Welcome to the BMS Undergraduate Research portion of our Meet the Faculty Night!

We started this tradition in order to talk with interested students about the opportunities in undergraduate research.

Why does undergraduate research matter?

- Work with faculty/mentors one-on-one. Learn how to work in a professional capacity with someone who wants to teach you their craft.
- Apply what you know. In a lab, you will apply classroom knowledge in a way that is focused on a singular problem.
- Make discoveries! You can develop and design your own experiments and analysis.
- Find out if you like research. Maybe you’ll love it, maybe you won’t. Either way, your mentors understand that this educational process is important.

How do I start?

- Investigate. Check out the list of possible topics; peruse the posters and listen to the talks. Check out links to other opportunities at GVSU and outside of GVSU.
- Talk. Ask the professors about their research. Tell them what interests you. It’s OK if you decide you don’t like that particular research topic. Just start the discussion. We know you are exploring and we would love to help.
- Connect. Get the necessary contact information and consult your class schedule to see if you can commit to dedicating time in a lab. Take that first step forward by asking the professor or mentor to see if there are any opportunities, or ask if they have recommendations for other openings.
- Commit. Expect that this will take some time and effort and will probably extend over the summer. It’s work, but it’s worth it!
Here are a few topics:

- Nutrition and Public Health (Nochera)
- Infectious Disease Epidemiology, Viruses and Informatics (Cleary, Graham, Thomas)
- Parasitology (Graham)
- Viral, Fungal, and Bacterial Physiology (Baxter, Cleary, Haley, Thomas)
- Developmental Biology (M. Burg, Delano-Taylor, Ramsson)
- Cardiovascular Physiology (Liu, Kurjiaka, Sylvester)
- Intracellular Signaling (Baxter, M. Burg, Capodilupo, Delano-Taylor, Fateye, Kurjiaka, Pearl, Ramsson, Sylvester)
- Immunology (D. Burg, Renkema)
- Physiological Biochemistry (Kipp, Ramsson)
- Neurobiology (Bergman, M. Burg, Capodilupo, Delano-Taylor, Ramsson, Shabani)
- Neurophysiology and Pharmacology (Bergman, Linn, Shabani)
- Neurophysiology and Behavior (Bergman, M. Burg, Ramsson)
- Muscle Physiology (Kurjiaka, Sylvester, Tows)
- Ancient Human Anatomy (Kegley, Laudicina, Tallman)
- Functional Anatomy and Bone Biology (Kegley, Laudicina, Reed, Stroik, Tallman)
- Feeding Ecology (Stroik, Thompson)
- Thermoregulation (Thompson)
- Reproductive Physiology (Pearl)
- Endocrine Toxicology (Fateye)

Resources for Finding Research Opportunities and Research Support

Grand Valley State University
Biomedical Sciences Website Faculty Research Interests: http://www.gvsu.edu/bms/

Office of Undergraduate Research and Scholarship (OURS): http://www.gvsu.edu/ours/

The Student Summer Scholars Program (S3 and MS3): This program provides funds for a student and faculty mentor to devote up to twelve weeks to a research and/or creative project during the spring/summer semester. Generally, S3 Grants provide a student stipend, faculty stipend, and a small budget for supplies.

OURS Project Supplies Grant: This OURS grant program is designed to encourage collaborative scholarly research and creative work between undergraduate students and faculty. These grants provide students with financial support of up to $500 for supplies and equipment.

Academic Conference Fund (ACF): The ACF is available to students to present at an academic conference related to their professional goals. Support is up to $500 for domestic travel, $750 for international travel.

Academic and Professional Enrichment Fund (APEF): APEF is available to all students to attend (presentation not required) an academic conference that is related to their professional goals, or to engage in a professional experience. Support is up to $400.

McNair Scholars Program: www.gvsu.edu/mcnair

The McNair Scholars Program is designed to help academically talented students from traditionally underserved backgrounds reach their potential by earning a doctoral degree. We work closely to help students navigate their undergraduate career through academic counseling, financial aid assistance, mentoring, summer research opportunities, seminars, tutoring, and more.
Frederik Meijer Office of Fellowships:
http://www.gvsu.edu/fellowships/

The purpose of this office is to advise and support students and alumni to achieve the extraordinary by matching their dreams to prestigious fellowship and scholarship awards and other opportunities. The website has an extensive list of non-GVSU scholarship and fellowship opportunities.

They have a “fellowship finder” which is very useful as well.
http://www.gvsu.edu/fellowships/award-opportunities-55.htm#Awards

CLAS Advising:
http://www.gvsu.edu/clasadvising/

Have you talked about your potential career plans with an advisor yet?

Outside Grand Valley State University:
Van Andel Research Institute
http://www.vai.org/research/trainingprograms/studentinternship.aspx

National Science Foundation Research Experience for Undergraduates
http://www.nsf.gov/crssprgm/reu/reu_search.cfm

American Society for Microbiology Summer Research Fellowship

University of Michigan Pipeline/Profiles for Success, Experience for minority pre-dental students
http://www.dent.umich.edu/research/training.html

Wayne State University SURE program in biomedical science
http://www.gradprograms.med.wayne.edu/sure.php

Perrigo Undergraduate Research Fellowship
http://www.lsi.umich.edu/facultyresearch/initiatives/perrigo

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My research revolves around understanding the pathogenic mechanisms of *Salmonella* and *Escherichia coli*. Currently, I am focused on two areas of interest. My first project involves looking for additional genes necessary for *Salmonella* pathogenesis. Studies have shown that the genes involved in *Salmonella* pathogenesis are often found in specific areas known as pathogenicity islands. *Salmonella* Pathogenicity Island 1 (SPI-1) contains numerous genes involved in the formation of the type III secretion system and other secreted effector proteins. Activation of this island allows for bacterial invasion of intestinal cells. A second critical island (SPI-2) is required for survival within macrophage after invasion. Due to the number of genes required for these processes, I have focused on the regulatory genes that control activation and repression of these islands in response to environmental signals. In the course of these studies I identified a repressor known as *hilE*, which represses the activation of SPI-1. Studies of the sequences around *hilE* suggest that this repressor falls in a 40 kb region of the chromosome that has all of the hallmarks of a pathogenicity island, yet very little is known about the function of the genes around *hilE*. Since its identification, we have created ten different polar mutations in open reading frames within this potential pathogenicity island. Work has commenced trying to analyze the effects these mutations have on *Salmonella* virulence by utilizing gene reporter, cell invasion, macrophage survival, bacterial adherence, *C. elegans* survival model, and cell motility assays under various inducing and noninducing environmental conditions. Any effects on *Salmonella* invasion could then be further characterized by identifying how each of the mutations leads to changes in *Salmonella* invasion in response to an environmental signal.

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My second project is looking for genes important for *Escherichia coli* biofilm formation under conditions that mirror its natural environment. Previous work has identified many genes needed for the activation and formation of a biofilm when the bacteria are grown under aerobic conditions. As *E. coli* is commonly found in the anaerobic conditions of the colon we are trying to identify regulator genes that are responsible for increasing or decreasing biofilm formation in response to oxygen. We have developed a biofilm assay to screen for biofilm formation that can be used under aerobic, microaerophilic and anaerobic conditions. A library of nonpolar mutants has been acquired in which we can screen them for their effects on biofilm formation in response to varying oxygen levels.

*Salmonella typhimurium.*

Image: Volker Brinkmann, Max Plank Institute for Infection Biology, Berlin.

Professor Bergman’s research lab is a multidisciplinary lab that works in the disciplines of neuroscience, physiology, ethology, ecology, toxicology, histology, and pharmacology. Much of the research in the lab is accomplished using crayfish. Using crayfish for biomedical research may not seem immediately applicable when considering human health, but basic biomedical research it turns out is largely about understanding organisms and their interactions with other organisms. Humans as you know are extraordinarily complex on many levels, yet we only understand a small fraction of the interactions, structures, chemicals, and pathways in our bodies. Therefore, the best way to determine the effect of a drug or disease on a living system is to study it first in an animal system. Drugs, vaccines and treatments in human medicine are largely based on years of physiological research with animals. To that end, the crayfish lab studies sensory system physiology, neurochemical modulation of aggression, neurogenesis via social enrichment, operant conditioning/learning, pollution effects on sensory receptors and development, nociception, growth/molting, orientation strategies when finding food or mates, the interactions of various invasive crayfish species, and feeding behaviors. A student joining this lab can expect to become knowledgeable in the scientific fields of neuroscience, animal behavior, physiology, biomechanics, toxicology, ecology, chemistry, and molecular biology.

Two blindfolded crayfish chemically communicating using urine that has a fluorescent dye added for visualization.
My area of expertise encompasses immunology, protein biochemistry and tyrosine kinase-mediated signal transduction. I am not taking any new research students at this time, but I am available for BMS399 (independent study) and HNR499 (honors senior project) projects that are related to my expertise.

My lab’s research focus is to identify processes that may be affected by the neurotransmitter histamine in the fruit fly *Drosophila melanogaster*. One gene that is required for synthesis of histamine encodes the enzyme, Histidine decarboxylase (*Hdc*). We are currently examining the role of the *Hdc* gene in establishing when and where the neurotransmitter substance, histamine, is synthesized. We are also continuing to examine what the function of histamine may be with regards to central brain function and its consequences on courtship behavior as well as effects of histamine elsewhere in the fly. As a result, projects in my lab range from molecular biology projects to behavioral neuroscience projects.

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We are examining a molecule called GAP-43 which is a brain protein that is expressed in a wide variety of species including humans and has been shown to become biochemically altered in the process of learning and memory. Specifically, levels of phosphorylated forms of GAP-43 have been shown to increase following a controversial paradigm of learning and memory in several animals including rat, mouse, and rabbit. We are interested to see if any differences in the profile of GAP-43 are associated with dementing illnesses that severely disrupt memory and learning. Since human brain tissue is difficult to obtain, we utilize brain tissue from a genetically altered mouse engineered to resemble Alzheimer’s disease, a human neurodegenerative disorder characterized by profound cognitive impairment. Therefore, to test the hypothesis that the profile of phosphorylated isoforms of GAP-43 are changed in the brains of a mouse used to model Alzheimer’s disease, GAP-43 will be examined by 1 and 2 dimensional SDS polyacrylamide gel electrophoresis. Isoforms of mouse brain GAP-43 will be detected by immunocytochemistry and silver staining and, further, quantified by computerized densitometry. Alterations in quantities of phosphorylated forms of GAP-43 might result from a pathological biochemical processes. Revealing molecular defects generates potential targets for the development of possibly more effective drugs to combat dementia.
Neurodevelopment/Genetics/Stem Cell Biology:

Our group uses the chicken and mouse embryo as model systems to determine how neural stem cell differentiation is influenced by intrinsic factors (such as gene expression) and extrinsic factors (such as factors secreted by other cells). The accessibility of the chick embryo to experimental manipulation allows us to screen for the effect of experimental manipulation on stem cell differentiation using quantitative PCR and anatomical approaches. With this approach, undergraduate and master’s level students have determined that the basic helix loop helix protein Nato3 is sufficient to promote expression of markers for dopamine producing neurons. The clinical significance of this finding is that dopamine neurons are the target of degeneration in the pathophysiology of Parkinson’s Disease, so our current studies are focused on understanding the mechanism of this effect with the hope of informing therapeutic strategies towards this disease. Additionally, our lab is using the same model system to analyze the effect of factors outside of the neural stem cell (cell-extrinsic factors) such as polyunsaturated fatty acids. These factors have been shown to be important signaling components in development and can affect stem cell differentiation in culture, but have not been analyzed in the living embryo.

The Fateye lab utilizes a multidisciplinary approach (analytical, genomics and bioassays) to study the mechanistic basis of toxicity and/or ecological impact of xenobiotics in vertebrate and invertebrate models. Current projects in the lab focuses on pharmacology of volatile organic compounds, the environmental impact of pollutants in surface waters in Crockery Creek, Michigan.
My research program is somewhat unique in that there isn’t really an overarching disciplinary theme that all my work falls neatly under. My tendency is to seize on an interesting question, recruit students to help answer it, then after a year or two move on to something (often completely) different. Most of the projects in my lab, in one way or another, have employed molecular markers to infer past demographic and evolutionary events in populations of parasites and human pathogens. Past projects have looked at intragenic recombination in rotavirus, positive selection in viral hemorrhagic septicemia virus, microevolution of rabies virus in Michigan bat populations, the population dynamics of raccoon roundworm in West Michigan, modeling Ebola diffusion in West Africa, and social evolution in bacteria. Currently, my lab is using the nematode *C. elegans* to investigate how the gut microbiome modulates the severity of viral infection.

### Professor Graham

**Research Interests:**
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During the infectious process a battle ensues between the human host and the bacterial pathogen over access to various nutrients including metals. The focus of my research is to gain a better understanding of how bacteria acquire nutrients during an infection and how availability of various metals influence disease outcomes. One bacterial pathogen I am currently studying is *Staphylococcus lugdunensis* which is a coagulase-negative Staphylococcal species that has the potential to cause aggressive and progressive disease. Currently little is known regarding the molecular mechanisms deployed by *S. lugdunensis* that enable it to transition from a harmless component of the skin flora to a deadly pathogen. I am interested in identifying genes involved in metal acquisition, metal detoxification, and biofilm formation in *S. lugdunensis*.

### Professor Haley

**Research Interests:**
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*Helicobacter pylori*
I am actively involved in both laboratory and field research. My current lab-based projects include assessing various aspects of hominin (e.g. humans, two species of chimpanzee, their ancestors, and the extinct lineages of their common ancestor) evolutionary anatomy through dissection and non-invasive Magnetic Resonance Imaging (MRI). I have been examining the insertion of the pectoralis minor muscle in the chimpanzee (Pan troglodytes), as various interpretations of this attachment have been reported throughout the anatomical literature. Clarity of this issue is fundamental for not only understanding the evolutionary structural and functional pathway(s) of the muscle, but also for producing a better understanding of the evolution of the hominin shoulder.

Another research area that I have focused on is assessing spatio-temporal variation of stress and developmental stability among extant and extinct mammalian taxa through fluctuating asymmetry (FA). The aim of this research area is to continue exploring the utility and advancement of FA to a variety of modern and prehistoric mammalian species. Deviations from symmetry in bilateral characters have achieved some prominence as measures of developmental (in)stability, revealing greater levels of asymmetry under adverse settings and mirrored target phenotypes under optimal extrinsic (environmental) and intrinsic (genetic) conditions. Increased FA has been associated with dietary, thermal, audiogenic and chemical stresses, but has been reported to decrease when genetic heterozygosity is elevated. Identifying the distribution and expression of FA among (paleo)species that have an extensive and well documented biological history (i.e. through time and space) provides a context for understanding how evolutionary processes and events potentially impact development.

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Fascial release is a popular technique in the strength and performance community. Several methods are reported to increase strength, mobility, and recovery (Body Tempering, roam rolling, Graston®, Reflex Performance Reset, Rolfing). My current interests are in assessing the efficacy of these methods.

The initial investigations will focus on Body Tempering. In Body Tempering, static or shear heavy compressive forces (20-220 lbs.) are applied to tissues to stimulate changes that will stimulate adaptations that render it more resilient to heavier loads. It is proposed that tempering initiates tissue remodeling according to Wolff’s Law, Davis Law, and the Specific Adaptations to Imposed Demands (S.A.I.D Principle). Wolff’s Law states that mechanical stimulus stimulates bone remodeling (strengthening) and Davis’s Law states that soft tissue will adapt and heal in response to a given mechanical stress. The S.A.I.D. principle is applied to explain that all tissue will respond to mechanical stress by increasing strength and resistance. Tissue tempering is initially applied to stimulate tissue to adapt to heavier loads in order to prevent injury. Secondarily it is used to reduce tissue tightness and improve blood flow through local reactive hyperrhemia. With these purported benefits of tempering, there is a lack of scientific data to back up the claims.

Initial investigations of the range of motion around joints will be used to assess tissue tightness, as well as the measurement through specific exercise movements. This will be accomplished using goniometers and a linear displacement accelerometer (OpenBarbell V3). The effects of tempering on muscular strength will be assessed using a Biodex Balance System SD.

My aim is to understand and learn the methodology of tissue tempering in order to measure its effectiveness for increased mobility and muscular strength. Future research into the other named procedures will be done and their efficacy compared and contrasted, and we will work to explain results we may obtain.

Professor Kipp
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My research evaluates the role of the inflammatory response in blood vessel function. This involves directing student research evaluating the response of cultured blood vessel cells (endothelial or smooth muscle) to pro- or anti-inflammatory compounds. We first treated endothelial cells with omega-3 fatty acids and measured changes in the gap junction protein connexin 43 (Cx43). Cx43 is responsible for communicating between endothelial cells and is usually increased by inflammation. After 24 hours, saturated fatty acids (palmitic acid) increased Cx43 while omega 3 fatty acids (eicosapentaenoic and docosahexaenoic acids) decreased Cx43. Blockade of the receptor proposed to mediate the anti-inflammatory effects of omega 3 fatty acids (free fatty acid receptor 4) had the opposite affect (the inhibitor appeared anti-inflammatory). We are currently evaluating inflammatory signaling molecules (NF-kB) to determine whether this unexpected result is reproducible and to explain it.

In addition to the specific question above, my broad training in exercise and comparative physiology has provided me with the background to address many other questions. Although on sabbatical this fall (2021), I would be happy to talk with a student this semester about research questions that they might be interested in addressing in the lab in the winter semester and beyond. I have mentored many student projects (both research and writing) for the Honors College.

Professor Kurjiaka
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My research focuses on how bony morphology can inform us about behavior. Using 3D animation modeling, I reconstruct birth mechanisms in human, fossil, and extant primate species in order to better understand how and why human childbirth can be difficult. Students working with me can learn these virtual reconstruction processes while working on pelvic morphology projects.
My research at GVSU has largely been an extension of my previous work at Pharmacia/Upjohn/Pfizer. We have been exploring the possible therapeutic benefits of nicotinic compounds in the treatment of visual diseases, specifically glaucoma. ACh can activate several subtypes of nicotinic ACh receptors (nAChR) and we have been interested in the alpha7 subtype. Our previous studies have shown that selective activation of the alpha7 nAChR can provide neuroprotection to the cells that are the target of neurodegeneration in glaucoma. We have also shown that a selective ‘positive allosteric modulator’ (PAM) of the alpha7 nAChR can be of potential benefit. In addition, we have provided evidence that a compound originally developed to increase ACh release in Alzheimer’s disease was effective in our experiments. Most recently, we have shown that activation of the alpha7 nAChR can also lead to regeneration of cells lost during the disease process. We are currently investigating if this regeneration leads to functional recovery of visual function lost during the disease process. Experiments in my lab have included cell culture, neurotransmitter release studies, and confocal microscopy. Future experiments could include labeling existing cells to differentiate from ‘regenerated’ cells, applying compounds to rodents as eye-drops, and following functional recovery (via electroretinograms).

Confocal image of a pig retinal slice in response to DMP-543

Heart disease is the leading cause of death worldwide. According to the data published by American Heart Association in 2015, ~610,000 people die of heart disease in the United States every year—that’s 1 in every 4 deaths. Research in my lab focuses on understanding how a group of protein phosphatases called dual specificity phosphatases (DUSPs) regulate extracellular signal-regulated kinases 1/2 (ERK1/2) signaling in the heart. Recently we have identified DUSP8 as a critical regulator of ERK1/2 activity in the heart. Knockout of Dusp8 gene in mice leads to increased ERK1/2 activity, which protects the mice from progression towards heart failure in two surgery-induced disease models. Cardiac specific overexpression of DUSP8 in mice results in decreased ERK1/2 activity, ventricular dilation, and heart failure. These data suggest that targeting DUSP8 might be a therapeutic approach for heart disease. We are currently investigating whether knockout of both Dusp6 and Dusp8, two DUSPs specific for ERK1/2, will protect the heart from disease. In this study, we propose two specific aims to study the effect on ERK1/2 signaling upon loss of DUSP6 and DUSP8 proteins in the heart. Specific aim 1: generation of knockout mice with loss of both Dusp6 and Dusp8 genes. Specific aim 2: determine the effect of loss of both Dusp6 and Dusp8 genes on MAPK signaling, myocyte proliferation, and cardiac function at both rest and stimulation conditions.

Confocal image of a pig retinal slice in response to DMP-543
Dr. Nochera is interested in developing research with breadfruit not only as an alternative but also as a "functional food" product for the public. Breadfruit is high in starch, rich in antioxidants, and is also gluten free. New breadfruit products can provide nutritious, appealing and inexpensive gluten free food sources based on locally available breadfruit in areas of the world where it can be easily grown.

The overall focus of my research is to investigate the role of hormone signaling in the male reproductive tract with the goal of better understanding male fertility and infertility. Control of spermatogenesis and sperm production is hormonally regulated through the hypothalamic-pituitary-gonadal axis. Classically, testosterone and other androgens have been associated with the male, while estradiol and other estrogens have been associated with the female. However, the testes express functional aromatase and produce significant amounts of estradiol in addition to testosterone and males unable to produce estrogens are infertile. Results from my research, and other groups, reveal that estrogen receptor alpha (ERα) and beta (ERβ) are expressed within the testis and epididymis of multiple species. This indicates that the male reproductive tract in mammals is both a source and target for estrogen regulation. The mechanisms by which estrogen regulates sperm production and maturation remain largely unknown, but this knowledge is essential for further progress in understanding male fertility. Elucidating these mechanisms is the long-term objective of my research program. Active projects in my lab are investigating the effects of estrogen signaling during aging and the effects of endocrine disruption of the reproduction tract.

A secondary line of research in my lab involves investigation of the effects of leptin-deficient-related obesity on male fertility. Leptin deficient mice are obese and show varying levels of testicular dysfunction. However, extremely low sperm counts in the epididymis are more likely responsible for the infertility reported in these mice. My lab is investigating potential causes for this reduced epididymal sperm count.
My research interests center around a functional, real-time measure of neurotransmission. Neurons send and receive information through chemical means, transducing electrical signals into chemical signals. These transmissions occur on a very fast time-scale, in the millisecond timeframe.

One of the best methods for monitoring neurotransmission in real time is called Fast-Scan Cyclic Voltammetry (FSCV). Fast-scan because it is happening fast: every 100 ms; cyclic because it happens repeatedly; and voltammetry because it deals with voltage changes. In brief, when a carbon surface reaches a certain voltage, and a neurotransmitter is next to it, the neurotransmitter will oxidize (like metal rusting). You can measure this reaction and use it to look at changes in neurotransmitter concentration.

The goals of my lab: 1) continue to improve neurotransmitter recording techniques. 2) characterize the effects of various substances on dopamine neurotransmission in the mouse brain, such as melatonin and CBD.
My lab’s research revolves around two themes;  
1) Environmental signals govern the immune system during homeostasis and disease.
2) Adaptive immune cells maintain a state of “innate-like readiness” which benefit host response to infection and/or cancer. 

My lab weaves these two themes to determine how environmental cues regulate innate immune “readiness”. Specifically, we measure how environmental exposure, including microbial experience, shapes immune cell signaling pathways and ability to respond to activation signals. Previous research has shown that traditional inbred specific pathogen free (SPF) adult mouse immune systems resemble human infant immune systems, and may not be an ideal model for studying adult human immunity. Locally purchased pet store mice, which harbor a number of naturally acquired murine pathogens, have immune systems that resemble those of human adults. We will use this model to answer several remaining questions, including how the microbial environment shapes the immune cells throughout development, and how microbial exposure translates into signals that are interpreted by immune cells, ultimately leading to an enhanced state of “readiness: to respond to pathogens.

Impact: Allergies, including allergic asthma, diagnoses have exploded in the last 30 years. For example, the frequency of asthma sufferers has tripled since 1980 (CDC). We hypothesize that increased hygiene practices may prevent critical signals from shaping the immune system, potentially allowing it to overreact to allergens. We hope that our research will help find ways to safely prime the immune system during childhood, and therefore help prevent potentially deadly allergic responses.
My broad research interests are on genetic risks of drug use disorders and the associated neural substrates that influence specific aspects of drug use such as, drug taking, seeking and relapse. Methamphetamine (MA) use like that of opioids is a widespread problem in US and is highly addictive drug with devastating consequences to the individual and society at large. My research program explores binge MA use, MA withdrawal and relapse using a genetic mouse model for high and low MA intake. The main aim of my research is to identify and explore druggable targets for future development of therapeutic interventions. Extensive work by my collaborator at Oregon Health & Science University, and her group, who developed this mouse model system have identified at least two quantitative trait loci (QTL) associated with MA intake, located in chromosome 10 and X. In particular, two gene candidates located in the chromosome 10 QTL, namely a u-opioid receptor and a g-protein coupled receptor, known as trace-amine associated receptor TAAR1, seem to play an important role in the MA intake, and other correlated traits. Correlated behavioral traits of interest involve: sensitivity to rewarding and aversive effects through procedures such as, conditioned place preference, conditioned place aversion, conditioned taste aversion; drug reinforcement such as the operant self-administration paradigms; drug withdrawal in form of anxiety and depression-like symptoms tests, such as, zero or plus-maze, forced-swim, and tail-suspension. Recent pharmacological manipulations of TAAR1 receptor in a number of these experiments seem to support the hypothesis that TAAR1 receptor plays an important protective role in MA use and therefore is a prime druggable target to explore in the future. In sum my lab pursues neuroscience related questions mostly at the behavioral genetics, physiological, neurochemical, and pharmacological level.

Professor Stroik
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My primary research interest lies in understanding the ecological mechanisms that drove changes in community composition and structure throughout mammalian, specifically primate, evolution. In other words, I am interested in determining “why” and “how” mammalian groups arose, diversified, and went extinct by studying their interactions with their physical environment and with one another. In mammals, one of the most impactful species interactions is competition, and species most likely to compete with one another are those who occupy the same ecological niche, or “role” in the community. In the fossil record, ecological niches can only be examined using the anatomical features preserved in fossil specimens, namely teeth and bones. As teeth are the point of contact between a mammal and its food, I use fossil teeth to reconstruct the dietary niches, and ultimately patterns of dietary competition, of mammals living in North America between 65 and 40 million years ago.

Students working in my lab have the opportunity to explore different aspects of mammalian evolution and dietary reconstruction through the study of dental anatomy. This process can include preparing dental molds for casting, casting dental specimens from these molds, curating molds and casts, mounting dental casts for microCT-scanning, and processing digital microCT scans and collecting two- and three-dimensional data using imaging software. Students working in my lab also have the opportunity to conduct paleontological fieldwork in Utah.
The cardiovascular system plays an essential role in enabling the body to adequately perform daily activities such as spending time studying for an exam or setting a new personal best on a university athletic field. Pathologic alterations in the function of the cardiovascular system can be catastrophic as impairments in this system are the leading cause of morbidity and mortality in the United States. In humans, the cardiovascular system is a closed system composed of a pump, i.e. the heart, and a lengthy series of tubes that carry blood from the heart to the cells of the body and, ultimately, back to the heart – these tubes (blood vessels) are specifically known as arteries, veins, capillaries, arterioles, and venules. Our lab is interested in the function of the blood vessels, as they are primarily responsible for regulating blood flow to precisely meet the needs of the body’s tissues across a wide range of metabolic states. Additionally, blood vessels play a pivotal role in the regulation of blood pressure as changes in blood vessel diameter directly impact this important physiological parameter. Since blood vessels are partially comprised of smooth muscle which is known to be sensitive to a variety of stimuli, our lab studies the responses of blood vessels to numerous physiological/pharmacological stimuli including temperature, drugs, and nutritional supplements. We conduct in vitro and in vivo experiments using animal models (usually pigs or mice) to observe acute or chronic changes in cardiovascular function. Many of our experiments involve the isolation of specific arteries that are studied under controlled conditions in an attempt to observe and measure the response of living arteries to relevant stimuli. It is hoped that insights gained from these studies will increase our understanding of the regulation of the human cardiovascular system.

I’m broadly interested in the evolution of locomotor diversity in primates. My research uses three-dimensional geometric morphometrics (analysis of shape in three dimensions) to examine patterns of postcranial variation in the forelimb and hindlimb of human ancestors. I am using this data to investigate the underlying processes that drive shape variation in the postcranial skeleton of primate (e.g., patterns of integration/modularity, potential ontogenetic shifts, functional constraints) and the environmental factors that could have been instrumental in past selective events. Students working with me have the opportunity to either dissect primate cadavers and collect data on patterns of musculature in living primates or to collect three dimensional data using computer rendered models of bony material.
My research focuses on using Candida albicans as a model fungal pathogen. C. albicans is a frequently acquired nosocomial infection both within the US and worldwide. It is an increasingly common threat to human health as a consequence of AIDS, steroid therapy, organ and tissue transplantation, cancer therapy, broad spectrum antibiotics and other immune defects. These infections carry unacceptably high morbidity, mortality rates (30-50%) and important economic repercussions (estimated total direct cost of approximately 2 billion dollars in 1998 in US hospitals alone).

The objectives of my research are: (i) the application of state-of-the-art yeast cell biology and genetics to the study of Candida albicans pathogenesis and commensalism, (ii) the use of proteomics, genomics, and bioinformatics in the analysis of the lifecycle of C. albicans, (iii) studies of C. albicans virulence in vivo, and (iv) signal sensing and transduction particularly with reference to disease related and quorum sensing pathways in C. albicans.

Currently, we are focused on studying a subset of proteins whose level appears to need to change to allow the shape transition that is associated with disease to occur. We are studying this subset on multiple levels including: Which need to change to allow the transition, how this subset influences the ability to cause disease and how the proteins modulate their effect.

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My research investigates how primates integrate behavioral and physiological adaptations to overcome ecological challenges in their natural environment. I aim to understand how these different facets of animals’ biology work together at the organismal level. My lab provides opportunities for undergraduate and Master’s students to gain experience in hormone analysis, behavioral observation methods, thermoregulatory research, and international field work.

One main focus of my research program is the sensory ecology of foraging decisions. I am interested in how primates use cues, such as olfactory signals, in order to select foods and communicate information about resources. This research has investigated exudate feeding by common marmoset monkeys and seeding eating in pithecids.

Secondly, I am interested in how animals utilize behavioral and physiological adaptations to maintain a stable body temperature, as well as cope with the energetic demands of thermoregulation. My research in this area has covered behavioral mechanisms such as microhabitat choice and use of postures, hormonal mechanisms of thermoregulation, and non-invasive assessment of body temperature via infrared thermography.

Lastly, I maintain ongoing research projects that examine the biological basis for social behavior in primates, particularly white-faced saki monkeys. I am broadly interested in the ecological, demographic, and physiological factors that drive variation in monogamous social systems.
My research interests involve understanding the supply and demand for oxygen in tissues such as skeletal muscle and brown adipose. To this end I use a variety of noninvasive techniques such as Doppler ultrasound, magnetic resonance imaging, and spectroscopy to study human research subjects across a range of ages (college-aged to octogenarians) and physical activity levels (sedentary to highly trained athletes). The goal of my research is to better understand the interplay between the demand and supply for oxygen by these tissues and the impact of chronic physical activity and different disease states.