TEXAS A&M UNIVERSITY
Naresh K. Vashisht
College of Medicine

RESEARCH DAY

Summer Research Program
July 25, 2025

Medical Research & Education Building 2
Bryan, Texas

Schedule of Events

9:00AM-12:00PM	**Poster Viewing & Judging** MREB 2 Lower Level
12:00PM-12:30PM	**Lunch** MREB 2, Room 1403
12:30PM-1:30PM	**Keynote Speaker** J. Thomas Cunningham, PhD Professor & Senior Associate Dean for Research **“Homeostasis and the Hypothalamus: Lessons Learned”** MREB 2, Room 1403
1:30PM-2:00PM	**Presentation of Certificates** MREB 2, Room 1403
2:00PM	**Group Photo & Adjourn**

Keynote Speaker

J. Thomas Cunningham, PhD
Professor & Senior Associate Dean for Research

Title: **“Homeostasis and the Hypothalamus: Lessons Learned”**

Tom Cunningham did his graduate training in the Department of Psychology and Cardiovascular Research Center at the University of Iowa with Dr. Alan Kim Johnson and his postdoctoral training was with Leo Renaud, MD, PhD, at McGill University and the University of Ottawa followed by work with Francois Abboud, MD, at the University of Iowa. At the University of Missouri, he was Block Director for the Neuroscience block for first year medical students for 7 years and was a member of the Dalton Cardiovascular Research Center. He moved to UT Health San Antonio in 2006 to join the Department of Pharmacology and Neuroscience and was chair of their Committee for Graduate Studies for 2 years. In 2009, he moved to the University of North Texas Health Science Center as Professor of Integrative Physiology and Director of their Cardiovascular Research Center. Prior to joining Texas A&M, Dr. Cunningham was the Associate Vice President for Research Administration and Regents Professor of Physiology and Anatomy at UNTHSC. His areas of teaching include neuroanatomy, neurophysiology, endocrinology, and the pharmacology of anesthetics and analgesics. His laboratory studies the role of the central nervous system in body fluid homeostasis and blood pressure regulation and has continuous funding from NIH since 1995. His laboratory’s long-term goal is to determine how changes in CNS network function contribute to chronic human diseases. He has been active in reviewing grants for the American Heart Association and the National Institutes of Health and served as chair of a standing NIH study section (Neuroendocrinology, Neuroimmunology, Rhythms, and Sleep). He currently belongs to the American Physiological Society, the Society for Neuroscience, the American Heart Association, the American Society for Pharmacology and Experimental Therapeutics, the Pan-American Neuroendocrine Society, and the International Society for Regulatory Peptides.

Abstracts

TEXAS A&M HEALTH
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EVALUATING THE EFFECTS OF INDUCED HEART FAILURE WITH PRESERVED EJECTION FRACTION IN CALCIUM FLUORESCENT CGCAMP8 AND WILD-TYPE MOUSE MODELS

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Heart failure with preserved ejection fraction (HFpEF) contributes to nearly 50% of all heart failure diagnoses. HFpEF entails diastolic dysfunction, preserved ejection fraction (EF ≥ 50%), and limited therapeutic options. A well-established method to induce the HFpEF phenotype in mice involves feeding them a high-fat diet (HFD; 60% kcal from fat) and administering L-NAME (0.5 g/L in drinking water), a nitric oxide synthase inhibitor. We have developed a mouse model expressing the genetically encoded fluorescent calcium sensor cGCaMP8 specifically in cardiac myocytes. We hypothesize that HFD+L-NAME treatment will induce HFpEF in cGCaMP8 mice similarly to wild-type (WT) mice. Demonstrating that cGCaMP8 mice respond comparably would validate this model for studying calcium transients in relation to crossbridge cycling during diastolic dysfunction in HFpEF. To test this, both WT and cGCaMP8 mice were fed HFD and given L-NAME in their drinking water, then underwent echocardiography at baseline and every 5 weeks to closely monitor the progression of diastolic dysfunction. Diastolic function was assessed using peak early diastolic myocardial relaxation velocity (e') and the E/e' ratio, where E represents early transmitral flow velocity. A decline in e' and an increase in E/e' indicate worsening diastolic function. At baseline and 5 weeks, no significant differences were observed between WT and cGCaMP8 mice. These preliminary results suggest that cGCaMP8 mice respond similarly to HFD+L-NAME treatment. However, continued observation through the full 15-week protocol is necessary to confirm that cGCaMP8 mice develop the HFpEF phenotype in the same manner as WT controls.

This work was supported in part by NIH grant HL145534 and Frank W. Mayborn Endowment to CT. NA was supported by the Texas A&M College of Medicine Office of Research.

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MICROGRAVITY SIMULATION BY HINDLIMB UNLOADING LEADS TO INCREASED LIVER INFLAMMATION

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Microgravity alters human physiology and homeostasis, impeding long-term space travel. Obtaining samples from human subjects in outer space is costly and limited. Hindlimb unloading (HLU) is a readily available mouse model that simulates microgravity conditions. We hypothesized that HLU mice exhibit increased liver inflammation and ductular reaction due to microgravity effects, leading to elevated pro-inflammatory cytokines and proliferating cholangiocytes. C57BL mice were assigned to either regular control cages (SHAM) or HLU for four weeks before tissue procurement. Paraffin-embedded liver samples were stained for macrophages (F4/80) and cholangiocytes (CK19), while snap-frozen samples underwent Proteome Profiler Mouse Cytokine Array analysis and Oil Red O staining. HLU mice demonstrated heightened inflammation, as indicated by strong F4/80 immunohistochemistry staining and elevated pro-inflammatory cytokines, including CDL20, CCL5, and IL-17A, in the cytokine array. Ductular reaction was evident via strong CK19 immunohistochemical staining, consistent with increased wound-healing cytokines, such as MMP-9, EGF, and Endoglin, in HLU mice compared to control mice. However, Oil Red O staining revealed reduced fat deposition in the livers of HLU mice, contrasting with the spaceflight mouse liver samples, which showed increased fat accumulation, likely due to full-body microgravity exposure, cosmic radiation, and/or high fat content of the rodent space diet. Future investigations should focus on specific cytokines, including MMP-9, IL-17A, and IL-6, using Western Blots or ELISA techniques, as well as more age- and sex-focused research. Although HLU is not representative of fat deposition, it remains an accurate model for inflammation and should continue to be utilized for that purpose.

SC was supported by the Texas A&M College of Medicine Office of Research.

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ENHANCING AAV TRANSDUCTION WITH CATIONIC AMPHIPHILIC DRUGS IN ASTROCYTES

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Cationic amphiphilic drugs (CADs) have been proposed as enhancers of intracellular delivery by triggering membrane permeabilization, facilitating escape from endosomal entrapment. In gene therapy, the endocytic pathway presents a major barrier, as internalized vectors often become trapped in endosomes and fail to reach their target, reducing therapeutic effectiveness. While increasing viral load can improve delivery, it also leads to cytotoxicity, limiting its use. This project evaluates the effect of CADs on adeno-associated virus (AAV) transduction efficiency in astrocytes across a range of viral concentrations. GFP expression is used to quantify AAV delivery in CAD-treated versus untreated cells at increasing concentrations of virus. By promoting endosomal escape, CADs may help prevent degradation in downstream lysosomal pathways and enhance transduction. These findings support the potential role of CADs in overcoming intracellular barriers to AAV-mediated gene delivery.

KD was supported by the Texas A&M College of Medicine Office of Research.

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FINASTERIDE MODULATION OF HIPPOCAMPAL NEUROGENESIS AFTER TRAUMATIC BRAIN INJURY IN MICE

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Traumatic brain injury (TBI) often leads to lasting neurological problems such as cognitive impairment and post-traumatic epilepsy (PTE), significantly affecting quality of life. One important consequence of TBI is disrupted neurogenesis in the hippocampal dentate gyrus (DG), a brain region associated with memory and learning. Neurogenesis can be assessed by immunostaining for doublecortin (DCX), a protein expressed in immature neurons, providing a measure of newborn neurons. Finasteride, a 5α-reductase inhibitor that inhibits neurosteroid biosynthesis in the brain, may interfere with neuronal pathways that play a key role in brain repair and neurogenesis. However, its effects on hippocampal neurogenesis following TBI have not been well characterized. In this study, we investigated the effect of finasteride on neurogenesis in mice subjected to mild-to-moderate TBI, using a controlled cortical impact model. Four months post-injury, brain tissue was collected, and DCX(+) cells in the DG were quantified using an unbiased stereological method to measure newborn neuron number, cell density, and total DG volume. Results showed that TBI caused a 22% reduction in the number of DCX(+) newborn neurons in the DG subfield. Finasteride-treated TBI mice exhibited an even greater 28% decrease in DCX(+) cells compared to controls, with parallel decreases observed in neuronal density. Moreover, the DG volume was significantly lower in the finasteride-treated group. These findings suggest that finasteride treatment is associated with reduced neurogenesis after TBI and may, in fact, exacerbate hippocampal structural damage following brain injury.

CE was supported by the Texas A&M College of Medicine Office of Research.

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ADRENALECTOMY PRIOR TO SPINAL CORD INJURY REDUCES LESION SEVERITY AND PROVIDES NEUROPROTECTIVE EFFECTS BY WEAKENING THE ENDOGENOUS GLUCOCORTICOID ELEVATION, THEREBY ALSO MITIGATING SCI-INDUCED TRABECULAR BONE LOSS

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Spinal Cord Injury (SCI) greatly impacts an individual's quality of life, producing widespread consequences that affect both neurological and systemic health. Among many outcomes, SCI results in autonomic dysregulation, pain, loss of bladder and bowel function, depression, and often leads to paralysis. The mechanisms that link SCI and secondary consequences of injury are not clear. For instance, it is unknown how spinal cord tissue damage could trigger changes in the brain that result in depression and/or cognitive decline. We hypothesize that SCI-induced activation of the hypothalamus-pituitary adrenal gland (HPA) axis immediately following injury, and the subsequent elevation of corticosterone, could contribute to secondary outcomes including impaired locomotor function and depression. To test this, we gave young adult male Sprague Dawley rats a bilateral adrenalectomy 5 days prior to a moderate spinal contusion injury. For 28 days post injury, we scored the rats locomotor function, assessed depression, and recorded weight gain as an index of overall health. First, using ELISAs, we confirmed that adrenalectomy successfully reduced corticosterone levels following SCI. Despite reduction of serum corticosterone levels, however, there appeared to be no effect of adrenalectomy on depression, locomotor function, or weight gain. We have now sectioned the spinal cord tissue and stained the spinal sections with Luxol Fast Blue for future lesion analysis. Based on the null effects on locomotor recovery we do not expect neuroprotection from adrenalectomy. Overall, our study challenges the dogma that high levels of corticosterone are a major cause of adverse consequences of SCI.

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CORRELATION BETWEEN PODOPLANIN LOCALIZATION AND INNERVATION AT NEUROMUSCULAR JUNCTIONS IN ALS MICE

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Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disease caused by the loss of motor neurons. ALS is characterized by the loss of innervation at neuromuscular synapses in skeletal muscles. Podoplanin (PDPN) is a glycoprotein that is involved in lymphatic and inflammatory responses. Recent findings from our lab show that PDPN expression increases both in the spinal cord and muscles in the SOD1-G93A mouse model of ALS. However, it is unknown if there is a link between PDPN localization and neuromuscular junction (NMJ) innervation in ALS. To begin exploring this question, we collected the tibialis anterior (TA) muscle from age- and sex-matched symptomatic SOD1-G93A and control mice, fixed, and then cryosectioned them. Sections were stained with fluorescently-coupled probes to mark NMJs (α-Bungarotoxin, BTX), nerves (anti β3-tubulin), and PDPN. Confocal microscopy showed PDPN staining near NMJs, as if capping the synapse at a distance. Quantitative analysis revealed: (i) In control TA, 78% of NMJs were innervated and 50% of those had overlapping PDPN staining, and (ii) In ALS TA, 64% of NMJs were denervated and 52% of those lacked overlapping PDPN. We conclude that these preliminary results suggest a link between NMJ innervation and PDPN localization near the synapse. Future work will extend our observations to more animals, different muscle groups, and different disease stages. We will also investigate the spatial correlation between NMJ innervation and the localization of LYVE1, another lymphatic marker.

CF was supported by a Fellowship from NIH R25 DK126642.

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A COMPARISON BETWEEN METHYLATION RISK SCORES USED TO PREDICT ALCOHOL USE

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Several studies have developed Methylation Risk Scores (MRSs) to predict alcohol consumption, however, the overlap in CpGs between MRSs has not been examined. Most MRSs are developed using LASSO or Elastic Net Regression that select only one CpG site from regions with many highly correlated sites. Hence, there is a high likelihood that these MRSs may capture overlapping signals. To investigate this, we first annotated CpGs that form 5 different alcohol MRSs to examine both positional and genic overlap. The Corrected Cover Area (CCA) method was used to quantify the overlap in individual sites between the three MRSs based on Generation Scotland, sites for the Liu and Xiaoyu MRSs which utilized the 450K array, and gene overlap between all 5 MRSs. The highest overlap was the genic overlap between Lohoff and McCartney at 13.037% while the highest CpG overlap was between Lohoff and McCartney at 8.39%. A permuted Fisher's Exact Test was computed for each pair of MRSs within the three sets of comparisons. All tests were significant (p-value $\leq 1x10^{-4}$) suggesting a significant overlap in both position and genes for all examined MRS pairs. However, it is unclear if this overlap is due to all MRSs predicting alcohol consumption, or if it is due to the same cohorts being used in some MRSs. Future studies should utilize independent cohorts to disentangle potential cohort specific effects and may also investigate overlaps on a regional level.

DG was supported by the Texas A&M College of Medicine Office of Research.

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INFLUENCE OF DIET AND GENETIC BACKGROUND ON MACROPHAGE RESPONSE TO SALMONELLA ENTERICA TYPHIMURIUM IN MICE

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Salmonella enterica serotype Typhimurium (STm) causes 1.35 million food-borne illnesses annually in the United States (CDC, 2024). Bacterial exposure elicits a different reaction in each individual. Using a genetically diverse mouse model called the Collaborative Cross (CC), we investigated how individually variable host genetics and diet alter macrophage response to bacterial exposure. We hypothesized that variation in genetics, combined with dietary modulation of the immune system, influence the ability of macrophages to support STm growth. Mice from several CC lines were put on three distinct diets: Ketogenic, Vegan, and Mediterranean, to evaluate (1) the influence of diet on macrophage state, and (2) the way diet and genetic background may influence susceptibility to STm infection. We isolated bone marrow-derived macrophages (BMDMs) from two lines of CC mice to examine innate immune responses and exposed them to STm using a gentamicin protection assay. The gentamicin protection assay allowed quantification of bacterial adhesion as well as intracellular bacterial replication and survival. This approach highlights the interaction between host genetics, nutritional inputs, and pathogen behavior to provide clarification on how distinct gene-diet combinations structure susceptibility to bacterial infection. In our assays, we found that there are diet-based and genetic-dependent differences in macrophage efficacy which may contribute to host susceptibility to infection.

FGL was supported by a Fellowship from NIH R25 DK126642.

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AAV-VEGFD GENE THERAPY IMPROVES LYMPHATIC FUNCTION AND REDUCES INFLAMMATION IN A MOUSE MODEL OF DUCHENNE MUSCULAR DYSTROPHY

Dheeraj Govu, Bhuvaneshwaran Subramanian, Akshaya Narayanan, Shedreanna Johnson, Ilse Paredes Mares, Ahana Majumder, Aditi Mohankumar, Anjali Ghosh, Mendell Rimer, Peter Nghiem, Mariappan Muthuchamy
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Duchenne Muscular Dystrophy (DMD) is a chronic neuromuscular disorder caused by mutations in the DMD gene, leading to loss of dystrophin, a scaffold protein essential for myofiber integrity. Without dystrophin, sarcolemma instability results in repeated cycles of muscle degeneration, inadequate regeneration, fibrosis, and progressive muscle wasting, ultimately leading to premature loss of ambulation and shortened lifespan. Our previous studies demonstrate that lymphatic dysfunction is linked to chronic inflammation and DMD pathogenesis. Hence, we hypothesize that inducing therapeutic lymphangiogenesis in dystrophic muscle would improve lymphatic function, reduce inflammation, and ameliorate muscle pathology. We delivered an AAV-VEGFD gene vector under control of a muscle creatine kinase (MCK) promoter to control and D2.mdx mice, a well-established DMD model. Results demonstrated that the control AAV-MCK-GFP injection showed expression of GFP in all the skeletal muscles. Microlymphangiography experiments showed that AAV-VEGFD treatment significantly enhanced lymph transport in D2.mdx mice. Additionally, inflammatory cytokines and chemokines, and lymphangiogenesis marker genes were significantly decreased in AAV-VEGFD-treated D2.mdx mice. Histological analysis of tibialis anterior, extensor digitorum longus, gastrocnemius, soleus, and quadricep muscles using Hematoxylin and Eosin (H&E) and Masson's Trichrome staining indicated a distinct polygonal shape of myofibers in the AAV-VEGFD-treated D2.mdx animals when compared with the GFP-treated group, indicating the active proliferation phase of the myocytes. Thus, our data demonstrates that VEGFD-mediated lymphangiogenesis improves lymphatic function and reduces inflammation, thereby enhancing muscle histopathology in DMD animals, highlighting its promise as an adjunctive therapeutic strategy targeting the inflammatory and fibrotic components of DMD disease progression.

DG was supported by the Texas A&M College of Medicine Office of Research.

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METTL14-MEDIATED RNA M6A METHYLATION IS REQUIRED FOR HEALTHY CARDIAC COMPOSITION AND FUNCTION

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Heart failure (HF) is a global health crisis affecting over 64 million people and remains a leading cause of hospitalization and death. Despite available treatments, the underlying molecular mechanisms of HF are not fully understood, limiting the development of curative therapies. Emerging evidence suggests that N6-methyladenosine (m6A) RNA modifications, regulated by enzymes such as METTL14, play critical roles in cardiac function. We hypothesized that METTL14 is essential for maintaining cardiomyocyte homeostasis and that its deletion leads to maladaptive remodeling via IRGM1 upregulation. To test this, cardiomyocyte-specific METTL14 knockout (KO) mice were generated using the Cre-loxP recombination system. These mice exhibited early mortality, disorganized myofibrils, cardiac fibrosis, and dilated cardiomyopathy. To understand the underlying molecular changes, we performed RNA sequencing and methylated RNA immunoprecipitation sequencing (MeRIP-seq). Among the 224 genes found to be both hypomethylated and upregulated, enrichment analysis identified the cGAS-STING innate immune pathway as most significantly activated, with IRGM1 exhibiting the highest fold-change. IRGM1 upregulation was validated by Western blot and additionally found to localize primarily to mitochondria. Additionally, METTL14-/IFNR- double KO models also maintained the IRGM1 overexpressed phenotype, substantiating that IRGM1's modulated expression was indeed a consequence of METTL14-KO. These results identify METTL14 as a critical epitranscriptomic regulator of cardiac integrity and position IRGM1 as a potential therapeutic target linking RNA methylation loss and mitochondrial dysfunction in HF. Future studies may focus on targeting the IRGM1–cGAS-STING axis to mitigate cardiomyocyte loss in epitranscriptomically dysregulated hearts.

DH was supported by a Fellowship from NIH R25 DK126642.

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IL-17A AS A CAUSAL AGENT OF ISCHEMIC STROKE IN MIDDLE-AGED SPRAGUE DAWLEY RATS

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Stroke affects 1 in 32 individuals in the United States, with middle-aged females experiencing a higher incidence than age-matched males. Our previous work demonstrated that stroke increases white matter neurodegeneration and circulating IL-17A levels in serum. Furthermore, prior work has found a treatment with miR-20a-3p that attenuates neuroinflammation and stroke-induced cognitive impairment. This study focuses on Interleukin-17A (IL-17A) as a proinflammatory cytokine implicated in microglial activation and post-stroke neurodegeneration and cognitive outcomes. We hypothesize that IL-17A elevation and subsequent microglial activation are key mediators of stroke-induced neuroinflammation in reproductively senescent (RS) female rats. To test this, intact 12-month-old female Sprague Dawley rats (N=19) received stereotaxic intracerebroventricular (ICV) injections of IL-17A at concentrations of 0, 50, 100, or 200 ng/µl into the left ventricle. Brains were sectioned (30 µm) and stained for Iba1 (a microglial and macrophage marker) and CD68 (an activated microglia and macrophage marker), then imaged using a FV-3000 Olympus confocal microscope to assess the number of microglia. The quantity of microglia was used to determine the most effective dose of IL-17A, which we found was 100 ng/µl. In the future, we will conduct a chronic study (90 days) to assess the cognitive effects of IL-17A–induced neuroinflammation using validated behavioral assays, including the Novel Object Recognition Test (NORT) and Barnes maze.

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A SYSTEMATIC REVIEW OF METHYLOME-WIDE ASSOCIATION STUDIES EXAMINING CANNABIS USE

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Research suggests DNA methylation, an epigenetic modification that impacts gene expression, may be altered by cannabis. This systematic review sought to synthesize the literature on methylome-wide association studies (MWAS) of active cannabis use in humans. We screened 675 studies returned by a Google Scholar search in June 2025 and identified 10 studies for inclusion. Analysis of the included studies revealed that the current literature is burdened by low sample sizes and restrictive cohorts, with five studies comparing fewer than 100 participants and 8 studies focusing primarily on cohorts with majority European ancestry. However, 3 studies overcame sampling limitations via meta-analysis of multiple cohorts. Another challenge unique to cannabis MWAS is tobacco use, as co-use is common. There was significant variation across studies when accounting for tobacco use. One consistently identified gene across studies was *AHRR*, a gene heavily associated with cigarette use. *AHRR* was identified in studies even when smoking was accounted for via stratification. Additionally, 4 studies identified significant enrichment in the dopaminergic synapse pathway. This enrichment was found in studies examining both current and former cannabis use. These findings highlight a need for larger sample sizes or meta-analyses in diverse populations to generate results with methylome-wide power that are generalizable to non-European populations. Additionally, future studies must utilize consistent definitions of cannabis use alongside consistent controls for smoking to better identify cannabis specific genes and pathways.

TJ was supported by the Texas A&M College of Medicine Office of Research.

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METABOLICALLY ENGINEERED *BRUCELLA MELITENSIS* AS A POTENTIAL THERAPY IN A MOUSE MODEL OF MULTIPLE SCLEROSIS

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Multiple sclerosis (MS) is a neurodegenerative disease characterized by the breakdown of the protective myelin sheath surrounding the brain and spinal cord. A major driver of MS pathology is the dysregulation of immune cells, particularly the overactivation of T helper 17 cells and impaired function of regulatory T cells, which leads to chronic inflammation and neural damage. Current treatments for MS often involve broad immunosuppressive strategies, which can be nonspecific and increase the risk of side effects. The engineered bacterium *Brucella melitensis* carries the *tnaA* gene, enabling the production of indole, a microbial metabolite known to suppress inflammation of T helper 17 cells and promote regulatory T cells, which help mitigate autoimmune responses. Using the engineered bacteria for in vivo model could provide a more localized and sustained therapeutic effect for MS. This project investigates using a metabolically engineered strain of *Brucella melitensis* as a potential therapeutic agent for Experimental Autoimmune Encephalomyelitis (EAE), a mouse model of MS, observing the therapeutic efficacy through a combination of clinical scoring and histological analysis of tissue. Preliminary findings suggest that treatment with indole-producing *Brucella melitensis* reduces neuroinflammatory markers and attenuates disease severity in EAE mice. This approach highlights the potential of engineered microbial therapies to modulate the immune response in a more targeted and effective manner than conventional treatments. This research underscores the therapeutic promise of microbial-based immune modulation for autoimmune neuroinflammation. It may inform the development of more precise interventions for MS and related disorders.

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INVESTIGATING THE ENIGMATIC ROLE OF NF-κB-INDUCING KINASE (NIK) IN THE PROMOTION OF NEUROINFLAMMATION WHEN PRESENT IN INBORN ERRORS OF IMMUNITY

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Inborn errors of immunity (IEI) are severe genetic disorders that disrupt the innate and adaptive immune systems causing frequent, chronic bacterial and viral infections, aberrant inflammation, blood disorders and delayed physical development. Importantly, central nervous system (CNS) inflammation and neurological impairments are increasingly recognized as components of IEI, yet are not ameliorated by current treatment paradigms, underscoring a need for new therapeutic approaches. Given NIK's high expression in brain immune cells, including astrocytes, we investigated the hypothesis that NIK is an important mediator of neuroinflammation and neurological dysfunction in IEI. Using two newly established mouse models of IEI patients with NIK loss-of function mutations, $NIK^{Val347Met}$ ($NIK^{PID\text{-}VM}$) and ($NIK^{PID\text{-}PR}$) mutations (collectively referred to as NIK-IEI), we tested the effect of systemic administration of the bacterial endotoxin lipopolysaccharides (LPS) on the brain. We observed increased expression of the interleukin-6 (IL-6) cytokine, as well as glial acidic fibrillary protein (GFAP), a marker of astrocyte activation (astrogliosis). Increased IL-6 and GFAP expression are often associated with neuroinflammation and neurodegenerative processes and indicate an aberrant, elevated neuroinflammatory response in NIK-IEI mice. Thus, we have generated two clinically relevant models of NIK-mutant IEI that recapitulate patient disease phenotypes in the periphery and brain that will not only advance mechanistic understanding of IEI pathology, but also enable the development and screening of new therapeutics.

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EFFECTS OF K-OPIOID RECEPTOR ANTAGONISTS ON RECOVERY FOLLOWING SPINAL CORD INJURY

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There are 300,000 people in the United States living with a spinal cord injury (SCI). This condition impacts a patient's quality of life through loss of motor and sensory control, hormone irregularities, bone loss, and bladder incontinence. 80% of patients with SCI are treated with opioids within 24 hours of injury; however, opioids have been shown to undermine long-term locomotor recovery and induce chronic pain. Our research is focused on testing the efficacy of the Kappa Opioid Receptor (KOR) antagonist norbinaltorphimine (nor-BNI) as a method to curb the adverse effects of opioid administration. For this study, we used adult male Sprague Dawley rats. We inflicted some of the rats with a spinal contusion and operated on the rest without administering the contusion. We administered these rats with one of four opioids or saline and tested their sensory and locomotor functions for seven days post-surgery. We also administered the rats with nor-BNI one- and three- days post-surgery. We continued to periodically test the rats' sensory and locomotor functions until 28 days post-surgery, at which point they were euthanized and their spinal cords harvested for histology or proteomic study. Contrary to our hypothesis, nor-BNI administered in conjunction with opioids promoted the development of chronic pain. There is an indication that nor-BNI helps, in the case of certain opioids, with the recovery of locomotor function but undermines it in others. Moving forward, we will continue to investigate the mechanisms that underly the exacerbation of chronic pain in SCI rats using proteomics and rt-PCR.

AL was supported by a Fellowship from NIH R25 DK126642.

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ANALYSIS OF VARIABILITY IN TUBERCULOSIS VACCINE SUBSTRAINS THROUGH IMAGING

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Tuberculosis (TB), caused by *Mycobacterium tuberculosis* (Mtb), remains a threat to humans and is considered a leading infectious cause of death globally. The only vaccine against TB, bacille Calmette Guérin (BCG), displays variable efficacy in preventing pulmonary TB. BCG has been distributed to different laboratories worldwide, where it has continued to be subcultured. Consequently, multiple BCG strains have emerged with genetic and phenotypic differences. Evaluation of vaccine efficacy depends upon protection studies in animals that take months to years, due to the slow growth rate of Mtb. The absence of rapid ways to dissect mechanisms of pathogenesis, evaluate therapeutics, and determine vaccine efficacy is a major roadblock to progress. Currently, colony forming unit (CFU) determination remains the standard for evaluating therapeutic and vaccine efficacy. Preliminary studies suggest imaging can be used to rapidly evaluate vaccines, greatly reducing time for analysis. However, it is important that the dynamic range is sufficient to measure differences between vaccines and provides comparable or better data than CFU. Here, we developed a new reporter system using the extremely bright NanoLuc luciferase and bioluminescence resonance energy transfer (BRET) with NanoLuc and large Stokes shift fluorescence proteins, designated NanoBRET. NanoBRET imaging is expected to have a threshold of 1-10 CFU, allowing detailed analysis of therapeutics and vaccines. A sensitive imaging method allows for identification of infected tissues, quantification of infected cells, and localization of the bacteria. Imaging can provide new insight into the kinetics of host cell infection and protective efficacy of the vaccine.

This work was supported in part by NIH grants AI187440, AI186092, and AI165913 to JC. AM was supported by the Texas A&M College of Medicine Office of Research.

TEXAS A&M HEALTH
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2025 SUMMER UNDERGRADUATE RESEARCH PROGRAM

EFFECTS OF IGF-1 TREATMENT ON ASTROCYTIC ACTIVATION FOLLOWING EXPERIMENTAL TRAUMATIC BRAIN INJURY

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Traumatic brain injury (TBI) is a leading cause of death and disability that is estimated to occur in nearly 70 million people each year worldwide. TBI is associated with a myriad of physiological consequences and the hypothalamic-pituitary axis (HPA) is particularly vulnerable. Aberrant HPA signaling is linked to reduced growth hormone (GH) and insulin-like growth factor (IGF-1) levels following TBI. Phase II clinical trials and preclinical studies which supplemented IGF-1 after TBI have found this intervention to be beneficial. However, the therapeutic mechanisms underlying this response have not been fully elucidated. Astrocytes are glial cells that are involved in immune responses, tissue repair, and maintaining brain homeostasis, among other functions. In our previous studies, we have identified alterations to astrocyte activation following TBI. The effect of IGF-1 on this astrocytic response has yet to be explored. We hypothesized that IGF-1 treatment would ameliorate TBI-induced astrocytic alterations. To test this hypothesis, the lateral fluid percussion injury (FPI) model of TBI was employed. Male rats received FPI or sham, followed by intraperitoneal (i.p.) IGF-1 at 4- and 24-hours post-injury. Brains were collected 3- and 35-days post-FPI, processed, and stained with anti-GFAP to visualize astrocytes in the hippocampus. Utilizing unbiased stereology, GFAP+ cells were quantified in the hilus, molecular layer, and granule cell layer of the dentate gyrus. Quantitative and qualitative assessments of GFAP+ cells are ongoing. Together, these findings will determine the effects of IGF-1 treatment on the astrocytic response to TBI.

This work was supported by the Texas A&M Neural-Gut Immune Axis group and the WoodNext Foundation. LM was supported by the Texas A&M College of Medicine Office of Research.

TEXAS A&M HEALTH
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EXPLOITING DROSOPHILA TO EXPLORE THE GENETIC BASIS OF BACTERIAL INFECTION TOLERANCE

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When exposed to pathogenic infection, host organisms rely on two defense strategies: eliminating microorganisms (resistance) and minimizing damage from pathogens and immunopathology (tolerance). Using *Drosophila melanogaster* and *Pseudomonas entomophila* - a lethal entomopathogenic bacterium - as a host-pathogen model, we examined the defense strategies adopted by flies and the underlying immune-metabolic profiles shaping their responses. Prior work revealed two phenotypic cohorts: high-resistance flies, showing low levels of innate immune activation but efficiently cleared bacteria through defecation driven by lipid mobilization; and low-resistance flies, which had elevated immune signaling that limited lipid turnover and defecation, having higher infection susceptibility. Here, we performed a screen and identified a tolerance-favoring founder line (B6) from the *Drosophila* Synthetic Population Resource that defies typical resistance-related energetic trade-offs. Following 20 hours of oral bacterial infection, we assessed survivability, midgut bacterial load and defecation. Fitness and metabolic traits were evaluated through pre-infection egg count and lipid staining of dissected adipose. B6 exhibited heightened survivability with bacterial load and defecation comparable to other lines, suggesting a low-resistance yet high-tolerance phenotype. Remarkably, B6 maintained high fecundity and large lipid reservoir, without mobilizing fat upon infection. Given B6's unique immune-metabolic profile, future work aims to perform genomic Quantitative Trait Locus (QTL) mapping in an F2 population derived from a cross between B6 and a low-tolerance line. After assessing dominance of tolerance traits in F1s, backcrossing to the recessive parent will generate a recombinant population that, when combined with QTL mapping, will be used to identify candidate genes driving infection tolerance.

This work was supported by an NIH NIDDK grant to JK. NN was supported by the Texas A&M College of Medicine Office of Research.

TEXAS A&M HEALTH
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IMPACT OF AUTOCRINE SPHINGOSINE-1-PHOSPHATE SIGNALING ON LYMPHANGIOGENESIS

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Lymphatic vessels play essential roles in fluid homeostasis, inflammation resolution, immune cell trafficking, and tissue remodeling. Lymphangiogenesis, the expansion of the lymphatic vasculature, is often driven by inflammation to support these roles. A key regulator of lymphatic immune functions is sphingosine-1-phosphate (S1P), a chemokine secreted by lymphatic endothelial cells (LECs) via the transporter Spinster-homolog-2 (Spns2). Interestingly, past studies demonstrated the loss of LEC S1P signaling through loss of S1P receptor (S1PR1) signaling inhibits lymphangiogenesis. To test whether this inhibition was due to a potential autologous feedback loop, the Rutkowski laboratory developed a mouse model with inducible LEC-specific deletion of Spns2, inhibiting LEC S1P secretion. To test whether the hypothesis that LEC-secreted S1P was necessary for lymphatic remodeling, several in vivo and in vitro models of lymphangiogenesis were tested. In a tail lymphedema model, mice with Spns2 deletion are expected to develop more severe swelling due to defective lymphatic remodeling. Tail volume was measured over 2 weeks to assess lymphedema. In a kidney injury model that normally shows lymphangiogenesis, kidney tissues were analyzed by immunofluorescence for LYVE1 and podoplanin. For in vitro analysis, LECs were isolated from wildtype and Spns2-deleted mice. These LECs were subjected to various assays testing aspects of lymphangiogenesis: migration, proliferation, and tubulogenesis. If autologous S1P signaling is important in LEC biology, deficiencies in these assays are expected in LECs lacking S1P secretion. Preliminary results from the kidney injury model suggest that Spns2 loss may not strongly impact lymphatic remodeling in comparison to the wildtype mice. Immediate work to confirm genetic Spns2 deletion on induction will be necessary to ensure differences between LECs as the ongoing studies are finalized for data analysis.

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TEXAS A&M HEALTH
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OPTIMIZING HPLC TO DETERMINE KINETIC PARAMETERS OF NOVEL CEPHALOSPORIN ANALOGUES FOR RAPID DIAGNOSIS OF TUBERCULOSIS

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Tuberculosis (TB) is an infectious disease that has persisted partially due to the extremely slow growth rate of the causative agent, *Mycobacterium tuberculosis* (Mtb), complicating treatment and diagnosis. Globally, TB remains a major public health threat, ranking as the leading cause of mortality in humans due to a single pathogen. Existing diagnostic methods are time-consuming, often lack specificity, and are costly, collectively contributing to substandard point-of-care (POC) diagnostics. The demand for innovative diagnostic techniques has led to the development of reporter enzyme fluorescence (REF), which employs BlaC, an enzyme constitutively secreted by Mtb, in conjunction with CDG-3, a modified cephalosporin that, upon cleavage, produces fluorescence. This research seeks to identify the rate of breakdown of CDG-3 by BlaC. Initially, BlaC was purified via affinity chromatography while substrates were synthesized and purified. Enzymology was then conducted using high performance liquid chromatography (HPLC) to determine the rates of CDG-3 cleavage at different concentrations of CDG-3 with BlaC. Our findings indicate that, when optimized via HPLC, BlaC may only turnover CDG-3 once, making it necessary to use short timepoints to determine the catalytic efficiency of BlaC. Additionally, this work has also suggested that the fluorescent dye produced upon cleavage of CDG-3 strongly binds to BlaC, yielding a new strategy to optimize probes for Mtb detection. These studies have provided insight into the kinetic parameters for BlaC cleavage of CDG-3, facilitating progress in development of a novel POC test for diagnosis of tuberculosis.

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TEXAS A&M HEALTH
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2025 SUMMER UNDERGRADUATE RESEARCH PROGRAM

iPSC-MSC EXTRACELLULAR VESICLES (iEVs) FOR TREATING SJÖGREN'S SYNDROME, A COMMON AUTOIMMUNE DISEASE

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Primary Sjögren's syndrome (pSS) is characterized by extensive inflammation and damage to salivary glands (SG) and lacrimal glands via activation of immune cells, especially an imbalance of Treg and Th1/Th17. Damage leads to dry mouth, dry eyes, and reduced quality of life, for which there are only momentary relief treatments. Extracellular vesicles from iPS cell-derived mesenchymal stem cells (iEVs) have shown therapeutic effects on pSS by promoting M2 polarization and decreasing Th17 cells in the spleen, but the molecular mechanisms remain unknown. Therefore, we performed RNA sequencing of spleens from healthy mice, non-treated pSS mice, and mice at 2 or 14 days after iEV treatment. Gene Set Enrichment Analysis (GSEA) and EnrichmentMaps were utilized to compare gene expression of notable gene sets. GSEA confirmed that multiple inflammatory gene sets, such as IFNG signaling, are downregulated in the iEV Day 14 group, as we reported before. Meanwhile, GSEA revealed that glutathione, one-carbon metabolism, and other redox/antioxidant pathways were upregulated in iEV-treated groups, suggesting a scavenging of ROS. Amino acid metabolism, tryptophan metabolism, OXPHOS, and FAO were also upregulated in iEV groups, especially in the Day 2 group, suggesting that iEVs modulate immune responses through these cell metabolism pathways. Together, these data reveal potential molecular mechanisms of iEVs in treating pSS. Future research is needed to determine the iEV contents responsible for modulating these pathways.

This work was supported by NIH grant DE032028 to FL. EW was supported by a Fellowship from NIH R25 DK126642.

TEXAS A&M HEALTH
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2025 SUMMER UNDERGRADUATE RESEARCH PROGRAM

EFFECTS OF PRENATAL ALCOHOL AND/OR CANNABINOID EXPOSURE ON CECAL AND PLASMA METABOLITES IN ADULT MICE OFFSPRING

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Prenatal alcohol exposure (PAE) impairs gestational development, affecting neurodevelopmental and gastrointestinal structure and function. Occasionally, alcohol is paired with psychoactive substances, like cannabinoids, creating exacerbated singular effects on development. Prenatal cannabinoid exposure (PCE), like PAE, compromises gestational development, affecting neurocognitive function and gut metabolism. While studies on gut microbiota and metabolites in offspring with PAE are emerging, the long-term effects of combined alcohol and cannabinoid exposure are unknown. This study investigated long-lasting alterations in gut and systemic metabolites from fecal and plasma samples, identifying metabolite-behavior associations. C57Bl/6J mice were exposed on gestational days 12-15, and adult offspring (approximately 8 months old) were assessed for anxiety-like and alcohol-seeking behaviors. Plasma and cecal samples were collected for metabolite profiling, where distinct cecal and plasma metabolite signatures were observed across exposure groups, with lipid, amino acid, and xenobiotic metabolites as major contributors to the group's separation. Volcano plot analysis showed increased nucleotide metabolites in cecal samples and decreased microbial-host co-metabolites (e.g., enterolactone sulfate, genistein derivatives) in plasma with combined exposure. Correlation analysis linked these metabolic changes with increased alcohol-seeking behavior and impaired motor coordination in adulthood, suggesting that these metabolic signatures may reflect altered gut-brain communication shaped by prenatal exposure. Our findings show that prenatal alcohol and cannabinoid exposure induce long-lasting changes in systemic and cecal metabolites, potentially influencing neurobehavioral health outcomes. Understanding these alterations could provide nutrition-based interventions to manage addiction and help identify gut-derived biomarkers for early intervention.

This work was supported by NIAAA grants AA028406 and AA029594 to RM. MY was supported by a Fellowship from NIH R25 DK126642.

TEXAS A&M HEALTH
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2025 SUMMER UNDERGRADUATE RESEARCH PROGRAM

THE ROLE OF PERIPHERAL INFLAMMATION IN MICROGLIAL ACTIVATION FOLLOWING ISCHEMIC STROKE IN MIDDLE-AGED FEMALE RATS

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Ischemic stroke is a neurological disorder that happens when blood flow to the brain is blocked due to the formation of a blood clot. Currently, tissue plasminogen activator (tPA) is the only medication for ischemic stroke that utilizes a protein to dissolve blood clots. Post-menopausal women have a higher risk of stroke and more severe strokes due to declining levels of estrogen and other peptide hormones such as Insulin-like Growth Factor 1 (IGF1). Our lab has shown that repairing the gut barrier via intraperitoneal IGF1 reduced peripheral inflammation and improved cognitive function after an ischemic stroke. We hypothesized that if peripheral inflammation is reduced, then microglia will be less activated. Female middle-aged rats were subjected to Middle Cerebral Artery Occlusion (MCAo) via Endothelin-1 (ET1) to induce an ischemic stroke. Immunostaining was conducted on IGF1-treated rat brains for Iba1 and co-stained with either IL-17R, IGF1R, or CD68. Additionally, we did microglia isolation on non-IGF1 treated female adult rat brains, and conducted flow cytometry using CD45, CD32, CD11b, pHrodo Red Zymosan, and Live/Dead. There was no gross difference in immunostaining of Iba-1 with IL-17R or IGF1R. Flow cytometry showed that stroke brains have high CD11b and pHrodo Red Zymosan levels. Future immunostaining will include CD68, which is a more direct marker to show activated microglia. Further adjustments to the flow cytometry voltages, as well as titrations on antibodies, are needed to obtain more optimal readings.

CY was supported by the Texas A&M College of Medicine Office of Research.

Acknowledgements

The Texas A&M University College of Medicine's Summer Research Program (SRP) continues to attract the top undergraduate students from the best colleges and universities across the country. This year we had 22 participants who completed the 10-week program and 2 Air Force Academy Cadets who participated for 6 weeks. These students were selected from a large pool of applicants based on their research experience, desire to attend graduate/medical school, grades, and recommendations. I would like to thank the selection committee who dedicated their time.

I would also like to thank the faculty that gave their time as mentors. You have provided each of these students with a valuable experience that will undoubtedly help them achieve their career goals.

The SRP was made possible by the following who provided funding and programmatic support:

- **Amy Waer, MD, FACS, MPSA – '23 Jean and Tom McMullin Endowed Dean**
- **J. Thomas Cunningham, PhD – Professor & Senior Associate Dean for Research**
- **National Institute of Diabetes and Digestive and Kidney Diseases (R25DK126642)**
- **Air Force Academy Cadet Summer Research Program**

The following faculty and their colleagues selected eligible students to represent their Texas A&M University System school in the SRP:

- **Chamindika Siriwardana, PhD – Texas A&M University – Central Texas**
- **Venu Cheriyath, PhD – East Texas A&M University**
- **Riccardo Mozzachiodi, PhD – Texas A&M University – Corpus Christi**
- **Richard Laughlin, PhD – Texas A&M University – Kingsville**
- **Dennis Daniels, MPH, DrPH – Prairie View A&M University**
- **Chris Mares, PhD – Texas A&M University – San Antonio**
- **Michael Kidd, PhD – Texas A&M International University**
- **Max Sanderford, PhD – Tarleton State University**
- **Nurul Alam, PhD – Texas A&M University – Texarkana**
- **Neil Terry, PhD – West Texas A&M University**

Each week we had roundtable discussions and professional development sessions in which participants got to engage with faculty, guest speakers, and alumni. Participants also learned about Texas A&M University College of Medicine's various graduate and medical programs.

Finally, I would like to thank the Associate Director for Research, **Stacy De Leon**, Senior Administrative Coordinator, **Shelly Daughters,** and student assistant, **Meray Lewis,** who did a lot of work arranging the arrival, housing, registration, and processing of the participants as well as the weekly meetings. Thank you to our poster judges who had an extremely difficult task of picking the best of the best. Thank you, students, for your hard work and for a memorable summer – Gig 'em!

Brett Mitchell, Ph.D., F.A.H.A.
Director, Texas A&M University College of Medicine Summer Research Program

2025 Texas A&M University College of Medicine
Summer Research Program Participants

Nidhi Alle	Cal State University – Los Angeles	Dr. Carl Tong
Sarah Cioffi	Case Western Reserve University	Dr. Shannon Glaser
Kayla Dixon	Texas A&M University	Dr. Cedric Geoffroy
Caleb Elarabi	Texas A&M University	Dr. Samba Reddy
Lauren Featherson	Prairie View A&M University	Dr. Michelle Hook
Celeste Fuentes	Texas A&M University – San Antonio	Dr. Mendell Rimer
Dennis Gao	University of Texas – Austin	Dr. Shaunna Clark
Fatima Gonzalez Laureano	East Texas A&M University	Dr. Helene Andrews-Polymenis
Dheeraj Govu	Texas A&M University	Dr. Mari Muthuchamy
Daniela Hernandez	Texas A&M University – Texarkana	Dr. Xu Peng
Aubrey Jackson	Air Force Academy	Dr. Farida Sohrabji
Troy Jones	Texas A&M University – Kingsville	Dr. Shaunna Clark
Timyee Leung	Tarleton State University	Dr. Jim Song
Khyla Lewis	Texas A&M University – Texarkana	Dr. Raquel Sitcheran
Angel Lozano	Tarleton State University	Dr. Michelle Hook
Ayleen Mendoza	University of Texas – El Paso	Dr. Jeffrey Cirillo
Luke Mosca	Case Western Reserve University	Dr. Lee Shapiro
Nguyen Nguyen	Gettysburg College	Dr. Jason Karpac
William Price	Air Force Academy	Dr. Sarah Bondos
Dragui Salazar	Utah Valley University	Dr. Joseph Rutkowski
Sabine Tovar	East Texas A&M University	Dr. Jeffrey Cirillo
Emma Wewel	West Texas A&M University	Dr. Fei Liu
Madison Ybarra	Texas A&M University – Central Texas	Dr. Rajesh Miranda
Cyrai Young	Prairie View A&M University	Dr. Farida Sohrabji

2025 Texas A&M University College of Medicine

Summer Research Program Seminar Series

Date	Time	Topic	Presenter
5/19	9:00 AM	Orientation and Laboratory Safety Training	
5/22	9:00 AM	Professional Development #1 - Icebreaker	Dr. Brett Mitchell
5/23	9:00 AM	RCR – Data Management	Dr. Brett Mitchell
5/27	12:00 PM	TAMU College of Medicine MD Program	Mr. Fernando Vasquez
5/29	9:00 AM	Professional Development #2 – Career Q&A	Dr. Brett Mitchell
5/30	9:00 AM	RCR – Transparency & Reproducibility	Dr. Brett Mitchell
6/3	12:00 PM	CST*R Grand Rounds	MD/PhD Students
6/5	9:00 AM	Professional Development #3 – Alumni Q&A	Abigail Bauder & Abigail Rodriguez
6/6	9:00 AM	RCR – Animal Subjects	Dr. Ryan Buhrer & Kelsey Johnson
6/10	12:00 PM	TAMU College of Medicine MD/PhD Program	Dr. Carolyn Cannon
6/12	9:00 AM	Professional Development #4 – MD Interviews	Dr. Brett Mitchell
6/13	9:00 AM	Roundtable Discussion – Writing an Abstract	Dr. Brett Mitchell
6/17	12:00 PM	TAMU College of Medicine MS and PhD Programs	Dr. Gillian Bartlett-Esquilant
6/19		Holiday	
6/20	9:00 AM	Roundtable Discussion – Marketing and Interviewing	Dr. Brett Mitchell
6/24	12:00 PM	A&M Rural and Community Health Institute	Dr. Kia Parsi
6/26	9:00 AM	Professional Development #5 – Conflict Resolution	Dr. Vincent VanBuren
6/27	9:00 AM	Roundtable Discussion – Organizing Your Poster	Dr. Brett Mitchell
7/1	12:00 PM	RCR – Scientific Misconduct	Dr. Brett Mitchell
7/3	9:00 AM	Professional Development #6 – Professional Writing	Dr. Barbara Gastel
7/4		Holiday	
7/8	12:00 PM	Roundtable Discussion – Giving a 10 Minute Talk	Dr. Brett Mitchell
7/10	9:00 AM	Professional Development #7 – PhD & MD/PhD Interviews	Dr. Brett Mitchell
7/11	9:00 AM	Roundtable Discussion – Presenting at Conferences Air Force Academy Poster Session	Dr. Brett Mitchell Cadets
7/15	12:00 PM	Roundtable Discussion – Applying to Schools	Dr. Brett Mitchell
7/17	9:00 AM	Professional Development #8 – Communication Styles	Dr. Brett Mitchell
7/21	9:00 AM	Student Presentations	
7/22	9:00 AM	Student Presentations	
7/23	9:00 AM	Student Presentations	
7/24	9:00 AM	Texas A&M University Walking Tour	
7/25	9:00 AM- 2:00 PM	Research Day Poster Presentations, Reception, Keynote Speaker, Awards Ceremony, and Pictures	

Program Director

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Please keep us updated with your contact information and career or school decisions after graduation.

Thank you for your hard work this summer!

TEXAS A&M UNIVERSITY

Naresh K. Vashisht

College of Medicine