Women in Science and Engineering and Pratt's Office of Diversity and Inclusion invite you to the

2018 WiSE Symposium: STEM Equity and Access

Increasing Diversity in Senior Positions in Academia and Beyond

April 27, 2018 Field Auditorium, Environment Hall





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12:30 pm: Lunch is served

12:45-2:00 pm: Keynote Address by Dr. Renetta Tull, University of Maryland Baltimore County Talk Title: "Academic Leadership: Prepare to Take Your Seat at the Table"

2:00-4:00 pm: Panel Discussion Topic: Why are there so few women in senior positions in academia and across industries? (RCR credit)

Panelists include:

Dr. Renetta Tull, Associate Vice Provost for Strategic Initiatives, UMBC

Dr. Jacqueline Looney, Senior Associate Dean for Graduate Programs and Associate Vice Provost for Academic Diversity, Duke University

Dr. Elaine Cohen Hubal, Acting Director, Computational Exposure Division, U.S. Environmental Protection Agency Ms. Victoria Thio, Watson Financial Services Sector Cloud Delivery and Operations Manager, IBM

4:00-5:00 pm: Student Poster Session and Happy Hour

Keynote Speaker Dr. Renetta Tull

Dr. Renetta Garrison Tull is Associate Vice Provost for Strategic Initiatives at UMBC and the Founding Director of PROMISE: Maryland's Alliance for Graduate Education and the Professoriate (AGEP).

Dr. Tull presents across the U.S. and Puerto Rico on topics ranging from graduate school recruitment, retention, and dissertation completion, to faculty development. She serves as a national coach and mentor for prospective and current graduate students at universities outside of Maryland through STEM conferences such as GEM, NSBE, SACNAS, SHPE, and AISES. She is a former Board Member of the Northeastern Association of Graduate Schools.

Dr. Tull earned the B.S. in Electrical Engineering from Howard University, and both the M.S. in Electrical Engineering and the Ph.D. in Speech Science from Northwestern University. She was an Anna Julia Cooper Postdoctoral Fellow and Assistant Professor of Communicative Disorders at the University of Wisconsin-Madison (UW), and a researcher in the both the Waisman and Trace Centers (Rehabilitation Engineering) prior to coming to UMBC. In addition to academic experience, she has been involved with entrepreneurship projects and meetings in Silicon Valley, New York, Raleigh, as well as Illinois and Maryland. She has also worked with the Washington DC Technology Council.

Dr. Tull works to increase community and professional development opportunities for graduate students in Maryland through targeted PROMISE programs that include: Professors-in-Training (PROF-it), Dissertation House, the Community Building Retreat, Fall Harvest, Research Symposium, Reflections Health and Wellness Seminars, and others. These programs, along with PROMISE's recruitment efforts and the growing recognition of Maryland's commitment to diversity at the graduate level, have contributed to increases in applic

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Metals as Mediators in the Cross-Talk Between Drug and Fungal Pathogen

Elizabeth J. White* and Katherine J. Franz

A well-established aspect of the host immune response is the manipulation of metal availability at the host-pathogen interface. While transition metals like copper and iron are essential for fungal survival, they can also be toxic due to their redox-active nature and ability to disrupt Fe-S clusters. Here, we investigate the complex interplay between widely used antifungal drug fluconazole and ubiquitous fungal pathogen Candida albicans in the context of metal homeostasis. Specifically, the effect of different metal environments on the efficacy of azole antifungals will be discussed, as well as efforts to elucidate cellular adaptations to these drugs in varying metal environments. Utilizing a unique combination of chemical and genetic approaches, this work will provide insight into how the dynamic metal environment at the site of infection may play a part in the cross-talk between drug and pathogen with ultimate implications in drug resistance and fungal pathogenesis.

Optical Imaging Reveals Metabolic Phenotypes of Breast Cancer Dormancy and Recurrence

Megan Madonna*, Douglas Fox, Brian Crouch, Jihong Lee, James Alvarez, Nimmi Ramanujam

We demonstrate metabolic changes in HER2+ tumors' life cycle. Decreased glucose uptake in recurrent vs. active cells suggests a shift in fuel source to survive oncogene inhibition. This platform can identify pathways key to recurrence and improve long-term patient outcome.

Predicting the effect of mutations in the KRas/c-Raf-RBD protein-protein interface

Anna U. Lowegard*, Marcel S. Frenkel, Bruce R. Donald

KRas is a small GTPase commonly implicated in several difficult-totreat cancers such as pancreatic ductal adenocarcinoma (PDAC). KRas normally cycles between an active, GTP-bound form and an inactive. GDP-bound form. Active, GTP-bound KRas functions by forming protein-protein interactions (PPIs) with multiple effector proteins in order to regulate various important signal transduction pathways. However, when KRas is mutated it is constitutively active which leads to signal transduction pathway dysregulation that subsequently increases and sustains tumorigenicity and invasiveness. KRas has long been considered an "undruggable" target due to its picomolar affinity for its substrate, GTP. However, blocking the PPIs between KRas and its effectors eliminates harmful downstream effects. The tightest known binder of KRas is c-Raf, an enzyme in the ERK1/2 pathway. The Ras-binding domain (RBD) is the minimal binding domain of c-Raf that selectively binds to active, GTP-bound KRas. Previous work has measured Kd for various (mostly point) mutations in the KRas/c-Raf-RBD interface [1, 2]. We use OSPREY [3] (Open Source Protein REdesign for You), a state-of-the-art software package for computational structure-based protein design (CSPD), along with K* [4], an algorithm that estimates the binding constant for a given protein complex, to computationally predict the effect of these mutations. We compared our computational predictions to the experimental measurements and found that we can accurately predict the effect of these mutations. These results validate the accuracy of CSPD with OSPREY to target protein-protein interfaces and give us confidence that we can accurately redesign the KRas/c-Raf-RBD interface for future work towards targeting "undruggable" proteins.

[1] M. Fridman, et al. Journal of Biological Chemistry, 275(39):30363–30371, 2000.

[2] C. Kiel, et al. Journal of Biological Chemistry, 284(46):31893–31902, 2009.

[3] P. Gainza, et al. Methods in enzymology, 523:87–107, 2013.

[4] I. Georgiev, et al. Journal of computational chemistry, 29(10):1527–1542, 2008.

Importance of vinculin tension in epithelial cells during collective cell migration

Evan M. Gates*, Aarti Urs, and Brenton D. Hoffman

Collective cell migration (CCM) is a fundamental biological process that plays a prominent role in both developmental events (e.g. gastrulation and neural crest migration) and various pathophysiologies (e.g. congenital heart defects and cancer metastasis). In CCM, cells are both perturbed by and exert forces on adjacent cells and the extracellular matrix through two primary structures, focal adhesions (FAs) and adherens junctions (AJs), respectively. The underlying processes which regulate these structures and subsequently enable a group of cells to migrate collectively remain poorly understood.

In the simple case of 2D sheet migration, CCM is thought to arise from a gradient of FA and AJ forces. At the leading edge, cells are believed to generate large forces that propagate into the ECM through FAs and into the cell layer through AJs. Vinculin is a potential regulator of underlying mechanical mechanisms mediating CCM as it is known to bear load and localizes to both FAs and AJs. Therefore, we hypothesize that vinculin has key roles in transmitting forces during CCM.

To study the role of vinculin tension in CCM, we expressed a previously described Forster resonance energy transfer (FRET)based vinculin tension sensor (VinTS) into two strains of the Madin-Darby Canine Kidney (MDCK) cell line. Comparing the parental and type IIG strains, we discovered significant differences in both global and spatial distributions of vinculin tension at both FAs and AJs. Examination of 2D migration speed also demonstrated significant differences between the cell strains. These results suggest that increased vinculin tension at AJs, but not FAs, is correlated with increased migration speed, but how vinculin is regulated in this context remains to be elucidated.

Bimodal decompression sickness onset times are not related to dive type or event severity

Amy E. King*, F. Gregory Murphy, Laurens E. Howle

Human decompression sickness (DCS) is a condition associated with depressurization during underwater diving. Human research dive trial data containing dive outcome (DCS, no-DCS) and symptom information are used to calibrate probabilistic DCS models. DCS symptom onset time information is visualized using occurrence density functions (ODF) which plot the DCS onset rate per unit time. For the BIG292 human dive trial data set, a primary U.S. Navy model calibration set, the ODFs are bimodal, however probabilistic models do not produce bimodal ODFs. We investigate the source of bimodality by partitioning the BIG292 data based on dive type, DCS event severity, DCS symptom type, institution, and chronology of dive trial. All but one variant of data partitioning resulted in a bimodal or ambiguously shaped ODF, indicating that ODF bimodality is not related to the dive type or the DCS event severity.

Rather, we find that the dive trial medical surveillance protocol used to determine DCS symptom onset time may have biased the reported event window. Thus, attempts to develop probabilistic DCS models that reproduce BIG292 bimodality are unlikely to result in an improvement in model performance for data outside of the calibration set.

Can massively parallel fluid simulations guide coronary stenting procedures?

Madhurima Vardhan*, John Gounley, James Chen, Andy Kahn, Jane Leopold, Amanda Randles

Coronary stent implantation remains the leading percutaneous intervention to treat coronary artery disease with an estimated 454,000 patients undergoing this procedure annually. In stent restenosis occurs in 12-15% of stents and is associated with an increase in adverse cardiovascular events and healthcare costs. This pathophysiological phenomenon arises when neointimal hyperplasia progressively obstructs the vessel lumen at the edges and/or within the body of the stent. Stents alter coronary artery geometry and increase rigidity leading to areas of perturbed (i.e., low) shear stress; low shear stress promotes neointimal formation. In this study, we are investigating the role computational fluid dynamics in surgical planning and clinical decision-making for coronary stenting procedures.

Our hypothesis is that access to patient-specific wall shear stress data derived from computational fluid simulations would influence where a clinician would place the stent and potentially reduce the likelihood of stent restenosis. Accurate 3D representations of left and right coronary arteries are reconstructed from anonymized CT data of 10 patients. Each patient CT data sets consists of approximately 2000 DICOM slices; coronary arteries were segmented on each slice using commercial software, Mimics. Reconstructed 3D coronary geometries after smoothing are validated by cardiologists.

Wall shear stress (WSS) in these anatomically validated models is calculated using HARVEY - a massively parallel computation fluid dynamics application. It is a C++ code that uses the Message Pass Interface for parallelization and has been shown to scale efficiently to 1.6 million cores of the IBM Blue Gene/Q supercomputer. To determine WSS physiological input parameters such as inlet velocity and fluid viscosity are derived from averaged patient data. The WSS results are then used in a formal quantitative user study with experts in the field of interventional cardiology across different geographical regions. The statistically significant results from the user study would demonstrate that the knowledge of low WSS regions in coronary arteries could guide stent placement. Modified stent configurations could potentially help reduce instent restenosis and thereby translate to improved surgical outcomes.

Bacterial Vesicles as Novel Plant Immune Activators: Plants Take the W in the Fight for Defense Response Hannah M McMillan* and Meta J Kuehn

Since the evolution of land plants, plants and pathogens have been locked in an epic battle for survival. Bacterial pathogens, which evolve and adapt much more quickly than their plant hosts, pose a serious threat to plant health, thereby threatening global food security. Because of this mismatch in evolutionary speed, plants have developed flexible and broad-reaching mechanisms of bacterial pathogen recognition including resistance proteins that recognize bacterial effectors and bacterial modifications of plant proteins. While many facets of plant innate immunity have been well-studied, the plant immune response to an emerging area of bacterial virulence through bacterial outer membrane vesicles has been critically overlooked. Bacterial outer membrane vesicles bud from the outer membrane of all Gram-negative bacteria and are composed of a select set of lipids, proteins, and molecules. In mammalian systems, these vesicles are known to deliver toxins and other immunogenic molecules to host cells, thus triggering host immune responses. To probe bacterial vesicle-plant interactions, I am using Arabidopsis thaliana and the model bacterial pathogen Pseudomonas syringae (Pst). My data show that Pst vesicles induce a kinetically different immune response than Pst cells. Furthermore, pre-treatment with vesicles from Pst reduce bacterial growth upon subsequent infection in a dose-dependent manner. Based on these data, I hypothesize that Pst vesicles induce and enhance plant innate immune responses to bacterial infection. This would suggest that presently plants may be winning the battle with bacterial vesicles; however, it remains to be discovered whether they will win the war. Understanding the mechanisms behind all facets of bacterial plant infection will lead to better control of agricultural pathogens and improved ability to supplement the plant immune system, thus helping plants win the war and ensuring food security worldwide.

Conversing with the Unknown: The Effects of Racial & Gender Ambiguity on Interaction Anxiety

Teresa Frasca*, Sarah Gaither, Laura Babbitt, Samuel Sommers

As evidenced by the continued discussions surrounding interracial marriage and policies such as HB2, interactions with a person of ambiguous race or gender continue to cause anxiety. Previous research shows that undoing ambiguity by providing social category labels may attenuate some of this tension. In Study 1 (N = 115), we assessed whether the anxiety experienced when interacting with a racially ambiguous individual could be mitigated by the use of a racial label that marked a common ingroup. Results showed that White participants had significantly reduced anxiety when knowing their interaction partner was half-White compared to when race remained ambiguous. In Study 2 (N = 259), the anxiety faced concerning gender ambiguity was also reduced with a gender ingroup label, but participants reported feeling warmer towards outgroup interaction partners. These effects spark needed discussion surrounding the common-ingroup identity model and potential differences between race and gender when ambiguity is present.

Targeting Drug-Resistant Bacteria with Enzyme-Activated Prochelators

Abigail Jackson*, Jacqueline Zaengle-Barone, Katherine Franz

Antibiotic resistance is a growing threat, partially due to expression of β -lactamase enzymes by bacteria. These enzymes hydrolyze antibacterial drugs in the β -lactam class to make them inactive. This work aims to take advantage of this resistance by utilizing β lactamase-activated prodrugs of an antimicrobial chelator. We report progress toward the development of these prochelators as agents to selectively kill resistant strains of bacteria by interfering with cellular homeostasis of essential metals, including copper. Results include the design, synthesis, measurement of enzymemediated chelator release, and antibacterial activity of prochelators. These results demonstrate that the antibacterial prochelator strategy is a promising method for treatment of drug-resistant bacterial infections by manipulating metals at the host-pathogen interface.

Histograming Privately Ever After

Maryam Fanaeepour* and Benjamin I. P. Rubinstein

It is recently observed that existing histogram release mechanisms under differential privacy do not provide satisfactory privacy protection. Existing work either tunes on sensitive data to optimise parameters without consideration of privacy; or selection is performed arbitrarily and independent of data, degrading utility. We address this open problem by deriving a principled tuning mechanism E2EPriv that privately optimises data-dependent error bounds. Theoretical analysis establishes privacy and utility, while extensive experimentation demonstrates that E2EPriv can practically achieve true end-to-end privacy.

Ultra-rare variants in RNH1 are associated with leukodystrophy and immune deficiency

Julie Korda Holsclaw*, Kristin Cleveland-Childs, Heidi Cope, Azita Sadeghpour, Alyssa Stephany, Philip Roehrs, Neal Sondheimer, Task Force for Neonatal Genomics, Erica E. Davis, Nicholas Katsanis

The Task Force for Neonatal Genomics (TFNG) is a collaborative effort between physicians, genetic counselors and researchers at the Center for Human Disease Modeling (CHDM) and Duke pediatric specialty clinics to accelerate the diagnosis and treatment of disorders with suspected genetic etiology in young children. Using a combined approach involving trio-based whole exome sequencing (WES) and functional studies in model systems, the TFNG seeks to identify rare causal mutations, and when possible, to uncover novel therapeutic opportunities. We enrolled a family with two affected siblings: a one-year-old female with seizures, leukodystrophy, anemia and immunodeficiency, and a deceased male sibling with a similar disease presentation with onset following a respiratory illness at age 3, suggesting the disorder might be activated by infection. Quad-based WES on both siblings and their healthy parents identified inherited compound heterozygous missense mutations in RNH1, encoding ribonuclease inhibitor. Through a public data sharing platform, we identified an additional, unrelated family with an inherited rare homozygous missense variant in RNH1. Two affected siblings presented a similar phenotypic spectrum; leukodystrophy and immune deficiency was observed in the proband and a deceased older sibling supporting further the candidacy of RNH1 deficiency in the etiology of the disorder.

The molecular pathomechanism underpinning RNH1 dysfunction remains unclear. RNH1 is a ubiquitous protein with multiple cellular and developmental functions, including inhibition of ribonucleases and regulation of the angiotensin-mediated stress response. We observed near complete loss of RNH1 in primary fibroblasts from all four affected children yet they were asymptomatic through their first year of life suggesting residual levels of RNH1 are sufficient to prevent catastrophic ribonuclease cytotoxicity under normal conditions. We hypothesize that activation of the stress response might overwhelm the limited quantity of cellular RNH1 in affected individuals, resulting in disease. To test this hypothesis, we developed a conditional Rnh1 floxed mouse. We will cross our floxed strain to Nestin-cre and CD2-cre mice to model patient-relevant phenotypes. To test if leukodystrophy is the result of RNH1 deficiency in neurons or immune cells, we will characterize myelination and oligodendrocyte formation in p25 mouse brains via stereology in challenged and unchallenged animals. To investigate the hematological and immune consequences of Rnh1 ablation, we will perform complete blood counts, cytokine analysis via ELISA and splenic immune cell population analysis using FACS. These studies will contribute to our understanding of RNH1 deficiency disorders and provide information critical to the treatment of affected individuals.

Investigation of Eotaxin Production by Lung Fibroblasts in Obese Asthma

Karen Zhao*, Dr. Jennifer Ingram, Victoria McQuade

In obese allergic asthma, increased levels of leptin and Interleukin-13 (IL-13) recruit eosinophils to lung airways, which causes inflammation of airway tissue and contributes to airway fibrosis. Several studies have observed that increased numbers of eosinophils are found in the airway tissue of obese asthma patients, indicating more inflammation. It is hypothesized that in obese patients with allergic asthma, increased airway fibrosis contributes to increased tissue eosinophilia through increased production of eotaxin, a chemoattractant for eosinophils, by lung fibroblasts. This hypothesis was studied by collecting lung tissue, blood, and BAL fluid from 3 lean wildtype mice and 3 obese leptindeficient mice. Through quantitative real-time PCR, ELISA, and immunohistochemistry for CCR3 (a receptor present in eosinophilia) and CCL11 (eotaxin), the relationship between fibroblasts and eosinophils was investigated. Cell differential analysis and airway examination through PAS, H+E, and trichrome staining were also used to determine baseline relationships.

Varying PEGDA Stiffness with Concentration and Importance of Surface Stiffness in Cell Culture

Chinar N Wakhaloo*, Jennifer L. West

Substrate stiffness is a key factor influencing cell growth. Cells sense the modulus of their environment and respond appropriately. In general, stiffer substrates promote proliferation, cell adhesion, cell spreading and decrease apoptosis, subject to native tissue modulus. Stiffness also directs differentiation of cells. One way to modulate substrate stiffness is by changing concentration of polymer used. This study uses PEGDA to make photopolymerised hydrogels of varying stiffness, since PEGDA is a non-fouling, hydrophilic substance often used as a scaffold for tissue culture.

Evaluation of Skeletal Muscle Injury Biomarkers panel on 3-D biomimetic tissue constructs

Amulya Kaza B.S*., Alastair Khodabukus PhD, Nenad Bursac PhD

Drug administration can cause a direct myotoxic effect, leading to primary structural or functional impairment i.e. skeletal muscle (SKM) injury or Myopathy. Statin cerivastatin, was withdrawn from the market due to reported severe musculoskeletal toxicity. rhabdomyolysis. A diagnostic method that can safely & sensitively detect drug induced skeletal muscle injury is critical for pre-clinical safety assessment & monitoring of myotoxicities of therapeutic targets in human subjects during clinical use. Novel accessible biomarkers are required to supplement the routinely used creatine kinase, aspartate transaminase and lactate dehydrogenase for diagnosis of drug-induced myopathy as they lack SKM tissue specificity, sufficient sensitivity to SKM degeneration/necrosis and does not distinguish between fast-twitch and slow-twitch myofibers. A collaborative study conducted by Pfizer and Predictive Safety Testing Consortium, showed their novel muscle injury biomarker panel (MIP) outperformed routine biomarkers across 34 rat in vivo toxicological studies. The contractile SKM tissues of primary human myogenin cells engineered at Bursac Lab are electrically and chemically responsive, and are a potential platform as 3-D biomimetic tissue constructs to conduct correlation of MIP biomarker response with progression/regression of SKM injury or myopathy. The objective is to perform a dose response and time course studies of five clinically relevant SKM toxicants and nontoxicants on 3-D biomimetic tissue constructs and correlate MIP biomarker response to functional (contractile force) & structural (histopathology) measurements.

Computational Analysis of Randomized Secondary Structure RNA Libraries

Rachel K. Dveirin*, Sarah L. Wicks, B. S. Morgan, Amanda E. Hargrove

RNA is implicated in important biological processes and often misregulated in various diseases. To study disease-driving RNAs, small molecule targeting of RNA secondary structures have been proven to be a viable method. To date, secondary structure binding preferences have been primarily identified for non-selective small molecules, and these small molecules have been screened against a limited subset of secondary structures. We herein describe a computational analysis of randomized secondary structure RNA libraries, which will expand the diversity of RNA motifs screened, provide a global evaluation of small molecule binding preferences, and identify unique RNA:small molecule interactions. To achieve these goals, several scripts were developed to rapidly: 1) generate all possible sequences for each library; 2) predict an ensemble of secondary structures for each sequence; and 3) calculate the percentage of occurrence for each structure. These scripts were used to evaluate three literature-based randomized libraries, which revealed several patterns that led to improper folding. Current work is focused on designing an in-house cassette to maximize the number of sequences that represent the secondary structure of interest. Once the cassette is optimized, randomized RNA libraries will be synthesized, sequenced to assess bias, and used to identify secondary structure selective small molecules. These small molecule probes will lead to rational approaches to study the role of RNA in various diseases and aid in the discovery of therapeutic leads.

Fluorescent Indicator Displacement Assay to Identify and Characterize RNA:Small Molecule Interactions

Sarah L. Wicks*, Brittany S. Morgan, Jordan E. Forte, N. N. Patwardhan, Amanda E. Hargrove

Non-coding RNAs (ncRNAs) are a novel class of biomolecules implicated in important biological processes and are often misregulated in various diseases. To study disease-driving ncRNAs, small molecule chemical probes have been proven to be a viable avenue. Commonly, fluorescence-based labeling and immobilization techniques are used to study RNA:small molecule interactions, which hinders the types of RNAs and small molecules that can be screened. In order to fundamentally understand RNA:small molecule recognition, we propose to utilize highthroughput screening assays that can be used with a variety of small molecules and RNAs without the need for ligand or target modification. We herein describe a TO-PRO-1 displacement assay as an appropriate screening tool along with current efforts to identify and characterize RNA:small molecule interactions. To investigate several assay parameters, the HIV-1 Transactivation Response Element (TAR) was used as a model system. Using TAR, the dissociation constant of TO-PRO-1, signal-to-background ratio, and effective RNA concentration for displacement were assessed. To confirm these optimized conditions, small molecules that bind TAR were used to evaluate displacement of TO-PRO-1 and binding curves were obtained. Currently, the TO-PRO-1 displacement assay is being utilized with biologically relevant RNAs to screen diverse, RNA-biased small molecules and reveal structure-activity relationships. Derivatives of a lead RNA-privileged amiloride-based small molecule with moderate affinity for TAR have been compared to identify substituents that enhance binding. In addition, commercially available small molecules that encompass RNA-privileged chemical space are being selected and screened to identify new RNA-binding scaffolds and leads for therapeutically-relevant RNAs. Concomitantly, the physicochemical properties of small molecules are being analyzed and compared to elucidate guiding principles that bias recognition. At the conclusion of this work, hundreds of RNA:small molecule interactions will be assessed and the physicochemical properties that lead to specific interactions will be determined. Furthermore, by expanding the diversity of RNA targets, small molecules, and methods used to identify and analyze these interactions, rational approaches for targeting RNA will be developed. Such approaches can result in selective small molecule probes to study the role of RNA in disease and aid in the discovery of therapeutic leads.

Continuous Focal Plane Translation Enhances Rate of Point-Scan Volumetric Imaging

Courtney Johnson* and Kevin Welsher

Traditional Point-Scanned Microscopes capable of optical sectioning perform 3D imaging by sequentially scanning a volume frame-by-frame. To improve the rate of this slow process, we introduce a method to improve the rate of point-scanned volume imaging microscopes through continuous focal translation.

Change in Stress Levels During Adolescence

Ceren Ebrem*, Nourhan Elsayed, Dougles E. Williamson

Exposure to stress during adolescence increases risk for anxiety and depression in adulthood (Blaustein & Holder, 2013). Little is known about how exposure to different domains of stress (e.g., education, work, money) changes over the course of adolescence, or how demographic characteristics differentially subject individuals to stressful events. In this study, 331 adolescents (168 females) were assessed annually for 6 years from the approximate age of 13 at interview 1 (I1) (Range = 11.61-15.84, M = 13.48, SD = 0.96) to the approximate age of 16 at interview 6 (I6) (Range = 11.69 -20.04, M = 16.56, SD = 1.70). Stress levels were measured using the adolescent version of the interview-based Stressful Life Events Schedule (SLES). In this study, we describe changes in stress levels across adolescence, as well as differences due to race and gender. In this cohort, we find that the four most prominent stressors at I1 were education (M =2.57, SD = 3.04), health (M = 3.11, SD = 4.35), death (M = 2.08, SD = 3.76), and other relationships (M = 3.14, SD = 4.93). In contrast, at study cessation these were education (M = 2.0, SD = 3.51), health (M = 2.17, SD =3.90), romantic relationships (M = 1.76, SD = 2.98), and other relationships (M = 1.82, SD = 4.03). We report that females are more stressed in domains of money (F (1, 1271) = 6.576, p < .05), housing (F (1, 1271) = 8.850, p < .01) and health (F (1, 1271) =27.406, p < .001), and non-romantic relationships (F (1,1271) = 28.638, p < .001) in comparison to their male counterparts. Racial groups differ in stress about health (p < .05) and death (p < .01). These findings support the need to further understand the development of stress in the context of race and gender.

Keywords: stress, adolescence, gender, race, longitudinal study

Duke Graduate School Department of Biochemistry Department of Cell Biology Department of Civil and Environmental Engineering Department of Computer Science Department of Electrical and Computer Engineering **Department of Mathematics** Department of Mechanical Engineering and Materials Science Department of Molecular Genetics and Microbiology Department of Pathology Department of Pharmacology and Cancer Biology **Department of Physics Department of Statistical Science** Graduate Program in Neurobiology University Program in Genetics and Genomics