated DBP reactivity (Fioroni et al., 2006).
- Previous studies of the effects of exercise on blood pressure responses during psychological stress have generally failed to directly measure cardiopulmonary fitness (VO2 peak) 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
- VO2 peak

Psychological Stressors
- modified Stroop task: loss feedback was provided on 40% of responses
- Serial Subtraction task: Continuous subtraction of 2-digit number (17, 13, and 18) from a random 4-digit number

Cardiovascular Autonomic Measures
- HR and BP were continuously assessed during the stressor tasks. Through impedance cardiology, pre-ejection period (PEP) and high frequency (0.15-0.5 Hz) heart rate variability (HF-HRV) were assessed as primary measures of sympathetic and parasympathetic cardiac control, respectively.

RESULT

- 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.
- High anxiety group displayed higher SBP and DBP for the Stroop task, trending towards significance, p < .05.
- No significant relationships between hostility and CVR or recovery from stress were found, p > .05.

CONCLUSION

- 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 Exercise Research Center for Undergraduates, and the School of Arts and Sciences for their funding and assistance with this project.
Oral Presentation

• Use body language
  - motion to the section you're explaining
  - explain all pictures and diagrams

• Keep your presentation under 5 minutes

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• explain diagrams

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