Three Minute Thesis Competition
University of South Carolina
November 7, 2025

COMPETITION SCHEDULE

FINALS

3:00-4:30 PM **Close-Hipp 750**

Presenters

- Samin Ashjaei, Communication Sciences and Disorders
- Courtney Carter, Chemistry and Biochemistry
- Devan Gavin-Von Roue, Media Arts
- Mahtab Ghasemi Dogaheh, Drug Discovery and Biomedical Sciences
- Mina Karami, Communication Sciences and Disorders
- Michael Kaven, Biomedical Engineering
- George Merhej, Drug Discovery and Biomedical Sciences
- Benedicta Nashiff, Pharmaceutical Sciences
- Ebenezer Seesi, Mechanical Engineering
- Grace Thaggard, Chemistry and Biochemistry
- Courtney Wright, Biomedical Sciences
- Jia Zheng, Drug Discovery and Biomedical Sciences

Judges

- **Dr. David Banush**, Dean of University Libraries
- **Dr. Stacy Fritz**, Associate Vice President for Student Health and Well-Being
- **Dr. Justin Lawhead**, Assistant Vice President for Career Readiness and Post Graduate Success

Three Minute Thesis Competition
University of South Carolina
November 7, 2025

FINALIST PRESENTERS

Samin Ashjaei
Title: Beyond the Ear: The Brain's Role in Understanding Speech in Noise
Abstract: Background: Age-related hearing loss (ARHL)—the third most prevalent chronic condition—affects ~50% of U.S. adults by age 60 and 80% by 85, limiting speech understanding in background noise and increasing risk of social isolation and cognitive decline. Yet, the brain mechanisms linking aging and communication difficulties remain unknown.
Research objectives: Speech-in-noise (SiN) recognition is a marker of real-world communication challenges beyond audiometric thresholds. We examined whether regional brain atrophy mediates age effects on SiN performance.
Methods: In 208 adults (20–78 y) from the University of South Carolina Aging Brain Cohort, we collected pure-tone audiometry (PTA), word-in-noise (WIN) scores, and T1-weighted MRI. We used the Graphical Brain Association Tool to assess whether gray matter (GM) and white matter (WM) volumes statistically mediated the relationship between age and WIN performance, controlling for total intracranial volume.
Results: Age accounted for 39% of WIN variance. Mediation effects were more widespread across GM than WM, suggesting that age-related decline in WIN performance is driven more by local cortical atrophy than by disruption in the connections between brain areas. Mediating GM regions (thalamus, superior frontal gyrus, dorsal anterior cingulate, precentral gyrus) support auditory-cognitive areas responsible for auditory processing, attention, working memory, and top-down modulation. Mediating WM pathways (superior parietal gyrus, middle frontal gyrus, pontine brainstem) support networks for spatial attention, cognitive control, and temporal resolution.
Discussion: ARHL is not solely a peripheral condition. Age-related atrophy in auditory, attentional, and sensorimotor hubs mediates declining SiN recognition. These findings highlight neural targets for interventions supporting communication and cognitive health in older adults.

Courtney Carter
Title: Electrochemical Sensor for Stress Biomarker Monitoring
Abstract: The continual monitoring of critical stress biomarkers is essential for the diagnosis and treatment of stress-related conditions. Electrochemical methods offer sensitive, selective, rapid, cost-effective, and long-term measurement of such biomarkers. This presentation highlights our recent advancements and future objectives in developing an electrochemical biosensing platform for continuous monitoring of cortisol as a stress biomarker. Our design employs structure-switching nucleic acids, or aptamers, as biorecognition elements to selectively bind to cortisol. While current electrochemical aptamer-based biosensors (E-ABs) show promise, traditional designs that rely on large, planar gold electrodes face challenges in stability and reproducibility due to poor aptamer surface coverage. To overcome

these challenges, we present a novel E-AB design using aptamer-gold nanostructure-modified carbon ultramicroelectrode arrays (Apt-AuNS-CUAs). CUAs contain approximately three billion carbon-based ultramicroelectrodes (~90 nm radius) per square cm, arranged in an array and surrounded by a metal oxide layer. These unique CUA features result in high signal-to-noise ratios, rapid sensor response times, and reduced electrode adsorption. Recent progress has demonstrated the successful electrodeposition of gold nanostructures (AuNPs) onto CUAs via our novel alternating potential pulse (APP) method, enabling aptamer immobilization. Optimization of APP electrodeposition parameters—such as deposition potential, time, and gold ion concentration—has achieved effective AuNS functionalization with large electroactive surface area and improved electrode coverage. Subsequent immobilization of cortisol-specific aptamers on the AuNS-CUA platform has enabled the detection of biologically relevant, nanomolar (nM) concentrations of cortisol. Future studies will further evaluate the analytical performance of this AuNS-CUA E-AB platform for continuous cortisol monitoring in biological fluids.

Devan Gavin-Von Roue
Title: Beyond the Hero's Journey: A Metamodern Approach to Narrative
Abstract: This thesis proposes A Narrative Approach to Metamodernism, an academic and creative exploration of how narrative frameworks can evolve to meet the challenges of a fractured world. It argues that traditional heroic narratives, best exemplified by Joseph Campbell’s monomyth, are an outdated modernist model that prioritizes social obligation and self-sacrifice over personal agency. While postmodernism has offered a critique of these "grand narratives" and their totalizing solutions, it has often led to narrative cynicism and a lack of constructive alternatives.
My creative work, the animated screenplay Rave Wave: Face the Music, serves as a case study for a metamodern approach. The film’s protagonist, a superhuman musician, subverts the traditional heroic call by refusing to sacrifice himself. Instead, he forges a new path, using his abilities not for individual glory but for collective action.
Through a critical analysis of animation theories from scholars such as Michel Chion and Curtis Scott, this thesis demonstrates how animation's unique symbolic elasticity makes it a powerful medium for exploring this metamodern shift. By blending theory with creative practice, this project presents a new framework for storytelling that moves beyond the hero’s journey, offering a pragmatic and hopeful narrative that resonates with contemporary audiences and provides a roadmap for creators seeking to redefine heroism for a new age.

Mahtab Ghasemi Dogaheh
Title: Synthesis of peptides as cyclin F inhibitors: A new approach to cancer treatment
Abstract: Cyclin F, a key cell cycle regulator, mediates the ubiquitination of numerous target proteins through E3 ubiquitin ligase complex (SCF), leading to their degradation by proteasome. These substrates, including RRM2, CP110, NUSAP, Cdh1, CDC6, B-Myb, E2F1-8, RB, P130 and P107 (called RBL proteins), EXO1 and

SLBP are targeted by Cyclin F for ubiquitination and degradation, contributing to cell cycle progression, DNA replication and repair and maintaining genome stability. The degradation of these substrates through Ubiquitination-Proteasome System (UPS) plays a significant role in pathology of several human malignancies, including breast cancer, hepatocellular cancer, lung cancer, renal cell carcinoma, Familial Hodgkin Lymphoma and pancreatic cancer. Therefore, Cyclin F may function as a prognostic biomarker and a promising therapeutic target in cancer.
Retinoblastoma protein (RB) is a prototypical tumor suppressor. This protein and its paralogs (p107 and p130) are critical negative regulators of the cell cycle, primarily by associating with E2F family members to repress gene expression related to cell proliferation, apoptosis, and differentiation keeping them inactive, thereby preventing unscheduled cell cycle entry 9.
Based on the interaction between Cyclin F and P130 and the role of cyclin F in degradation of P130, our hypothesis is that by understanding these interactions and identifying their binding sites, we can design and synthesize peptides which binds to Cyclin F and through inhibiting
Cyclin F, they can prevent its interaction with the substrates. Thereby, preventing substrates degradation would enhance their tumor-suppressing effects in cancer.

Mina Karami
Title: How Does Our Social World Shape the Way We Speak?
Abstract: Older adults often face reduced emotional support and increased social isolation, both of which negatively impact quality of life and cognition. Although social connection has been linked to brain health and cognitive outcomes, its relationship to naturalistic language use remains underexplored. This study examined the association between self-reported social connection and spoken discourse in 117 healthy adults over age 50, drawn from the Aging Brain Cohort at the University of South Carolina.
Participants completed PROMIS measures of social isolation and emotional support, along with two discourse tasks: the Cookie Theft picture description and Cat Rescue narrative. Transcribed language samples were analyzed with CLAN software for features such as propositional density, semantic errors, false starts, and noun usage. Correlational analyses showed that higher emotional support was associated with fewer semantic errors, lower noun proportions, and a trend toward greater propositional density. Greater social isolation correlated with more false starts. Group comparisons indicated that low emotional support was linked to increased errors and differences in noun use, while isolation effects were weaker. Regression models adjusting for demographic factors revealed limited predictive power, with small amounts of explained variance.
Overall, findings suggest emotional support exerts a stronger influence on discourse performance than social isolation. Enhanced emotional support was linked to more accurate and efficient language production. These results underscore discourse analysis as a valuable tool for identifying communication changes related to social-emotional health in aging and highlight the need for longitudinal research and potential applications in remote patient monitoring.

Michael Kaven
Title: Peptoid mimic of Alzheimer's disease amyloid-β protein reduces neurotoxicity of inflammation in vitro
Abstract: In 2024, loved ones and caregivers of individuals with Alzheimer's disease (AD) contributed over 19 billion hours of unpaid assistance. The seventh leading cause of death in the United States, AD is a neurodegenerative disorder distinguished by the deposition of amyloid-β (Aβ) into senile plaques in the brain. Among the few available therapeutics, cholinesterase inhibitors strictly target symptoms like neuroinflammation: another medical hallmark of AD pathology in the brain. New monoclonal antibodies aim to counteract the aggregation of Aβ but are expensive.

Peptoids are protein-like molecules distinguished by centering around an amine nitrogen instead of a chiral carbon. Patented at the University of South Carolina by Dr. Melissa Moss, JPT1 is a peptoid that mimics the amino acid sequence of Aβ and has demonstrated promise as an aggregation inhibitor of Aβ. Prior to this project, effects of JPT1 on differentiated, neuronal-like human cells were unexplored.

Cultures of human macrophage cells in vitro were conditioned with an endotoxin (LPS) in the presence or absence of JPT1 treatment, and the pro-inflammatory cytokine-rich media collected as an agent for treating human neuronal-like cells in vitro. Phase contrast microscopy and metabolic neurotoxicity assays were completed to evaluate the neuronal effects. Images capture greatest death and damage in the "endotoxin alone" group, and assays reveal statistically significant reduction in cytokinemediated neurotoxicity by JPT1. In anticipation of future projects with JPT1 in animal studies, the present results of therapeutic potential in JPT1 usher in new hope for the millions of individuals with AD and their families.

George Merhej
Title: Finding the Right Target – Developing Smarter, Safer Cancer Drugs
Abstract: Polo-Like Kinase 1 (PLK1) is elevated in prostate cancer and is linked to higher tumor grade. Elevated PLK1 is critical to adapt to mitotic stress caused by deletion of a tumor suppressor gene called Pten. This suggests that PLK1 is a promising drug target for prostate cancer patients harboring Pten deletions(Liu et al., 2012). PLK1 has 2 main domains: a highly conserved N- terminal catalytic Kinase Domain (KD), and a less conserved, functionally essential C-terminal Polo Box Domain (PBD), separated by a interdomain linker (Strebhardt & Ullrich, 2006). In the McInnes/Wyatt labs, novel nonpeptidic PBD- binding drugs have been developed, namely Abbapolins (ABBAs). Abbapolins were shown to demonstrate potent antiproliferative activity in prostate and other cancer cell lines, to inhibit PLK1 activity, and to induce PLK1 degradation (Chapagai et al., 2021). Among the several abbapolins that were developed, two were selected to be tested in an animal xenograft model of prostate cancer, based on potent antiproliferative activity in cellular assays and in the NCI-60 tumor panel. Both abbapolins showed significant in vivo anti-tumor activity depicted by reduced tumor volume in treated relative to untreated mice, and decreased PLK1 expression. Related to their anti-cancer activity, abbapolins have been shown to promote conformational changes in PLK1 that are different than those induced by their KD inhibitor counterparts. We have shown that Abbapolins produce ligand induced thermal stabilization of PLK1, while KD inhibitors produce a less thermally stable protein (Chapagai et al., 2023). Current

work is focused on understanding the cellular implications of the different conformations and their effects on PLK1 dimerization. Better understanding of PLK1 conformational dynamics will enhance our knowledge of PLK1 functions in normal and in cancer settings and of PLK1 interaction with other proteins. Moreover, recent data from the NCI-60 panel shows sensitivity of other cancer cell lines to our PLK1 inhibitors and currently, abbapolins are also being tested against some of these. In conclusion, our work investigates novel PLK1 inhibitors in prostate cancer and aims at using those inhibitors as probes for understanding conformational dynamics of PLK1 in cancer and in normal settings.

Benedicta Nashiff
Title: Understanding Reversible and Irreversible Binding Mechanisms of Novel Lysyl Oxidase Inhibitors through Computational Modeling
Abstract: Lysyl oxidase (LOX) is a copper-dependent amine oxidase responsible for crosslinking collagen and elastin in the extracellular matrix. Dysregulated LOX activity contributes to pathological fibrosis and tumor progression, making it an important therapeutic target. Previous studies, including those on β-aminopropionitrile (BAPN) and pyridine-based inhibitors such as PAT-1251, have shown that inhibition can occur through irreversible covalent binding to the lysine tyrosylquinone (LTQ) cofactor in the enzyme, forming a stable Schiff-base adduct that locks the enzyme in an inactive state. Our research aims to determine whether newly synthesized LOX inhibitors follow this same irreversible mechanism or bind in a reversible, non-covalent manner. Using computational approaches, including molecular docking (Schrödinger Glide), AlphaFold-modeled LOX structures, and molecular dynamics simulations, we analyze the interaction of our compounds within the LOX active site. The focus is on the spatial orientation of reactive amine groups relative to the LTQ carbonyl, hydrogen bonding, and metal coordination that could support Schiff-base formation. Comparative analyses with known irreversible inhibitors (BAPN, PAT, and HCTL) are used to identify mechanistic similarities and predict reversibility. Through these studies, we aim to provide molecular-level insight into the binding behavior of our novel inhibitors and establish computational criteria for predicting reversible versus irreversible LOX inhibition. These findings will guide further biochemical validation and support the rational design of selective, controllable LOX inhibitors for the treatment of fibrotic and metastatic diseases.

Ebenezer Seesi
Title: Tiny Oxygen Gaps, Big Power: Turning Waste Heat into Electricity
Abstract: Every day, engines, factories, and power plants release vast amounts of waste heat into the air. Our goal is to capture that heat and quietly convert it into electricity. Thermoelectric generators can do this, but the best existing materials rely on rare or toxic elements, which are unsuitable for large-scale use. We develop a safer, low-cost ceramic called calcium manganese oxide, formed into ultra-thin layers that are thousands of times thinner than human hair. Thin films are ideal for discovering design rules because they allow atomic-scale control and reveal immediate electrical changes, which are harder to observe in bulk materials. Insights from these films can then guide coatings or bulk ceramics for manufacturing.
When oxide films grow, they stretch, which creates tiny missing oxygen atoms. A few of these tiny gaps help electricity flow, but too many act like potholes that slow it

down. Since they cannot be avoided, our goal is to find the right balance. Our experiments show that less stretching yields better performance: relaxed films retain enough missing oxygen atoms to carry charge without damaging the crystal, producing a much stronger electrical response. In fact, relaxed films outperform highly stretched ones by orders of magnitude in key electrical measures.
This gives us a clear path forward: use thin films to tune and optimize these tiny gaps, then carry those winning conditions to scalable coatings or bulk ceramics. The result is a practical route to abundant, non-toxic ceramics capable of converting industrial waste heat into clean, reliable electricity.

Grace Thaggard
Title: Designing a "Synthetic Leaf" through Photochromic Metal-Organic Frameworks
Abstract: As global demands for more sustainable energy sources continues to grow, research and development of synthetic light-harvesting materials for organic photovoltaics (OPVs) and photocatalysts has become a dominant field of research. Despite recent impressive improvements in solar cell efficiency, current state-of-the-art OPVs have only reached ~20% energy transfer (ET) efficiency. In contrast, the natural photosystem, which harvests sunlight to provide energy for plants, relies on the precise arrangement of hundreds of chromophores participating in directional ET pathways in a single leaf to achieve nearly 100% efficiency. Inspired by the highly effective hierarchical arrangement of chromophores in the natural photosystem, this presentation explores how metal-organic frameworks (MOFs), which are crystalline structures composed of inorganic metal nodes connected by organic linkers, can be used as a scaffold to achieve precise organization of light-harvesting molecules resulting in a "synthetic leaf". In particular, photochromic molecules, which undergo isomerization between two distinct forms upon exposure to appropriate excitation wavelengths, can not only harvest light, but also direct ET processes based on their photoisomerization. As a result, photochromic MOFs can harvest light and then funnel energy in pre-designed pathways, thereby mimicking the natural photosystem. Moreover, integrated photochromic moieties behave as "switches" which can be used to tailor ET efficiency using light as a non-invasive external stimulus. Thus, integration of photochromic compounds in well-defined structures creates a "synthetic leaf" that is composed of hierarchically organized chromophores cooperating to harvest, store, and transfer solar energy.

Courtney Wright
Title: Kynurenic Acid, Sleep, and Inflammation During Pregnancy Are Important Therapeutic Targets for Mental Health
Abstract: Maternal inflammation, from infections or prolonged sleep disturbances, during pregnancy is a highly recognized risk factor for compromised offspring neurodevelopment and psychiatric illness. Infections and sleep disruptions elevate tryptophan degradation via the kynurenine pathway and increase kynurenic acid (KYNA) levels, an inhibitor of glutamatergic and cholinergic neurotransmission, both critical to neurodevelopment. KYNA is elevated centrally in adults with psychiatric disorders (schizophrenia, bipolar disorder). Thus, we hypothesize that prenatally elevated KYNA is a mechanistic link between disturbed maternal sleep, inflammation, and poor offspring health.

Wistar rats were manipulated the last week of pregnancy (embryonic day 15-21). Experiment 1: Chronic sleep fragmentation occurred via a novel paradigm that disrupted maternal sleep 18hrs/day (N=4-6). Experiment 2: Maternal diet was supplemented with kynurenine (100 mg/day), direct KYNA bioprecursor, to elevate kynurenine metabolism only (N=6-7). On embryonic day 21, we assessed maternal sleep, maternal plasma cytokines, and metabolites (kynurenine, KYNA) in maternal (plasma, brain) and fetal (placenta, brain) samples.
Sleep fragmentation reduced sleep ($P<0.0001$) and significantly elevated plasma cytokines like IL-1□ ($P<0.01$), a kynurenine metabolism activator. Sleep fragmentation did not alter maternal metabolites, yet elevated fetal brain KYNA ($P<0.05$). Kynurenine diet impaired sleep quality (-10% sleep duration, $P<0.05$), yet plasma cytokines were unaffected. Kynurenine diet elevated KYNA systemically (maternal brain: $P<0.01$; fetal brain: $P<0.0001$). Kynurenine pathway activation was more pronounced in male versus female fetal brain across experimental paradigms. Our models identify elevated fetal KYNA as a molecular consequence of inflammation and sleep disturbances translationally relevant for psychiatric illness and sex-specific neurobiological outcomes.

Jia Zheng
Title: Building with Blocks to Erase Cancer Proteins
Abstract: Many cancers are driven by harmful proteins that have long been considered "undruggable." For decades, our main strategy was to block these proteins, but what if we could completely erase them instead? This is the exciting promise of new medicines called protein degraders, which cleverly hijack the cell's own garbage disposal system to eliminate these harmful targets.
Our research use a novel "building block" method to create these potential drugs. This modular approach allows us to easily design drugs from scratch, much like putting LEGOs together. Excitingly, one of our first molecules shows powerful anti-cancer activity in the lab. But another key question remains: how exactly does it work?
To investigate this, we turn to the power of AI. We use state-of-the-art AI-predicted protein structures to build 3D simulations. This modeling allows us to visualize at how our molecule might be behaving with proteins. This helps us understand its potential and guides our future experiments. This combined strategy of modular chemistry and computational analysis creates a powerful platform to accelerate the discovery of next-generation cancer therapies.