2026 ANNUAL SENIOR SYMPOSIUM

FLORIDA ATLANTIC UNIVERSITY HIGH SCHOOL
JUPITER CAMPUS,
IN PARTNERSHIP WITH
MAX PLANCK ACADEMY

Welcome and thank you for attending the 5th Annual Florida Atlantic University High School - Jupiter Campus, in partnership with Max Planck Academy Senior Research Symposium. This event showcases the high-caliber research projects of the Class of 2026. The students have worked countless hours in laboratories and field research sites to further scientific knowledge and our understanding of the world around us.

Throughout their participation in this program, these 18 senior researchers have secured $25,880 in grant funding and research awards to bolster their research pursuits. The students have also delivered 36 presentations at university, state, and national conferences, and contributed to the publication of three peer-reviewed research articles. One of our seniors, Danylo Fedkiv, was also recently recognized nationally as a Goldwater Scholar for his undergraduate research accomplishments. Shyla Grant was selected as the Wilkes Honors College Undergraduate Researcher of the Year, an honor awarded annually to one student from each college at FAU. This is only the beginning for these amazing students.

The Senior Research Symposium event showcases numerous remarkable research and community collaborators. Among these partnerships, we wish to acknowledge and express gratitude for the outstanding support of FAU High School:

- Max Planck Society
- Max Planck Florida Institute for Neuroscience
- Florida Atlantic University Max Planck Honors Program
- Florida Atlantic University Harriet L. Wilkes Honors College
- Florida Atlantic University College of Science
- Florida Atlantic University Stiles-Nicholson Brain Institute
- Florida Atlantic University Institute for Human Health and Disease Intervention (I-HEALTH)
- Florida Atlantic University Harbor Branch Oceanographic Institute
- The Herbert Wertheim UF Scripps Institute for Biomedical Innovation and Technology

The mentorship provided by the researchers at these institutions has significantly influenced the professional growth of our emerging student researchers. We extend our gratitude to all attendees tonight, as your presence underscores the significance of our students' research efforts in their respective communities.

As we commemorate this significant event, we invite you to immerse yourself in the world of research, inquire about the students' projects, and congratulate our graduating scholars for their invaluable contributions to their research communities.

SYMPOSIUM AGENDA

3:30 pm	|	Check In | Lobby
4:00 pm	|	Research Poster Session | Dreyfoos Atrium
5:05 pm	|	Welcome Remarks | Elmore Auditorium
5:15 pm	|	Research Talks | Elmore Auditorium
5:45 pm	|	Closing Remarks & Photos | Elmore Auditorium

PROGRAM OVERVIEW

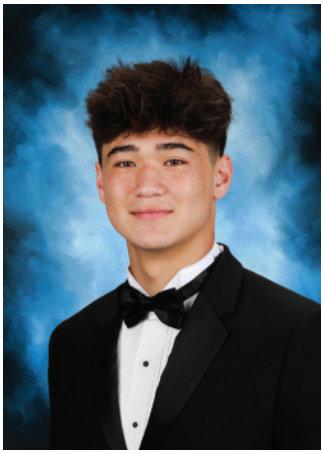
In November 2018, a visionary partnership between Florida Atlantic University (FAU) and Max Planck Academy was formalized with the signing of a Memorandum of Understanding, leading to the establishment of the FAU High School - Jupiter Campus. This unique accelerated pre-collegiate program provides students with the opportunity to earn both a high school diploma and a tuition-free bachelor's degree simultaneously. The school opened its doors in Fall 2020 and has since expanded its impact.

This groundbreaking initiative is the result of the collective vision and dedication of a distinguished group of leaders, including Dr. Patrick Cramer, President of The Max Planck Society; Dr. John Kelly, President Emeritus of Florida Atlantic University; President Adam Hasner of Florida Atlantic University; Dr. Martin Stratmann, former President of The Max Planck Society; Dr. Bill Hansson, former Vice President of The Max Planck Society; Dr. David Fitzpatrick, CEO and Scientific Director of The Max Planck Florida Institute for Neuroscience; Dr. Joe Schumacher, Associate Vice President of Scientific Operations at The Max Planck Florida Institute for Neuroscience; Dr. Robert Stackman, Associate Vice President for Academic Affairs at the John D. MacArthur Campus and Dean of the Graduate College; Dr. Justin Perry, Dean and Professor at the Harriet L. Wilkes Honors College; and Dr. Joel Herbst, Superintendent of FAU Lab Schools. Their unwavering support, along with the invaluable contributions of our distinguished research and community partners, has made this initiative possible. Additionally, the dedicated efforts of the teams at FAU High School and the Max Planck Florida Institute for Neuroscience have been crucial in bringing this vision to life.

As the only program of its kind, FAU High School - Jupiter Campus, in collaboration with Max Planck Academy, stands as a pioneering model for STEM education. Located on the John D. MacArthur Campus at FAU in Jupiter, this innovative program offers high school students the rare opportunity to engage in cutting-edge research at Max Planck and other prestigious partner institutes, actively contributing to scientific discovery as part of their academic journey.

The program integrates seamlessly with the Max Planck Academy's enrichment curriculum, providing students with advanced research skills and specialized knowledge in neuroscience and data science. Through the Wilkes Honors College, students earn significant college credits, pursue STEM majors, and engage in independent research at FAU and other esteemed institutions, preparing them for admission to top-tier graduate and professional programs. Additionally, students contribute to the broader research landscape in South Florida and beyond, enriching the global scientific community.

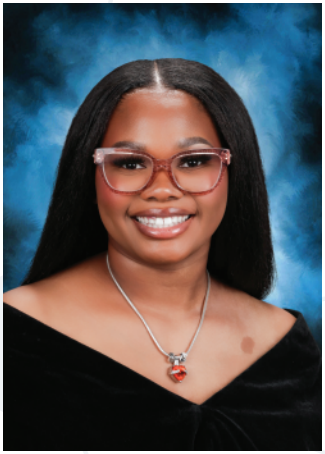
STEVEN *Amorosino*



Research Mentor: Dr. William Ja, The Herbert Wertheim UF Scripps Institute for Biomedical Innovation and Technology

Project Title: Influence of Dietary Sugar on Feeding Behavior of Cropectomized *Drosophila*

Abnormal eating behaviors contribute to metabolic disorders such as obesity. Understanding how feeding behavior is regulated and altered by disease, diet, and treatment is essential for elucidating disease mechanisms and developing interventions. Quantifying human eating behavior is challenging, making *Drosophila melanogaster* a valuable model due to its powerful genetic tools for dissecting neural and genetic mechanisms underlying behavior and metabolism. We have developed a surgical approach, the cropectomy, where the crop—a digestive storage organ—is removed to model aspects of gastric bypass surgery in adult flies. Preliminary results indicate cropectomized flies display altered meal patterns without changes in total daily intake. Using the Activity Recording Capillary Feeding (ARC) assay, we quantify meal structure and total food consumption to assess the combined effects of cropectomy and dietary sugar concentration on feeding behavior. This study's findings will provide new insights into the interplay between *Drosophila* digestive physiology, diet, and ingestive behavior.



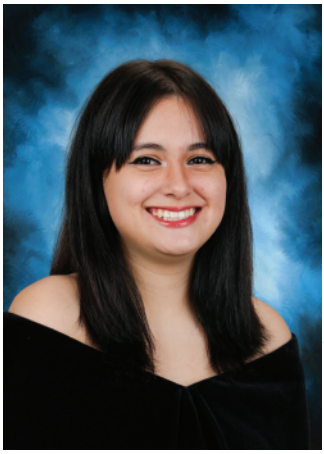
JOHNIYA *Atterbury*

Research Mentor: Dr. Lucia Carvelli, Florida Atlantic University

Project Title: Quercetin modulates the Amphetamine-induced expression of the *sod-3* gene in *Caenorhabditis elegans*

The human body produces an antioxidant enzyme called superoxide dismutase (SOD) to reduce reactive oxygen species (ROS) produced during oxidative stress. Quercetin, a flavonoid antioxidant, is a natural supplement that, like SOD, reduces oxidative stress by depleting ROS. Amphetamine (AMPH) is a drug that when abused can generate addiction, and is also known to increase oxidative stress, causing neurological damage. This project aims to determine whether quercetin can reduce the increase of oxidative stress caused by embryonal AMPH exposure. Using a genetically modified line (GFP-mCherry) of Caenorhabditis elegans, which expresses the green fluorescent protein (GFP) at the promoter of the sod-3 gene (P_{sod-3}::GFP) and the red fluorescent protein (mCherry) at the promoter of the dopamine transporter (dat-1) gene (P_{dat-1}::mCherry), we performed confocal imaging studies to visualize changes of sod-3 expression. Because P_{dat-1}::mCherry is expressed only in dopaminergic neurons, we were able to measure *sod-3* expression specifically in the dopaminergic neurons. We found that embryonal exposure to AMPH significantly increases *sod-3* expression in the dopaminergic neurons of adult animals. Whereas embryonal exposure to quercetin did not have any effect on *sod-3* expression. Interestingly, embryonal co-exposure to AMPH and quercetin caused a slight increase of *sod-3* in adult animals with respect to animals treated with AMPH or quercetin alone. To test if the AMPH-induced increase of *sod-3* had behavioral consequences, we tested animals for a dopamine-dependent behavior named Swip (Swimming induced paralysis). We found that the GFP-mCherry strain behaves the same as the wild-type despite the fluorescent proteins. Using the GFP-mCherry strain, we are currently testing if the Amph-induced behaviors in adult animals are changed by embryonal treatments with quercetin alone or in combination with AMPH. These findings help determine if quercetin could be used as a supplement to reduce dopaminergic deficits during or after AMPH abuse.

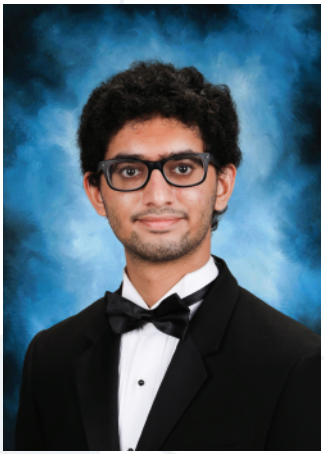
NATALIA *Builes*



Research Mentor: Dr. Robert Stackman, Jr., Florida Atlantic University

Project Title: Modulating Fear Extinction Using the 5-HT_{2C} Antagonist SB-242084

Traumatic experiences can often fuel the development of fearful responses to a once non-threatening cue, leading to persistent fear and anxiety disorders, such as post-traumatic stress disorder (PTSD). Repeated exposure to fear-associated cues in the absence of a threat can reduce fear responses, a process known as fear extinction. Serotonin (5-hydroxytryptamine; 5-HT) is a modulator of brain circuitry implicated in many behaviors, including emotional learning. Our lab has found that 5-HT acting at the 5-HT type 2A receptor facilitates fear extinction. The 5-HT type 2C receptor (5-HT_{2C}R) has also been linked to anxiety-related behaviors and fear memory; yet, its role in fear extinction remains unclear. In this study, we investigated whether pharmacological inhibition of 5-HT_{2C}Rs alters fear extinction. To test this hypothesis, adult male C57BL/6J mice underwent delayed fear conditioning in which each mouse received three presentations of a 30-s tone stimulus paired with a 0.5 mA, 1-s footshock (US). Each mouse subsequently underwent extinction in a distinct context. Prior to extinction training, mice received the selective 5-HT_{2C} antagonist SB-242084 (0.25mg/kg, or 0.5mg/kg), or vehicle (i.p.), followed by 20 non-reinforced CS presentations. SB-242084 treatment did not significantly alter freezing–the measure of fear, extinction rate, or the number of trials required to reach extinction criteria compared with vehicle controls. The findings suggest that inhibiting the 5-HT_{2C}R does not robustly modulate the retrieval of conditioned fear memory or the development of fear extinction under the tested conditions. These results further the understanding of the roles of specific receptors in fear memory and extinction, which is critical for the development of therapeutics that can remediate maladaptive fear present in anxiety-related disorders.



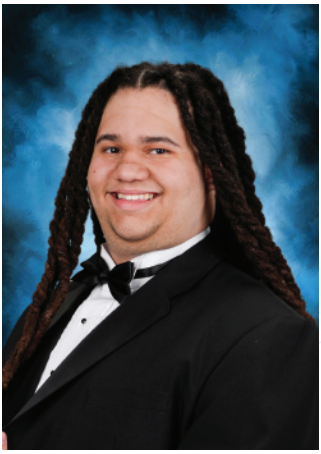
RISHI *Chhibber*

Research Mentor: Dr. Matthew Disney, The Herbert Wertheim UF Scripps Institute for Biomedical Innovation and Technology

Project Title: Covalent Screening of Compounds that Bind to Disease Causing RNAs

Genetic diseases arise from diverse molecular mechanisms, including nucleotide repeat expansions that disrupt cellular function through protein sequestration, misfolding, and formation of toxic RNA or protein structures. Two major examples are myotonic dystrophy type 1 (DM1) and type 2 (DM2), which are caused by expanded repeats in the noncoding regions of the DMPK and CNBP genes and lead to the production of toxic RNAs that sequester RNA-binding proteins such as muscleblind-like 1 (MBNL1), resulting in abnormal pre-mRNA splicing and symptoms such as muscle weakness. Small molecules that bind these expanded RNAs can block protein sequestration and alleviate disease-related defects. To identify such compounds, a high-throughput UV-induced cross-linking assay was developed and optimized to detect RNA-binding molecules, enabling the screening and validation of compound libraries and the discovery of binders for both DM1 and DM2. This platform is now being expanded to other diseases, including prostate cancer, amyotrophic lateral sclerosis, and frontotemporal dementia, and can be completed rapidly to support efficient early-stage discovery. Furthermore, the assay can be completed within a relatively short timeframe, allowing for quick preliminary discovery of binders. Overall, this approach provides a scalable method for identifying RNA-targeting small molecules for repeat expansion disorders and advancing new therapeutic strategies.

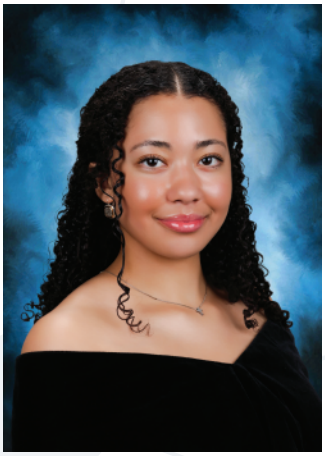
LUCAS *Deese*



Research Mentor: Dr. Jordon Beckler, Florida Atlantic University

Project Title: Finding Sediment Derived CDOM in the Northern Gulf of Mexico

Oceanic sediments represent the largest known carbon store on the planet; In coastal waters, said carbon can be disturbed, and flux out into the ecosystem. Colored dissolved organic matter (CDOM), the visible portion of dissolved organic carbon (DOC) in the water column, has many sources, particularly in coastal ecosystems: fluvial (derived from rivers), biological, and sediment-derived (SD). SD CDOM substantially impacts carbon levels in coastal ecosystems, such as the Northern Gulf of Mexico (NGoM). Due to satellite visibility limitations related to water depth and clarity, SD CDOM is challenging to measure & differentiate via satellite remote sensing (RS). We, using a combination of NASA RS data products and information about the state of the NGoM and surrounding environment, identify SD CDOM, forcibly surfaced by high-energy events in dynamic nGoM waters, by a number of optical and physical qualities of the water. In conjunction with previously developed algorithms to estimate CDOM via RS data, our goal is to develop machine learning approaches to identify SD CDOM. Preliminary results show promise in identifying SD CDOM surfaced by hurricanes. This should allow for the accounting of SD CDOM in global carbon budgets, a key factor in comprehending the substantial yet poorly understood impacts of carbon in coastal sediments on marine ecosystems.



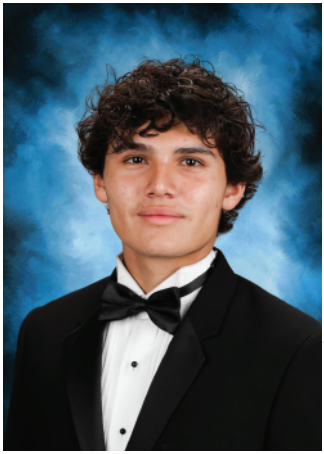
MORIAH *Holland*

Research Mentor: Dr. Eli Chapman, The Herbert Wertheim UF Scripps Institute for Biomedical Innovation and Technology

Project Title: A NanoBRET Platform for the Discovery of NRF2 Inhibitors to treat NRF2 Addicted Cancers

Nuclear factor erythroid 2-related factor 2 (NRF2) is a bZIP Cap'n'Collar transcription factor that protects cells from oxidative and electrophilic stress. NRF2 forms a heterodimeric complex with a small MAF (sMAF) protein and binds to antioxidant response elements (ARE) to initiate production of cellular protection genes. However, like many protective proteins, cancer cells can hijack NRF2 to gain a survival advantage. This is especially true in lung cancer, which has been shown to have high NRF2 (called NRF2-addicted) in 33% of cases. NRF2 overexpression in lung cancer leads to cancer cell proliferation and chemoresistance. For this reason, we are interested in the discovery and development of NRF2 inhibitors. To facilitate this discovery, we created an assay that measures the formation of the NRF2-sMAF-ARE complex. We fused a nanoluciferase to the N-terminus of the Neh1 and Neh3 domains of NRF2 and used an ARE containing a fluorophore. This forms a bioluminescence resonance energy transfer (BRET) pair that gives a signal when NRF2 heterodimerizes with MAFG and this heterodimer binds the ARE. Future studies will identify NRF2 inhibitors that are hypothesized to lead to lung cancer cell death either as a single therapy or as an adjuvant therapy for other cancer drugs.

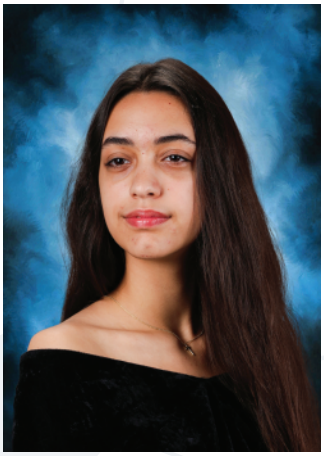
SEBASTIAN *Lopez*



Research Mentor: Dr. Gregg Fields, Florida Atlantic University

Project Title: Effects of MBLAC1 Overexpression and Copper Modulation on Glioblastoma Invasion in 3D Spheroid Models

Aggressive Glioblastoma (GB) is the most dangerous and common type of brain tumor that grows from supporting glial cells in the brain. In patients with this cancer, life expectancy is very poor and treatments remain very limited. MBLAC1 (Metallo-β-lactamase domain-containing protein 1) is a copper-converting enzyme that converts Cu^{2+} to Cu^{+} and a key player in supporting mitochondrial respiration and redox balance. MBLAC1 and its orthologs have shown to regulate Cu(I) homeostasis, mitochondrial ATP production, and oxidative stress, particularly in glial cells, thereby influencing neuronal viability and systemic metabolic function. MBLAC1 expression has also been shown to be associated with favorable outcomes in pancreatic cancer cells, however its role within glioblastoma remains understudied. Some evidence suggests a potential relationship between MBLAC1 and poorer prognosis in glioblastoma, however the mechanism for this is still being studied. MBLAC1's involvement in copper redox biology and mitochondrial function leads researchers to hypothesize that MBLAC1 will modulate tumor growth and can have potential implications in bioenergetics, oxidative stress, as well as invasiveness of glioblastoma. To study the effect of MBLAC1 on the invasion and metastatic potential of glioblastoma, our work has been focused on creating a stable cell line overexpressing MBLAC1 (HEK/GFP-MBLAC1) as well as generating co-culture spheroids of glioblastoma and HEK/GFP-MBLAC1. Preliminary results indicate that overexpression of MBLAC1 leads to enhanced spreading and invasion of several glioblastoma cell lines. Additionally, perturbing copper availability with a copper chelator BCS, as well as treatment with and inhibitor of MBLAC1 (ceftriaxone), significantly reduces glioblastoma matrix invasion, suggesting that MBLAC1-linked copper biology may be a modifiable driver of metastatic potential and could potentially serve as a therapeutic target.



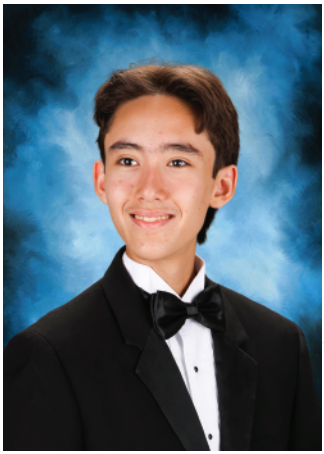
CAMILA *Oliver Sampaio*

Research Mentor: Dr. Nicholas Baima, Florida Atlantic University

Project Title: Roman Spectator Takes on Violence and Bloodshed in the Combat Sports of the Colosseum

Roman gladiatorial games were an important part of Roman culture, particularly in their use of public violence. The games served not just as entertainment for the public but as a way of celebrating heroism through displays of strength, skill, and endurance. This project aims to analyze Roman views on the violent bloodshed in the Colosseum games. A literature search was performed using texts such as David Potter's The Victor's Crown: A History of Ancient Sport from Homer to Byzantium. Evidence from these sources was compared to identify both support for violence and disdain toward bloodshed. Textual evidence suggests that Romans found enjoyment in the violence of the games to an extent, though not always purely for its own sake. The bloodshed was more of a feature in the games that helped display the courage of its participants and add dramatic effect to the event than it was the primary reason audiences attended. Ultimately, the behavior of the crowds and their attitudes toward violence reflect broader Roman cultural values and the foundations of Rome as a war-forged empire.

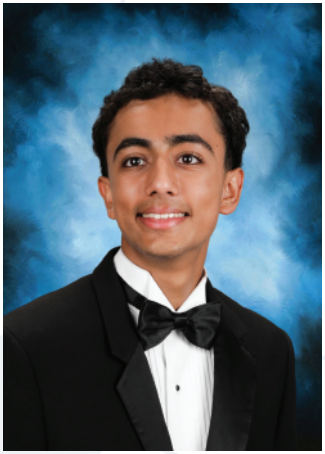
JASON Pindell



Research Mentor: Dr. Lin Tian, Max Planck Florida Institute for Neuroscience

Project Title: A ML Approach for Investigating Phasic Relationships between 5-HT and Fear Responses

Serotonin (5-HT) signaling is critical for fear learning and regulation, but its phasic relationship with fear responses remains poorly understood. We analyzed phasic relationships between 5-HT activity and freezing behavior in the orbitofrontal cortex. Undergoing audio-cued fear conditioning, mice learned to associate a previously neutral stimulus (a 20-s tone) with a 1-second foot shock. Experimental conditions included low shock (0.3 mA), high shock (1.7 mA), and low shock treated with fluoxetine (20 mg/kg). Z-scored 5-HT signals were recorded using fiber photometry and aligned with computer-scored freezing. We initially tested linear regression, generalized linear models, and random forests, but none showed high predictive accuracy. While convolutional neural networks (CNNs) yielded moderate improvement, they failed to capture longer-scale temporal structure. Therefore, we developed a dual-head region proposal network (RPN) designed to model how sustained 5-HT dynamics influence phasic freezing. This approach achieved modest predictive performance, with intersection over union (IoU) scores of 0.54 for low shock, 0.68 for fluoxetine, and 0.74 for high shock. To interpret model behavior, we applied integrated gradients and occlusion-based saliency analyses, revealing distinct temporal epochs contributing to prediction accuracy. While the biological significance of these model-predicted windows remains unknown, their temporal specificity suggests structured relationships that are not captured by standard analyses. Additionally, our analysis indicates that increasing shock magnitude strengthened the serotonin-freezing association, while pharmacologically increasing serotonin availability with fluoxetine replicated a similar effect in the low-shock condition. These findings indicate machine learning approaches can identify latent temporal structure in 5-HT-fear dynamics, supporting a view of serotonin as a context-dependent modulatory system.



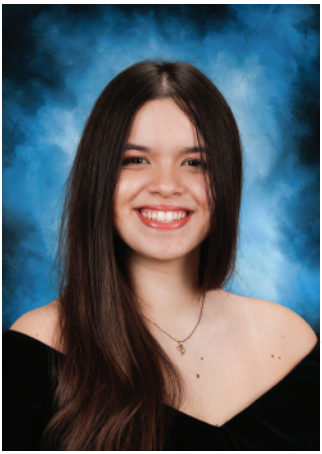
EKANSH *Puri*

Research Mentor: Dr. Ayako Makino, The Herbert Wertheim UF Scripps Institute for Biomedical Innovation and Technology

Project Title: FKBP5 as a Potential Driver of Coronary Endothelial Dysfunction during Menopause

Menopause occurs at the age of approximately 50, and women during the menopause transition exhibit profound disruption of sex hormone levels, accompanied by increased cardiovascular risk. Coronary microvascular disease (CMD) is known to be highly prevalent in menopausal women; however, the molecular mechanisms of menopause-mediated CMD remain poorly understood. Our previous data from bulk RNA sequencing using cardiac endothelial cells isolated from control and menopausal mice indicated that FK506 binding protein 5 (FKBP5) was at most decreased in menopausal mice. FKBP5 is a negative glucocorticoid receptor (GR), and glucocorticoid levels are increased under stress as well as during menopause. Therefore, the objective of this study is to investigate the role of the FKBP5-GR axis in regulating endothelial function. Human coronary microvascular endothelial cells (HCMVECs) will be plated at 2×10^4 cells per well in a 24-well plate and transfected with FKBP5-targeting siRNA (100 nM) or control siRNA for 48 hours. Cells will then be treated with cortisol at 0.01, 0.1, 1, and 10 μM for 24 hours. Endothelial cells release nitric oxide (NO) which helps reduce vascular tone and promote anticoagulation. Therefore, we will measure NO production after treatment. Twenty-four hours after the treatment, NO production will be assessed using DAF-FM diacetate staining and quantified by fluorescence microscopy with a 40× objective. This experiment will help identify the pathophysiological role of FKBP5 in menopause-mediated coronary microvascular dysfunction. We will also examine how FKBP5 levels are regulated by sex hormones and whether overexpression of FKBP5 restores endothelial dysfunction. The completion of our work will lead to a novel treatment for menopause-induced CMD.

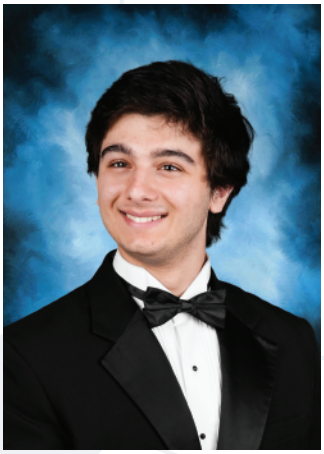
FERNANDA *Salomão Del Bianco*



Research Mentor: Dr. Tanja Godenschwege, Florida Atlantic University

Project Title: Attractin homolog (Dsd) role in metabolism regulation and neuronal function

Attractin (Atrn) is a transmembrane protein linked to neurodegeneration, obesity, and metabolic dysfunction. G-protein coupled receptors (GPCRs), regulated by the Atrn protein family, are essential for cellular signaling and the modulation of metabolism, feeding behavior, and sleep. Dysregulation of GPCR signaling is implicated in metabolic and neurodegenerative disorders including obesity, diabetes, and Alzheimer's disease. Atrn family structure is highly conserved, its homolog in our model organism Drosophila melanogastor is Distracted (Dsd). Previous research has shown Dsd is post-transcriptionally regulated by a large number of micro-RNAs (miRNAs), suggesting that its expression is tightly controlled in a spatio-temporal manner, potentially in response to intrinsic or extrinsic signals such as circadian rhythms. However, how this regulation influences GPCR trafficking and metabolic responses to glucose in insulin-producing cells (IPCs) and the gut remains unclear. This project examines Dsd function in IPCs and gut cells following glucose ingestion using immunohistochemistry, western blotting, and confocal microscopy to assess protein expression, localization, and GPCR regulation. Preliminary data demonstrate that Dsd influences GPCR regulation in IPCs and in the nervous system in response to glucose ingestion, suggesting a role in glucose-dependent signaling pathways. These findings support a model in which Dsd modulates GPCR-dependent mechanisms that regulate glucose metabolism, metabolic homeostasis, and lifespan, potentially through spatio-temporal control of GPCR trafficking.



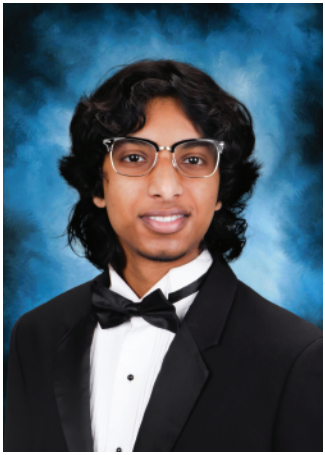
DANIEL *Scher*

Research Mentor: Dr. Deguo Du, Florida Atlantic University

Project Title: MnO_2 Hollow Nanoparticles Modulate Amyloid-Beta Aggregation Pathways in Alzheimer's Disease

Amyloid-beta (Aβ) aggregation into toxic polymers is a hallmark of Alzheimer's disease (AD). This project investigates whether engineered manganese dioxide (MnO_2) hollow nanoparticles can modulate Aβ40 and Aβ42 aggregation pathways. Nanoparticles were synthesized through a redox method and characterized using transmission electron microscopy, dynamic light scattering, and Fourier-transform infrared spectroscopy. Peptides were incubated with and without nanoparticles to assess changes in aggregation kinetics and structural morphology. MnO_2 nanoparticles reduced total fibril formation while increasing the time it took for aggregation to start, altered aggregate structure towards less toxicity compared to untreated controls, and reduced harmful oligomerization byproducts. These findings demonstrate that nanoparticles can function as tunable platforms for influencing protein. If proven safe and efficient, the nanoparticle platform can serve as both a delivery method and part of a treatment for not only Alzheimer's Disease, but adjacent neurological conditions.

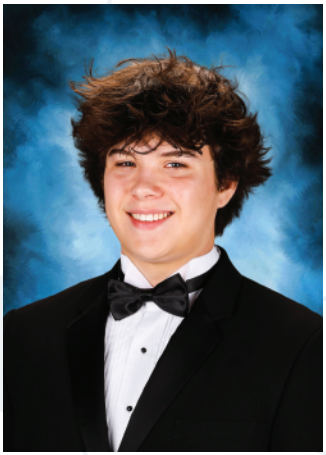
NAKUL *Balaji*



Research Mentor: Dr. Ilyas Yildirim, Florida Atlantic University

Project Title: Computational Construction of a Structural Database for Small Molecules Targeting Disease-Causing RNA Internal Loops

Many neurological and neuromuscular disorders, such as myotonic dystrophy type 1, are caused by expanded RNA repeats in non-coding regions of the genome. These repeats form abnormal structures with dynamic hairpin loops that trap musclebind proteins, disrupting gene regulation. Designing small molecules to target these RNA structures offers a potential therapeutic approach, but experimental screening is challenging due to the highly flexible nature of RNA. Therefore, physics-based simulations provide an accurate and efficient framework for studying such complex systems.



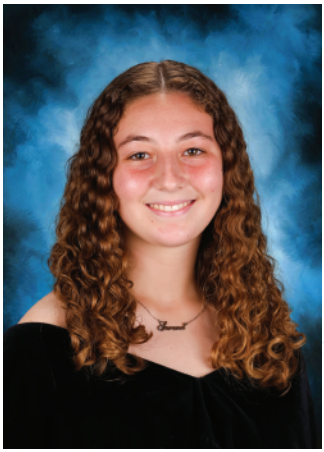
JAKE Thornberry

Research Mentor: Dr. Lawrence Toll, Florida Atlantic University

Project Title: IL-1β Expression and Behavioral Changes in Chronic Inflammatory Pain

Inflammation is a critical defense response that removes harmful stimuli, protects tissue from further injury, and initiates repair. However, chronic inflammation contributes to further pain and pathogenesis. The Interleukin-1 receptor 1 (IL-1R1) mediates inflammatory signaling in response to infection and injury, including pain. IL-1β, a pro-inflammatory cytokine, binds to IL1R1, which is critically involved in pathophysiological pain states through amplifying neuroinflammation. To examine IL-1β expression dynamics during inflammatory pain, we used a mouse line called IL-1-β-TRAP (β-TRAP) that enables temporal and cell-specific labeling of IL-1β-expressing cells via Cre-dependent tdTomato expression. Mice received Complete Freund's Adjuvant (CFA), a pro-inflammatory adjuvant that induces persistent inflammatory pain, followed by 4-hydroxy-tamoxifen to label IL-1β-expressing cells during defined time windows. Following the administration of the inflammatory agent Complete Freund's Adjuvant (CFA), we found that β-TRAP expression increased 1–3 days later. Next, to assess the behavioral correlation to the IL-1β expression pattern, pain behavior was examined on running-wheel after CFA or saline injection. Preliminary data suggest a temporal correlation between IL-1β expression pattern and the use of the running wheel, supporting a potential role of IL-1β signaling in the development of inflammatory pain. Altogether, these findings demonstrate the usefulness of β-TRAP mouse strain for dissecting the in vivo spatiotemporal and cell-type-specific contributions of IL-1β and its receptor system in the development of inflammatory pain.

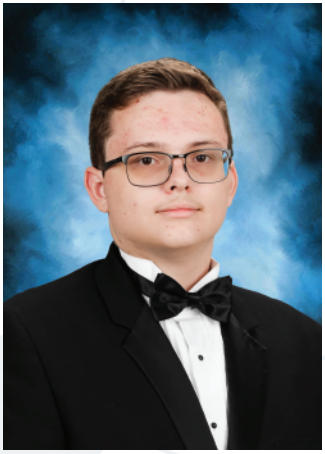
SERENA
Amro Gazze



Research Mentor: Dr. Éric Duboué, Florida Atlantic University

Project Title: Investigating the Neuroanatomy of Adult Astyanax mexicanus Using Fully Segmented, 3-Dimensional Brain Atlases

Animals must adapt to novel environments to meet new behavioral demands, but how brains are shaped by these evolutionary adaptations remains unclear. Astyanax mexicanus provides an effective model for researching these evolutionary changes because its surface-dwelling and cave-dwelling morphs exhibit pronounced differences in neuroanatomical organization while belonging to the same species. Surface fish live in open water and possess functional eyes, while the cavefish live in caves and lack functional eyes. Adult brain atlases were created in this study because they are spatially organized maps of neuroanatomy that allow for the quantitative comparison of brain regions between the morphs. Accordingly, fully segmented, three-dimensional brain atlases were generated for the adult surface fish and Pachón cavefish morphs. To generate these atlases, adult brains were dissected, rendered optically transparent, stained with nuclear dye, and imaged using light sheet fluorescence microscopy. Three-dimensional image stacks were then registered and averaged to produce brain volumes for each morph. Manual segmentation of the brain volumes was then performed using established neuroanatomical landmarks and references, resulting in the labeling of sixteen brain regions for the surface fish and seventeen brain regions for the cavefish. Volumetric measurements were then extracted from all segmented regions, and comparative analyses using fold change calculations quantified the neuroanatomical divergence between the morphs. The results revealed reductions in cavefish visual-processing structures, including the optic tectum, periventricular gray zone layer, and torus longitudinalis, consistent with their evolutionary eye loss. Conversely, cavefish exhibited enlargement of the olfactory bulbs, supporting enhanced reliance on non-visual sensory processing. Additional volumetric differences were observed in the hypothalamus, while many cerebellar and forebrain regions displayed similar volumes between morphs. Together, these atlases provide a quantitative and anatomical framework for characterizing evolutionary changes in vertebrate neuroanatomy.



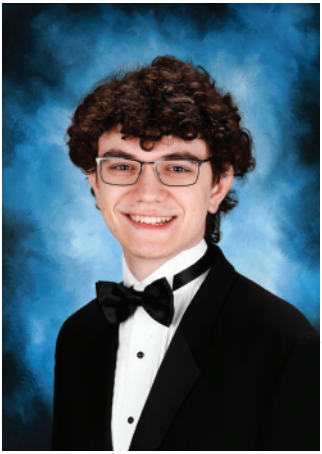
DANYLO *Fedkiv*

Research Mentor: Dr. Gregg B. Fields, Florida Atlantic University

Project Title: Discovery, Synthesis, and Evaluation of Selective Inhibition of MT1-MMP through Novel Small Molecule Inhibitors

Proteins are macromolecular amino acid chains that perform various functions in life, such as maintaining homeostasis and regulating cellular replication; however, when dysregulated, they can contribute to disease. Matrix Metalloproteinases (MMPs) are endogenous zinc peptidases found in the extracellular matrix that degrade extracellular proteins, such as collagen. Membrane Type-1 MMP (MT1-MMP) is a well-studied membrane-anchored protease implicated in tissue remodeling and prognosis; its overexpression can lead to cancer progression and tissue damage through its enzymatic activity, requiring inhibition. This study reports the development of six novel MT1-MMP small-molecule inhibitors, their synthesis, and biological evaluation. The inhibitors were designed and planned for solid-phase synthesis. The resulting inhibitors were purified and analyzed for identity; of the synthesized inhibitors, fractions containing only the desired inhibitors were isolated. Biological evaluation is currently proceeding with MT1-MMP and related proteases to assess inhibitor selectivity. To date, 3 of the 6 inhibitors have been synthesized, and their evaluation is currently underway; the full synthesis of the 6 inhibitors, including their evaluation, is expected to conclude by the end of March. This study establishes scaffolds for MT1-MMP inhibitors that can be modified by replacing select moieties, expanding the frontiers of future MT1-MMP small-molecule inhibitors. This study contributes to the broader development of enzymatic inhibitors that can function as drugs to prevent disease progression and initiation, advancing the field of medicinal chemistry.

ALEXANDER *Castronovo*



Research Mentor: Dr. Naomi Kamasawa, Max Planck Florida Institute for Neuroscience

Project Title: A Semi-Automated Method to Measure Dendritic Spine Head Geometry in 3D Electron Microscopy Reconstructions

Dendritic spines are mushroom-like protrusions that receive synaptic input, making them critical for communication between neurons. Dendritic spine structure is closely linked to synaptic function and plasticity. Prior studies found that dendritic spine head volume is strongly correlated with postsynaptic density (PSD) surface area, an indicator of synaptic strength. High-resolution serial block-face scanning electron microscopy (SBF-SEM) enables imaging and segmentation of dendrites. However, measuring dendritic spine head geometry from a segmentation is a labor-intensive and manual task, limiting analysis scalability and reproducibility. We present a semi-automated pipeline to compute dendritic spine head radius from SBF-SEM dendrite segmentations. The pipeline first finds dendritic spine head center candidates by locating the dendrite's widest point. Candidates are conservatively filtered to remove invalid candidates while prioritizing a low false-negative rate to reduce human proofreading effort. In one dataset, 53% (111 of 209) of candidates were accepted, reflecting intentionally conservative filtering designed to minimize false negatives. Finally, the pipeline uses a ray-tracing algorithm to compute spine head radius from the accepted head centers. We found that dendritic spine head radius was correlated with established proxies of synaptic strength. Preliminary results showed a correlation between dendritic spine head radius and PSD area (Spearman correlation $r = 0.742$, 95% CI = 0.536 to 0.851, $n = 33$). Radius was also strongly correlated with the cube root of spine head volume (linear regression: $R^2 = 0.907$, $n = 56$). These results support that spine head radius could be a biologically informative metric. Our pipeline substantially reduces analysis time of dendritic spines in SBF-SEM applications. These findings suggest that our approach enables higher-throughput analysis of dendritic spines while maintaining biological relevance.

SHYLA *Grant*

Research Mentor: Dr. Tracy Mincer, Florida Atlantic University

Project Title: Understanding Microbial Adherence to Plastics

Marine plastic pollution now exceeds 5 trillion pieces, and as this massive influx of synthetic material has emerged only in the last century, it is rapidly offsetting the balance of the marine ecosystem. These plastics provide ample novel abiotic surfaces for bacteria to form biofilms where substrate competition is fierce. Adhesive properties are the driving force behind successful biofilm formation; thus, studying adhesion is at the forefront of understanding microbial evolution. The Mannose-Sensitive Hemagglutinin (MSHA) operon is the primary gene locus contributing to this process, specifically the mshA gene, which encodes the fiber in direct contact with substrates and determines biofilm formation on hydrophobic surfaces. Furthermore, AI programs can now generate amino acid sequences from text prompts based on biochemical properties, offering a powerful tool for protein discovery. This study characterizes mshA sequences and structures while evaluating AI-generated predictions. Through adhesion assays, phylogeny, and protein modeling, we will explore relationships among adhesion strength, protein structure, and AI predictions. This project also explores how different microbial strains and mshA alleles affect hydrophobic interactions. Overall, proteins with similar adhesive properties share structural motifs, and AI-generated sequences show high similarity to natural ones. These results bridge biotech, natural history, and basic science. By analyzing the mshA genes responsible for adhesion, we can identify ways to inhibit them, potentially preventing the spread of pathogens in the medical and food industries, while examining how bacteria have adapted to a changing marine environment over time.

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