Research Poster Session

2025 Central Florida Brain Bee

Date: February 8, 2025
Time: 9:00 AM - 6:00 PM
Location: Psychology, UCF Main Campus

Projects

The Role of MDA-9/Syntenin in Glioma INvasion and Metastasis

Presenter: Alex Lieberman, Pine View High School

Abstract:

Glioblastomas are defined as stage 4 gliomas; gliomas are the most common type of primary central nervous system tumors (1, 2). They are invasive and prolific and may overexpress melanoma differentiation associated gene-9 (MDA-9), also be referred to as syntenin (2). MDA-9 interacts with c-Src (proto-oncogene tyrosine-protein kinase Src) to regulate progression and metastasis of human melanoma, and it has roles in cellular functions like adhesion (2, 3, 4). MDA-9 is important in nervous system development, and increased levels of the protein have been detected in various breast and gastric cancer cell lines (2, 5). Importantly, MDA-9 is over-expressed in some glioblastoma/glioma and neuroblastoma tumors and intensifies invasion (2, 5). Gliomas, especially glioblastomas, can be lethal, and at a higher rate for elderly patients; thus, researching treatments such as MDA-9 inhibition is vital.

Müller Glia: Unlocking the Secrets of Retinal Regeneration

Presenter: Selena Shen, Pine View High School

Abstract:

An estimated 401.6 million people around the world suffer from some form of retinal disease which remains a significant clinical challenge due to the retina's limited natural capacity for repair.9 While therapeutic interventions, such as cell transplantation and prosthetic devices, being explored, several challenges persist. These approaches often require the introduction of exogenous genes or cells as well as invasive surgical procedures, which carry the risk of immune rejection and potential tumor formation. 6 Recent research has highlighted an alternative solution: harnessing the regenerative potential of Müller glial cells (MGCs), the primary glial cell in the retina that demonstrates remarkable reparative abilities in non-mammalian species (e.g. zebrafish). By exploring the mechanisms that enable MGCs in non-mammalian species to reprogram and regenerate in zebrafish, this literature review aims to identify potential strategies for overcoming these barriers in mammalian systems. Such insights could inform future therapeutic approaches to address the unmet needs of retinal diseases.

Beta Amyloid Plaques in Alzheimer's Disease in the Brain

Presenter(s): Mikaela Tereshkov & Isabella Zmiyiwsky, Pine View High School

Abstract:

One major effect of Alzheimer's Disease is the accumulation of beta-amyloid plaques which are clumps of molecules that originate from improper cleavage of the amyloid prosecutor protein (APP). APP is typically split apart by y-secretase and a-secretase, resulting in a soluble molecule being let out to be reused to form other

molecules. If another enzyme called -secretase splits apart the protein instead of a-secretase, it will result in an insoluble molecule that forms beta-amyloid plaques. (1) These beta-amyloid plaques are insoluble and are toxic to synapses. This will cause issues synaptic plasticity, resulting in weakened learning and memory. (3) These plaques also activate microglia and astrocytes, causing inflammation. (1) These plaques also interfere with tau proteins, causing issues in the microtubules of nerve cells. This is another major effect of Alzheimer's disease. (6)

How Dopamine Receptor Variation Affects Eidetic Memory

Presenter: Liam Smit, Windermere High School

Abstract:

Genetic variations of dopamine receptors in the brain can be correlated to the processes of eidetic memory. Dopamine is the primary hormone/neurotransmitter in the brain which directly controls how memory works and its connections to the other parts of the brain. The receptors which process dopamine can have slight genetic variations among them, changing their function, this variation also affects memory as a result. I used data sets from various educational, and research-focused websites to analyze the relationship between dopamine and memory. Specifically, data about activating these receptors and analyzing how they affect memory. This allowed me to better understand how memory and dopamine work in tandem, while simultaneously locating how they change regarding eidetic memory. Utilizing this information, I was able to conclude potential ways to improve memory, to a quality that matches or comes close to eidetic memory. My conclusions arrived at the use of different methods to stimulate the dopamine receptors in the brain, leading to improved, almost photographic-like memory.

Crossing blood brain barrier: Transcytosis and bifunctional IgG fusion proteins

Presenter: Rishik Yellu, Pine View High School

Abstract:

Selective permeability of the blood-brain barrier (BBB) is key to protecting the internal environment from toxins and pathogens. Endothelial cells lining blood vessels in the brain play a key role in the regulation of entry into the BBB. Systemic antibody dosing results in CNS exposures with only 0.2%–0.4% of serum/plasma concentrations. Development of antibodies that have affinity to certain proteins on endothelial cells has been studied but their utilization to treat human diseases is still being investigated. Bispecific and other antibodies may play a key role in the treatment of CNS diseases if selective permeability can be achieved. Receptor mediated transcytosis (RMT) utilizes various nutrient transporters including TfR and CD98hc. However, antibodies created against these antigens do not penetrate well through the BBB possibly due to lack of affinity and selectivity by endothelial cells. Therefore, transcytosis enabling module (TEM), a single chain variable fragment fusion was created to improve binding kinetics. Studies have shown desirable levels of antibodies were achieved in the brain using TEM. Among various targeted proteins on the endothelial cells lining BBB, CD98hc binding mAb has better brain

concentration of antibody. Fc mutation was created to prevent FcyR binding to glial cells/macrophages leading to reduced neuroinflammation. It may also extend the half-life of the antibody by enhancing absorption at acidic pH. Various studies have shown moderate affinity may result in optimal concentration as attachment and sequestration can be balanced. In conclusion, receptor mediated transcytosis using TEM is a step forward in improving the brain concentration of certain antibodies that may have utilization in the treatment of various brain disorders. Bifunctional antibodies are now used clinically to treat various cancers but their utilization in the treatment of human brain disorders needs to be studied.

Csnk1e/d Inhibition: A Possible Treatment for Familial Parkinson's Disease?

Presenter: Isabella Diaz Escobar, Timber Creek High School

Abstract:

Parkinson's Disease (PD) is a common neurodegenerative disorder that results from loss of dopaminergic neurons[1]. The disease is characterized by progressive resting muscle tremors, rigidity, bradykinesia, gait disturbances, postural instability, and dementia[1]. In recent studies researchers have investigated the pathology of familial type PD, which is currently unknown. However, it is most commonly linked to a T61I mutation which occurs in the CHCHD2 gene. In this literature review we investigated the relationship between CHCHD2 and the Csnk1e/d protein while also investigating the T61I mutation's effect on Csnk1e/d. It was observed that the CHCHD2T61I then appointed casein kinase 1 epsilon/delta (Csnk1e/d) to phosphorylated α-synuclein. When α-synuclein begins to misfold forming cytosolic aggresomes (aggregation of misfolded proteins in the cell, formed when the protein degradation system of the cell is overwhelmed[3]). However, when using Csnk1e/d inhibition, there was a significant suppression of phosphorylation of neurofilament and α-synuclein. In this literature review we investigated the T61I mutation's effect on Csnk1e/d. Finally, we demonstrated possibility of Csnk1e/d inhibition which is a potential treatment that would, in theory, decrease misfolding in α -synuclein, thus treating Parkinson's disease.

Comparative Analysis of Caffeine and Dietary Polyphenols in Mitigating the Cognitive Effects of Sleep Deprivation in *Drosophila melanogaster*

Presenter: Clara Martins De Bellis Silva, Horizon High School

Abstract:

Sleep deprivation has an extensive negative impact on cognitive function which leads to both a decrease in productive and overall health. This research investigated the effect of caffeine and dietary deprivation in minimizing cognitive functions associated with sleep deprivation in Drosophila melanogaster. Eight experimental groups were examined which combinations between control, sleep-deprived, caffeine-treated, polyphenol-treated to determine the effects. The Drosophilia melanogaster experienced sleep deprivation via an orbital shaking platform and several olfactory tests were utilized to evaluate their cognitive performance based off of response times and memory retention.

The results demonstrated that the caffeine or polyphenol receiving Drosophilia melanogaster had increased cognitive performance when compared to the control

groups. Additional quantitative analysis displayed decreased response times in the treated groups which suggests that dietary polyphenols may serve to address cognitive impairments from sleep deprivation. The potential applications are many as it could be applied to shift workers, students, military personnel, healthcare workers, and more to improve their cognitive performance and enhance their quality of life as well as their productivity. Future research should aim to denude the molecular mechanisms and evaluate the long-term efficacy of polyphenol supplementations.

BNC2 Neurons: The Missing Link in Appetite Regulation

Presenter: Trisha Gokhale, Seminole High School

Abstract:

Leptin, a hormone released by fat cells plays a key role in regulating appetite, energy expenditure and body weight. The appetite-suppressing and metabolism boosting effects of leptin were previously believed to be mediated by two groups of neurons- orexigenic agouti related protein (AGRP) neurons, which rapidly induces appetite and anorexigenic pro-opiomelanocortin (POMC) neurons, which promotes satiety, though more slowly. However, recently, a novel neuronal cluster was identified in the arcuate nucleus of hypothalamus, which expresses both lepR and bnc2 genes, through single-nucleus RNA (SnRNA-seq) sequencing of neurons. These leptin-responsive neurons, when activated, rapidly suppress hunger by directly inhibiting AGRP neurons precisely counteracting the hunger-promoting effect. Deletion of LepR receptors from BNC2 neurons resulted in significant weight gain in mice. The research findings indicate that AGRP and BNC2 neurons function in a yin-yang balance or hunger and satiety. The study expanded the known neural circuit for feeding and offers potential drug targets for treatment of obesity and other metabolic disorders.

Exploring the Therapeutic Potential of Probiotics in Treating Multiple Sclerosis

Presenter: Allison Lowe, Windermere High School

Abstract:

Multiple sclerosis (MS) is a chronic neurodegenerative disease characterized by a heightened immune response that attacks components of the central nervous system. This results in a wide range of neurological deficits that tend to worsen over time. In recent decades, there has been an upsurge in disease-modifying treatments (DMTs) for multiple sclerosis. Many of these treatments must suppress the immune system to combat the overactive immune response present in this disease. Because of this, multiple sclerosis patients receiving immunosuppressants are more susceptible to infections and other severe complications. To minimize these adverse effects, it is crucial to explore less risky alternatives. One recent approach involves probiotics, which have been shown to provide anti-inflammatory and immunoregulatory benefits. I found that administering probiotics to experimental mice (who were induced with an MS-like disease) reduced clinical severity, delayed symptom onset, and decreased inflammation and demyelination—key markers of multiple sclerosis. Treatment also increased expression of the biomarker Foxp3,

suggesting high activity of regulatory T-cells that combat autoimmunity. Research surrounding probiotic therapy for multiple sclerosis is considerably novel. Given these promising results, further investigation could be conducted to discover the specific biological mechanisms that make probiotics a feasible therapeutic agent for this disease. Increased attention to this subject area could support the development of innovative, low-risk treatment options that could ultimately improve the quality of life for multiple sclerosis patients. Until then, probiotics may offer various supplementary benefits to individuals who are already undergoing multiple sclerosis treatment.

Transcranial Magnetic Stimulation Therapy for Cocaine Abuse Disorder

Presenter(s): Dhanya Rao, Zoya Shahzad, Katie Wu, Windermere High School

Abstract:

Cocaine Use Disorder is a disorder that is defined by the compulsive utilization of cocaine, regardless of severe medical and psychological consequences. The presence of drug addiction is associated with cognitive control deficiency - specifically, a decreased range of activity in the dorsolateral prefrontal cortex (DLPFC). The DLPFC is involved in overall cognitive function, such as emotion, decision-making, and goal-directed behavior. It is important to realize that while not all individuals who use cocaine develop CUD, statistics show that out of the 2.2 million people who regularly used cocaine in 2022, more than half fall under CUD criteria. Risk factors include genetic, environmental, and a variety of other demographic and individual factors. It is considered as one of the most heritable mental health conditions, with the heredity risk for females at 65 % and males at 78 %. The diagnostic criteria for Cocaine Use Disorder is grouped into four main areas: 1) physiologic -withdrawals, cravings 2)no control over cocaine usage 3) priority loss - cocaine taking precedence over all other activities/responsibilities 4) other miscellaneous . negative consequences from cocaine usage. As of right now, there are no medications approved by the US Food and Drug Administration that specifically counter Cocaine Abuse Disorder. That's approximately 1.5 million people left without a plausible solution. Transcranial magnetic stimulation is a procedure that operates magnetic fields to stimulate nerve cells in the frontal cortex of the brain, increasing or decreasing neuronal activity (depending on the desired effect). It's a personalizable, noninvasive technique that does not require sedation, and has no lingering side effects. TMS devices operate outside of the body, as a treatment coil is applied to the head, above the designated area. It delivers powerful pulses of magnetic energy. Patient's simply sit on a recliner, stay awake the whole period (length of time various among treatment plans) and resume daily activities after treatment. TMS can be combined with medication and other therapies to maximize recovery time for patients.

Determining the Efficacy of Disease Modifying Immunotherapy in Slowing the Progression of Alzheimer's Disease

Presenter(s): Arianny Jimenez, Timber Creek High School

Abstract:

Alzheimer's disease is a progressive neurodegenerative disorder mainly affecting the hippocampus and cerebral cortex, resulting in low cognitive function, memory loss, attention deficit, dementia development, behavioral changes, and functional impairment (Masters et al., 2015; Aprahamian et al., 2013). AD occurs when amyloid-ß (Aß) is accumulated in plaques among extracellular spaces including blood vessels. AD can also come from protein tau (microtubules) creating intracellular neurofibrillary tangles in neurons (Masters et al., 2015). Patterns show that AD is a result of age, while there have been recent questions rising on other possible causes of AD development (Aprahamian et al., 2013; Ghezzi et al. 2013). Currently, some common forms of symptomatic treatments include cholinesterase inhibitors and memantine(Salomone et al., 2011). Scientific researchers are conducting several clinical trials of disease-modifying therapies (DMTs) in order to prevent the development of AD by decreasing Aß production before accumulation (Ghezzi et al., 2013). Passive immunotherapy is said to be one of the most favorable approach when it come to developing effective DMTs (Salomone et al., 2011). Passive immunotherapy involves aquiring monoclonal antibodies to directly target Aß. Solanezumab is a humanized monoclonal antibody that binds to the Aß peptide central region (Ghezzi et al. 2013; Aprahamian et al., 2013). Specifically, this literature review aims to explore how effective solanezumab, an immunotherapy DMT, is in slowing the progression of AD.

Investigating Abnormalities of the Brainstem's Serotonergic System in cases of Sudden Infant Death

Presenter(s): Noora Abusoua, Timber Creek High School

Abstract:

Sudden Infant Death Syndrome, or SIDS, can be defined as the sudden death of an infant under one year of age that is typically associated with sleep and that remains unexplained after a complete autopsy and death scene investigation [1]. Today, SIDS is among the leading causes of mortality in infants, with reports suspecting numerous cases to be caused by irregular breathing or suffocation during sleep periods [1]. While the cause of such things are not entirely defined, it has been discovered that prone sleeping positions and unsafe sleep environments are possible risk factors for SIDS [2]. When advised to parents to avoid prone sleeping in campaigns such as "Safe to Sleep", incidences of Sudden Infant Death decreased [2]. However, not all infants that sleep prone will experience sudden death nor do all cases of SIDS include babies that were found to have slept in a prone position [3]. Thus, it is possible that underlying factors may attribute risk for Sudden Infant Death Syndrome in infants [3]. With this in mind, I initially constructed a general hypothesis suggesting that the structure of the brain responsible for breathing and sleep, the brainstem, paired with the risk of an infant's anatomical immaturity, has a possible correlation to this fatal condition. Moreover, Serotonin, also known as 5 HT, is a neurotransmitter nearly entirely produced within the brainstem that

follows complex pathways throughout the brain and towards the spinal cord [3-4]. Among its numerous functions, 5 HT contributes to respiratory and other autonomic regulations within the medulla for homeostatic conditions [3-4] . 5 HT's effects are exclusively reliant on its binding to synaptic receptors with 5-HT 1A receptors being amongst the most abundant [3]. However, it's been established that abnormalities within the 5 HT medullary system is due to 5 HT binding deficiency, involving roughly 70 % of SIDS deaths [3].