MEDICAL SCHOOL COUNCIL
RSO Funding Allocation Report

Meeting Date: 10/23/19
Meeting Time: 5:00 pm
Meeting Location: College of Medicine
MSC Members Present: Vinita Akula, Mayla Oyola, Mohsan Khan, Shalom Chege,
Eduarda Machado, Caitlyn Blake-Hedges, Jenny Warnock

<table>
<thead>
<tr>
<th>Organization</th>
<th>Event/Conference Name, Date, #of students impacted</th>
<th>Contractual Services (speaker, DJ)</th>
<th>Expense (travel, rentals, supplies)</th>
<th>Food (food, food supplies)</th>
<th>Clothing (tshirts)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biomed Graduate Student Association</td>
<td>64th Annual Meeting for Biophysical Society</td>
<td></td>
<td>$2000</td>
<td></td>
<td></td>
<td>$2000</td>
</tr>
<tr>
<td>American Medical Woman’s Association</td>
<td>Crisis Pregnancy Centers 12/03/19 12:00-1:00PM Open to all students</td>
<td></td>
<td></td>
<td>$150</td>
<td></td>
<td>$150</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td>$2000</td>
<td>$150</td>
<td></td>
<td></td>
<td>$2150</td>
</tr>
</tbody>
</table>

Organizations receive funds for the particular conference of event, any funds not spent will be swept back to the funding council and eventually to COGS at the end of the fiscal year. COGS approval is required for all expenditures, and funds are only granted to organizations who follow policies outlined in the SGA/COGS Financial Manual, COGS Code, and SGA Statutes.

Meeting Minutes
(please include a breakdown of the funding request, costs, and what allocated funds are being allocated for. Should also include the vote for each request).
The Medical School Council (MSC) reserves the right to sweep any funds back to us.
If an organization wishes to use the funds for a reason other than the one they described at the meeting, they must get approval from MSC.

DATE 10/23/19
Attendance → Vinita Akula, Mayla Oyola, Mohsan Khan, Shalom Chege, Eduarda Machado, Caitlyn Blake-Hedges, Jenny Warnock
Organization: American Medical Women’s Association

<table>
<thead>
<tr>
<th>Amount requested</th>
<th>Amount Allocated</th>
<th>Votes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Budget Categories and Amount Requested</td>
<td>Budget Categories and Amount Requested</td>
<td>8-0, approved</td>
</tr>
<tr>
<td>Contractual Services 0</td>
<td>Contractual Services 0</td>
<td></td>
</tr>
<tr>
<td>Expense General 0</td>
<td>Expense General 0</td>
<td></td>
</tr>
<tr>
<td>Expense Travel 0</td>
<td>Expense Travel 0</td>
<td></td>
</tr>
<tr>
<td>Food 150</td>
<td>Food 150</td>
<td></td>
</tr>
<tr>
<td>Clothing/Awards 0</td>
<td>Clothing/Awards 0</td>
<td></td>
</tr>
</tbody>
</table>

Questions:
- What are the funds for?
  - This is an awareness-raising event for Crisis Pregnancy Centers (CPCs), which often pose as prenatal care or abortion clinics. Here in Tallahassee, Florida we have a number of CPCs in our own backyard – come out to learn what services they do or don’t provide, and why. We will be providing a safe space for discussion, and questions to follow the presentation. This event will include catering from one of the approved vendor lists. This event will also be a follow up to our movie screening of 12th and Delaware so it will hopefully be well attended.
- Note: The RSO gave estimates for multiple vendors in their Qualtrics survey, however the representative of the RSO confirmed that they wanted to use Publix as their vendor.
- Note: The request also meets our guidelines of being below $150 for a lunch & learn while also not conflicting with other events so it was approved.

Organization: Biomed Graduate Student Association

<table>
<thead>
<tr>
<th>Amount requested</th>
<th>Amount Allocated</th>
<th>Votes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Budget Categories and Amount Requested</td>
<td>Budget Categories and Amount Requested</td>
<td>8-0, approved</td>
</tr>
<tr>
<td>Contractual Services 0</td>
<td>Contractual Services 0</td>
<td></td>
</tr>
<tr>
<td>Expense General 0</td>
<td>Expense General 0</td>
<td></td>
</tr>
<tr>
<td>Expense Travel 5088</td>
<td>Expense Travel 2000</td>
<td></td>
</tr>
<tr>
<td>Food 0</td>
<td>Food 0</td>
<td></td>
</tr>
<tr>
<td>Clothing/Awards 0</td>
<td>Clothing/Awards 0</td>
<td></td>
</tr>
</tbody>
</table>

Questions:
- What are the funds for?
  - As science becomes increasingly interdisciplinary, the Biophysical Society Annual Meeting continues its long-held reputation for bringing together leading scientists from the all over the world who work at the interface of the life, physical, and computational sciences. The dynamic five-day Meeting provides attendees with opportunities to share
their latest unpublished findings and learn the newest emerging techniques and applications. Despite its nearly 6,500 attendees, the Meeting is noted for maintaining its “small meeting” feel beginning with the Saturday subgroup symposia, which allow attendees to meet within their scientific communities. It is also known for its vitality, demonstrated by the over 900 highly interactive daily poster presentations, the more than 500 speakers selected from submitted abstracts, the many career development programs for those working in academia, industry, and agencies throughout the world, and its advocacy and education programs.

- Note: There will be a total of 4 attendees (3 females and one male) and each of these attendees are presenting. (See Figures 1-4 for each student’s confirmation of registration/abstract)
- Note: That MSC has allotted a total of $2000, with a total of $500 per person (this maximum is set by COGS). There will be the COGS maximum of $150 per hotel room per night. Since there will be with 2 hotel rooms and a total of 5 nights gives a total of $1500 (See Figure 5 for estimated hotel cost). A total of $320 for registration (80$ per person registration multiplied by 4 gives a total of $320). See Figure 6 for cost of registration. The remainder $45 per person for transportation (multiplied by 4 people gives the total of $180). (See figure 7 for cost of transportation)
- Note: The students that were to be funded by MSC were chosen due to their demonstrated interest in the conference. When opportunities like this arise then the presidents of the respective RSO’s send out the information to the whole medical school. This is true for all events being funded by MSC.
- Note: The conference was not fully funded due to the fact that none of the students that will be attending the conference are presenting research or giving a speech at the conference.

Figure 1: Abstract of One Student Presenting

---

Figure 1: Abstract of One Student Presenting

**Abstract**

**Title:** Measurement of active thin filament in partially stabilized heart muscle reveals novel structural properties of the cardiac thin filament

**Authors:**

- Melissa Landen Warsa
- Jami Blanken
- James Johnson
- Prescott E. DeChiel
- Thomas C. Lin
- Daniel O. Pepe
- Dina H. Aziz
- Frank A. Friesen
- David B. Soslow

**Institution:**

- University of Florida, Gainesville, FL, USA
- Department of Anatomy, University of Florida, Gainesville, FL, USA
- Department of Physiology and Biophysics, University of Florida, Gainesville, FL, USA
- Department of Biochemistry and Molecular Biology, University of Florida, Gainesville, FL, USA
- Department of Chemistry, University of Florida, Gainesville, FL, USA
- Department of Biochemistry and Molecular Biology, University of Florida, Gainesville, FL, USA
- Department of Physiology, University of Florida, Gainesville, FL, USA
- Department of Biochemistry and Molecular Biology, University of Florida, Gainesville, FL, USA

**Abstract:**

The relative filament periodicity within the individual filaments is poorly understood due to the challenges of obtaining high signals with quick image acquisitions. Small angle x-ray diffraction (SAXS) conducted on partially stabilized heart muscle revealed novel structural properties of the cardiac thin filament. Furthermore, the use of partially stabilized muscle allows us to probe the role of Ca2+ and myosin crossbridges to thin filament arrangements during contraction. The study shows that the long pitch helical periodicity of the actin filament intensity increased due to Ca2+ contraction rather than myosin cross-bridges. The actin filament intensity increased during contraction with equal contribution from Ca2+ alone and both cross-bridges and Ca2+ x myosin cross-bridges observed under both conditions. These data suggest that both Ca2+ alone and myosin cross-bridges contribute to the thin filament length changes. Furthermore, compared to isolated muscle, cardiac muscle shows an overall contribution in the long pitch helical periodicity of the actin filament intensity. It is similar to the role of inositol 1,4,5-triphosphate (IP3) in cardiac muscle. These findings suggest that the role of IP3 in cardiac muscle and the role of inositol 1,4,5-triphosphate (IP3) in cardiac muscle may play a crucial role in modulating the length changes of the thin filament. This mechanism is altering the cardiac muscle. In addition, the role of isolated muscle and an isolated myofibril in conventional myofibril measurements was seen during contraction (Ca2+ x myosin cross-bridges intensity), similar to what was observed with cardiac muscle here. We speculate that the role of myosin in cardiac muscle and the role of inositol 1,4,5-triphosphate (IP3) in cardiac muscle may play a crucial role in modulating the length changes of the thin filament. This mechanism is altering the cardiac muscle. In addition, the role of isolated muscle and an isolated myofibril in conventional myofibril measurements was seen during contraction (Ca2+ x myosin cross-bridges intensity), similar to what was observed with cardiac muscle here. We speculate that the role of myosin in cardiac muscle and the role of inositol 1,4,5-triphosphate (IP3) in cardiac muscle may play a crucial role in modulating the length changes of the thin filament. This mechanism is altering the cardiac muscle. In addition, the role of isolated muscle and an isolated myofibril in conventional myofibril measurements was seen during contraction (Ca2+ x myosin cross-bridges intensity), similar to what was observed with cardiac muscle here. We speculate that the role of myosin in cardiac muscle and the role of inositol 1,4,5-triphosphate (IP3) in cardiac muscle may play a crucial role in modulating the length changes of the thin filament. This mechanism is altering the cardiac muscle. In addition, the role of isolated muscle and an isolated myofibril in conventional myofibril measurements was seen during contraction (Ca2+ x myosin cross-bridges intensity), similar to what was observed with cardiac muscle here. We speculate that the role of myosin in cardiac muscle and the role of inositol 1,4,5-triphosphate (IP3) in cardiac muscle may play a crucial role in modulating the length changes of the thin filament. This mechanism is altering the cardiac muscle. In addition, the role of isolated muscle and an isolated myofibril in conventional myofibril measurements was seen during contraction (Ca2+ x myosin cross-bridges intensity), similar to what was observed with cardiac muscle here. We speculate that the role of myosin in cardiac muscle and the role of inositol 1,4,5-triphosphate (IP3) in cardiac muscle may play a crucial role in modulating the length changes of the thin filament. This mechanism is altering the cardiac muscle. In addition, the role of isolated muscle and an isolated myofibril in conventional myofibril measurements was seen during contraction (Ca2+ x myosin cross-bridges intensity), similar to what was observed with cardiac muscle here. We speculate that the role of myosin in cardiac muscle and the role of inositol 1,4,5-tripha...**
Figure 2: Abstract of One Student Presenting

Biophysical Society
2020

64th Annual Meeting of the Biophysical Society
February 15-19, 2020 - San Diego, California

Please review your work, no changes may be made after the revision deadline, October 4, 2019 at 11:59 PM EST.

Control/Tracking Number: 26-A2507-BPS
Activity: Abstract
Current Date/Time: 10/1/2019 1:54:21 PM

REDUCED BETA MYOSIN HEAVY CHAIN K213 ACETYLATION AND T215 PHOSPHORYLATION IN HUMAN HEART FAILURE

Author Block: Amanda Wacker1, Michelle C. Rodriguez Garcia2, Maicon Landim Vieira3, Rakesh K. Singh4, Elizabeth A. Bundeage5, Bryan A. Whitton6, Paul M. Janssen7, Prescott B. Chase8, Brandon J. Biesiadecki9, Michelle S. Parvatyan10, J. Renato D. Pinto11
1Florida State University, Tallahassee, FL, USA, 2Biomedical sci, Florida State University, Tallahassee, FL, USA, 3Biomedical Sci, Florida State Univ, Tallahassee, FL, USA, 4The Ohio State University, Columbus, OH, USA, 5Dept Physiol/Cell Biol, Ohio State Univ, Columbus, OH, USA, 6Dept Biol Sci, Florida State Univ, Tallahassee, FL, USA, 7Dept Physiol/Cell Bi, The Ohio State Univ, Columbus, OH, USA, 8Nutrition Food, Florida State University, Tallahassee, FL, USA, 9Dept Biomed Sci Col, Florida State Univ, Tallahassee, FL, USA.

ABSTRACT:
Post translational modifications (PTM) modulate cell signaling and protein function within cells. In the heart, protein phosphorylation found on several sarcomeric-proteins has been shown to be crucial for fine-tuning of myocardial contractility. Acetylation has an important role regulating histone proteins in many cell types including cardiomyocytes, but was subsequently found as a protein modification outside of the nucleus. Our interest is to further characterize the K-acetylation and S-, T-, and Y-phosphorylation found on the beta-myosin heavy chain (B-MHC) protein in donor, ischemic, and non-ischemic human heart samples with the long-term goal of understanding the potential role of PTMs on cardiac function. Using bottom-up proteomics and label-free quantification, we identified two high-confidence peptides exhibiting high PTM scores on the B-MHC protein that were ather in failing human hearts. One peptide showed significant changes in HM levels involving the simultaneous acetylation of K213 and the phosphorylation of T215 within the myosin subfragment-1 (head) domain. Both PTMs were found at a bend/loop in

www.abstractonline.com/Print/PrintFriendlyVersion.aspx?ControlKey=94CF2168-3648-4680-8960-B0398640E3D5&MaddrigActivityKey=%7B2A130E8-B038-4B84-A29E-3F4D5E1...
CONNECTING CARDIAC SARCOLEMMMA PROTEIN CONTENT WITH SARCOMERIC FUNCTION

Author Blocks: Isabella Leite Coscarella¹, Maicon Landim Vieira¹, Isela C. Valera², Amanda L. Wacker², Prescott B. Chase³, J. Renato D. Pinto³, Michelle S. Parvatiyar⁴.
¹Biomedical Sciences, Florida State University, Tallahassee, FL, USA, ²Nutrition, Food and Exercise Sciences, Florida State University, Tallahassee, FL, USA, ³Biological Sciences, Florida State University, Tallahassee, FL, USA, ⁴Nutrition, Food and Exercise Sciences, Florida State University, Tallahassee, FL, USA.

Abstract:
Background: Mechanotransduction is important for regulation of cellular function, whereby external mechanical signals are translated into biochemical pathways that can alter striated muscle function. Evidence of communication by the proteins at the cardiac cell membrane with proteins of the sarcomere has been slowly emerging. This study is examining the consequences of reduced dystrophin–glycoprotein complex (DGC) abundance on contractile parameters of the cardiac myofilament. Methods: Left ventricular papillary muscle bundles were used to determine the effects of DGC reduction on cardiac myofilament function in wild-type and homozygous–null (Homo) mice. Sarcomere length was set at 2.1 μm at pCa 8.0 using a HeNe laser. Sinusoidal stiffness (0.2% PIP length oscillation) and rate of tension redevelopment (kTR) were obtained during Ca²⁺ activation when force was at least 20% of maximal force. All experiments were carried out at ~21°C. Analysis of mathematical modeling of force is in progress. Results: No significant changes were observed in neither myofilament Ca²⁺ sensitivity nor cooperativity of thin filament activation between WT and Homo. Maximal force values were observed to be significantly increased in the Homo group as compared to WT. Although Homo exhibited a significant faster kTR in submaximal activation, no significant changes in maximal kTR were observed. Homo fibers displayed an enhanced sinusoidal stiffness in both submaximal and maximal activation. To explain these results we have utilized immunofluorescence, echocardiography, qRT–PCR and immunoblotting. Homo mice have larger hearts by 4 months of age and manifest concentric hypertrophy. It remains unknown whether the cardiac enlargement is driven by the increased maximal force of cardiac myofilaments. We will also determine whether the reduction in sarcolemma DGC protein content in the heart alters myofilament function by alterations in sarcomeric isoform expression.

Presentation Preference (Complete):
Poster Only
No

Sponsorship (Complete):

Topic (Complete): 6C Cardiac Muscle Regulation; 6B Cardiac Muscle Mechanics & Structure

Technique (Complete):
First Selection: Fluorescence
Figure 3: Abstract of One Student Presenting

**Control/Tracking Number:** 20-A-619-BPS  
**Activity:** Abstract  
**Current Date/Time:** 9/25/2019 11:05:31 AM

**SEX DIFFERENCES IN REGULATING THE CARDIAC TRANSCRIPTOME WITHIN A MURINE MODEL FOR HYPERTROPHIC CARDIOMYOPATHY**

**Author Block:** Karissa M. Dieseldoff Jones1, Cynthia Vied2, Isela C. Valera3, Prescott B. Chase4, Michelle S. Parvatiyar5, J. Renato Pinto3.

1Department of Biomedical Sciences, Florida State University, Tallahassee, FL, USA. 2Translational Science Laboratory, Florida State University, Tallahassee, FL, USA. 3Department of Nutrition, Florida State University, Tallahassee, FL, USA. 4Department of Biological Sciences, Florida State University, Tallahassee, FL, USA.

**Abstract:**
Heart disease remains the number one killer of women in the United States. Nonetheless, studies in women and female animal models continue to be underrepresented in cardiac research. Susceptibility to hereditary hypertrophic cardiomyopathy, the most commonly inherited cardiac disorder, has been tied to sarcomeric protein variants. Among the susceptible genes, TNNC1—encoding cardiac troponin C (cTnC)—has contributed to a substantial hypertrophic cardiomyopathy (HCM) phenotype in mice. In this study we sought to characterize the sexual dimorphism observed within cardiac hemodynamics, morphology, and gene expression of a TNNC1 HCM mouse model. Adult age-matched male and female mice bearing the knock in HCM associated point mutation of alanine to valine in position 8 of cTnC (cTnC_8AV) were studied. The hemodynamics and cardiac morphology of the HCM mice were significantly altered. Isovolumetric contraction time was significantly higher in the female HCM mice, and female measurements for the majority of parameters trended toward the more severe disease presentation. RNA sequencing revealed several altered canonical pathways among the HCM vs WT groups including an increase in the EIF2 signaling, ILK signaling, actin nucleation by ARP-WASP complex, regulation of actin-based motility by Rho, VDR/RXR activation, and glutathione redox reactions pathways within the HCM mice. In contrast, the valine degradation, TCA Cycle II, Methionine Degradation, and Inositol Phosphate Compound pathways were notably down regulated in the HCM mice. Interestingly, seven of the genes that were differentially expressed in both the WT and HCM male vs female comparisons changed directions in fold change between the sexes. These data suggest a sexually dimorphic HCM phenotype and identify several key pathways and genes that could be critical to sex differences seen in disease manifestation.

**Presentation Preference (Complete):**

: Poster Only

**Sponsorship (Complete):**

**Topic (Complete):** 6C Cardiac Muscle Regulation ; 2B Transcription

**Technique (Complete):**

First Selection: Cell/Tissue Imaging & Mechanics  
Second Selection: None/Other

**Payment (Complete):** Your credit card order has been processed on Wednesday 25 September 2019 at 10:53 AM.

**Status:** Complete
Biophysical Society
2020 BPS Annual Meeting

Abstract Fee Receipt
Your payment has been received.
Please save this receipt for your records.
Thank you for your submission.

Current Date: 9/25/2019
Name: Dieseldoff Jones, Karissa
Submission Type: Abstract
Control/Invoice #: 20-A-419-BPS
Title: SEX DIFFERENCES IN REGULATING THE CARDIAC TRANSCRIPTOME WITHIN A MURINE MODEL FOR HYPERTROPHIC CARDIOMYOPATHY

Billing Information:
Gwen R Drake
1115 West Call St.
Tallahassee, FL 32306
Payment Posted On: 9/23/2019
10:53:39 AM
Payment Type: Visa *** *** *** ***
3864
Amount Due: $150.00
Amount Paid: $150.00
Amount Billed: $150.00
Balance Due: $0.00

Please preview your abstract by clicking the link in the left-hand column entitled Review My Work.
Figure 5: Estimated cost of Hotel

Stay Alfred at Alexan ALX

San Diego, CA
Comfortable and welcoming apartments close to the waterfront and convenient for hitting San Diego's best bars and restaurants.

<table>
<thead>
<tr>
<th>Studio</th>
<th>$201.00 per night (3 night minimum)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleeps 1-2 Guests</td>
<td></td>
</tr>
<tr>
<td>Just 2 Left!</td>
<td></td>
</tr>
<tr>
<td>Add</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>1 Bed/1 Bath</th>
<th>$206.00 per night (3 night minimum)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleeps 1-4 Guests</td>
<td></td>
</tr>
<tr>
<td>Just 1 Left!</td>
<td></td>
</tr>
<tr>
<td>Add</td>
<td></td>
</tr>
</tbody>
</table>

Figure 6: Estimated Cost of Registration

<table>
<thead>
<tr>
<th>Product Name</th>
<th>Quantity</th>
<th>Unit Cost</th>
<th>Line Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>2020 Annual Meeting Early Registration - Student Member</td>
<td>1</td>
<td>$80.00</td>
<td>$80.00</td>
</tr>
</tbody>
</table>
Figure 7: Estimated Cost of Transportation