



# Undergraduate Research Symposium May 17, 2019 Mary Gates Hall

## Online Proceedings

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### POSTER SESSION 1

MGH 258, Easel 185

11:00 AM to 1:00 PM

#### **Uncovering Protein Interactions for Learning and Memory Using a Novel Synaptic TAG**

*Karen Brittney Immendorf, Senior, Biology (Molecular, Cellular & Developmental)*

*Mentor: Stephen Smith, Pediatrics*

*Mentor: Whitney Heavner, Center for Integrative Brain Research*

Memory formation, or plasticity, is the brain's ability to change and adapt in response to new information through altering the strength and effectiveness of communication at the synapse. Synaptic plasticity plays a major role in the brain's capacity to incorporate past experiences into stored memories and it has become one of the most intensively researched subjects in neuroscience. Neuroscientists typically evaluate synapse function using enriched, homogenized preparations of synapses, called synaptosomes. However, synaptosome preparations contain a mix of neuronal and non-neuronal contamination along with active and inactive synapses that are impossible to separate, making it difficult to draw specific conclusions about synapse activity. To address these challenges and understand the molecular mechanisms behind synaptic plasticity, my lab has developed a synaptic TAG- an engineered protein that localizes to recently active synapses. The TAG construct, containing an extracellular CD4 protein whose expression is driven by activity-dependent regulatory elements, will be electroporated into the developing motor cortex of mice in utero. The mice will later be trained to run on a rotarod, a test commonly used to assess motor learning. After the mice have learned this task, the differences in their synapses will be evaluated by sectioning and staining the motor cortex with fluorescent antibodies. The number of distinct fluorescent TAGs on the synapses on a given number of neurons will be counted through confocal imaging. My hypothesis is that I will see an increase in the number of TAGs on the synapses of a trained mice, which will demonstrate that the TAG is synaptic in vivo and activity dependent. The completion of this experiment will allow my lab to identify learning-associated proteins using magnetic cell sorting, mass spectrometry, and RNA sequencing. Understanding learning-associated protein interactions will shed light on how their malfunctions contribute to Autism Spec-

trum Disorders or neuropsychiatric disorders.

### POSTER SESSION 1

MGH 258, Easel 184

11:00 AM to 1:00 PM

#### **Behavioral Rescue of Transgenic Ube3a Autism Model Mice with Rapamycin**

*Ryan Mendel, Senior, Biochemistry, Public Health-Global Health*

*UW Honors Program*

*Mentor: Stephen Smith, Pediatrics*

Autism spectrum disorder (ASD) are a group of neurodevelopmental disorders characterized by impairments in social interaction and repetitive behaviors. One of the most common mutations leading to ASD is 15q11-q13 duplication, a CNV mutation where extra copies of a chromosomal region are expressed. The major gene within this region is ube3a. My lab has generated a mice model with increased copies of this gene and demonstrated a novel deficit in the mTOR/AKT signaling pathway. Other mice models of autism have exhibited deficits in this pathway and have rescued behavioral deficits with a drug, rapamycin. Rapamycin is an inhibitor of a key protein of the mTOR/AKT pathway suggesting a similar behavioral rescue could be observed with ube3a transgenic autism model mice. Rapamycin is administered to ube3a transgenic mice to normalize deficits in social interaction and repetitive behavior, two main behavioral hallmarks of autism. Mice are assessed using a three-chamber social interaction and repetitive self-grooming test. Successful behavioral rescue by rapamycin would be seen as increased social interaction and decreased repetitive grooming, similar to behaviors seen in wild type control mice. This would suggest that therapeutic treatment of the mTOR/AKT signaling pathway could be a viable target for patients with 15q11-q13 duplication.

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## SESSION 1J

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### UNDERSTANDING OUR WORLD: DATA-BASED APPROACHES

*Session Moderator: Walter Andrews, Near Eastern  
Languages and Civilization*

**MGH 251**

*12:30 PM to 2:15 PM*

\* Note: Titles in order of presentation.

#### **Torn In Transition: the Decolonization of New Caledonia**

*Ethan Thomas Walkley, Senior, French, Human Centered  
Design & Engineering*

*UW Honors Program*

*Mentor: Maya Smith, French and Italian Studies*

New Caledonia is a French collectivity in the South Pacific that transitioned from colony to territory in the 1940's, like many other former French colonial holdings. However, unlike other overseas territories of France, New Caledonia has been on an unofficial transition towards decolonization that began in 1998 with the Nouméa accords, stipulating a vote to take place in twenty years that would decide whether or not the islands would remain French. Despite a slim loss for independence when this long-awaited vote took place last year in November 2018, future referendums to challenge this decision are on the horizon. The objective of this research is thus to understand why the situation in New Caledonia is so different from that of other French territories. In particular, how do contemporary issues in the archipelago and the question of race complicate the decolonization process? To effectively answer this question, this study will analyze academic literature comprising subjects on foundational postcolonial theory, New Caledonian history, and race in the French-controlled South Pacific. Peculiarities of New Caledonia include its relatively late colonization compared to other French territories and the fact that the native society residing on the island continues to hold a strong presence. These factors along with a turbulent past may explain in part why New Caledonia finds itself in a slow process of decolonization. However, rich mineral reserves and the rise of neocolonialism may also complicate this event. Ultimately, this research hopes to bring a better understanding of New Caledonia to a wider public while serving as a foundation for continuing conversations around neocolonialism and efforts to combat it in the French territories and elsewhere.

### POSTER SESSION 2

**MGH 241, Easel 138**

*1:00 PM to 2:30 PM*

#### **Phased Array Wireless Power Optimization on a Planar Array of Coupled Resonators**

*Usman M. (Usman) Khan, Sophomore, Electrical  
Engineering*

*Mentor: Joshua Smith, Computer Science & Engineering,  
Electrical Engineering*

Wireless power transfer has many applications, from powering biomedical implants to wireless sensors. For more practical use, however, several challenges must be overcome, such as a lack of efficiency and power leakage to other nearby electronics. These issues become especially difficult to tackle with a moving receiver. To combat these problems, an array of magnetically coupled coils was designed. Previous work has shown the capabilities of this system when one coil of the array is supplied with power. In this work, I explore the possible benefits of having two coils in the array driven with power instead, studying the interaction between the different coils. By adjusting parameters such as the phase relationship between the two transmitters' signals, we aim to optimize power delivery to specific targets and simultaneously minimize leakage to other areas. I tested different configurations of the system in a series of experiments and analyzed measured data to determine which setup is most favorable. Afterwards, I evaluated the efficiency of the configuration compared to the previous single-transmitter case. This provides better insight into how the coils in the array magnetically interact with one another, which will inform future design decisions. It can also eventually lead to better solutions for delivering high power to selected targets within a given space. This can create flexible, efficient, and safe wirelessly charged electronic implants for a variety of biomedical applications, enabling further research in the field and the development of novel solutions.

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## SESSION 2C

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### ASSESSING THE SOURCES: WOMEN, IDENTITY, AND PRACTICES OF EMPIRE

*Session Moderator: Mira Green, History*

**MGH 231**

*3:30 PM to 5:15 PM*

\* Note: Titles in order of presentation.

#### **Cultural Amnesia: Decolonization of Indochina and the Vietnamese Diaspora**

*Kimberly Meilin Yee, Junior, French*

*Mentor: Maya Smith, French and Italian Studies*

Twenty-seven years ago, the movie *Indochina* debuted and brought up interesting questions about the direction and future of France's colonies. Almost three decades later, the continuing presence of France's colonies and influx of immi-

grants to France from countries known formerly as Indochina also raise questions about the effects of colonialism, especially pertaining to immigrant identities both past and present. This essay examines the lasting legacy of French colonization on the Vietnamese diaspora through both a literary analysis of Linda Lê's novel *Les Trois Parques*, which offers an observed history of Vietnamese restaurant workers and scholars and their agency regarding the fight against colonization, as well as a sociological exploration of a study conducted on the success of international students' integration into French universities. Additionally, the paper seeks to illustrate the significance of cultural amnesia—the assimilation into a new culture by rejecting one's former identity—as well as the benefits and motivations that may have guided first-generation immigrants to do so instead of rebelling and continuing their cultural traditions.

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## SESSION 2E

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### ANIMAL RESPONSES TO THEIR ENVIRONMENT

*Session Moderator: Jay Parrish, Biology*  
**MGH 238**

3:30 PM to 5:15 PM

\* Note: Titles in order of presentation.

#### **Effects of Early Life Adversity in Gelada Monkeys**

*Lia Koklic, Senior; French, Biology (General)*

*Mary Gates Scholar, UW Honors Program*

*Mentor: Noah Snyder-Mackler*

Exposure to traumatic events during infancy can lead to adverse health effects later in life, stemming from an imbalance between the innate and acquired arms of the immune system. Trauma experienced at an early age, such as childhood abuse or environmental stress, is categorized as early life adversity. As the immune consequences of early life adversity are unclear, our study explores how early life adversity affects immunological development and parasite susceptibility in gelada monkeys (*Theropithecus gelada*). Geladas are an ideal species to study early life adversity due to the similarity of their immune response to that of humans and the fact that they may face adversity early in life. Male geladas compete for reproductive access to females, which often leads to attempted infanticide. Surviving juveniles experience trauma that may have similar physiological consequences to early life adversity in humans. Thus, we determine if there are differences in the immune development of the acquired immune system between geladas exposed to takeovers as infants and those who were not. Acquired immune system development is measured by the identification of gastrointestinal (GI) parasites species that commonly infect geladas, as these parasites trigger the adaptive immune response. We identify these GI

parasites using high throughput methods to sequence a region of DNA with known variation across nematode species, allowing us to identify parasites at the level of genus. We expect to see greater GI parasite species diversity in geladas with underdeveloped acquired immune systems as we continue to identify GI parasite species in geladas that were exposed to early life adversity and those who were not.

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## SESSION 2F

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### ADENOVIRUSES AND MALARIA VACCINE

*Session Moderator: James Mullins, Microbiology*  
**MGH 242**

3:30 PM to 5:15 PM

\* Note: Titles in order of presentation.

#### **Mechanisms of Defensin-Mediated Enhancement of Adenovirus Infection**

*Danielle Williams, Non-Matriculated, Biology, University of Washington*

*UW Post-Baccalaureate Research Education Program*

*Mentor: Jason Smith, Microbiology*

Human alpha defensins, a component of the innate immune system, are small cationic peptides that possess antiviral activity against non-enveloped viruses. The effect of defensins on human adenoviruses (HAdV) is serotype-dependent, infection by some serotypes is enhanced while for others it is neutralized. Enhanced infection correlates with increased cell binding; however, the mechanism of increased binding is unclear. One hypothesis is that defensins mediate receptor-independent binding. Inhibitor studies support this hypothesis, although formal proof is still needed. To test this hypothesis, we used CRISPR/Cas9 lentivirus to knockout the primary receptor, coxsackie adenovirus receptor (CAR), in A549 lung cells. In order to vet these cell lines, they were infected with different HAdV serotypes that use either CAR or an unrelated molecule, sialic acid, as their primary receptors. As expected, the sialic acid-utilizing but not the CAR-utilizing serotype was able to infect the CAR KO A549 cells. We have used these cell lines in combination with integrin coreceptor inhibitors to measure binding and infection of wild-type and mutant adenoviruses in the presence and absence of defensins. These experiments allowed us to determine the extent to which defensin-mediated attachment and entry is receptor-independent.

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## SESSION 2F

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### ADENOVIRUSES AND MALARIA VACCINE

Session Moderator: James Mullins, Microbiology  
MGH 242

3:30 PM to 5:15 PM

\* Note: Titles in order of presentation.

#### Identifying the Major Determinants of Mouse Adenovirus Tropism

Yasmine Arbob, Senior, Biology (Molecular, Cellular & Developmental), Microbiology  
Mentor: Jason Smith, Microbiology

Mouse adenoviruses (MAdVs) are non-enveloped double stranded DNA viruses. There are two different types of MAdVs with different tropisms or ability to infect particular cells or tissues. MAdV-1 infects macrophages and endothelial cells and causes encephalitis. MAdV-2 infects intestinal cells but causes no disease. Although, the MAdV fiber capsid proteins are important for attachment of the virus to host cells, it is not known if they are the major determinant of tissue tropism in the mouse. To address this question, I use recombination-mediated genetic engineering to make chimeric MAdVs, wherein I keep most of the genome of one strain but replace the fiber protein with that of the other strain. I then use transfection to introduce the DNA of the chimeric virus into a mouse cell line to allow the virus to replicate. I am currently designing and testing the proper chimeric fiber construct that will result in a replication-competent virus. Ultimately, I compare infection of the chimeric virus to that of the parent viruses in intestinal organoids, a tissue culture model that allows us to faithfully test tropism without the need for mouse studies. These experiments may reveal general principles of AdV tropism that will allow us to understand why different human AdVs cause disease in specific organs.

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## SESSION 2F

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### ADENOVIRUSES AND MALARIA VACCINE

Session Moderator: James Mullins, Microbiology  
MGH 242

3:30 PM to 5:15 PM

\* Note: Titles in order of presentation.

#### Receptor Usage Does Not Determine the Tissue Tropism of Mouse Adenovirus

Veronica Carruthers, Senior, Microbiology  
Mentor: Jason Smith, Microbiology  
Mentor: Karina Diaz

Mouse adenoviruses (MAdV), like human adenoviruses (HAdVs), have specific tissue tropisms. MAdV-1 infects macrophages and vascular endothelial cells, which can result in encephalitis, while MAdV-2 infects epithelial cells of the intestine but does not cause overt disease. The viral protein that determines MAdV tropism is unknown; however, for many viral families it is the viral attachment protein that is critical. For MAdVs, fiber is the viral attachment protein, and the receptors used by MAdV-1 and MAdV-2, although unknown, are distinct. To test whether MAdV receptor usage dictates tissue tropism, I constructed a MAdV-2 chimeric virus, replacing its fiber protein with that of MAdV-1 using a gene-editing recombination system. The chimera was used to infect a 3D culture model of the intestinal epithelium called "enteroids." As expected, MAdV-1 does not replicate in enteroids and MAdV-2 does, consistent with their *in vivo* tropisms. Remarkably, the chimera replicated efficiently, indicating that the fiber protein is not the sole determinant of MAdV-2 intestinal tropism. Although fiber is not the main contributor to tropism, its interactions with host factors are still likely important for productive infection. A recent study identified N-acetylglucosamine (GlcNAc) as a specific ligand for MAdV-2 fiber. We have shown that GlcNAc is not the primary receptor for MAdV-2; however, binding to GlcNAc may aid in adhesion of MAdV-2 and penetration through the mouse intestinal mucus layer. To test this hypothesis, I mutated the GlcNAc interacting residues in MAdV-2 fiber to prevent GlcNAc binding. I am currently comparing the infectivity of this mutant virus to wild type MAdV-2 in both epithelial tumor cells and enteroids. Unlike tumor cell cultures, enteroids contain mucus-secreting goblet cells which will recreate the *in vivo* context more accurately. Together, these studies of MAdV may help us to understand why different HAdVs infect specific tissues.

## POSTER SESSION 3

MGH 241, Easel 155

2:30 PM to 4:00 PM

#### Analyzing the Effects of Arhgap29 and Arhgap35 on Zebrafish Embryonic Morphogenesis and Mesodermal Cell Migration in Posterior Body Elongation

Charlotte An, Senior, Biochemistry, Applied & Computational Mathematical Sciences (Biological & Life Sciences)

UW Honors Program

Mentor: David Kimelman, Liberal Arts

Mentor: Natalie Smith, Biochemistry

Studying zebrafish embryos allows us to understand features of vertebrate embryonic development. Neuro-mesodermal progenitor cells at the very posterior end, or tailbud, of an embryo are bipotential. This is because the presence or absence of Wnt signaling commits them to either neural or mesodermal fate. Directed by environmental cues, mesodermal cells exit the tailbud, migrate anteriorward, and become somites, structural segments from which muscles differentiate. The Kimelman lab has found that Tbx16/Spadetail, a major driver of mesodermal morphogenesis, downregulates Arhgap29 and Arhgap35, members of Rho family GTPase activating proteins. This suggests Arhgap29 and Arhgap35 may be involved in mesodermal cell movement. My work in the lab is focused on finding out what roles these two genes play. I used heat shock promoter *hsp70* to overexpress Arhgap29 and Arhgap35 in transgenic fish lines. Previously, our lab showed that sustained Arhgap35 affected somite morphology, and that sustained Arhgap29 also decreased the number of somites. In my experiments, I carried out in-situ hybridization in wild-type, Arhgap29- and Arhgap35-expressing embryos to examine genes regulating specification/differentiation of muscle cells and genes involved in transmembrane cell adhesion. I will present data on cell tracking and cell protrusions collected from Arhgap29- and Arhgap35-expressing embryos. These results will help me compare cell migration between Arhgap-expressing and wild-type embryos. The purpose of these analyses is to understand how these two proteins control cell movement in the embryo. In the future, I will continue to investigate cellular mechanisms underlying vertebrate posterior elongation.

### POSTER SESSION 3

MGH 241, Easel 140

2:30 PM to 4:00 PM

#### Linguistic Knowledge and Transferability of Contextual Word Representations

*Nelson Liu, Senior, Linguistics, Computer Science  
Goldwater Scholar, Mary Gates Scholar, UW Honors Program, Undergraduate Research Conference Travel Awardee, Washington Research Foundation Fellow  
Mentor: Noah Smith, Computer Science & Engineering*

Contextual word representations derived from large-scale neural language models are successful across a diverse set of natural language processing (NLP) tasks, suggesting that they encode useful and transferable features of language. To shed light on the linguistic knowledge they capture, we study the representations produced by several recent pretrained contextualizers (variants of ELMo, the OpenAI transformer LM, and BERT) with a suite of sixteen diverse probing tasks. We find that linear models trained on top of frozen contextual representations are competitive with state-of-the-art task-specific models in many cases, but fail on tasks requiring fine-grained

linguistic knowledge (e.g., conjunct identification). To investigate the transferability of contextual word representations, we quantify differences in the transferability of individual layers within contextualizers, especially between recurrent neural networks (RNNs) and transformers. For instance, higher layers of RNNs are more task-specific, while transformer layers do not exhibit the same monotonic trend. In addition, to better understand what makes contextual word representations transferable, we compare language model pretraining with eleven supervised pretraining tasks. For any given task, pretraining on a closely related task yields better performance than language model pretraining (which is better on average) when the pretraining dataset is fixed. However, language model pretraining on *more data* gives the best results.

### POSTER SESSION 3

Commons West, Easel 31

2:30 PM to 4:00 PM

#### Diet and Gene Regulation in Arterial and Fat Tissues

*Matthew R Harrington, Senior, Biology (General)*

*UW Honors Program*

*Mentor: Noah Snyder-Mackler*

Diet influences mammalian physiology and has been linked to obesity and the development of carotid artery disease. Carotid artery disease (CAD) is a result of plaque buildup in arteries leading to the brain and other parts of the head, reducing blood flow and increasing the risk of stroke. Rates of obesity and carotid artery disease are higher in countries with Western diets (higher saturated fat and carbohydrate composition) compared to those with Mediterranean diets (higher produce and monounsaturated fat composition). Although there is evidence demonstrating the effect of diet on overall health and physiology, the mechanisms by which these changes arise could be better understood. These health consequences of diet are thought to be mediated by changes in gene expression caused by eating different diets. Understanding how diet influences gene regulation in different tissue types could give critical insight into our understanding of diet's effect on physiology. To this end, I measured gene expression via RNA-Seq in 37 cynomolgus macaques (*Macaca fascicularis*) that were randomly assigned either a Western or Mediterranean diet. By analyzing gene expression in samples collected from two types of fat (subcutaneous and visceral) as well as two arterial tissue types (iliac and carotid), I aim to gain insight into the transcriptomic changes due to diet. Further, by understanding diet's role in gene regulation, I aim to construct a clearer picture of the mechanisms through which western diet might heighten the risk of obesity and the development of CAD. I predict that the differentially expressed genes in fat tissues will be associated with obesity and that the differentially expressed genes in the arterial tissues will

be associated with plaque build up and CAD. This study will illuminate how diet mediates gene regulation in a variety of tissues, providing a mechanistic link to diet-related diseases such as obesity and CAD.