Spring 2023

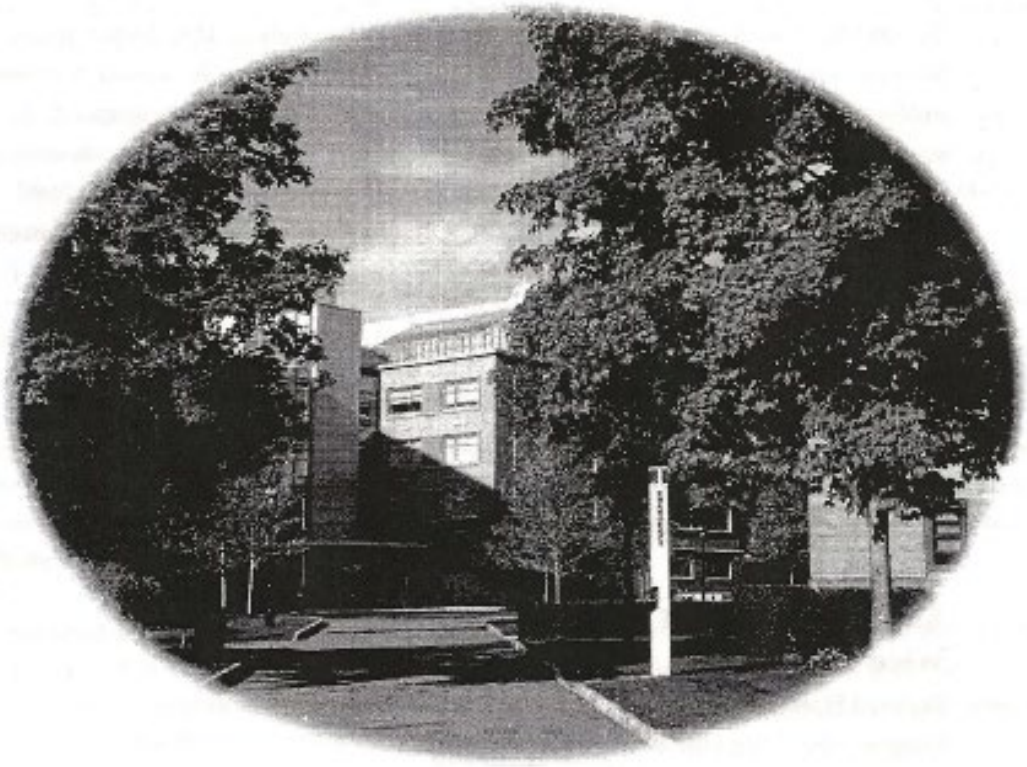
Book of Abstracts

Seton Hall University

SETON HALL UNIVERSITY

College of Arts & Sciences

Department of Biological Sciences



15th Annual Biological Sciences Symposium

Observe, Explore, Achieve

Abstract Booklet

Spring 2023

The Biological Sciences Symposium is a proud participant in the Annual Petersheim Exhibition at Seton Hall University

Schedule of Events

Jubilee Hall – 4th Floor Atrium

3:00 pm **Opening Remarks**

Dr. Jessica Cottrell, Chair of Biological Sciences

3:05 pm **Poster Session**

Graduate Research
Undergraduate Research
Senior Seminar Capstone Projects

McNulty Sciences Center – SC-101 Amphitheater

5:25 pm **Keynote Seminar (*Introduction by Dr. Sulie L. Chang*)**

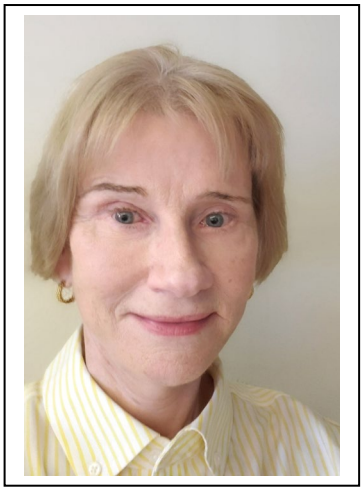
Jean M. Bidlack, PhD
Professor of Pharmacology and Physiology and Associate Chair
Dept. of Pharmacology and Physiology
University of Rochester School of Medicine and Dentistry
Rochester, NY

Title: "Ga subunits selectively modulate opioid receptor signaling"

6:25 pm **Closing Remarks**

Dr. Edward Tall, Biosymposium Committee Chair
Acknowledgements
Announcement of poster winners

Keynote Lecturer



Jean M. Bidlack, PhD

Professor of Pharmacology and Physiology and Associate Chair
Dept. of Pharmacology and Physiology
University of Rochester School of Medicine and Dentistry
Rochester, NY

Jean M. Bidlack, Ph.D. has been a Professor of Pharmacology and Physiology at the University of Rochester School of Medicine and Dentistry since 1997 and Associate Chair since 2013. She graduated from Skidmore College in 1975 with a B.A. in Biology-Chemistry. Immediately following graduation, Dr. Bidlack became a Biophysics Ph.D. graduate student at the University of Rochester. She received her M.S. degree in 1977 and her Ph.D. in 1979. After receiving her Ph.D., Dr. Bidlack joined the Center for Brain Research at the University of Rochester, starting her research on opioid receptors as a postdoctoral fellow. She became an Assistant Professor in the Center for Brain Research in 1982 and an Associate Professor of Pharmacology in 1987.

Dr. Bidlack is Past President and Past Interim Secretary of the Society on NeuroImmune Pharmacology (SNIP). One of her seminal scientific contributions was demonstrating that thymocytes and activated macrophages expressed the kappa opioid receptor, which signaled just like the brain kappa receptor. She received the Herman Friedman Founder's Award from SNIP. Dr. Bidlack was a K05 Senior Scientist supported by the National Institute on Drug Abuse from 1998-2008. From 2004-2012, Dr. Bidlack was Treasurer of the International Narcotics Research Conference. During her career, Dr. Bidlack collaborated with many medicinal chemists. With Dr. Mark Wentland from Rensselaer Polytechnic Institute and scientists at Alkermes, Inc., the Bidlack lab helped characterize and advance the opioid antagonist samidorphan to FDA approval in 2021. Dr. Bidlack has published over 170 papers and she holds four patents.

Dr. Bidlack's current research focuses on understanding how the coupling of the opioid receptor to different Gα subunits of the G protein influences the pharmacological properties of opioids. The Bidlack lab is using bioluminescence resonance energy transfer (BRET) technology to understand how positive allosteric modulators regulate opioid receptor signaling. In addition, the Bidlack lab is studying how fibroblast growth factor 21 decreases morphine preference and withdrawal symptoms without affecting antinociception in mice.

ABSTRACTS

GRADUATE RESEARCH

1) ROLE OF GLUT2 S-PALMITOYLATION AT CYS239 IN MEMBRANE TRAFFICKING
Paul Jun, Connor Jaedicke, Jade Hadley, Humza Khawaja, and C.J. Urso
Department of Biological Sciences, Seton Hall University

Palmitoylation is a reversible post-translational modification wherein palmitic acid is covalently added to a cysteine residue of a protein, increasing the molecule's hydrophobicity and membrane affinity, leading to a variety of effects on signal transduction and other cellular functions. Recently, the facilitated glucose transporters GLUT1 and GLUT4, both class I GLUT isoforms, have been found to be palmitoylated. However, it is unknown if other class I GLUT isoforms are also palmitoylated. We noted patterns of amino acid sequence homology among several class I isoforms including evolutionarily conserved cysteine residues analogous to the confirmed palmitoylation sites in GLUT1 and GLUT4, suggesting that other class I GLUT isoforms may also be regulated by palmitoylation. Our bioinformatic data suggested a high probability that GLUT2, the primary transporter of glucose in the liver, may be palmitoylated at Cys239.

Therefore, we first generated recombinant expression vectors to assess the subcellular localization and trafficking of wild-type (WT)- and C239S mutant GLUT2-mCherry fluorescent fusion proteins in human HepG2 hepatocytes by confocal microscopy. We further investigated the effect of C239S substitution on cellular glucose uptake by flow cytometry. Additionally, we compared cell viability and proliferation, metabolic activity, and gene expression among untransfected HepG2 cells and those stably transfected with either WT GLUT2 or C239S GLUT2 constructs. Furthermore, since GLUT2 is able to bidirectionally facilitate glucose transport either into or out of hepatocytes in different metabolic paradigms, we performed these assays in the context of various glycemic states at physiologically and pathologically relevant glucose concentrations. Our data support the hypothesis that GLUT2 membrane trafficking and function may be regulated by palmitoylation at Cys239. Taken together, our study sheds light on a potentially novel mechanism mediating hepatic glucose transport.

2) AN INVESTIGATION OF THE EFFECTS OF INSULIN AND MAGNESIUM ON BONE HOMEOSTASIS IN A 3-DIMENSIONAL MODEL
Katherine Lefferts
Department of Biological Sciences, Seton Hall University

Bone is a dynamic tissue that undergoes formation, degradation, and repair in a constant state of remodeling. Bone remodeling is performed and regulated by three cell types: osteoblasts, osteocytes, and osteoclasts. Bone homeostasis, the balance of bone formation and resorption, is regulated by biologically active substances secreted by the three bone cell types, as well as circulating hormones and proteins in the body. Insulin is the main anabolic hormone in the body, and its effects on bone homeostasis and regulation are still being uncovered. Magnesium is a trace element that is critical to bone homeostasis. It affects the secretion of hormones that regulate bone cell function and affects structural formation of mineralized bone. However, the exact effects of magnesium on bone cells, as well as ideal concentrations of magnesium in the body are still being elucidated. In this research, we hypothesized that insulin or magnesium would have dose-dependent positive effect on bone formation in a 3D model, based on current knowledge regarding both substances. To evaluate this hypothesis, 3D model bone organoids were developed and treated with increasing doses of insulin or magnesium chloride for 21 days. Spent cell media was collected at 7, 14, and 21 days of post-treatment to evaluate osteoblast and osteoclast function via alkaline phosphatase activity and CTX-liberation, respectively. Bone organoids were harvested at days 14 and 21 to evaluate COX-2 protein expression and calcium deposition. Our data shows that insulin and magnesium showed some dose-dependent effects on osteoblast activity. Overall, our data shows that the effects of insulin and magnesium may differ in a 3D organoid model compared to a 2D monoculture or in vivo conditions, which is important to establish for future research using 3D organoid models to evaluate these key substances.

3) **PALMITOYLATION OF GLUT3 AT CYS205 MEDIATES GLUT3 MEMBRANE LOCALIZATION AND FUNCTION**

Hannah Peters, Vanessa Guo, Kassidy Beauzil, Christa Semexan, and C.J. Urso
Department of Biological Sciences, Seton Hall University

GLUT3 belongs to class I of the facilitative glucose transporter family and is the main glucose transporter in neurons. Yet, despite its physiological importance, there are limited studies characterizing the molecular mechanisms of GLUT3 regulation. Recently, others have identified palmitoylation as a regulator of GLUT1 and GLUT4. At present, a role of palmitoylation in regulating GLUT3 biological activity remains unexplored. Our bioinformatic analyses suggested that GLUT3 may be palmitoylated at Cys205. This cysteine residue is highly conserved among all class I GLUT isoforms (GLUT1, 2, 3, 4, and 14) and corresponds to the analogous cysteine residues in GLUT1 and GLUT4 recently confirmed in the literature as palmitoylation sites.

In this study, we experimentally investigated whether GLUT3 is regulated by palmitoylation at Cys205 by first generating a recombinant DNA vector encoding wild-type (WT) GLUT3-mCherry fusion protein. We next performed site-directed mutagenesis to also generate a vector expressing a C205S-substituted GLUT3-mCherry mutant. Data generated in neuronal cell lines stably expressing these vectors support our hypothesis that GLUT3 may be palmitoylated at Cys205. Under confocal microscopy, we observed that WT, but not C205S GLUT3, was membrane-localized, suggesting an essential role of Cys205 palmitoylation in GLUT3 translocation from the cytoplasm to the cell membrane, and consequently in facilitating cellular glucose uptake. The functional significance of GLUT3 palmitoylation at Cys205 was investigated by comparing glucose uptake in WT- and C205S-transfected vs. untransfected cells by flow cytometry. Our data supported a role of GLUT3 palmitoylation in glucose uptake. The biological relevance of Cys205 palmitoylation was further interrogated by assaying other cellular processes. Results of these studies implicate GLUT3 palmitoylation in cell viability and proliferation, neuronal metabolism, and gene expression. Taken together, our study provides several lines of evidence to suggest that the biological activity of GLUT3 is regulated by palmitoylation at Cys205, revealing a novel mediator of glucose homeostasis in the brain.

4) INVESTIGATION OF ZINC CHLORIDE & CAFFEINE AND THEIR ECTS ON BONE HOMEOSTASIS IN A 3D BONE MODEL

Pooja Shah, M.S.
Department of Biological Sciences, Seton Hall University

Approximately fourteen percent of the human body is composed of bone. The adult human skeleton is made of 206 bones, which make up the internal framework of the body. Osteoblast and osteoclast cells are essential in maintaining the structure and function of bone, for bone homeostasis to occur. Previous data shows that caffeine can potentially have a negative effect on bone homeostasis. Whereas, $ZnCl_2$ has been found to have a positive effect on bone homeostasis in controlled doses. Published data for treatments of caffeine and $ZnCl_2$ are inadequate. The goal of this study was to determine the optimal concentration range for these treatments in maintaining bone metabolism. $ZnCl_2$ and caffeine were provided in increasing concentrations to a 3D-bone organoid model (3D-BOM) for 21 days. Treatment concentrations were determined based on WHO/EPA guidelines. Alkaline phosphatase activity and alizarin red staining was completed to assess osteoblast differentiation and function respectively. Data shows that in the concentration range of 3μM-30μM, $ZnCl_2$ maintains alkaline phosphatase activity. 3μM and 10μM $ZnCl_2$ concentrations enhanced calcium deposition while 30μM $ZnCl_2$ showed impairment. Caffeine (0.005μM, 0.01μM and 0.1μM) had negative effects on alkaline phosphatase activity or calcium deposition. Data demonstrates that $ZnCl_2$ and caffeine has concentration-dependent effects on osteoblast differentiation/function. In the future, the effects of trace elements on osteoclast function and gene/protein expression will be evaluated. Determining optimal levels of trace elements for bone metabolism can be used to assess the range of disorders and disease caused by disruption in bone homeostasis.

UNDERGRADUATE RESEARCH

5) IDENTIFICATION OF STAT3 TARGET GENES THAT PROMOTE BREAST CANCER METASTASIS USING QIAGEN INGENUITY PATHWAY ANALYSIS (IPA)

Fatima Galicia, Jennifer Pena, Ningberi Z. Tchontchoko, Christina Kim and Marylynn Snyder
Department of Biological Sciences, Seton Hall University

Stat3 is a transcription factor that is a member of the STAT (Signal Transducers and Activators of Transcription) family of genes that has been shown to play a role in various cellular processes including tumorigenesis, cell migration, cancer metastasis, and stem-cell self-renewal. Stat3 has been shown to promote breast cancer metastasis through the regulation of the fascin gene, which is an actin-bundling protein. In response to interleukin-6 (IL-6) and oncostatin M (OSM), treatment Stat3 forms a complex with the transcription factor Nuclear Factor Kappa B (NFκB) which binds to the fascin promoter to regulate its expression. Furthermore, Stat3 has been shown to bind NFκB to regulate a subset of Stat3 target genes. The objective of this study was to identify potential Stat3 and NFκB target genes that play a role in breast cancer metastasis. Using Qiagen Ingenuity Pathway Analysis (IPA), we identified a subset of genes that could be potentially regulated by Stat3 to promote breast cancer metastasis. We also identified a subset of genes that could be regulated well by NFκB to promote breast cancer metastasis. Six of these genes are predicted to be regulated by both Stat3 and NFκB, suggesting they may be directly regulated by Stat3/ NFκB to promote breast cancer metastasis. We will further analyze the expression of these genes in cancer cell lines treated with IL-6 or OSM to determine if Stat3 and NFκB regulate the transcription of these genes in the processes of cell migration and metastasis.

6) IDENTIFICATION OF STAT3 TARGET GENES THAT PROMOTE CARDIOMYOPATHY USING QIAGEN INGENUITY PATHWAY ANALYSIS (IPA)

Eric W. Helmer, Carlos E. Aguirre and Marylynn Snyder
Department of Biological Sciences, Seton Hall University

Cardiomyopathy is a major cause of heart failure and death. While hypertrophic cardiomyopathy causes thickening of the heart muscle, dilated cardiomyopathy causes enlargement of the heart chambers. Chronic alcohol consumption is a common cause of dilated cardiomyopathy and can lead to heart failure. Identification of signaling molecules that lead to both types of cardiomyopathy is critical to further understand cardiac physiology as well as identify potential drug targets. It has been shown that Stat3 (Signal Transducer and Activator of Transcription 3) plays a role in both types of cardiomyopathy. Stat3 is a transcription factor that is a member of the STAT family of genes that has been shown to function in various cellular processes. In addition to cardiomyopathy, Stat3 regulates other cellular processes including oncogenesis, cell migration, cancer metastasis and stem-cell self-renewal. Using Qiagen Ingenuity Pathway Analysis (IPA), we identified a subset of genes that could be potentially regulated by Stat3 to promote hypertrophic cardiomyopathy. We also identified a subset of genes that could be regulated by Stat3 to promote dilated cardiomyopathy. Five of these genes are predicted to be regulated by Stat3 to promote both hypertrophic and dilated cardiomyopathy. We will further analyze expression of these genes in cardiomyocyte cell lines to determine if Stat3 regulates transcription of these genes to promote cardiomyopathy.

7) FROM DIRT TO MEDICINE: ISOLATION AND CHARACTERIZATION OF TWO ANTIBIOTIC-PRODUCING BACILLUS SPECIES FROM SOIL

Alexa Minniti, Louis Prinzo, Judy Sagha, Orlene Raymond, and C.J. Urso
Department of Biological Science, Seton Hall University

Antibiotic resistance is spreading rapidly due to the misuse and over-distribution of antibiotics. The development of new antibiotics is crucial because resistance is growing at a faster rate than novel antibiotics are discovered. Many antibiotics have been discovered from soil owing to the abundance and diversity of soil bacteria populations. The multitude of bacteria present in soil exert ecological pressure on bacterial species to compete against nearby species for resources, and therefore, many have evolved to produce naturally-occurring antibiotic molecules to compete within this niche. We can isolate these producers and extract their antibiotic molecules for use in medicine. To this end, we collected soil samples in our local area in hopes of finding a new antibiotic that would be effective in inhibiting growth of six safe "ESKAPE" pathogens, or non-pathogenic relatives of the six most clinically concerning multi-drug resistant. Our results indicate that we isolated two strains of antibiotic-producing bacteria with anti-ESKAPE activity: *Bacillus proteolyticus* and *Bacillus wiedmannii*. We further characterized these isolates to better understand their biological and chemical properties. Herein we present the results from our studies including ESKAPE screening, Gram-staining, 16S ribotyping PCR and DNA sequencing, biochemical testing, chemical extraction and characterization, and many more!

8) THE 'POSITIVE SIDE' OF GRAM-NEGATIVE BACTERIA FROM SETON HALL SOIL

Raees Rana, Madison Loza, Brooke Loza, Juliyah Bautista, Khadeja Uddin, and C.J. Urso
Department of Biological Sciences, Seton Hall University

Antibiotic resistance is emerging as one of the top threats to global public health. Antibiotic resistance develops when bacteria acquire means to resist antibiotic medications, for example, by degrading or exporting the medication. Due to the misuse and overuse of antibiotics, many bacteria have acquired resistance to several commonly prescribed antibiotics. Of concern, some strains have acquired multi-drug resistance while other strains have now been identified as resistant to all known antibiotic medications. With this, the rapid discovery of new, cost-effective antibiotics is crucial to the future of our collective health and to avoid, or at least delay, the dawn of the Post-Antibiotic Era. In response, we isolated soil bacteria, which often naturally produce antibiotic molecules, and screened these isolates for antibiotic activity against non-pathogenic bacteria closely related to multi-drug resistant strains of clinical concern. Our isolates originated from Seton Hall University soil. We employed several molecular and microbiological techniques to identify and characterize these isolates. All three antibiotic-producing bacterial isolates were identified as Gram-negative: Acinetobacter venetianus is a species of bacteria notable for degrading n-alkanes, Enterobacter sichuanensis has been associated with chronic renal insufficiency patients, and a strictly aerobic bacterium of the genus Sphingobacterium has been observed in isolates from the lichen cladonia on Geogeum Island in Korea. Overall, our goal for this project is to contribute new antibiotic-producing soil bacterial species to the Tiny Earth Database for further chemical and pharmacological screening as potentially new molecules for therapeutic use.

9) DIGGING INTO THE UNKNOWN: DISCOVERING ANTIBIOTIC-PRODUCING BACTERIA IN SOIL

Brianna Urquico, Gilbely De Sala Vasquez, Samira Allen, Bervanie Jules, and C.J. Urso
Department of Biological Sciences, Seton Hall University

Many infections are caused by antibiotic-resistant bacteria. The proliferation of these microorganisms has become an urgent clinical threat, intensified by the slow rate at which new, effective antibiotic medications are discovered. Among the antibiotic-producing bacteria, six species are recognized as being especially concerning. These multi-drug resistant bacteria have been dubbed the "ESKAPE" pathogens – an acronym for their respective genus names and nod to their ability to escape our currently available medications. Therefore, in an attempt to identify new antibiotics effective in treating these resistant stains, we cultured bacteria from soil in our community. We isolated 4 strains of antibiotic-producing bacteria by collecting several different samples of soil, culturing, then patching library plates, and screening these libraries for antibiotic activity against non-pathogenic strains of ESKAPE bacteria. We performed several biochemical analyses, 16S ribotyping PCR with gel electrophoresis and DNA sequencing, and other tests to identify and characterize these producers. Results of these experiments identified our antibiotic-producing isolates as: Pseudomonas hunanesis, Pseudomonas otitis, Pseudomonas glycinae, and Sphingobacterium caeni. Herein we present our methods and results in pursuit of discovering new antibiotics that can be effective in treating different types of bacterial infections.

10) INHIBITORY EFFECTS OF ANTIBIOTIC COMPOUNDS OBTAINED FROM SOIL BACTERIA

Natalie Irwin and Dr. Hill, Ph.D.
Department of Biological Sciences, Seton Hall University

Antibiotic resistance is a critical concern in which infective bacterial pathogens gain an evolutionary advantage to defeat antibiotic function. novel antibiotic compounds were chemically extracted from antibiotic producing bacterium sourced from New Jersey soil samples. Following serial dilution plating of the soil cultures, hundreds of bacterial colonies were screened against one known Gram-positive and one known Gram-negative species on LB Agar plates. Ten soil bacteria colonies that produced a zone of inhibition (producer colonies) on the known plates, were further screened against additional Gram-positive and Gram-negative species. These species are relatives to six highly virulent and antibiotic resistant bacterial pathogens (ESKAPE pathogens). Individual producer colonies were then streaked on an LB agar plate and incubated for 72hrs at 20C. Then, the agar was frozen to -80C for 10min. The frozen agar was moved to 100mL bottles and rocked for 48hrs in excess Ethyl Acetate. The organic fraction was then separated, and ethyl acetate solvent removed by evaporation over 72hrs. Each crude dried antibiotic extract was solubilized in 100uL of methanol. Using a pour plate method, 10uL of extracts were screened against different safe relatives of ESKAPE pathogens. Zones of inhibition confirmed the inhibitory effects of extracted compounds when compared to the original producer colony. Lastly, using serial dilutions of the extracted compounds, zones of inhibition were measured and used to produce a dose-response curve, identifying a possible IC50 for the extracts. The extracts can now be analyzed by HPLC and MassSpec to identify the chemical compounds produced. The information acquired from this study shows that soil bacteria can produce antibiotics that can hopefully counteract the antibiotic resistance crisis.

11) INVESTIGATION OF ELEVATED ASCORBIC ACID CONCENTRATION'S EFFECTS ON CHONDROCYTE VIABILITY AND PROLIFERATION

Matthew McGlynn & Ricardo Gomes-Garcia
Seton Hall University, College of Arts and Sciences, Biological Sciences Department

Pharmacologically-dosed ascorbic acid (PAA) is a proposed cancer therapy that entails the intravenous administration of high concentrations of ascorbic acid to selectively kill the tumor cells of multiple myeloma (Xia 41). It is imperative to evaluate the safety and viability of this proposed therapy before it can be applied in a clinical setting. In this investigation of PAA, ATDC5 cell lines were plated and exposed to ascorbic acid concentrations similar to those proposed in the original study of PAA (Xia 41). 0 mM (negative control), 10 mM (low concentration), and 40 mM (high concentration) of ascorbic acid were applied to ATDC5 cells. The effects of PAA on chondrocyte proliferation and function were assessed via a 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-2H-tetrazolium bromide (MTT) cell proliferation assay, Alizarin Red S staining quantification assay, and cellular reactive oxygen/nitrogen species(ROS/RNS) detection assay. The qualitative and quantitative data collected by these assays support that low doses of ascorbic acid (10mM) do not significantly impair ATD5 viability and proliferation, while high doses of ascorbic acid (40mM) correlate with impaired ATDC5 viability and proliferation.

12) ANTIFOULING PROPERTY OF PHTHALOCYANINE DIMETHYLFORMAMIDE

Javid Saleh-Esa, Ammar Elshikh, and Tinchun Chu
Department of Biological Sciences, Seton Hall University

Algae play a critical role in aquatic ecosystems by providing oxygen for many organisms to survive. However, algal blooms, which are rapid increases in algae populations, can have catastrophic effects on ecosystems as they quickly consume large amounts of oxygen, leading to significant wildlife depletion. In recent years, algal blooms have become more widespread, causing detrimental effects on ecosystems all over the world. This study aimed to investigate the effectiveness of a novel antifouling agent, phthalocyanine dimethylformamide (PC-DMF), in stopping algae proliferation. The efficacy of PC-DMF was tested over an eight-week experiment using two tanks that contained GloFish. Both tanks were subjected to ambient light at specified time points throughout the day. However, the activated tank was fixed with lumen projectors, which provided a constant supply of luminescent light required for the activation of PC-DMF. The environmental parameters, such as temperature, pH, ammonia, nitrate, and nitrite, were carefully monitored to maintain a stable experimental environment for the GloFish to survive adequately. Glass slides coated with PC-DMF, along with epoxy positive control slides coated and uncoated slides, were collected from each tank weekly to observe the antifouling properties of PC-DMF. 100 mL water samples were also collected to conduct microscopic identification, cell enumeration, and DNA extraction. Microscopic identification revealed the presence of various algal species, including Chlorella, Diatoms, Chlamydomonas, Eudorina, Chlorococcum, and filamentous bacteria, with a higher abundance of such organisms observed in the uncoated and epoxy sectionals. The microscopic analysis was carried out for Day 28 and Day 49 which contained significant algal growth. DNA extraction of those selected samples was conducted and optimized. Sequencing will be carried out to identify the species in the collected samples. The preliminary results suggested that PC-DMF could be an effective anti-fouling agent in aquatic environments.

13) ANTIBACTERIAL PROPERTIES EVALUATION OF CARVACROL

Rich K. Patel and Tinchun Chu
Department of Biological Sciences, Seton Hall University

As the issue of antibiotic resistance increases in the medical field, there grows a greater need to develop natural antibacterial alternatives. The aim of this study is to investigate the antimicrobial activity of carvacrol against two Gram-negative bacteria, Escherichia coli (E. coli), Pseudomonas spp., and two Gram-positive bacteria, Bacillus subtilis (B. subtilis), and Staphylococcus spp. Carvacrol, found in Oregano and other essential oils, has shown antimicrobial properties on a wide range of bacterial species. E. coli is known to cause many bacterial infections, including urinary tract infections (UTI), cholecystitis, bacteremia, cholangitis, and other clinical infections such as pneumonia. Pseudomonas spp. can cause diseases such as respiratory tract infections, UTIs, and gastrointestinal infections. B. subtilis can also cause infections such as bacteremia and pneumonia but also endocarditis and septicemia. Staphylococcus spp. is a common causing agent for endocarditis and dermatitis. Microplate antibacterial assay and colony-forming unit (CFU) assays were carried out to evaluate the antibacterial activity of carvacrol. The antibacterial assay results indicated that 0.1% carvacrol significantly inhibited four bacteria species. Moreover, the potential synergistic effect of carvacrol with various antibiotics was also examined. The Kirby-Bauer assay results suggested antibacterial synergism of tetracycline and carvacrol. Additional antibiotics and possible synergism will be included in future studies to assess the best combinatorial effect of antimicrobial agents.

14) EVALUATION OF ANTIBACTERIAL ACTIVITY OF PATCHOULI AND LIPOPHILIC GREEN TEA POLYPHENOL-CONTAINING FORMULATIONS

Augusta Saverimuttu and Tinchun Chu
Department of Biological Sciences, Seton Hall University

One of the most prevalent global health concerns is antibiotic resistance. Very limited new antibiotics are being developed and approved. Our research aims to look towards two natural products, both of which are plant derivatives, as potential alternatives to antibiotics. Patchouli essential oil (PEO) is extracted from the leaves of the Pogostemon cablin, while lipophilic green tea polyphenol (EGCG-P) is derived and modified from Camellia sinensis. Three bacteria, Klebsiella spp., Pseudomonas spp., and Staphylococcus spp. are included in this study. The synergistic effects of antibiotics (erythromycin, streptomycin, tetracycline, and penicillin) were also evaluated. Patchouli alone had limited antibacterial activity but some synergy with antibiotics was observed. The microplate assay showed a minimum inhibitory concentration (MIC) of 1.66 mg/ml, which inhibited bacterial growth for up to 24 hours. The colony-forming unit (CFU) results indicated that PEO could inhibit bacterial growth from 97.24% to almost 100%. Fluorescence-based cell viability assay showed 81.23% inhibition for S. epidermidis, 98.56% for P. fluorescence, and 82.59% for K. aerogenes. F2, a novel EGCG-P-containing formulation showed more significant antibacterial properties. Kirby-Bauer assay results showed that F2 had a better synergistic antibacterial effect with antibiotics. Further experiments are needed to elucidate their antibacterial mechanisms.

15) qPCR Confirmation of Meta-Analysis of Viral Interplay with Necroptotic Pathways

Elizabeth Vydra and Erik Hill
Department of Biological Sciences, Seton Hall University

The poxviridae family (dsDNA group 1) is a family of viruses that evades host immune responses (apoptosis/necroptosis). Poxviridae member Molluscum Contagiosum Virus (MCV) has two genes, MC159 and MC160, both known to inhibit host cell immune processes. MC159 is a viral FLIP (FLICE inhibitory protein), which inhibits NF-κB induced by stimuli, such as activated tumor necrosis factor receptor (TNFR). MC160 works as derivative of a putative vFLIP protein and reduces both TNF-alpha activation in NF-κB and IKK activation, including inhibiting the cGAS/STING pathway that induces IFN-a expression. While the related MC159 and MC160 proteins play a role in host immune evasion, our research specifically focuses on the MC159 gene to uncover common activated/deactivated host necroptotic gene pathways and further our understanding of the evasion of host cell-defense mechanisms employed by these viruses.
To identify putative host pathways, a meta-analysis was performed on public datasets of induced vFLIP proteins from dsDNA genome viral infected cells. Ingenuity Pathway Analysis (IPA) software analyzed available GeneChip data from different dsDNA viral vFLIP expressing cells (Kaposi Sarcoma Herpesvirus and Vaccinia virus) to identify necroptosis related gene targets. The meta-analysis identified 7 putative genes of interest in regulating necroptosis in host cells: MLKL, IFN-G, NF-κB, IL1-A, RIPK3, CASP8, and APAF. For statistical accuracy we implement a fold-change cutoff value of 0-1 across all datasets. We found this value to fit our datasets best. To confirm if the IPA dataset changes were also observed in MC159 expression systems, transcription expression changes MC159 transfected cells were compared to vector with and without necroptotic inducing and apoptosis inhibitor compounds. For this study, HT-29 cells were transfected with either 1ug of MC159 or vector DNA, incubated for 12hrs, then treated with inhibitor cocktails for 12hrs (MC159+N). After transfection and incubation, RNA was collected and was converted into complementary DNA (cDNA) using dT and random hexamers. Next, primers for the 7 genes were designed using Primer-BLAST from NCBI. Quantitative PCR using SYBR-Green with cDNA and designed primers for the 7 genes was performed. The relative quantity of transcripts was calculated using the ΔΔCt values when compared to both GAPDH expression and mock vector transfections. The qPCR data confirms some of the IPA analysis. Both the qPCR and IPA data identifies changes in certain host cell gene transcription regulation elements when apoptosis systems are halted. Results revealed increased expression levels of NFKB and RIPK3, whereas MLKL and CASP8 showed decreased expression levels in both MC159 and MC159+N transfected and treated cells. These findings suggest that MC159 may modulate the expression of key genes involved in the regulation of necroptosis, offering potential insights into viral evasion of host immunity mechanisms designed to prevent viral infections.

16) GROWTH OPTIMIZATION AND CHARACTERIZATION OF LACTOBACILLUS SPP. IN YOGURT

Abdulrahman Abduallah, Connor Dolan, Noah Ibasitas, and Tinchun Chu
Department of Biological Sciences, Seton Hall University

Attributed to having a myriad of health benefits relating to gastrointestinal health, Lactobacillus spp. are common microorganisms in yogurt. Labeled as the largest genus of lactic acid bacteria, Lactobacillus spp. are categorized as facultative anaerobes that are Gram-positive and rod-shaped. Seeking to determine the properties of Lactobacillus spp. within store-bought yogurt, samples were collected from four yogurt brands to conduct growth analysis using various growth media. Throughout this study, in vitro techniques were performed in order to further characterize the Lactobacillus spp. in Chobani®, Siggi'sTM, Nature's Promise®, and LiGHT + FiT® yogurts. Cytological techniques, isolation, and purification were performed to confirm the growth of Lactobacillus spp. In addition, distinct colonies present in the incubation cultures were isolated on Brain Heart Infusion (BHI), Man, Rogosa, Sharpe (MRS), and Tryptic Soy Agar (TSA) plates to optimize the growth condition. DNA was extracted from mixed yogurt samples and isolated colonies for further species identification. Comparative analysis of Lactobacillus spp. will be carried out to provide insights into the probiotic community.

SENIOR SEMINAR CAPSTONE PROJECTS

17) THE ROLE OF CXCL10 AND STAT3 IN THE MODULATION OF COLORECTAL CANCER BY INFLAMMATORY BOWEL DISEASE

Alyssa M. Bulhão and Sedra T. Alabed
Department of Biological Sciences, Seton Hall University

Colorectal cancer (CRC) is the fourth leading cause of cancer death. Inflammatory bowel disease (IBD) has been implicated as a cause of the onset of CRC. IBD is an umbrella term for conditions that induce persistent inflammation of the gastrointestinal tract. We identified two genes, STAT3 and CXCL10, associated with IBD. These genes are significantly upregulated in Crohn's disease and ulcerative colitis, the top two most common types of IBD. Mutated STAT3 activity in CRC promotes cell proliferation and tumorigenesis, while also playing a role in regulating cancer pathways in tumor cells and immune escape. CXCL1 is implicated in angiogenesis, inflammation, wound healing, and cancer. The upregulation of CXCL10 is associated with a higher proportion of protumor immune cells. Both diseases induce an inflammatory response in the colon, increasing the production of pro-inflammatory cytokines that disrupt normal gut stem cell function. This research will investigate the relationship between IBD and CRC. Our hypothesis is that targeting STAT3 and CXCL10 can slow down the progression of CRC. Through an in-vivo study using mice, the STAT3 and CXCL10 genes will be knocked out, a xenograft tumor will be injected into the mice, and the total tumor growth will be measured. We will also inhibit STAT3 and CXCL10 activities using pharmacological inhibitors in SW1417 human CRC endothelial cell line and the cell growth will be measured by cell number. Our research will shed light on the relationship between IBD and CRC and investigate whether STAT3 and CXCL10 can be used as potential cancer therapeutic targets to slow down the progression of CRC in patients. *This research project is proposed as part of our Senior Biology Seminar capstone course*.

18) ROLE OF CCR5 ON THE IMMUNE DEFENSE AGAINST VARICELLA ZOSTER VIRUS INFECTIONS

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C-C Chemokine receptor type 5 (CCR5), a member of the chemokine receptor family, has been reported to mediate cellular immunity. The CCR5 delta 32 (CCR5Δ32) mutation prevents the receptor from being expressed on the cell surface. CCR5Δ32 mutation has been correlated with decreased IgG levels in mice infected with herpes simplex virus-2 (HSV-2). Although HSV-2 and the varicella-zoster virus (VZV) share a similar entrance pathway into the cell, limited research has looked at how the entry of varicella-zoster virus into the cell may be affected by the lack of CCR5. This study proposes to examine the effects of CCR5Δ32 mutation on cellular immunity against the VZV. The study hypothesizes that the CCR5Δ32 mutation would offer resistance against the virus. To test this hypothesis, an in-vivo mouse model will be used to obtain mice with the CCR5Δ32 mutation. Five-month-old mice will then be inoculated with the VZV, and an enzyme-linked immunosorbent assay (ELISA) will be run to measure the amount of IgG present in the blood. The results of this assay would indicate whether the presence of a functioning CCR5 receptor impacts VZV's, and possibly other herpesviruses, entry into the cell. This could provide a new area of interest for the treatment or management of herpesvirus infection. *This research project is proposed as part of our Senior Biology Seminar capstone course*.

19) TREATMENT OF WHITE MATTER DEGENERATION AMONG ALCOHOLICS: MARCHIAFAVA BIGNAMI DISEASE

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Marchiafava Bignami Disease (MBD) is characterized by the demyelination of the corpus callosum and subcortical white matter leading to various symptoms such as ataxia, dementia, dysarthria, gait abnormalities, seizures, and even coma. The disease is found in patients with alcoholism and a corresponding vitamin B deficiency. Part of the debate is if MBD in alcoholics is explicitly caused by ethanol damage to white matter structures, ethanol-induced intestinal damage (leading to decreased intestinal absorption of Vitamin B), or both. This study aims to find the most effective way of treating MBD in alcoholics by fixing structural components of the nervous system along with physical comorbidities resulting from white matter degeneration. We hypothesize that abstinence and vitamin b supplements will attenuate white matter regeneration and facilitate the return of normal body functions such as voluntary control of muscles, the ability to solve problems, think, and have recollection of memory. A rat model with MBD will be treated via abstinence, thiamine supplements, and a combination of both. After treatment, white matter regeneration will be measured through diffusion tensor imaging (DTI), and the return of cognitive function and motor skills will be assessed through memory tests, and rotarod tests. This research into thiamine as a treatment for MBD, will aid in the design of more effective therapeutic measures for those who suffer from detriments of the disease, by allowing for the well-being of the individual to return and eliminate the risk of death. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

20) INTERGENERATIONAL TRAUMA AND EPIGENETIC CHANGES ASSOCIATED WITH NR3C1 GENE AND CORTISOL LEVELS

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Epigenetic changes to one's DNA can occur as a response to factors such as trauma. DNA methylation is a type of epigenetic change that has been found to inhibit the transcription of the nuclear receptor subfamily 3 group C member 1 (NR3C1). Trauma can also induce hormonal changes in cortisol levels. Although numerous research indicates that epigenetic changes can be passed on to first-generation offspring, it fails to address the possibility of epigenetic changes down to the third generation. This proposal aims to assess whether trauma-induced epigenetic changes in the NR3C1gene and alterations in cortisol levels down to the third generation using a mouse model. The experiment will consist of a control group made up of naïve mice and an experimental group that would experience trauma. To enforce trauma, the experimental methods will involve the use of caging the mice and administering electric shocks 60 times a day with each shock increasing each day for 10 days. The data will be collected in each generation through saliva samples and/or blood samples. To analyze DNA methylation, sodium bisulfite sequencing technology will be used, and a cortisol blood test will be used to measure cortisol levels. The significance of doing this is to test the hypothesis which determines if trauma can induce epigenetic changes in the NR3C1 gene and changes in the cortisol levels in third-generation mice. *This research project is proposed as part of our Senior Biology Seminar capstone course*.

21) OPTIMIZING THE EFFECTIVENESS OF ONCOLYTIC VIROTHERAPY THROUGH THE USE OF MODIFIED ADENOVIRUSES

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The use of infectious viruses to treat cancers made a breakthrough with the use of modified Herpes Simplex Virus-1 (HSV-1) to treat melanomas with great effectiveness in reducing metastases and overall tumor size. From these promising results, other infectious viral pathogens have been explored for their anti-tumor properties, most notably the use of adenoviruses. Oncolytic adenoviruses (oAds) display great characteristics for this application, activating the immune system for systemic immunotherapies. oAds are extremely cytotoxic to cancer cells and have the potential to lyse tumor cells upon infection. Cell death is caused through various antitumor immune responses. Onyx-015 is one of the most advanced oAds due to its efficacy in replication in cancer therapies. Onyx-015 lacks the E1B55k protein, and only will replicate in genes where p53 inactive. The p53 gene is a tumor suppressor gene that is degraded by E1B55k protein, therefore should be controlled for increased oAd effectiveness. Increasing the effectiveness of oncolytic adenoviruses is done by the incorporation of heterologous genes into oAds treatment to optimize both infection and cytotoxicity to tumor cells. The integration of these genes improve immune stimulation through effector functions, antigen presentation, T-cell priming, and combatting the tumors immunosuppressive abilities that impact penetrance of the oAds. Currently, promising research is being performed to optimize the integration of heterologous genes that provide oAds with increased penetrance for greater effectiveness in systematic immunotherapies. We hypothesize the use of a novel adenovirus, Onyx-053, a facilitative adenovirus that suppresses the E1B55k gene, in conjunction with Onyx-015, will show that replication of Onyx-015 can occur where p53 is present. Our research will consist of testing the effectiveness of each adenovirus individually and in combination on different colonies of Human Small Airway Epithelial cells. This study can show that this mechanism, the combination of Onyx-015 and Onyx-053 can be used to govern oAd replication in tumor cells, therefore increasing penetrance into tumor cells and oncolytic selectivity. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

22) EFFECTS OF ESTROGEN ON SEROTONIN SYSTEMS AND PSYCHOLOGICAL STATE

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Estrogen and serotonin are key hormones involved in the regulation of menstrual and psychological conditions respectively. Current scientific literature indicates that changes in psychological state occur during pre-menstrual stages (PMS), and that serotonin levels affect psychological state; however, there is not extensive research upon the link between the two. This research project proposes that estrogen level impacts serotonergic signaling and serotonin uptake in the brain. The hypothesis is that an increase in estrogen levels promotes serotonin production or receptor capacity. Inversely, a decrease in estrogen levels is predicted to reduce serotonin production or uptake. To test this hypothesis, the experimental method proposed is to administer varying doses of estrogen to healthy female rats. Experimental groups would be given 0, 0.5 and 0.75mg of oral estradiol supplements daily for a period of three months. Serotonin levels in the brain would be assessed through PET (positron emission tomography) scans, fMRI (Magnetic Resonance Imaging), and behavioral changes will be examined. The results will help determine the presence and strength of a link between estrogen and serotonin. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

23) TREATING HUNTINGTON'S DISEASE THROUGH REMOVAL OF THE HTT GENE USING CRISPR CAS-9

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Huntington's Disease is an autosomal dominantly-inherited neurogenerative disease that is caused by the CAG trinucleotide expansion in exon 1 of the Huntingtin (HTT) gene. CRISPR, or clustered regularly interspaced short palindromic repeats, is a gene editing tool that targets specific sections of genetic code to remove or replace DNA. We hypothesize that the elimination of the polyQ domain of mHTT on the N-terminus, using CRISPR Cas-9 in the striatum, may provide therapeutic effects of Huntington's Disease with increased survival rate. A standard laboratory experiment will be done using heterozygous HTT mutant mice, at the age groups of 3 months, 6 months, and 9 months at the start of the experiment. mHTT gene in the striatum of each age group will be removed using CRISPR Cas-9 procedure. The control group of heterozygous mutant HTT mice, at each age group, will keep the HTT gene, along with a second control group of wild type mice. After removal of the HTT gene, immunostaining with anti-DARRP-32 will be done every two weeks to observe brain activity, and brain and body performance tests will be done every two weeks. The brain and body performance tests will be done through measuring body weight, a balance beam, grip strength, and a rotarod. The results will help to determine if the removal of the polyQ domain of the mHTT gene, using CRISPR Cas-9 in the striatum, is effective in entirety or if it lessens the symptoms of Huntington's Disease. *This project is proposed as part of our Senior Biology Seminar capstone course*.

24) THE EFFECTS OF SOCIAL MEDIA USAGE ON THE HUMAN AMYGDALA, RESULTING IN DEPRESSION

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The amygdala is an area of the brain that is responsible for emotional processing. As the usage of social media increases, the brain's processing of it prevents the human amygdala from functioning at its highest level. We hypothesize that the increased use of social media may impact the function of the amygdala, causing people to be unable to process emotions effectively and resulting in depression. Our study design includes recruiting participants from college campuses who use low and high amounts of social media. The low social media use group spends less than 10 hours on social media each week. The high social media use group spends more than 75 hours on social media each week. The experiment we plan to conduct is a sex balanced case. During the experiment, we plan to observe the participants for four weeks. We plan to measure their gray matter volume using the Voxel Based Morphometry (VBM). We plan to measure the depression using the DSM-IV criteria and 2 interviews with clinical psychiatrists. The supposed gray matter volume reduction specifically in the bilateral amygdala, calculated by the VBM would be considered to be a reduction seen as a reduction of gray matter in the bilateral amygdala. If this holds true, it supports our hypothesis that increased social media usage can lead to depression which in turn affects the amygdala. *This project is proposed as part of our Senior Biology Seminar capstone course*.

25) USE OF INSULIN-LIKE GROWTH FACTORS 1 AND 2 IN TREATMENT OF MUSCULAR ATROPHY

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Insulin-like growth factors 1 (IGF-1) is a hormone that plays an important role in childhood growth and has anabolic effects in adults. Insulin-like growth factor 2 (IGF-2) is a hormone, in contrast to IGF-1, is responsible for fetal growth and development. IGF-2 is still present in the adult body but is viewed at significantly lower levels than at the prenatal stage. Preliminary studies have shown that IGF-1 can be used to fight against the effects of muscular atrophy, which is characterized as a decrease in size and wasting away of muscle mass. There is, however, some controversy surrounding IGF-2 due to the fact that some studies suggest that IGF-2 influences cell growth and survival in normal tissue development while other studies implicate it in the progression of muscle disease. This study will test the hypothesis that IGF-1 and an overexpression of IGF-2, individually and in combination, can be effective treatments that will reduce muscular atrophy and maintain muscle mass in patients. This will be achieved through IGF injections on mice biceps femoris muscle undergoing muscular atrophy. Minor segments of the mice's muscle will be removed through surgery before and after the injections to examine the effects of the IGF treatment on muscle mass. This study will help further the understanding on the effects of IGF-1 and IGF-2 on muscular atrophy. Dylan Aldrich and John Pasquerella contributed equally to this project. *This research project is proposed as part of our Senior Biology capstone course*.

26) BACTERIOPHAGE THERAPY AS A TREATMENT FOR ANTIBIOTIC-RESISTANT BACTERIAL INFECTIONS

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Given the rapid rise in multidrug-resistant bacteria, there is a growing need for alternative treatment methods to combat pathological bacterial infections. The therapeutic use of bacteriophages, phage therapy, is increasingly considered a potential option to treat bacterial infections. Bacteriophages have high host specificity and target only specific bacteria. The objective of this study is to examine the effectiveness of phage therapy in burn mouse models infected with Clostridioides difficile, a spore-forming, Gram-positive bacteria listed as an urgent threat by the Center for Disease Control (CDC) in a 2019 report as the species has high levels of antibiotic resistance and increased rates of mortality. This proposal involves examining the effect that different methods of administrating the phages, such as intramuscularly, intraperitoneally, or cutaneously, have on mice mortality. Based on the results of previous studies, mice receiving phage therapy will have a much higher survival rate than mice who have not. Additionally, intraperitoneal injection of phages will likely result in the highest survival rate across each group. Ultimately, the decreasing ability of antibiotics to effectively manage pathogenic bacteria requires additional research into phage therapy as an alternative treatment. *This research project is proposed as part of our Senior Biology capstone course*.

27) PLAQUE FORMATION EXACERBATED BY DIESEL EXHAUST PARTICLES IN ALZHEIMER'S MICE

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The increase of ambient air pollution and awareness of its impact on the environment over the last several decades has led to studies being conducted regarding the effects of air pollution on the nervous system. The main air pollution particle being studied will be Diesel Exhaust Particles (DEPs) from diesel engine exhaust (DEE), created by modern diesel combustion engines. These particles constitute a large portion of ambient air pollution in urban and industrialized areas leading to increases in human exposure. Exposure to this polluted air is known to result in chronic respiratory tract inflammation, cardiovascular abnormalities, and correlation with higher instances of worsening neurodegenerative disease in humans. Research conducted by Harvard, Columbia, and Emory University's schools of public health indicate strong association between air pollution levels and instances of hospital admissions for several neurological disorders including Alzheimer's Disease (AD), Parkinson's Disease, and dementia. Our current study investigates whether long-term inhalation of diesel engine exhaust (DEE) will increase the formation rate of beta-amyloid plaques and impact motor function in female 5X familial Alzheimer's Disease (5XFAD) mice and their wild type (WT) counterparts. Beta amyloid plaque buildup in the brain is a strong indicator of neurodegenerative disease and the total plaque load signifies the extent of potential brain injury in those affected by these types of diseases. The mice in the control group will be exposed to purified and HEPA-filtered air, whilst the dependent group will be exposed to DEE diluted air (0.95 mg/m^3) that is mixed with exhaust and purified air. The mice that will be exposed to DEE will be exposed via whole body inhalation chambers in separate chambers for 5 days per week and 6 hours a day for either 3 weeks or 13 weeks. After the initial exposure, the mice will be assessed in grip strength, motor coordination, and spatial working memory. The volume of beta-amyloid plaques will also be measured in the cortex and hippocampal brain regions using immunohistochemical analysis of brain slices. The results of this study should indicate whether AD-like nervous system pathology is exacerbated in AD mice models in relation to their DEE exposure, motor behavior, and total plaque load. *This research project is proposed as part of our Senior Biology capstone course*.

28) TREATMENT OF ANTIBIOTIC-RESISTANT DISEASES THROUGH BACTERIOPHAGES

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Bacteria are rapidly evolving to become more resistant to antibiotics. Recent studies suggested that a way to continue to combat these bacteria is through bacteriophages. A bacteriophage is a virus that targets bacteria with high specificity to its host. Bacteria can form a biofilm to shield themselves from harm, which is a contributing factor to antibiotic resistance. It can be a big factor in how hospital devices are colonized by them, and this leads to hospitalized acquired infections. This study aims to review recent studies that focus on phages with antibiofilm properties. This process of looking for phages to kill specific bacteria has been helped by genetic engineering to make phages more specific and more lytic to the kind of bacteria they are trying to kill. Through some in vitro models and in vivo mouse models, it was shown that phages could effectively kill multiple types of bacteria, lower biofilm formation, and with less to no cytotoxicity towards humans. Even in a human test, a phage cocktail of three different phages, with one of those being a genetically engineered phage with its repressor gene removed to make it more lytic to the GD01 strain of Mycobacterium abscessus, the patient showed clinical improvement associated with the phage treatment. *This research project is proposed as part of our Senior Biology capstone course*.

29) CRISPR/CAS9 GENE EDITING AS A POTENTIAL TREATMENT FOR DISEASES

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The Clustered Regularly Interspaced Short Palindromic Repeats-Cas9 (CRISPR/Cas9) system is a promising genetic engineering tool for scientists to edit DNA sequences and alter gene function. This genome editing device induces double-stranded breaks where it can add desirable and remove undesirable alleles in a single process. This property can serve as a therapeutic in several genetic diseases, including cystic fibrosis, cancer, and Huntington's disease. This review aims to explore CRISPR's potential in treating such conditions by using data from clinical trials. Most studies used mouse models generated for cancer, neurological diseases, and other complex diseases. Research mice are genetically altered or knocked out using the CRISPR/Cas9 technology in order to understand disease phenotypes and create new therapies. It has also been proposed that CRISPR/Cas9 genome editing may be employed to correct hereditary mutations in IVF embryos, eradicating genetic disorders from afflicted families by altering an embryo's DNA before implantation into the womb. By treating complex disorders, the prospective applications of CRISPR/Cas9 technology will enhance the quality of people's lives. Additional experiments must be conducted to evaluate this novel technology in various animal models before the clinical trials to optimize delivery methods and address ethical concerns. *This research project is proposed as part of our Senior Biology capstone course*.

30) CRISPR/CAS9: GUIDING INNOVATIONS IN GENE EDITING TECHNOLOGIES FOR CANCER TREATMENT

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Cancer is a leading cause of human death worldwide, with high incidence and mortality rates. Research has demonstrated that genetic mutations play a crucial role in the occurrence and progression of malignant cancers. Traditional cancer therapies are often accompanied by severe side effects due to their lack of selectivity. Conversely, gene editing technologies such as CRISPR (Clustered Regularly Interspaced Short Palindromic Repeats) have emerged as a promising approach for cancer treatment, offering the potential for precise and selective targeting of cancer cells while sparing normal cells. Originally a component of bacterial immune systems, CRISPR-Cas9 acts as a precise pair of molecular scissors that can cut a target DNA sequence when directed by a customizable guide. Cancer cells are defined genetically by the mutations they harbor, commonly single nucleotide substitutions, which allow for specific targeting by CRISPR-Cas9. Recent studies have shown that using specifically engineered CRISPR-Cas9 vehicles can significantly reduce the cell proliferation and survival of treated mutant cells while sparing wild-type cells, highlighting the viability of utilizing CRISPR-Cas9 technology for the selective and accurate targeting of cancer-defining mutations with high specificity (to the extent of differentiating the change of a single nucleotide) allowing for the possible treatment of a wide variety of cancers. This review evaluates the molecular mechanisms of CRISPR/Cas9, discusses novel experimental methods for treating cancers using CRISPR/Cas9, and addresses its limitations and drawbacks, such as off-target editing. *This research project is proposed as part of our Senior Biology capstone course*.

31) EXOSOMES – DIAGNOSTIC BIOMARKER FOR GLIOBLASTOMA

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Glioblastoma multiform (GBM), a malignant form of primary brain tumor, accounts for a high frequency of brain tumors and a high mortality rate. Exosomes, containing biological molecules, are nanoscopic extracellular vehicles (EVs) released from all cells of the body to send signals. An experimental group of six CD-1 nude mice injected with the GBM GL261 cell line is compared to a control group of six CD-1 nude mice. Every week the mice models are imaged for tumor growth, and blood plasma samples are taken. After 16 weeks, the blood plasma is purified using Extracellular Vesicle Capture by AnTibody of CHoice and Enzymatic Release (EV-CATCHER) assay in Western Blot. Quantitative polymerase chain reaction (Q-PCR) is used to determine the presence of the biomarker, which plays a role in tumor proliferation and metastasis. This study suggested that exosomes can be used to determine the presence of glioblastoma at early development stages. *This research project is proposed as part of our Senior Biology capstone course*.

32) WHY GENETICALLY MODIFIED CROPS ARE BAD FOR HUMAN HEALTH AND THE ENVIRONMENT

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A genetically modified organism, more commonly known as a GMO, is a living organism in which the DNA has been altered by genetic engineering. This genetic engineering changes the genome of the organism into something with a more desirable outcome than the natural result would be. This is a common technique in agriculture to help increase the production of crops and control weeds and other insects that can potentially negatively influence a crop's yield. Although GMO crops provide some benefits, such as reduced pesticide use, more nutrients, and more affordable costs, there are more long-term negative impacts that come with them. These negative impacts are on both human health and the environment. From a health standpoint, GMOs can cause a few issues, a large one being allergic responses to the added genomes in the crops. Some more health concerns include antibiotic resistance, toxicity, and organ damage. A study was conducted with three different types of genetically modified maize on rats, and the effects seen by the testers included: effects in the kidney and liver as well as in the heart, adrenal glands, and spleen. The study concluded that data points to hepatorenal toxicity symptoms that may be brought on by the new insecticides unique to each GM corn. Therefore, it is impossible to rule out unwanted direct or indirect metabolic effects of the genetic change. Genetically modified crops do not have many more impacts on the environment than standard crops, but there are still a few effects that occur. For instance, they release toxins into the soil, which can prevent the growth of bacteria that is essential to plant growth. Another negative side effect is the disruption of biodiversity by disrupting the natural process of gene flow. With these being some of the effects caused by GMOs, there are a lot of long-term impacts of GMOs that are now being studied by scientists that were previously overlooked in exchange for healthier and cheaper crops. *This research project is proposed as part of our Senior Biology capstone course*.

33) AN ANALYSIS OF OBESITY AS A MEDICAL CONDITION OR SOCIAL CONSTRUCT IN THE UNITED STATES

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According to World Health Organization (WHO), obesity is the excessive accumulation of fat which represents a risk to an individual's health. Over the years, there has been a huge debate about whether or not obesity should be treated as a medical condition. Some people argue that the medical aspect of obesity is a social construction that was used to justify the stigma around obese people. They also argue that the stigma affects the individual's health more than the excess weight. Among the fat acceptance movement, National Association to Advance Fat Acceptance (NAAFA) fights against the medicalization of obesity, stating that it feeds the belief that larger bodies are a burden to the already-taxed health care system. Others attempt to rationalize the view of obesity as a medical condition by studying and presenting the effects of excessive weight on an organism. It is hypothesized that obesity is a social construct and a medical condition; both aspects influence an individual's health. The objective of this study is to study the physiological aspect of obesity and its social perception on the health outcomes of an individual with obesity. A study conducted with 232 healthcare workers on the interaction between healthcare workers and people with obesity was used to show that many healthcare professionals have negative attitudes and stereotypes about people with obesity, which further influence the care that they give. "Fearing the black body" by Sabrina Strings explains how large bodies became pathologized in the healthcare system over time. Furthermore, an analysis of medical research articles shows that excessive weight negatively affects the body. In conclusion, both the physiological and the social perception of obesity equally impact the health of people with obesity. Considering the complexity of obesity, a multi-dimensional intervention is needed to approach the alarming rates of cases. *This research project is proposed as part of our Senior Biology capstone course.*

34) CONNECTION OF PRETERM BIRTH AND PARTUM MORTALITY IN AFRICAN AMERICAN WOMEN: THE ROLE OF RACIAL DISPARITIES AND STRESS

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Studies have sought to identify the correlation between race and low birth weight, and postpartum mortality rates. Low birth weight refers to a birth that occurs before 37 weeks of gestation, which lead to possible health concerns such as jaundice, low blood sugar, difficulty maintaining body temperature, lack of oxygen, and an increased risk of infection. These potential complications can also result in emotional and financial stress for families, resulting in possible post-traumatic stress disorder (PTSD). Recent reports recognized that African American infants are more than twice as likely as white infants to die within their first year of life. African American women are also more likely to experience preterm births due to living in disadvantaged neighborhoods and facing more racial discrimination than their white coequals. The social determinants of African American women were suggested to be linked explicitly to biological factors such as cortisol, systemic inflammation, proteome and lipidome profiles, and telomere shortening. The hypothalamic-pituitary-adrenal axis regulates the body's response to stressful events. This feedback system which controls corticotrophin-releasing hormones and adrenocorticotropic hormones, is correlated with a higher risk of preterm birth. In this study, we aim to evaluate how we can more accurately assess the risks of biological factors concerning racial disparities and stress. *This research project is proposed as part of our Senior Biology capstone course.*

35) ANTIVIRAL PROPERTIES OF BLACK TEA POLYPHENOL TF3 AGAINST MOUSE HEPATITIS VIRUS (MHV) IN DELAYED BRAIN TUMOR (DBT) CELLS

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In 2019, a novel B-coronavirus known as Severe Acute Respiratory Syndrome-Coronavirus-2 (SARS-CoV-2) emerged in Wuhan, China, giving rise to the disease COVID-19. Although similar in homology to SARS-CoV of 2003, SARS-CoV-2 quickly spread worldwide, causing symptoms ranging from a dry cough to pneumonia. Prior to the vaccine rollout in December 2020, COVID-19 was responsible for the deaths of over 1.6 million individuals. This high mortality prompted research into vaccines as well as alternative therapeutics. Prior literature from Chia-Nan Chen et al. had shown the polyphenol compound theaflavin-3,3'-gallate (TF3) - found in the black tea plant Camellia sinensis - to have protease inhibitory properties against coronaviruses. After performing four plaque assays, TF3 showed an average of 43.7% viral-replication inhibition in the delayed brain tumor (DBT) cells infected with the coronavirus mouse hepatitis virus (MHV). The results suggested that TF3 alone may not be sufficient as a therapeutic against SARS-CoV-2. Nevertheless, the molecule may serve as a scaffold for further modification to enhance antiviral properties. *This research project is proposed as part of our Senior Biology capstone course*.

36) HOW PROBLEMATIC PORNOGRAPHY USAGE AFFECTS THE BRAIN: A NEUROSCIENCE PERSPECTIVE

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Living in the modern digital age, to fulfill one's sexual needs via pornography consumption has become a normalized practice. The accompanying aspects of accessibility, anonymity, and affordability have popularized viewing pornographic materials to attract millions of users. The alarming rate at which pornography usage has increased has triggered health professionals to be greatly concerned and to act through research. In the past several decades, research studies have revealed a correlation between problematic pornography usage (PPU) and various detrimental consequences related to the neuroanatomy of humans. This study aims to outline the effects pornography has on the brain, particularly within the prefrontal cortex (PFC), gray matter volume, and other areas related to the PFC. As more evidence surfaces as to the adverse effects of pornography usage, scientists have tested new treatment methods that aim to reduce this problematic behavior that shows promise. One prominent recovery method is cognitive-behavioral therapy (CBT), a behavioral-based therapy that gives individuals tools to reshape their thoughts and action to overcome their addictive behavior. Another behavioral-based approach under CBT is acceptance and commitment therapy (ACT), which utilizes strategies that target internal experiences and alter behaviors to reduce pornography usage. Another tested treatment method is naltrexone, an opioid-receptor antagonist that can decrease sexual urges. In my review, I aim to compare these three emerging treatment techniques of CBT, ACT, and Naltrexone. With these three treatments on the rise, further research and longitudinal studies need to be carried out to combat the stigma so people who struggle with PPU can receive proper assistance. *This research project is proposed as part of our Senior Biology capstone course*.

37) T-CELL-TARGETED IMMUNOTHERAPY FOR AUTOIMMUNE DISORDERS

Karen Liu
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The human immune system has developed complex mechanisms of self-tolerance to prevent immune cells from targeting the body's own cells. Central tolerance occurs when T-cells undergo negative and positive selection in the thymus where T-cells that are self-reactive are destroyed. In the periphery, regulatory T-cells function has specialized cells that maintain tolerance. The onset of type 1 diabetes (T1D), an autoimmune disorder, occurs when autoantibodies against islet cells are produced by B-cells signal to CD4+ and CD8+ T cells that have become autoreactive. T1D results in the progressive destruction of beta cells in the pancreas. Individuals with T1D are treated with insulin analogs and monitor their blood glucose levels, but none of these treatments can prevent the further progression of the disease. Immunotherapies aim to specifically target the autoreactive T-cells without hindering the remaining immune system. The goal of antigen-specific induction of regulatory T-cells is to restore the body's natural tolerance mechanisms against autoreactive T-cells. Though the cure is not guaranteed, tolerizing therapies with the goal of inducing insulin-specific regulatory T-cells has demonstrated that this treatment could slow the progression of T1D. Induction of regulatory T-cells has proven difficult at the onset of T1D due to impaired gene regulation and DNA methylation. These discoveries have shown that targeting miRNA can be a promising avenue as more research into their role in immune activation and inhibition. This study would target a specific miRNA sequence (miRNA92a) found to be highly expressed in individuals with T1D. By performing in vivo inhibition of miRNA92a in mouse models, we could gain insights into how gene sequences impact self-tolerance in T1D. Results from this study could demonstrate that miRNA therapies can broaden the opportunity to induce regulatory T-cells in combination with antigen-specific therapies. *This research project is proposed as part of our Senior Biology capstone course*.

38) THE LACK OF DIAGNOSTIC BIOMARKERS IN CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY

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Chronic inflammatory demyelinating polyneuropathy (CIDP) is a rare neurological disorder that involves the chronic inflammation of peripheral nerves and damage to the myelin sheath. It is characterized by progressive muscle weakness and impaired sensory function, affecting a patient's ability to walk and perform daily activities. CIDP lacks diagnostic biomarkers that help identify and monitor the progression of the disease, leading to underdiagnosis and challenges in managing the disease. With current diagnostic criteria relying on clinical suspicions, laboratory findings, and electrophysiological tests, diagnoses can often be delayed or ambiguous. This review aims to explore several treatment options available for CIDP, including intravenous immunoglobin (IVIg), corticosteroids (CCS), immunosuppressant drugs, and therapeutic plasma exchange (TPE). Despite these options, the lack of diagnostic biomarkers makes it challenging to assess the efficacy and safety of each treatment in individual patients, highlighting the need for further research on CIDP and finding a biomarker that can reliably identify its presence. *This research project is proposed as part of our Senior Biology capstone course*.

39) SILDENAFIL (VIAGRA) AND ALZHEIMER'S DISEASE: MODES OF ACTION AND OVERALL EFFECTIVENESS

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Alzheimer's disease (AD) is a progressive neurodegenerative disease characterized by synaptic dysfunction and memory loss due to the accumulation of amyloid B-peptides (Aβ) and tau protein. Aβ elevation and the consequent synaptic deficits have been linked to the downregulation of the nitric oxide/cGMP/cGMP-dependent protein kinase/c-AMP responsive element-binding protein (CREB) cascade. Nitric oxide (NO) exhibits a dual nature in neuroprotection and neurotoxicity, while both cAMP and cGMP are essential in neuroprotection and neuroplasticity. Sildenafil, a phosphodiesterase (PDE) inhibitor commonly referred to as Viagra, was recently observed to correlate with the decreased incidence of Alzheimer's disease and proposed as a potential treatment. The objective of this study is to analyze the interactions between Sildenafil and Alzheimer's-related pathways to identify Sildenafil's modes of action and effectiveness. Additionally, it aims to compare Sildenafil to drugs such as diltiazem, glimepiride, losartan, metformin, newly synthesized quinoline derivatives, and NO-donating drug cohorts. The research was conducted using QIAGEN Ingenuity Pathway Analysis (IPA) to explore the relationships between molecules and observe cascade interactions. Through in vivo mice models with induced AD, Sildenafil was shown to upregulate and enhance the phosphorylation of CREB (which is essential in the process of learning and memory) and elevate cAMP and GMP levels by reducing their degradation. Additional changes included an increase in brain-derived neurotrophic factor (BNDF) without modification of brain amyloid burden and a decrease in tau hyperphosphorylation, glycogen synthase kinase 3β (GSK3β), and cyclin-dependent kinase 5 (CDK5). Sildenafil produces immediate and long-lasting amelioration of synaptic function, reduction in Aβ levels, neurite growth, and memory improvement. Considering that cGMP-mediated pathways regulate inflammatory responses in immune and CNS systems, Sildenafil could be a promising drug against other neurological pathologies such as stroke, multiple sclerosis, and focal brain lesions, as well as inflammatory immune disorders such as arthritis. *This research project is proposed as part of our Senior Biology capstone course*.

40) THE IMPACT OF COVID-19 ON THE RATE OF ANTIBIOTIC RESISTANCE AND THE REPERCUSSIONS

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The recent acceleration of antibiotic resistance is a growing concern in the healthcare community. Misuse and overconsumption of antibiotics are contributing to antibiotics losing their effectiveness. According to CDC, the pandemic has likely resulted in rising antibiotic-resistant infections. The acceleration during COVID-19 is caused by patients receiving antibiotic therapies without requiring them, antibiotics being consumed for viral infections, and stewardship policies being overridden during this time of crisis. A literature search has been conducted to organize and explain the risk factors that lead to increased antibiotic resistance, including the COVID-19 pandemic, and communicate the healthcare repercussions that result. Additionally, this study aims to identify and explore approaches to combat this problem and look for new, effective antibiotics that infectious bacteria have not yet built-up resistance to. Kirby-Bauer technique was performed on bacteria, including Bacillus megaterium (B. meg), Staphylococcus epidermidis (S. epi), Enterococcus raffinosus (E. raff), and Erwinia carotovora (E. caro). A larger zone of inhibition (ZOI) is associated with greater antibiotic susceptibility. The results demonstrate that the Kirby-Bauer assay is an effective way to determine the susceptibility of a bacterium to various antibiotics. Previous reports pinpointed risk factors and repercussions and contributed to antibiotic stewardship. Additionally, this study depicts methods to develop new antibiotics that have promising applications for healthcare professionals. *This research project is proposed as part of our Senior Biology capstone course*.

41) THE LINKS BETWEEN OBSTRUCTIVE SLEEP APNEA AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Chronic Obstructive Pulmonary Disease (COPD) is a chronic inflammatory disease that causes obstructed airflow from the lungs due to various causes, such as inflammation and excess mucus. While partially reversible on its own, when combined with symptoms caused by Obstructive Sleep Apnea (OSA), the effects can be irreversible. It has been hypothesized that this "Overlap Syndrome" contributes to the further worsening of overall health and can lead to additional pulmonary, cardiac, and muscular issues. Studies have shown that the correlation of symptoms such as poor quality of sleep, overall reduced sleep time, and trouble breathing while sleeping further exasperate both conditions. In addition, feelings of extreme anxiety when attempting to sleep, depression from lack of sleep, heightened pain intensity, and pulmonary hypertension are major factors in lowering physical activity, leading to muscular deterioration and cardiac diseases. While conclusive research has yet to be performed on the direct correlation between COPD and OSA, it is inarguable that when diagnosed with both conditions, the conjoining symptoms exasperate one another significantly. Furthermore, updated methods for the management of the overlap syndrome will be required in order to maintain the risk of increased mortality due to exasperation. *This research project is proposed as part of our Senior Biology capstone course*.

42) EFFECTS OF ELECTRONIC CIGARETTES ON CARDIOVASCULAR HEALTH

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As younger generations turn to electronic cigarettes, the demand for the long-term effects of this new technology is on the rise, and with good reason. Over the years, self-reported surveys have shown that ~450,000 individuals who have switched to electronic cigarettes have been diagnosed with a form of cardiovascular disease. The mechanism of electronic cigarettes involves heating metal to vaporize liquid chemicals. Electronic technology and nicotine have been shown to contain harmful substances, such as heavy metals, formaldehyde, or carbon monoxide, that exacerbate existing cardiovascular diseases or stimulate the risk of developing infections. This review aims to analyze two studies to evaluate the parts of the cardiovascular system most likely linked to electronic cigarette use. Through the microscopic analysis of myocardial cells to determine the cytotoxicity of e-cigarette aerosols and using biomarkers, such as the NOX2-derived protein, to indicate the amount of oxidative stress placed on myocardial cells, these studies were able to identify the main targets. The results indicated that although electronic cigarettes did not produce more harmful effects than traditional cigarettes, the significantly increased oxidative stress and cytotoxicity showed that electronic cigarettes still pose a substantial risk to the system. In summary, the results help further expand the knowledge of how electronic cigarettes may not be a great alternative to traditional smoking and do not benefit users as advertised. *This research project is proposed as part of our Senior Biology capstone course*.

43) THE IMPACT OF SUNSCREEN INGREDIENTS SUCH AS OXYBENZONE (BENZOPHENONE-3) ON AQUATIC LIFE

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Skin cancer is the most common form of cancer in the United States, caused primarily by UV radiation. To prevent the onset of this cancer and other skin complications such as photoaging, sunscreen has been the primary solution and suggested by many dermatologists. Unfortunately, the active UV filters in many sunscreen products, such as oxybenzone (benzophenone-3), octocrylene, and octinoxate, are found to be toxic and damaging to aquatic life. These ingredients can end up in ocean waters through contact with these natural waters in places such as the beach or through washing off in a sink or shower, which ends up in the water supply. Additionally, many of these ingredients are readily absorbed through the skin and excreted through urine, entering the plumbing. These sunscreen products, specifically oxybenzone, can be harmful to the early development of sea urchins (Paracentrotus lividus), clownfish (Amphiprion ocellaris), coral reefs (Stylophora pistillata), loggerhead turtles (Caretta caretta). The adverse effects of these ingredients were seen through mortality testing of an ocean sample of clownfish subjected to various quantities of oxybenzone with 100 mg/L oxybenzone-based sunscreen exposure giving 100% disruption to development. Behavior testing was also conducted to visualize the effects of the product, which showed immobilization and harm to aquatic life. Sunscreen ingredients such as oxybenzone have been proven to be damaging to sea animals, and further testing can be conducted to see its effects on different animal groups and bodies of water along with other ingredients, such as the mineral TiO2. *This research project is proposed as part of our Senior Biology capstone course*.

44) NEXT-GENERATION SEQUENCING AND ITS CLINICAL APPLICATIONS IN RENAL MEDULLARY CARCINOMA

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Next-generation sequencing (NGS), also known as high throughput sequencing, allows for the sequencing of many fragments of DNA in parallel. Specifically, NGS has been helpful in the treatment of Renal Medullary Carcinoma, whereby researchers were able to detect somatic alterations which contributed to the growth of tumors. NGS requires three main steps. More recently, NGS has been adopted in clinical applications, including oncology, to advance the personalized treatment of cancer. NGS is used to identify novel diagnostic and rare cancer mutations, as well as copy number variants, translocations, inversions, insertions, and deletions. It also identifies carriers of familial cancer mutations and provides the molecular basis for targeted, therapeutic, and prognostic interventions. As opposed to its predecessor, Sanger sequencing is limited to the discovery of substitution and small insertions and deletions. It was also far slower and could take a decade to get the final draft of a genome. Some of the platforms for NGS include Roche/454 sequencing, Ion Torrent/ Proton, ABI/ SOLiD, and Illumina sequencing. Currently, Illumina sequencing is the most popular NGS platform and is responsible for more than 90% of the world's data that is generated. NGS holds many advantages that will be able to advance the way cancer patients receive treatment due to the advances it can provide in personalizing the treatment options. *This research project is proposed as part of our Senior Biology capstone course*.

45) THE ROLE OF BISPECIFIC ANTIBODIES IN THE TREATMENT OF HEMATOLOGIC MALIGNANCIES

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Treatments for B-cell malignancies have improved considerably over the last few decades, beginning with the approval of rituximab as the first immunotherapy treatment. Since then, the introduction and approval of more effective immunotherapies such as chimeric antigen receptor T-cell therapy (CAR-T) have led to further advancement in the management of B-cell malignancies. More recently, bispecific antibodies (BsAb) have emerged as a new category of T-cell redirecting immunotherapies and are currently amongst the most promising treatments for B-cell malignancies. Bispecific antibodies have the ability to simultaneously bind both target tumor cells and T-cell antigens, allowing for T-cell activation and subsequent tumor cell destruction. The two currently approved bispecific products, blinatumomab, and mosunetuzumab, have been engineered to target B-cell-specific CD19 and CD20 tumor cell antigens, respectively. Blinatumomab was originally approved for the management of resistant acute lymphoblastic leukemia and mosunetuzumab for follicular lymphoma, although their roles in the management of B-cell malignancies continue to evolve. Bispecific antibodies have proven to be highly effective, even in heavily pre-treated patients. Common adverse effects include cytokine release syndrome (CRS) and neurological toxicities, both generally considered manageable. Other products with more specific binding capabilities and potentially greater therapeutic benefits are in various stages of development. *This research project is proposed as part of our Senior Biology capstone course*.

46) CONSIDERATIONS FOR THE SEARCH FOR LIFE IN THE OUTER SOLAR SYSTEM

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As humanity begins to look beyond Earth for future exploration and expansion, an important factor to consider is the presence of potential lifeforms on other celestial objects. When sending probes and eventually people to other planets, moons, or asteroids, it is vital that we not only protect ourselves from any potentially hazardous lifeforms but prevent terrestrial microbes from contaminating these foreign lifeforms. The aim of this review is to draw comparisons between the conditions present on an early Earth and those hypothesized to be present on the Jovian and Saturnian moons Europa, Titan, and Enceladus to establish the potential for life on these moons and to suggest a strategy for moving forward with the search for any biomarkers, such as homochirality, present on or beneath their surfaces. The forms that this life may take will be expanded upon as well; in the case of Europa and Enceladus, experiments simulating the conditions present on these bodies in the style of the Miller-Urey experiment alongside physical samples analyzed by the Cassini-Huygens mission will be examined. Regarding Titan, current theories on potential non-water-based life in the liquid hydrocarbon lakes on its surface will be examined in the context of physical data retrieved by the Huygens probe. The limits of life known to humans, such as extremophilic microbes and bacteria present underneath the Antarctic ice sheets, will also inform this survey of how to search for life. Furthermore, instruments that are necessary for future unmanned missions to these moons to confirm the presence of life will be suggested alongside considerations for avoiding any potential cross-contamination between terrestrial and extraterrestrial lifeforms. *This research project is proposed as part of our Senior Biology capstone course*.

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