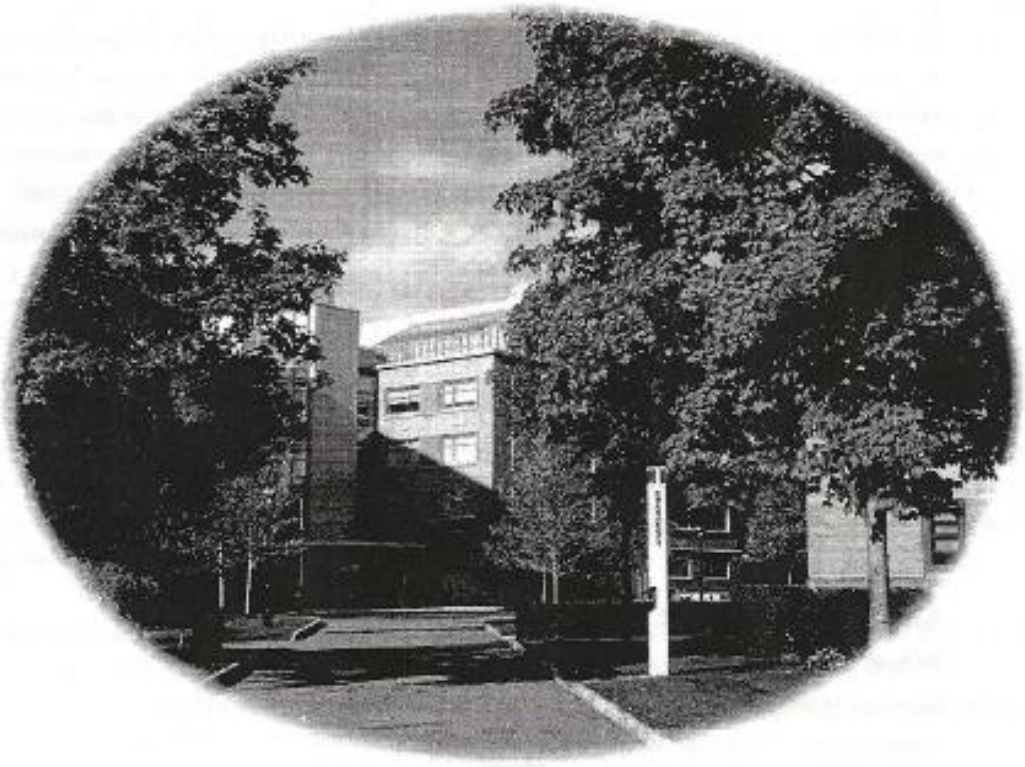
SETON HALL UNIVERSITY

College of Arts & Sciences

Department of Biological Sciences



13th Annual Biological Sciences Symposium

Observe, Explore, Achieve

Abstract Booklet

Spring 2021

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

Schedule of Events

3:30 pm **Opening Remarks**

Dr. Heping Zhou, Chair of Biological Sciences

3:40 pm **Poster Session (Virtual)**

Graduate Research
Undergraduate Research
Next Generation OMICS Research
Senior Seminar Capstone Projects

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

Professor Samuel H.H. Chan, PhD
National Chair Professor/Distinguished Chair Professor and Director
Institute for Translational Research in Biomedicine
Chang Gung Memorial Hospital
Kaohsiung 83301, Taiwan, Republic of China

Title: "Differential Clinical Impacts of Oxidative Stress and Nitrosative Stress: Therapeutic Implications"

6:25 pm **Closing Remarks**

Acknowledgements
Announcement of poster winners

Keynote Lecturer

Professor Samuel H.H. Chan, PhD

National Chair Professor/Distinguished Chair
Professor and Director
Institute for Translational Research in Biomedicine
Chang Gung Memorial Hospital
Kaohsiung 83301, Taiwan, Republic of China

Since graduating cum laude from the Chinese University of Hong Kong in 1968 and receiving his PhD of Physiology from Indiana University in 1971, Dr. Samuel H.H. Chan has dedicated his life to teaching others and researching biomedicine. After his postdoctoral training at the Mount Sinai School of Medicine in New York, he has held many academic positions at postsecondary institutions. These include the University of Hong Kong (1973-1977), Indiana University (1977-1982), National University of Singapore (1982-1985) and National Yang-Ming University in Taipei, Taiwan (1986-1998).

Dr. Chan has been the Chairman of the Medical Research and Development Board for the Chang Gung Medical Foundation since 2015. The Chang Gung Medical Foundation was founded in 1973 in the pursuit of providing the people of Taiwan with high quality healthcare which was severely lacking at the time. The focus of the foundation was to provide modern medical technology as well as the latest medical knowledge to all medical staff. Founders Mr. Yung-Ching Wang and Mr. Yung-Tsai Wang established the first Chang Gung Memorial hospital in 1976 in memory of their father, Mr. Chang-Gung Wang. Since then, the foundation has expanded to include five other hospitals, a University, and a nursing institute that still focuses on the mission of providing exceptional care to its patients.

Along with this, he has also been the Inaugural Director and Distinguished Chair Professor of the ITRBM (Institute for Translational Research in Biomedicine) at Chang Gung Memorial Hospital since 2009. During this time, he has overseen the foundation's mission of the integration of medical professionals and medical researchers. By uniting the knowledge from both medical staff and clinical researchers, the hope is to improve the quality of care across all of their medical centers in Taiwan. These centers care for an average of 8.2 million outpatients, 2.4 million inpatients, and 167,460 surgical patients per year.

Dr. Chan's current research focuses on cardiovascular regulatory functions, specifically translational research on brainstem death and neurogenic hypertension. By using a combination of the latest medical imaging technology, chemical analysis, and genetic phenotyping, Dr. Chan and his fellow researchers can visualize the affected nerve circuits and identify the affected cellular functions which induce neurogenic hypertension.

Dr. Chan's studies have led to over 270 publications and many prestigious accolades. These awards and titles include the Outstanding Researcher Award from the National Science Council in Taiwan (1986-1996) which he received over five consecutive 2-year terms, the Shih-Chun Wang Outstanding Investigator Lectureship in Neuroscience Award from the Professor Shih-Chun Memorial Scholarship Fund in the United States (1995) which no other person had received before, and the title of the Lifetime National Chair Professor of Neuroscience from the Ministry of Education in Taiwan (2000-), a position held by only six other professors.

ABSTRACTS

GRADUATE RESEARCH

1) YEAST 2 HYBRID ANALYSIS OF MCV HOST IMMUNE EVASION PROTEINS
Zachary A Cropley and Daniel Nichols
Department of Biological Sciences, Seton Hall University

Poxviruses are a wide variety of double stranded DNA viruses, including the now extinct smallpox. One such virus, Molluscum contagious virus (MCV), produces relatively benign skin papules in afflicted individuals. While non-life threatening in healthy individuals, it can present dangerous health complications for immunocompromised individuals. In these individuals, the disease can persist for months to years, up from the standard weeks to months in healthier patients. Seventy-seven MCV genes are predicted to produce products that function as immune evasion molecules. Amongst these are the MC159, MC160, and the recently described MC163. These three MCV proteins antagonize several important immune responses including the activation of apoptosis, TNF-induced NF-kB, and induction of type I interferons. Interestingly, all three MC159, MC160 and MC163 independently inactivate the NF-kB pathway. Understanding why this apparent redundancy occurs during an MCV infection is of interest to understand how MCV dampens host immune responses. In addition, these evasion genes may affect the elongated time for an afflicted individual to develop a natural immunity. MC159 and MC163 inhibit apoptosis, and MC159 also inhibits necroptosis. However, the molecular mechanism MC159 inhibits necroptosis remains unknown. Multiple MCV immune evasion genes allow the virus to thwart the cellular immune response and continue propagation. By determining the potential protein to protein interactions these viral genes exhibit, novel interactions with the host immune proteome may be discovered, and their effects on both apoptosis and necroptosis can be elucidated. Since the virus is an obligate human pathogen, whole viruses are impractical to culture in the lab as no tissue culture model exists to study MCV as of yet. To circumvent this problem partial MCV genome segments can be cloned and used to transfect mammalian tissue culture or transform yeast cells to elucidate protein-protein interactions. The goal of this dissertation is to further elucidate the molecular mechanisms of these MCV immune evasion proteins.

2) CLINICALLY RELEVANT DOSAGE OF VANCOMYCIN DOES NOT NEGATIVELY IMPACT PERIOSTEUM DERIVED OSTEOBLAST PRECURSOR CELLULAR FUNCTIONS
Alexis Hernandez and Jessica Cottrell
Department of Biological Sciences, Seton Hall University

Antibiotic therapy has been used to prophylactically prevent infection in immunosuppressed patients undergoing orthopedic surgery. Vancomycin is a commonly used glycopeptide antibiotic that successfully prevents infection in diabetic bone healing surgical sites. Research has been shown that clinically appropriate doses of vancomycin do not impede fracture healing in normal animals. However, the effect vancomycin has on aspects of diabetic bone regeneration is unknown. Our study aims to identify whether vancomycin treatment inhibits the viability/proliferation, calcium deposition, or function of periosteum derived precursor osteoblasts in diabetic bone healing. In this study, primary periosteum precursor osteoblasts were harvested from BB Wistar type 1 diabetic rats. The cells were cultured in growth media until confluency, approximately every 7 days. They were then seeded in 12-well plates and cultured using growth media supplemented with BMP-2, as control, and beta glycerophosphate to drive osteogenic differentiation. During culture, these cells were then treated with increasing doses of vancomycin (0, 50, 500, or 5,000 μg/mL) using single and continual dosing regimens. Cells were assayed for cell viability/proliferation via the MTT assay at 24hrs, calcium deposition via Alizarin Red S Staining at 14 and 28 days, and alkaline phosphatase activity at days 7 and 14. Periosteal precursor osteoblast cell proliferation, calcium deposition, and ALP activity was only significantly impaired at the 5000μg/mL vancomycin dose. There was statistically significant decreased AP activity following treatment with a continuous dose of vancomycin at 5000 ug/mL, but not at a single dose. Furthermore, calcium deposition was decreased at 5000 ug/mL, both single and continuous dosing. Statistical significance indicated with a $P<0.005$ was determine using 1 way ANOVA statistical testing. Our data shows that clinically appropriate doses of vancomycin do not negatively impact periosteum derived osteoblast cellular functions.

3) THE EFFECT OF PALMITATE ON THE EXPRESSION OF INFLAMMATORY CYTOKINES IN BV2 CELLS

Shiyu Ma, Heping Zhou
Department of Biological Sciences, Seton Hall University

Obesity has been associated with many pathologies including cognitive decline, atrophy of brain regions related to learning and memory, insulin resistance, cardiovascular diseases, and hypertension. Low grade chronic inflammation triggered by saturated fatty acids (SFA) such as palmitate (PA) has been suggested to contribute to the development of these conditions. In this study, time- and dose-dependent effects of PA on the mRNA expression of inflammatory cytokines, such as tumor necrosis factor (TNF)-α, interleukin (IL)-6, and IL-1β in BV2 microglia cells were examined. Pharmacological inhibitors were also used to examine the involvement of different mediators in PA-induced cytokine expression. Our studies found that 200 μM PA induced mRNA expression of cytokines such as IL-1β at 24 h post-treatment. Inhibition of PI3K and GPR40 by Wortmannin and DC260126 attenuated PA induced mRNA expression of inflammatory cytokine, suggesting that PI3K and GPR40 might be involved in PA-induced production of inflammatory mediators in BV2 cells.

4) EFFECTS OF SODIUM ORTHOVANADATE ON LPS-INDUCED SENESCENT RAW264.7 CELLS

Andrew Pugliese, Allan Blake, Jessica Cottrell

Cellular senescence is considered a signal transduction process where damaged cells arrest proliferation. Exogenous stressors include bacterial infection (oxidative stress), chemotherapy, and irradiation, can drive normal cells to senescence. Senescent cells are characterized by metabolic and morphological changes including the production of inflammatory mediators, flattened enlarged shape, and multinucleation. Literature has shown that senescence can exacerbate inflammation and contribute to disease progression. Sodium orthovanadate (Na_3VO_4) is a phosphatase inhibitor known to mediate the release of proinflammatory cytokine TNF-α. Preliminary analysis has shown that sustained LPS treatment of the RAW 264.7 monocytic cell line induced morphological changes that resemble those by senescent cells. In this study, we hypothesized that continual LPS treatment can induce senescence in RAW 264.7 monocytic cells. Further, we believe that Na_3VO_4 treatment will reduce the expression of inflammatory markers in these senescent cells.

In this study, RAW 264.7 cells were treated with control or LPS (10 μg/ml) for 96 hrs followed by treatment with and without Na_3VO_4 (100μM). Beta galactosidase activity staining was completed to measure senescence at time points before treatment and after treatment. Cell supernatants were then assayed for inflammatory cytokines via Luminex. Qiagen ingenuity pathway analysis (IPA) tool was also used to investigate the connection of exogenous stressors and their implication to senescence and tumor progression.

The data demonstrated that continual LPS treatment for 96 hours statistically induced senescence at 48 hrs ($P<0.002$). Preliminary cytokine analysis showed a difference in cytokine expression patterns between treatment groups. IPA analysis showed a connection between factors secreted by senescent cells, inflammation, tumor progression, and metastasis. Illustrating the duality that senescence plays in disease states such as cancer. Overall, our data shows LPS can induce senescence in monocytes and Na_3VO_4 may modulate senescent specific inflammatory cytokines and could be a potential therapeutic to combat senescence induced disease progression.

5) CHARACTERIZATION OF THE *MOLLUSCUM CONTAGIOSUM* VIRUS MC160 PROTEIN INHIBITION OF MAVS

Brian T. Reiss and D. Brian Nichols
Department of Biological Sciences, Seton Hall University

Molluscum Contagiosum Virus (MCV) is a member of the family *Poxviridae*. Of the viruses found within *Poxviridae,* the most well-known are variola virus, the causative agent of smallpox, and vaccinia virus (VACV), the model poxvirus of the family. Though smallpox was declared eradicated in 1980, several poxviruses including MCV remain a concern to human health. There has yet to be a working treatment for individuals who acquire molluscum contagiosum (MC). MC is a common skin infection that causes benign lesions. MC lesions are categorized as either inflammatory or non-inflammatory. The inflammatory infection appears as a raised lesion, while the non-inflammatory infection is more persistent due to the viral evasion of the innate immune system. MCV is known to encode a number of products that directly function in host immune evasion and the MC160 protein is thought to be one of these key viral proteins. MC160 protein has been characterized as a viral fas-associated death domain (FADD)-like interleukin-1β converting enzyme inhibitory protein (vFLIP), which shares homology to the cellular FLIPs (cFLIPs) like FADD, Procaspase-8, $cFLIP_S$, $cFLIP_R$, and $cFLIP_L$ that are known to govern the apoptotic activity of the cell. Expression of MC160 dampens several immune signaling pathways including interferon activation induced by the overexpression of the mitochondrial antiviral signaling (MAVS) protein, TBK1, and IKKε. The exact mechanism by which MC160 functions is not completely understood. I hypothesize that the MC160 protein targets and binds Hsp90, thereby destabilizing the TBK1/IKKε complex. Additionally, MC160 may inhibit MAVS by preventing the ubiquitination of key proteins within the pathway. MC159 inhibits NEMO polyubiquitination in TNF-α signaling. Since MC160 and MC159 share a similar structure, it is reasonable to hypothesize that these proteins prevent the ubiquitination of key adapter molecules in the MAVS pathway, which would prevent downstream antiviral signaling responses from occurring.

6) MOLECULAR DOCKING STUDY OF THEAFLAVIN ANTI-GERMINATION MECHANISM ON BACILLUS SPP.

Ayuni Yussof and Tinchun Chu
Department of Biological Sciences, Seton Hall University

Bacterial spores of various *Bacillus* spp. are associated with food spoilage and disease pathogenesis. The bacterial spore may remain dormant for years but can germinate when exposed to favorable growth conditions. Theaflavin-3,3'-digallate (TF3), extracted from fermented leaves of *Camellia sinesis*, has shown to have anti-bacterial and anti-spore properties. The aim of this study is to determine the anti-germination activity and explore the potential mechanism of TF3 on three *Bacillus* species, *Bacillus cereus* (*B. cereus*), *Bacillus megaterium* (*B. megaterium*), and *Bacillus subtilis* (*B. subtilis*). Biovia, Vega ZZ, AutoDock Tools, AutoDock Vina, and BIOVIA Discovery Studio were used to determine the binding location of TF3 on the conserved germination-associated proteins such as Gpr and Lgt (GerF). Previous reports indicated that -7.0 kcal/mol is considered as a cutoff for favorable binding energy as it removes weaker and non-specific binding. The AutoDock Vina revealed a range of -7.6 kcal/mol to -10.3 kcal/mol for the binding affinity of TF3 across all three *Bacillus* species. The result also showed that up to nine hydrogen bonds and up to three hydrophobic interactions between TF3 and the proteins. Those intermolecular forces signify favorable binding interactions. In conclusion, the molecular docking analysis suggested that TF3 could serve as a promising anti-spore agent in *Bacillus* spp.

UNDERGRADUATE RESEARCH

7) INDUCTION OF APOPTOSIS IN GLIOBLASTOMA CELLS THROUGH ACTIVATION OF PPARγ BY BIS INDOLYL METHANE COMPOUNDS

Margot Brown, Matthew Sunda, Isabella Somera, Zena M. Abukanan, Joseph Badillo, Suzanne Quartuccio Gantar
Seton Hall University, Department of Biological Sciences, South Orange, NJ

Glioblastoma is the most common and deadly type of malignant primary brain tumor in adults and has a rate of recurrence of around 90% in patients who receive treatment. Peroxisome Proliferator-Activated Receptor Gamma (PPARγ) is a prevalent transcription factor found in multiple cancers such as glioblastoma, liposarcoma, and carcinomas of the breast, colon, and prostate. PPARγ functions in lipid metabolism, cell growth, differentiation, and apoptosis. The purpose of this research is to determine if PPARγ activators, specifically 1,1-bis(3'-indolyl)-1-(aryl)methane and isatin-derived compound (oxindole) derivatives known as BIM compounds, can be an effective treatment for glioblastoma. A parallel project in the lab of Dr. Gantar has previously shown that these BIM compounds inhibit the proliferation of glioblastoma cell lines. However, this project aims to identify how cell proliferation is inhibited. Apoptosis is characterized as programmed cell death, or cell suicide. Other cellular processes, such as cell lysing, necrosis, or senescence are viable paths for reduced proliferation. Through the use of Western Blotting, the presence and abundance of PPARγ was examined in T98G glioblastoma cells. Then the ability of BIM compound treatment to induce apoptosis was measured through Western Blotting and qPCR analysis of pro-apoptotic factors including cleaved caspase-3 and BAX proteins. If BIM compounds induce apoptosis in glioblastoma cells, they can be used to treat this deadly cancer and improve overall patient survival.

8) THE ONCOGENIC ROLE OF AURKC IN MITOTIC CELLS

Zachary DiSanza, Adrian Bernal, and Suzanne Quartuccio Gantar
Seton Hall University, Department of Biological Sciences, South Orange, NJ

Aurora Kinase C (AURKC) is a part of the Aurora Kinase family that also includes Aurora Kinase A and B. While AURKA and AURKB are expressed in all cells throughout the body, AURKC is normally only expressed in germ line cells. Interestingly, AURKC is highly expressed in many cancer cell lines including bone osteosarcoma cells (U2OS). To evaluate AURKC as a potential therapeutic target, U2OS cells were edited with Crispr-Cas9 technology to knock out the gene. U2OS cells lacking *AURKC* showed no change in cell proliferation but did migrate less and formed fewer colonies in soft agar. Previous studies have shown that migration of cancer cells had been linked to the expression of collagen subunit *COL1A1* in certain cancers. The expression and transcription of COL1A1 in U2OS osteosarcoma cells with and without AURKC was tested through a real-time quantitative polymerase chain reaction (RT-qPCR) and western blot. Results from these experiments indicate the AURKC may regulate extracellular matrix proteins contributing to cancer cell metastasis and should be further evaluated as a potential new target in cancer therapies. We would like to thank Seton Hall University Research Council for supporting this work.

9) CHARACTERIZATION OF INSULIN MIMETIC EFFECTS ON GENE EXPRESSION DURING THE ATDC5 CELL LINE

Caitlin Gartley and Ruby Pasupuleti
Department of Biological Sciences, Seton Hall University

Previous data has shown that insulin mimetic such as vanadium compounds can enhance bone healing much like insulin can without the risk of glycemic changes. Our study used ATDC5 chondrocytes derived from mouse cells to analyze the potential regenerative effects of vanadium compound treatment during chondrogenesis differentiation, a key process in long bone healing. ATDC5 cells were treated with DMEM/F12 media (untreated, negative control), 10uM insulin (positive control), or vanadium compounds: vanadyl acetylacetonate (VAC) and vanadium (II) sulfate (VSO4) at concentrations of both 10uM and 100uM. Chondrocyte lysates were harvested at days 1, 2, 4, 7, 10, 14, 17, 21, and 28 for all treatment groups. After RNA isolation, RNA quantification was completed using a Biodrop, followed by reverse transcription for each sample. Successful conversion of RNA to cDNA was verified using polymerase chain reaction (PCR) and DNA gel electrophoresis with the housekeeping gene, glyceraldehyde 3-phosphate dehydrogenase (GAPDH). Finally, gene expression of key markers of chondrogenesis (i.e. Collagen 2a1, col2a1) was completed using quantitative real time polymerase chain reaction (qPCR) for each treatment group overtime. qPCR analysis demonstrated that col2a1 gene expression was more abundant on days 4, 7, and 10 when compared to insulin-treated or untreated samples. Our data also demonstrated that col2a1 expression increased over time. Together our data supports the hypothesis that vanadium compounds enhance chondrogenic differentiation which in turn can improve bone healing. Our study also demonstrates that vanadium compounds can serve as an alternative to insulin in modulating chondrogenesis and may be more impactful during the early stages of differentiation. In future experiments, we will continue to characterize the gene expression response to these vanadium compounds in our model and seek to determine if this enhancement occurs through the same molecular pathway as insulin.

10) ANTI-CANCEROUS BIS INDOLYL METHANE (BIM) COMPOUNDS PREVENT PROLIFERATION OF GLIOBLASTOMA CELLS

Karina Helou, Mackenzie McCann, Joshua Novello, Zena M. Abukanan, Joseph Badillo, Suzanne Quartuccio Gantar
Seton Hall University, Department of Biological Sciences, South Orange, NJ

Glioblastoma is the most belligerent type of brain cancer, with patients over 65 having a 2% survival rate over a 5-year period. 1,1-bis(3'-indolyl)-1-(aryl)methane and isatin-derived compound (oxindole) derivatives, known as BIM compounds, are characterized by their anticancer properties effective in multiple cell lines, including acute myelogenous leukemia, bladder cancer, colon cancer, and liver cancer. The present study tested the effects of BIM compounds on two different glioblastoma cell lines (T98G and Ln18). It was hypothesized that administration of the compounds would inhibit cellular proliferation in these cell lines. The cells were counted and plated in 96-well plates. After three hours, the 96-well plates were treated with 100 µL of different BIM compounds at concentrations ranging from 5µM to 50µM, or DMSO as the vehicle control. In total, 20 different compounds were tested on each cell line. The cells were fixed following 72 hours of treatment. A Sulforhodamine B assay was performed to determine the cytotoxic effects of the compound treatments. Absorbance was measured at 505 nanometers using a plate reader. Data from the plate reader was then interpreted in GraphPad Prism to determine the concentration of each BIM compound at half maximal response, or theEC50.. Results showed that all BIM compounds inhibited proliferation of both glioblastoma cells lines with similar EC50 values compared to DMSO control. This study is the first to test BIM compounds on glioblastoma cell lines and can lead to new therapeutic treatments to increase patient survival.

11) MECHANISMS OF OBESITY AUGMENTED COVID-19 PATHOLOGIES

Christie Joshi, Viren Jadeja, Heping Zhou
Department of Biological Sciences, Seton Hall University

The ongoing pandemic of coronavirus disease 2019 (COVID-19) that has claimed over 2.55 million lives globally as of March 2021 is caused by infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which not only causes pneumonia-like pathologies, but also damages endothelial cells in the heart, kidneys, liver, and intestines. Obesity is a significant risk factor for many pathologies, including type 2 diabetes, cardiovascular diseases, and nonalcoholic fatty liver disease. It also increases the acuity and mortality of COVID-19. COVID-19 and obesity have a synergistic effect on the production of pro-inflammatory cytokines, including Tumor Necrosis Factor alpha (TNF-α), Interleukin (IL-1), and IL-6. This study used Ingenuity Analysis Pathways software (IPA) to examine how excess levels of different fatty acids affect the progression of COVID-19. Our study found that Coronavirus Pathogenesis Pathway was one of the top ten canonical pathways activated by palmitic acid (PA), and this activation was mediated by key molecules including cytokines, such as IL-1β, IL-6, and C-C Motif Chemokine Ligand 2 (CCL2), transcription factors, such as nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) and Fos, signaling mediators, such as p38 mitogen-activated protein kinases (p38 MAPK) and extracellular signal-regulated kinases (ERK1/2). Our study also found that ω-3 unsaturated fatty acids may attenuate the PA's activation of the Coronavirus Pathogenesis Pathway by inhibiting the induction of key inflammatory mediators such as IL-1β and NF-κB.

12) MECHANISMS OF OBESITY-ASSOCIATED NONALCOHOLIC FATTY LIVER DISEASE BY META-ANALYSIS

Myeong Gil Jun and Heping Zhou
Department of Biological Sciences, Seton Hall University

Nonalcoholic fatty liver disease (NAFLD) has become more prevalent within recent years due to increasing poor diet habits, now recognized as the most common chronic liver disease in Western countries. NAFLD and its link to obesity demonstrates how the oversaturation of dietary components, such as triglycerides (TAGs), cholesterol, fructose, are key in triggering the pathogenic pathways for NAFLD as well as inducing insulin resistance (IR) that progresses the disease. Progression in the disease has been correlated with excessive lipid accumulation that drives elevated levels of free fatty acids (FFAs). This study conducted meta-analysis using the QIAGEN Ingenuity Pathway Analysis tool to examine the roles of different FFAs in the development of NAFLD. Saturated fatty acids such as PA was found to induce inflammatory pathways and produce reactive oxygen species (ROS) that causes oxidative stress. Exacerbated stress in the liver can also cause hepatocellular death, activating several apoptotic and necrotic pathways. PA also activates the hepatic stellate cell activation pathway, which may lead to excessive fibrogenesis.

13) LIFETIME ACCUMULATION OF RACISM-RELATED STRESS IN AFRICAN AMERICAN WOMEN AND ITS EFFECT ON ADVERSE BIRTH OUTCOMES

Jillian A Lazzara
College of Arts and Sciences, Department of Biological Sciences, Seton Hall University

The lifetime accumulation of chronic stress and psychosocial responses that accompany racism may be underlying causes of continual racial health disparities, including low birthweight, preterm birth, and infant mortality. The long-term effects of adverse birth outcomes, especially low birthweight, can persist into adulthood as kidney disease, hypertension, and Type 2 diabetes – all of which are commonly referred to as "underlying conditions." Chronic racial stress, also referred to as allostatic load, degrades organ systems including the immune system rendering women of color more susceptible to a variety of poor health outcomes. This degradation of basic physiological functioning in African American women is referred to as "weathering." Therefore, racism may directly affect the condition of a woman's reproductive system. Thus, pregnancy and parturition–biological events that rely on maintenance of stable regulatory body systems– are significantly impacted. Therefore, racism can be considered a physical stressor on the body, similar to poor nutrition and environmental toxins. The goal of the current work was to review the large body of literature available on adverse birth outcomes as they relate to culturally embedded racial bias.

14) THE EFFECTS OF NICKEL CHLORIDE ON CYANOBACTERIUM ANABAENA SPP.

Michael Lutz, Albert Dimaculangan Jr., Christian Martinez, Danielle Maragh, and Tinchun Chu
Department of Biological Sciences, Seton Hall University

Rapid industrialization and population growth has led to the contamination of freshwater bodies that serve as sources of drinking water as well as recreational purposes. Heavy metal pollutants from industry and farm runoffs are among the most troubling as they have negative downstream effects on both human and livestock health. Cyanobacteria, some of which can release harmful cyanotoxins are among the few species of aquatic microorganisms that can survive and tolerate heavy metal stress. This study aims to evaluate the response of the Cyanobacterium *Anabaena cylindrica* under nickel stress. Culture samples were exposed to various concentrations of nickel chloride (0, 1.0, 2.0, 4.0 mg/L). Cell growth and morphology changes were monitored over the course of 12 days. Cells exposed to 2.0 and 4.0 mg/L $NiCl_2$ showed the greatest morphology changes, including significant inhibition in the highest concentration tested. Filament length and pigment distribution was noticeable affected as early as day 1. QIAGEN Ingenuity Pathway Analysis (IPA) was used to explore the potential molecular interaction between key players and elucidate their likely impact on human health. From the network generated using IPA BioProfiler and Molecule Activity Predictor (MAP) tools, it is evident that there are possible connections between increased concentrations of nickel chloride and cyanotoxins and potentially harmful downstream effects including the activation of the formation of reactive oxygen species (ROS) as well as the potential for dopamine and sodium voltage-gated channer alpha subunit (SCN9A) inhibition. These results suggests that cyanotoxins and heavy metals both have largely negative downstream effects which should be a cause of concern for public health.

15) COVID-19 MODULATION OF NEUROINFLAMMATION IN MAJOR DEPRESSIVE DISORDER

Paige Murray and Heping Zhou
Department of Biological Sciences, Seton Hall University

Major Depressive Disorder (MDD) is one of the most common mental disorders affecting seven percent of adults in the United States, and there is evidence of neuroinflammation in MDD patients. COVID-19 patients have exhibited pathologies in a range of organ systems including the nervous system. COVID-19 pandemic itself is also a psychological stressor. This study was designed to examine neuroinflammation in MDD patients exposed to SARS-CoV-2. We analyzed the molecules associated with MDD and COVID-19 respectively using the Ingenuity Pathway Analysis (IPA) from QIAGEN. We found that neuroinflammation signaling pathway is one of the top canonical pathways involved in both MDD and COVID-19 patients. Further analysis identified key cytokines, including TNF-α, IL-1β, CXCL10, CXCL8/IL-8 and CCL2, involved in neuroinflammation of both MDD and COVID-19 patients. Documented research presents evidence for an increase of these key molecules in patients with MDD due to being a neuroinflammatory disorder. Further, our analysis suggests that the presence of the key cytokines is increased in COVID-19 infections, signifying an increase of neuroinflammation in COVID-19 patients. Our analysis suggests that neuroinflammation in MDD patients may be augmented by COVID-19.

16) USING IPA TO STUDY THE EFFECTS OF EXCESS ALCOHOL CONSUMPTION ON CARDIAC CONDITIONS

Tatiana Rengifo and Sulie Chang
Department of Biological Sciences, Seton Hall University

Alcohol abuse is a growing issue in the United States, especially in the young adult and college age populations. While alcohol is known to have many negative effects, the intermediary product found during ethanol breakdown in the human body known as acetaldehyde is a particular danger. Using Ingenuity Pathway Analysis, a bioinformatics network with access to many published studies, excess acetaldehyde levels and its effects on the cardiovascular system could be observed. The interaction network, disease and functions, and the molecular activity prediction (MAP) functions were used to observe the different body mechanisms that were affected by increased acetaldehyde levels. Increased acetaldehyde levels were found to be associated with many cardiac functions and diseases involving high blood pressure, premature cell death, myocardial infarction, and congenital heart disease. There were also associations found with genes that regulate these functions as increased acetaldehyde could increase or decrease the expression of these regulatory genes. These findings demonstrate that increased alcohol intake is detrimental to the human cardiovascular system, especially in the long term. By continuing to research and understand the negative effects of alcohol abuse, it may allow for the continued development of programs that raise awareness and help combat this harmful public health issue.

NEXT-GENERATION OMICS RESEARCH

17) A META-ANALYSIS OF THE EFFECTS OF EGCG ON BACILLUS SPP.

Sabrina Lopez and Tinchun Chu
Department of Biological Sciences, Seton Hall University

Having natural compounds as an alternative option can provide a different path for potential better preventative treatments. Epigallocatechin-3-gallate (EGCG), a green tea polyphenol which is one of the catechin-derivatives from the *Camellia sinensis* plant, possesses antimicrobial properties. Previous studies have reported the antiviral activity against herpes simplex virus and anti-spore activity against Gram-positive *Bacillus* species. *Bacillus* spp. can be detrimental to the food industry and in the medical field due to their production of enterotoxins and biofilm formation, which can be the main causes of food spoilage or cause major health issues within the human body. The aim of this project is to find a potential link between the effectiveness of EGCG on *Bacillus* spp. by QIAGEN Ingenuity Pathway Analysis (IPA). Molecules such as inosine and L-alanine that involved in the germination of *Bacillus* spores were identified with IPA BioProfiler. A pathway depicting the association between green tea polyphenol and bacterial diseases was generated. The core analysis results showed that toll-like receptor 4 (TLR4), a transmembrane protein which increases the susceptibility of bacterial sepsis, can be inhibited by EGCG. Results from the expression analysis revealed upregulation of TLR4 in bacterial respiratory infection and pneumococcal infection ($p = 4.51E-3$ and 2.68E-3, respectively). In summary, the meta-analysis of green tea polyphenols on bacteria provides further insights into the potential antibacterial and anti-spore mechanism of EGCG on *Bacillus* spp. *This research project is proposed as part of 2021 OMICS Program.*

18) EXPLORING THE ASSOCIATION BETWEEN CHRONIC ANATOXIN-A EXPOSURE AND SPORADIC AMYOTROPHIC LATERAL SCLEROSIS (ALS)

Danielle L. Maragh and Tinchun Chu
Department of Biological Sciences, Seton Hall University

Amyotrophic Lateral Sclerosis (ALS) is a neurodegenerative disease that primarily affects the motor neurons and results in progressive paralysis in patients due to the progressive weaken of voluntary muscles. Currently less than 5% of diagnosed cases for ALS is familial in nature with over 95% being considered Sporadic. Previous studies suggested that several environmental factors may play a role to the onset of ALS neurodegeneration. One potential environmental factor is cyanotoxins exposure. Cyanotoxins are toxins secreted by cyanobacteria harmful algal bloom (CHAB)-causing cyanobacteria. Anatoxin-a is a neurotoxin produced by several genera of freshwater cyanobacteria including *Anabaena* spp. The association between anatoxin-a, its analogs, and the progression of neurodegenerative diseases such as ALS have been explored. QIAGEN Ingenuity Pathway Analysis (IPA) BioProfiler was used to source genes and molecules associated with neurodegeneration such as solute carrier family 18 member A2 (SLC18A2), a dopamine transporter and prion protein (PRNP), a protein which plays a role in neuroprotection. IPA Network Analysis was utilized to explore the potential molecular interactions. IPA Molecule Activity Predictor (MAP) results indicated the dopamine inhibition in response to anatoxin-a elevation. Dopamine inhibition may contribute to the onset of ALS as dopamine deficiency results in the wasting of dopaminergic neurons and the eventually death of the neurons, these factors are said to contribute to the pathogenesis of sporadic ALS. *This research project is proposed as part of 2021 OMICS Program.*

19) IDENTIFYING TLR-3 ACTIVATING MOLECULES AS NEW ADDITIONAL ACTIVE INGREDIENTS FOR THE FLU VACCINE

Angelo Montero and Tinchun Chu
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The influenza viruses have caused serious public health crises in modern times. Though effective vaccines have been developed against them, adults of age 65 and older have been shown to not respond as well as younger people to the vaccination because of age-related changes in their immune system. Solutions to this problem include the use of an adjuvanted flu vaccine, as well as a high dose vaccine. However, the high dose and adjuvanted flu vaccines may generate more of the temporary, mild side effects that can occur with standard-dose seasonal shots. Therefore, it would be beneficial to investigate additional active ingredients to include in the vaccine preparation that could also boost immunity without the potential adverse effects already observed with the present alternative options. The molecules associated with Toll Like Receptor 3 (TLR3) were generated using the Grow and Trim tools of My Pathway in QIAGEN Ingenuity Pathway Analysis (IPA). Among them, 17 molecules that activate TLR3 were identified. Three of these molecules displayed direct activation of TLR3, namely rintatolimod, poly rI:rC-RNA, and Bruton's tyrosine kinase (BTK). As agonists of TLR3 that have been used successfully in mammalian systems, rintatolimod and poly rI:rC-RNA are potential candidates for new active ingredients to be used in flu vaccines to boost immunity in the elderly. *This research project is proposed as part of 2021 OMICs Program.*

20) GENES INVOLVED IN METASTATIC GASTRIC ADENOCARCINOMA AND THE NTRK2 EXPRESSION IN DIFFERENT CANCER TYPES

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Gastric cancer is aggressive and is often detected in an advanced stage. Every year this disease takes millions of lives worldwide and standard treatments such as chemotherapy are insufficient to combat this malignancy. Gastric adenocarcinoma is most common in individuals diagnosed with gastric cancer which furthers the need for additional research and targeted therapeutics for this type of cancer. Since most individuals diagnosed with gastric adenocarcinoma are in the late or metastatic stages, it is imperative to find target genes that can be exploited as potential treatment options. In the present study, the QIAGEN Ingenuity Pathway Analysis (IPA) software was used to identify the molecules that decreased activity affected metastatic gastric adenocarcinoma, including Neurotrophic Receptor Tyrosine Kinase 2 (NTRK2), ROS proto-oncogene 1, receptor tyrosine kinase (ROS1), Janus Kinase 2 (JAK2), Neurotrophic Receptor Tyrosine Kinase 3 (NTRK3) and anaplastic lymphoma kinase (ALK). Among them, NTRK2, a membrane-bound receptor that plays a role in various cancer types, was investigated further using the QIAGEN OmicSoft Land Explorer. The differential expression of the NTRK2 gene in disease vs. normal state from all reported studies revealed a 0.96 to 2.61-fold increase in gastric adenocarcinoma; a 1.09 to 4.23-fold decrease in colorectal cancer; a 1.18 to 4.62-fold decrease in invasive ductal carcinoma; and a 2.42 to 6.20-fold increase in glioblastoma. *This research project is proposed as part of 2021 OMICS Program.*

21) CANCER REOCCURRENCE: THE CONNECTION BETWEEN CELLULAR SENESCENCE AND CHEMOTHERAPEUTICS

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Cellular senescence, first described by Leonard Hayflick and Paul Moorhead in 1961, was observed at the time that a primary human fibroblast has a restricted lifespan of around fifty cell divisions in culture. It is now considered a signal transduction process where cells once they are damaged establish a stable proliferation arrest. Cells which become senescent are characterized by various metabolic and morphological changes, as well as the development of an inflammatory phenotype. This proinflammatory phenotype can be induced by chemotherapeutics, and as such have a potential to lead to cancer reoccurrence. Key factors from the SASP atlas developed by evaluating soluble protein SASP of primary fibroblasts (HCA2) 14 days after treatment with the protease inhibitor Atazanivir were analyzed using QIAGEN Ingenuity Pathway Analysis (IPA). This study aims to investigate the connection of chemotherapeutics, senescence, and tumor progression. It was hypothesized that there was a connection between factors secreted by chemotherapy induced senescent cells, and tumor progression. OmicSoft Land Explorer showed that log2 fold change of MCL-1 expression can be increased up to 14.2992 in cancer samples. My Pathway analysis and Molecule Activity Predictor (MAP) results showed that increased levels of myeloid cell leukemia 1 (MCL-1) can inhibit chemotherapy induced senescence and activate the proliferation of tumor cells. *This research project is proposed as part of 2021 OMICs Program.*

22) REGULATION OF ANTIMICROBIAL PEPTIDES HAMP, CAMP, AND ALPHA-DEFENSINS TO COMBAT ANTIMICROBIAL RESISTANCE

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Through evolutionary pressure from the development of antibiotics, bacteria have coevolved to overcome the mechanisms of action of many antibiotics most often by acquisition of resistance genes via horizontal gene transfer. Antimicrobial peptides (AMPs) provide a novel approach to combat antimicrobial resistance as alternative delivery molecules or synergistically with antimicrobials. In a sense the effector functions of the innate immunity, AMPs like hepcidin antimicrobial peptides (HAMP), cathelicidin antimicrobial peptides (CAMP), and alpha-defensins act as the body's natural broad-spectrum antibiotics against pathogens. QIAGEN Ingenuity Pathway Analysis (IPA) canonical pathway, OmicSoft, and core and comparison analyses were utilized to assess the bactericidal and bacteriostatic effects of genetic regulation of AMPs effects by nanoparticle delivery against Gram-positive *Staphylococcus aureus* (*S. aureus*) and Gram-negative *Escherichia coli* (*E. coli*). Canonical pathway analysis showed that heparin, toll like receptor 3 (TLR 3), bone morphogenetic protein 2 (BMP2), and heparin nanoparticles upregulated HAMP, which used the tumor necrosis factor (TNF) pathway to destroy *S. aureus* and *E. coli*. Chitosan nanoparticles, CCAAT enhancer binding protein alpha (CEBPA), and interleukin (IL-22) were major upregulators of CAMP, which killed *S. aureus* and *E. coli* by delivery molecules or directly. In alpha-defensins, which can be delivered by carbonate derivatives, Proteinase 3 antibody (PRTN3) and Matrix Metallopeptidase 7 (MMP 7), Interleukin1 (IL-1), and Fc gamma receptor 2a (FCGR2A) were identified as genes targets that either directly or via TNF-mediated pathways provide immune clearance of *S. aureus* and *E. coli*. In addition to confirming CAMP and HAMP's major role in the inflammation pathway, core and comparison analyses identified Reg3b (regenerating islet-derived 3β), Serpina3n (serine peptidase inhibitor A3N), and RNase I, which when applied to canonical analysis, use TNF-mediated or Interferon1 (IFN1)-mediated mechanisms to indirectly destroy *S. aureus* and *E. coli*. *This research project is proposed as part of 2021 OMICs Program.*

SENIOR SEMINAR CAPSTONE PROJECTS

23) META-ANALYSIS ON THE INVOLVEMENT OF STAT1 IN CH25H INHIBITION OF SARS-CoV-2 INVASION INTO THE HOST CELLS

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The Coronavirus disease (COVID-19) is caused by SARS-CoV-2 and is the cause of a global pandemic currently affecting our world. SARS-CoV-19 triggers the expression of many IFN-stimulated genes (ISGs). One of these ISGs is the STAT1 transcription factor, and it has been shown to be the most essential transcription factor involved in the induction of cholesterol 25-hydroxylase (CH25H). CH25H further converts cholesterol to 25-hydrocholesterol (25HC), which has been shown to block membrane fusion of the coronavirus. Using QIAGEN Ingenuity Pathway Analysis (IPA), a pathway was created between COVID-19 and the enzyme CH25H and using the "Grow" and "Connect" tools, the intermediate molecule STAT1 was found to be a connecting molecule between the virus and the enzyme. A Core Analysis of the molecules was performed, and it was found how they affect COVID-19. Through the Core Analysis I found an analysis on how STAT1 works and is involved in the induction of CH25H. These analyses were found through the IPA database and more analyses on how CH25H inhibits SARS-CoV-2 were found in the literature. STAT1 is involved in the interferon (IFN) antiviral response and works by binding to the promotor region of CH25H, and CH25H then converts cholesterol to 25HC. 25HC plays a big role in the antiviral response because activates the ACAT enzyme which depletes the amount of cholesterol in the lipid membrane. By depleting the cholesterol in the membrane, it reduces the ability of SARS-CoV-2 to bind to the cell and infect it, and thus stop virus replication, maturation, and secretion. STAT1 converts CH25H into the necessary molecules that will reduce the amount of cholesterol in the lipid bilayer and therefore, reduce the effect of COVID-19 on cells. These findings suggest that STAT1 and CH25H both play important roles in viral intervention, including the intervention of COVID-19. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

24) EFFECTS OF HYPOTHYROIDISM AND HYPERTHYROIDISM ON COVID-19

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COVID-19 from Coronavirus SARS-CoV-2 is a severe acute respiratory disease that presents itself with a range of symptoms including but not limited to fever, difficulty breathing, body aches, fatigue. Diseases such as obesity and diabetes have been known to put patients at higher risk of being infected with SARS-CoV-2 and are linked to increased severity of symptoms. Several thyroid conditions are known to cause a higher risk of diabetes, obesity, and a weakened immune system. We have hypothesized that having thyroid conditions such as hypothyroidism and hyperthyroidism may increase risk of infection from SARS-CoV-2 and may increase the severity of the symptoms. This study was done *in silico* with the use of QIAGEN Knowledge Base. Molecules which affect both hypothyroidism and Covid-19 were identified. Similarly, molecules which affect both hyperthyroidism and Covid-19 were identified. These overlapping molecules were then analyzed by QIAGEN Ingenuity Pathway Analysis. Our analysis indicated that Hypothyroidism and SARS-CoV-2 had 6 common molecules while hyperthyroidism and SARS-CoV-2 have 15 common molecules which affect them. These molecules included THRA and THRB. These overlapping molecules were mapped to several pathways including the TR/RXR Activation pathway. Our studies suggest that TR/RXR Activation pathway may affect the immune response to SARS-CoV-2. Using Ingenuity Pathway Analysis our findings suggest that hypothyroidism and hyperthyroidism may affect the severity of SARS-CoV-2 symptoms. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

25) META-ANALYSIS OF COVID 19 MODULATION OF ACE 2 LEADING TO SARCOPENIA

Thanelie Bien-Aime and Sulie L. Chang
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The global pandemic of Covid-19, caused by the virus, SARS-CoV2, has geared researchers to focus on Angiotensin Converting Enzyme-2 (ACE2), the receptor for the virus. Not only does the binding of SARS-CoV2 to ACE2 initiate virus entry, but it also compromises ACE2's protective function in affected organs. More specifically, recent research suggests that ACE2 protects against age-related muscle wasting (sarcopenia) through its ability to modulate molecules outside of the RAS system. We hypothesize that Covid -19's modulation of the ACE2 receptor increases the risk of sarcopenia in Covid-19 patients. This study used an in-silico approach to investigate potential links between ACE2 modulation (caused by Covid-19 infection) and sarcopenia. When SARS-CoV-2 binds to ACE2, it downregulates ACE2's expression. Utilizing the IPA Pathway Explorer and Molecule Activity Predictor (MAP) tools to obtain and explore the connections between ACE2 and Musculoskeletal disorders, I found that stimulating the inhibition of ACE2 ultimately predicts the activation of musculoskeletal disorders. The relationship between ACE2 and sarcopenia was more specifically explored, and I obtained the 6 shortest pathways between these two nodes. Utilizing MAP again, I observed the inhibition of ACE2, which predicted activation of the cytokines, Interleukin 6 (IL-6) and Tumor Necrosis Factor (TNF). The BioProfiler tool revealed increased activity for IL-6 and TNF affects sarcopenia, and these key mediators are biomarkers for diagnosis of sarcopenia in humans. Our studies using IPA and literature review demonstrate that Covid-19 modulation of the ACE2 receptor in skeletal muscle may augment sarcopenia by altering the activity of the key mediators. This might be due to a decrease in the protective function of ACE2 in skeletal muscle. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

26) ANTERIOR CRUCIATE LIGAMENT SURGICAL GRAFT OPTIONS TO OPTIMIZE RECOVERY

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Tearing of the anterior cruciate ligament (ACL) is one of the most common injuries in women's sports and in order to perform in one's sport again, a surgery is often required to ensure maximum quality of the knee. There has been a demand in determining which graft is the most beneficial in returning to one's sport with little to no complications. There are two main branches of surgery grafts: allografts and autografts. In this study, only autografts were focused on as they are more common and bring fewer complications of rejection. There are three main grafts then used for the surgery: bone-patellar tendon-bone graft (BPTB), the quadrupled hamstring tendon (HT), and the quadriceps tendon (QT). We proposed to recruit 40 patients: 10 being in each graft and 10 being in the control group of the nonsurgical route. The patients were examined and questioned for the pain levels, the strength of the quadriceps and hamstring as well as the major milestones in their recovery. The nonsurgical (control group) showed evidence of failing to fully recover, as well as experiencing inferior knee function. Individuals in the nonsurgical control group were able to return to sport eventually, however, the level of competitiveness was selective. The majority of the patients within the BPTB graph had excellent bone-to-bone healing through the tibial and femoral tunnels, with the addition of a faster healing process. Patients within the HT graph had minimal donor site morbidity compared to the BPTB autograph, and lastly, patients with the QT graph showed a decreased sign of pain throughout the recovery process and minimal analgesic consumption within the postoperative period. Results also suggested that individuals with hamstring graphs received higher doses of supplementary analgesic drugs, while individuals with quadricep graphs received little to no additional drugs. These results further enhance the theory that there is no superior option when it comes to the reconstruction of the knee, as the decision is ultimately dependent on the circumstances and preferences of the patient. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

27) NETWORK META-ANALYSIS OF COVID-19 MODULATION OF AMYLOID PRECURSOR PROTEIN IN BRAIN ENDOTHELIUM

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SARS-CoV-2 infection is responsible for COVID-19 in over 120 million people, many of whom present with neurological changes. SARS-CoV-2 infection begins with attachment to angiotensin-converting enzyme-2 (ACE2) which is involved in vasoconstriction, membrane trafficking, and inflammatory responses. SARS-CoV-2 has been reported to modulate expression of 83 genes in the brain microvascular endothelial cells (BMVEC) [Kaneko et al., 2020]. In this study, we explored how SARS-CoV-2-mediated gene expression in BMVEC might modulate the expression of Amyloid Precursor Protein (APP) using an in silico approach to identify potential connections between SARS-CoV-2 infection and APP expression. Among the 83 genes whose expression was modulated by SARS-CoV-2 infection, the 12 genes that were upregulated were analyzed for their effects on APP. The downstream targets affected by the 12 genes and the molecules associated with APP were identified via QIAGEN Knowledge Base (QKB). The compiled molecules were then evaluated using the QIAGEN Ingenuity Pathway Analysis (IPA), and the findings were statistically validated via quantitative analysis. Using the IPA networking and MAP tools on each of the 12 genes being upregulated, we found that 8 would activate (CD163, CXCL8, EBI3, IL9, IL15, ITGAL, OSM, TNFRSF17), 1 would inhibit (C3), and 3 would have no effect (APOBEC3G, CCL8, CCL24) on expression of APP. Moreover, the overall effects of concurrent activation of these 12 genes would result in strongly predicted activation of APP expression in BMVECs. This SARS-CoV-2 induced APP expression could involve the acute phase response, glucocorticoid receptor, and neuroinflammation canonical pathways, with -log(p-value) ranges of 26.1-41.2. Our in silico study utilizing IPA revealed that SARS-CoV-2 infection may cause a buildup of BMVEC APP through key inflammatory and vascular pathways, which may subsequently increase cerebral amyloid angiopathy prevalence. Our findings may help explain increased stroke prevalence in COVID-19 patients and may also warrant further exploration of potential long-term cognitive complications of COVID-19 including Alzheimer's disease. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

28) NETWORK META-ANALYSIS OF COVID-19 MODULATION OF ACE-2 LEADING TO ALZHEIMER'S DISEASE VIA SIMILARITIES IN INFLAMMATORY AND PLAQUE FORMATION PATHWAYS

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The coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome-related coronavirus (SARS-CoV-2), is responsible for the ongoing worldwide pandemic. Studies are currently being conducted to determine if the interaction between COVID-19 and angiotensin I converting enzyme 2 (ACE-2) is like that of severe acute respiratory syndrome coronavirus (SARS-CoV-1) where ACE-2 is internalized upon viral entry, therefore disrupting the Renin Angiotensin System (RAS) pathway. Post-mortem studies have also shown that COVID-19 induces acute hypoxic-ischemic brain injury and perivascular inflammation, especially in the white matter, which is important for cognitive function. This cerebral white matter damage and reduced activity of ACE-2 are both markers of Alzheimer's disease (AD). We hypothesized that if ACE-2 is internalized, viral infection of COVID-19 can thereby render a patient more likely to develop AD. An *in silico* approach was used to assess possible relationships between the modulation of ACE-2 in COVID-19 patients and the development of AD. The QIAGEN Coronavirus Network Explorer, QIAGEN Knowledge Base, and published literature were used to identify molecules affected by COVID-19 and ACE-2, especially those which play a role in the development of AD. QIAGEN Ingenuity Pathway Analysis (IPA) was then used to analyze each of these

molecules. Our analysis suggests that COVID-19 may modulate ACE-2 and increase the likelihood of developing AD as seen by their similar effects on cellular metabolism and neuroinflammation produced by common molecules including angiotensinogen (AGT), angiotensin I converting enzyme (ACE), amyloid precursor protein (APP), cytokines such as interleukin 1 beta (IL1β), interleukin 6 (IL6), and tumor necrosis factor (TNF), and the peptidases caspase 3 (CASP3) and caspase 8 (CASP8). Our *in silico* study findings using IPA suggest a link between COVID-19 and the modulation of ACE-2 leading to the development of AD. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

29) META-ANALYSIS OF THE MECHANISMS UNERLYING THE MILD PATHOLOGY OF COVID-19 OF CYSTIC FIBROSIS PATIENTS

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Cystic Fibrosis (CF) is a genetic disease that causes a build-up of mucus in the lungs and decreased activity of enzymes in the pancreas. It is characterized by the mutation or lack of the cystic fibrosis transmembrane conductance regulator (CFTR) protein. These patients normally get frequent severe lung infections. The research on SARS-CoV-2 infections in CF patients is limited because the occurrence of these patients getting sick with COVID 19 is small. Angiotensin converting enzyme 2 (ACE2) is the major receptor of the SARS-CoV-2 virus. In the research that has been done, I have found that patients with CF have experienced mild symptoms with COVID 19. I have hypothesized that the ACE2 regulator that binds with the virus is inhibited in patients with the CFTR protein mutation. In this study, I used Qiagen's Ingenuity Pathway Analysis (IPA) to analyze the pathway between the CFTR gene and the ACE2 regulator. The Molecule Activity Predictor (MAP) was used to predict the activation or inhibition of molecules in the pathway. The BioProfiler and Core Analysis tools were used to further understand the molecules and diseases worked with. In an expression analysis of molecules connecting CFTR and ACE2, 33 molecules were found to have an effect. When CFTR measurement is decreased, ACE2 is ultimately predicted to be inhibited according to the Molecule Activity Predictor in IPA, and activated with increased measurement of CFTR. Significant molecules in the upstream analysis include angiotensinogen (AGT), tumor necrosis factor (TNF), NFKBA, and Interleukin 1 beta (IL1β). BioProfiling of the cystic fibrosis disease showed that IL1β activity is increased with CFTR protein. The ACE2 regulator inhibition as seen in association with an decreased measurement of CFTR protein leads to a mild infection of COVID 19 in patients with cystic fibrosis. This finding is relieving for patients with CF, but precautions such as personal protective equipment and social distancing are still advised for these patients. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

30) SUSTENTACULAR CELLS INVOLVEMENT IN SARS-COV-2 VIRUS ENTRY, HYOSMIA & ANOSMIA

Cristina Casimiro

With the SARS-CoV-2 virus taking over the world creating a global pandemic there is one common symptom that seems to still have some unanswered questions. This one common symptom being, loss of taste and smell. It was found that many patients who suffered COVID-19 did experience this loss, the research on why/how this occurred was believed to have occurred one of four ways. I was able to thoroughly examine these four ways with IPA and the QIAGEN data base, through these examinations I was then able to eliminate and keep certain ones. Data gathered from RNAseq, RT-PCR, Western blot, and immunocytochemistry could be used to then determine the levels of ACE2 and TMPPRSS2 present on the actual sustentacular cells in the olfactory epithelium themselves. Understanding the location of where the ACE2 and TMPRSS2 is essential to then understanding the data gathered through IPA. There has been a plethora of data gathered about the natural processes of the human

body like how long it takes the body to replace dead olfactory receptor neurons and for cilia maturation. Looking at all the data it was found that large levels of ACE2 and TMPRSS2 were found on sustentacular cells of the olfactory epithelium. These levels were also very high in older individuals. There were very low if any levels of ACE2 and TMPRSS2 expressed on olfactory receptor neurons. It was also found that olfactory receptor neurons take about 8-10 days to be replaced with an addition of 5 days for cilia maturation. This will be demonstrated through pathways created on IPA. To conclude we were able to take a look at all the data gathered and eliminate three of the four possible ways COVID-19 patients lose their taste and smell. It is evident that SARS-CoV-2 has a target entry through the sustentacular cells of the olfactory epithelium because of the high levels of ACE2 and TMPRSS2, which means these cells could no longer support the olfactory receptor neuron. Which overall will effect the olfactory pathway leading to no smell. It was also found that these levels of ACE2 and TMPRSS2 increase as the individual's age increases, this could be a possible explanation as to why older individuals are more susceptible to the virus. I was also able to cut out loss of olfactory receptor neurons as the reason for this loss of smell because the process of recovering from dead olfactory receptor neurons is much longer than the time COVID-19 report that he/she had no smell. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

31) MODULATION OF CHLOROQUINE ON THE HEPATIC SYSTEM DUE TO COVID-19 TREATMENTS

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The coronavirus disease 2019 (COVID-19) has been the center of a worldwide pandemic since early 2019. This disease is categorized by its clinical presentation of pneumonia-like symptoms including fever, headache, and upper respiratory inflammation. Common treatments include anti-inflammatory medications, including chloroquine and its metabolite, hydroxychloroquine. Chloroquine has been linked to decreased interleukin 2 (IL-2) degradation; a cytokine previously linked to increased liver toxicity. I hypothesize that use of chloroquine and hydroxychloroquine in anti-inflammatory COVID relief responses is causing an increase in liver damage due to IL-2 activity. Molecules affected by chloroquine and IL-2 exposure were identified by analysis with QIAGEN Knowledge Base Molecules affected by COVID-19 were identified from both published literature on COVID-19 and the QIAGEN knowledge base. These molecules were then analyzed and compared by using the core analysis feature of QIAGEN's Ingenuity Pathway Analysis (IPA). A comparison between molecules regulated by Chloroquine suggests that Chloroquine, or hydroxychloroquine, exposure may contribute to liver damage or disease primarily by increasing the activity of IL-2, and also regulating the activity of key meditators including various cytokines and TNF-a, while also increasing levels of reactive oxygen species. *In silico* studies using IPA demonstrate that treatment with chloroquine or hydroxychloroquine may decrease COVID-19 induced inflammation but increase liver damage by influencing the activity of many different mediators. This finding suggests further investigation into the adverse effects of many treatments of COVID-19 that are currently being clinically administered. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

32) CRISPR-CAS GENE EDITING SYSTEMS AND POTENTIAL THERAPEUTIC USES

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CRISPR-Cas-mediated genome editing systems have increasingly become a highly relevant topic of scientific discourse. CRISPR gene editing systems have provided clinicians with a tool to effectively edit, modify, and replace targeted loci in a genome, allowing for the activation or silencing or particular gene expressions. Specifically, the CRISPR-Cas9 gene editing system has been proposed as treatment for several types of cancers, including lung cancer, leukemia, and gynecological cancers. CRISPR-Cas9 can be used to delete certain genes that have been previously determined to play a role in the proliferation and migration of cancer cells. Gynecological and lung cancer cell lines were used to test the effectiveness of the gene editing system, while CRISPR-edited stem cells were utilized in the treatment of an HIV and leukemia patient. Studies have also been conducted concerning CRISPR-Cas systems' ability to detect nucleic acids of various pathogens. Similarly, it has been proposed that the CRISPR-Cas9 system could be used to deliver immunity to COVID-19 using an approach called Prophylactic antiviral CRISPR in the human cells (PAC-MAN), as well as to detect the presence of SARS-CoV-2. The results from recent studies suggest that CRISPR-Cas9 could someday be used in gene therapy for neurological conditions, such as Huntington disease, fragile X syndrome, and others. While research into the aforementioned fields has been confined by ethical limitations, and has yet to yield definitively conclusive results, the current findings are promising. *This project is proposed as part of our Senior Biology Seminar capstone course.*

33) THE ANALYSIS OF THE SARS2 COVID-19 MRNA VACCINE AND ITS POTENTIAL EFFECTS ON FEMALE FERTILITY

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The global pandemic caused by the SARS-CoV-2 that began in 2019 was the beginning of a new type of vaccination known as an mRNA vaccine. This vaccine works by inserting mRNA, that codes for the surface proteins on the virus, into the human body. Once the mRNA is inside, the human cells will begin to make the protein directed by this mRNA. This protein, the spike protein, is seen as foreign, causing the body to produce an immune response against the spike protein; thus, leaving the body with antibodies to fight against the spike proteins on the coronavirus, should that vaccinated patient be exposed to the virus in vivo. In addition to coding for this glycoprotein, it has the potential to code for other glycoproteins, through transcriptional error. Syncytin-1 is a protein that is like the spike protein on the coronavirus, and it plays a significant role in the formation of the placenta in female mammals. This protein is theorized to have ancestral connections to the viral glycoproteins that the vaccine codes for, because they have very similar genomic sequences. With the use of the QIAGEN Ingenuity Pathway Analysis (IPA) and multiple scientific databases, information was collected on their mRNA and amino acid similarities, and the consequences that would occur if the vaccine was misinterpreted and transcribed to synthesize Syncytin-1. The potential for the mRNA to be mistranslated mirrors the potential for a cell to make the wrong protein, causing an immune attack on Syncytin-1 instead of SARS-CoV2 glycoproteins; thus, causing fertility complications due to an inability to form a placenta. *This project is proposed as part of our Senior Biology Seminar capstone course.*

34) COMPARING THE GENDER DIFFERENCES IN MELANOMA SURVIVAL, OUTCOMES, DEVELOPMENT, RISK FACTORS, AND PROGRESSION

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Melanoma is a form of skin cancer where melanocytes are mutated and proliferate uncontrollably. Melanoma can develop on any part of the body, but it often occurs in regions that are normally exposed to sun. Recent studies have found that there are differences in the outcome, metastasis, and incidence of melanoma between men and women. Some of the studies investigated the gender disparity in melanoma survival and development. Joose et al. reported that men tended to have less antioxidant enzyme expression compared with women. As a result, men had higher levels of reactive oxygen species which could possibly explain why men developed a more aggressive form of melanoma. In a study from the Munich Cancer Registry in Germany, melanomas which were localized in women had a lower chance of metastasizing compared to those found in men. This led to a greater survival rate for women. The 10-year overall survival rate for women was 76% compared to the 65% in men. These findings suggested that there is a gender difference in the development and survival of melanoma, however the difference may be caused by a collection of various factors. *This project is proposed as part of our Senior Biology Seminar capstone course.*

35) NETWORK META-ANALYSIS OF SARS-COV-2 INFECTION CAUSING MYOCARDITIS

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Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), caused a pandemic resulting in the coronavirus disease 2019 (COVID-19). Many are experiencing cardiac distress when succumbing to the SARS-CoV-2 virus. The cell signaling pathways used by SARS-CoV-2 to cause myocarditis, remain unclear. It is hypothesized that myocarditis is a result of the overactivation of cytokines and chemokines, rather than a direct attack on the cardiac system. This study used an *in silico* approach to examine how SARS-CoV-2 potentially causes myocarditis. Molecules affected by COVID-19 and myocarditis were identified using QIAGEN Coronavirus Network Explorer, and QIAGEN Knowledge Base. These molecules were analyzed using QIAGEN Ingenuity Pathway Analysis (IPA), the OIAGEN Knowledge Base, Molecule Activity Predictor, and Bioprofiler. Using IPA, a potential pathway has been produced. This pathway includes the activation or inhibition of ACE2, AGTR1, CCL2, IFNG, NR3C1, IL17A, IFNB1, F2R, IL1R1, IL1B and CLU. Data has shown that SARS-CoV-2 can upregulate CCL2, IFNG, IL1B, TNF-a, IL6 and can inhibit ACE2. The inhibition of ACE2 promotes the secretion of IL6. The activation of IFNG promoted by IL6 levels and SARS-CoV-2 leads to the inhibition of IL17A and NR3C1. The *in silico* studies using QIAGEN IPA suggest that the virus induces myocarditis by the enhanced activity of AGTR1, CCL2, IFNG, and IL1B while inhibiting ACE2, IFNB1, F2R, and CLU. Therefore, heart conditions are exacerbated and prolonged leading to extensive cardiac tissue damage by the overactivation of cytokines and chemokines. Acute cardiac injury and heart failure may not be symptoms of COVID-19, but they could be outcomes of the acute respiratory distress syndrome regardless of previous cardiovascular disease history. *This project is proposed as part of our Senior Biology Seminar capstone course.*

36) META-ANALYSIS OF GENETIC DELETION IN CAV 3 GENE AND CREATINE KINASE IN ITS EFFECT ON MORTALITY RATE IN COVID-19 INFECTED PATIENTS

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COVID-19 has been a worldwide epidemic since 2019. The disease is caused by the SARS-CoV-2 (acute respiratory syndrome coronavirus). The disease has been proven to be very contagious but have varying symptoms upon infection. Some infected individuals have been asymptomatic or experienced mild flu like symptoms. However, a significant percentage of infected individuals experienced severe and life-threatening symptoms as a result of certain predispositions. It was found that patients infected with COVID-19 had a lower survival rate if they had high creatine kinase (CK) levels. Analysis shows that unnaturally elevated creatine kinase levels in humans can be due to a deletion in the CAV 3 gene on chromosome 3. With the help of the QUIAGEN Knowledge Base, this gene was able to be analyzed in correlation with creatine kinase as well as its connection to COVID-19 patients. The virus enters by attaching to ACE 2 receptors on the cell membrane. ACE 2 is a protein found in respiratory cells primarily, but it is also found in muscle tissue in lesser amounts. As a result, muscle damage is done, and there is an interference in muscle energy production. These factors cause an increase in CK levels. Since the CK is already high in individuals with the CAV3 mutation, the symptoms worsen. Another contributor of muscle injury is immune-mediated pathway as a result of inflammation. In addition, dehydration and hypovolemia in COVID-19 patients may contribute to renal impairment and subsequent increase in CK levels. This study have addressed how CAV3 and CK might be involved in the pathologies of COVID-19. *This project is proposed as part of our Senior Biology Seminar capstone course.*

37) EFFICACY OF COVID-19 VACCINES BASED ON VACCINE TYPE AND ADMINISTRATION SCHEDULE

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Amidst a pandemic, pertinent questions and information have surfaced revolving around vaccine engineering and therefore the efficacy of the vaccines administered to the general population. Our research surrounds vaccines and their administration based on vaccine type and dosage. In this study, we first compared the efficacy of one dose versus two doses for each COVID-19 vaccine. Then we compared the effectiveness of three different COVID-19 vaccine types: Moderna, Pfizer, or Johnson & Johnson. Both experiments contained a control group in their respective categories. Following collection of data, statistical analysis was conducted to determine the significance of the results. The anticipated outcome should reveal that the greater number of doses given produced significantly greater immune response and antibody production as compared to the one dose group and control. In addition, the Pfizer vaccine should produce the greatest immune response and antibody production as compared to the Moderna or Johnson & Johnson. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

38) EFFECTS OF VANADIUM COMPOUNDS ON GENE EXPRESSION DURING BONE FRACTURE HEALING IN THE CHONDROGENIC ATDC5 CELL LINE

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The study used ATDC5 chondrocytes derived from mouse teratocarcinoma cells to evaluate the potential regenerative effects of vanadium compound treatment during chondrogenesis differentiation in fracture healing. ATDC5 chondrocytes were treated with DMEM/F12 media (untreated, negative control), 10 μM and 100 μM insulin (positive control), and vanadium compounds: vanadyl acetylacetonate (VAC) and vanadium (II) sulfate (VSO4) at concentrations of 10 μM and 100 μM. The ATDC5 lysates were harvested at days 1, 2, 4, 8, 10, 15, 18 and 22 for all treatment groups, RNA was isolated, quantified using a BioDrop™, followed by reverse transcription polymerase chain reaction (RT-PCR) and DNA gel electrophoresis. Lastly, gene expression of important markers of chondrogenesis (i.e. Collagen 2a1 and Collagen 10a1) and the housekeeping gene, glyceraldehyde 3-phosphate dehydrogenase (GAPDH) was assessed using quantitative real time polymerase chain reaction (qPCR) for each treatment group overtime. The qPCR analysis demonstrated that the gene expression of both Collagen 2a1 (COL2A1) and Collagen 10a1 (COL10A1) was more abundant in those treated with insulin or vanadium than the untreated at the specific time points. Our data affirmed the hypothesis that both insulin and vanadium promote bone fracture healing by increasing the concentration of COL2A1 and COL10A1 in the matrix during chondrogenesis which eventually leads to improved bone healing. The results suggest that vanadium compounds could potentially serve as an encouraging alternative to insulin in modulating chondrogenesis, especially in assisting and proliferating diabetic fracture healing. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

39) THE LONG-TERM EFFECTS OF PROLONGED SCREEN USE ON THE EYES AND PREVENTATIVE MEASURES TO MITIGATE THESE EFFECTS

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Increased technology use has been trending upwards for the past couple of decades and many of us rely on it in our daily lives. However, given the current state of the pandemic and most of our work is online, there are many concerns that prolonged screen use can have negative impacts on eye health. Maintaining healthy eyes is important for ensuring that we can view the world properly decades to come. Computer vision syndrome (CVS) and dry eye disease (DED) are two common conditions that are associated with screen use and affect millions globally. While treatment with artificial tears may be helpful, prevention is the best plan of action. By educating individuals on how to properly set up workstations with adequate lighting, proper ergonomic placement of the monitor, as well as optimal temperature and humidity conditions many of these eye problems can be avoided. Another preventative measure that is becoming more popular is the use of blue light-blocking glasses to help alleviate eye strain and promote better sleep. More research is needed to see if there is substantial merit to the blue light-blocking claims. With increased screen use, there is even more reason to stress prevention to avoid chronic eye conditions . *This research project is proposed as part of our Senior Biology Seminar capstone course.*

40) META-ANALYSIS OF IL-10 IN COVID-19 PATHOLOGIES

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Covid-19 is an infectious disease that is induced by a member of the family of coronaviruses, SARS-CoV2. This disease has brought upon a worldwide pandemic and has killed millions of individuals worldwide because of its nasty effects on the immune system. Some of the effects that this virus has on the immune system mimics those symptoms that present itself when an individual suffers from pneumonia, which is an infection that produces inflammation in the lungs. Present in both diseases, is a cytokine known as Interleukin-10, which acts an anti-inflammatory protein that suppresses immune and inflammatory responses, especially those that are present in both Covid-19 and Pneumonia. IL-10 is said to play a great role in gene therapy for patients suffering from pneumonia because of its ability to reduce inflammatory cytokine expression that occurs when a patient is diagnosed with bacterial pneumonia. Contrarily, in patients who have Covid-19, the elevation of IL-10 is said to play a pathological role especially in its pro inflammatory stages in the severity of the disease itself. The purpose of this study will be to do a meta-analysis of IL-10 in Covid-19 Pathologies and Il-10 in mediated gene therapy in Pneumonia. Specifically, I will be studying the specific molecules, JAK and SOCS1, as they are present in both diseases. We hypothesized that studying the molecular pathways will help us understand the difference in affects that IL-10 has on Covid-19. Using IPA's Bio-profiler tool, Canonical Pathway, and Molecular activity tool, I was able to determine that there is a direct relationship between the family of suppressor cytokines, specifically SOCS3, instead of SOCS1 and IL-10. After analyzing the canonical pathway of JAK/STAT using IPA, I was able to see that SOCS3 is more prevalent with a negative effect on IL-10. SOCS3 is a family of proteins that negative regulate JAK/STAT pathways, which is a pathway that IL-10 uses for receptor signaling and is the potential reason why there is a difference in patients who get pneumonia and Covid-19 pneumonia. *This project is proposed as part of our Senior Biology Seminar capstone course.*

41) THE RELATION BETWEEN NICOTINE AND SUSCEPTITILITY TO COVID-19

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There are two pandemics in the world, currently: COVID-19 and the use of e-cigarettes. In this study we will discuss both issues and how they relate to one another, along with the question of how nicotine may reduce or increase the risk of COVID-19 susceptibility. In December of 2019, in Wuhan, China, a pneumonia like disease was caused by a coronavirus (SARS-CoV-2). SARS-CoV-2, or severe acute respiratory syndrome coronavirus 2, is a single stranded RNA virus with particles that include structural proteins as well as genetic RNA material necessary for this virus to successfully invade a host cell. On the outside of the virus particle, the spike glycoprotein is found which is the main component of the covid infection due to its ability to recognize and attach to the human cell. Separately, the first electronic cigarette was successfully produced in Beijing, China, by a pharmacist/inventor and smoker, 52-year-old Hon Lik. E-cigarettes use mainly nicotine which binds to nicotinic acetylcholine receptors (nAChRs), usually causing depolarization of the neurons which are found along with elevated blood pressure and/or heart rate. The nAChRs become desensitized and less responsive with repeated usage resulting in a tolerance. The brain will upregulate or add more nAChRs as more receptors are desensitized. This study utilizes published literature from the QIAGEN Knowledge Base to determine the relation between nicotine and SARS-CoV-2 susceptibility. Using the "My Pathway" tool, a competition for the angiotensin-converting enzyme 2 or ACE2 receptor between SARS-CoV-2 and nicotinic acetylcholine receptors was identified by analysis. Studies have shown that users of E-cigarettes may have an increased rate of susceptibility to COVID-19 due to the aerosols irritating and damaging the lung cells, but utilization of IPA found nicotine may

worsen the interaction between COVID-19 and the ACE2 pathway. With the use of Core Analysis, relation between COVID-19 and nicotine was found to include TNF (tumor necrosis factor) along with IL-6 (interleukin-6). By using the comparison analysis tool in the QIAGEN Knowledge Base, it was found the ACE2 receptor correlated to Coronavirus Pathogenesis Pathway, Apelin Cardiac Fibroblast Signaling Pathway, Axonal Guidance Signaling and Coronavirus Replication Pathway. This significance of this study using IPA demonstrates the correlation of SARS-CoV-2 and the need of the ACE2 receptor to successfully bind to the human cell to cause infection. In addition, the study finds competition between nicotinic acetylcholine receptors and SARS-CoV-2 for the ACE2 receptor giving nicotinic acetylcholine receptors an advantage due to their abundance in numbers. Although addictive, the repeated use of nicotine, included from e-cigarette usage, may hinder the interaction between COVID-19 and the ACE2 pathway, concluding nicotine decreases susceptibility to COVID-19. *This project is proposed as part of our Senior Biology Seminar capstone course.*

42) CANCER IMMUNOTHERAPY: AN EXTENSIVE OVERVIEW OF THE PRESENT ADVANCES AND FUTURE POSSIBILITIES

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Cancer immunotherapy involves the common strategy of using therapeutic modalities to increase tumor immunity by manipulating the immune system using various immune agents. These immune agents can include the combined therapy of cytokines and monoclonal antibodies, vaccines, oncolytic viruses, adaptive T cell therapy, and checkpoint inhibitors. Immunotherapy treatment allows for a more specific approach to treating cancer by attempting to target the specific tumor through artificial stimulation of the immune system. It has become the most promising new cancer treatment approach since the 1940s when chemotherapy first emerged. Bladder cancer, breast cancer, and kidney cancer are among some of the several cancer types that have experienced success using cancer immunotherapy and are currently enrolling patients in clinical studies across the USA. In 1976, Morales et al. identified the success of Bacillus Calmette-Guerin (BCG) to treat superficial bladder cancer, and this is now a standard treatment. In recent years, cancer immunotherapy has made great strides and has become the fifth pillar of cancer therapy. It has wide adaptability with less potent side effects than other treatments, but there is high selectivity for patients, potential for negative regulation to occur, and high treatment costs among other challenges. Patients containing tumors with higher mutation burden seem to benefit most from this type of treatment while patients with less immunogenic tumors have struggled to achieve success. The future of cancer immunotherapy relies on the advances of further understanding which patient types will benefit most from various types of cancer immunotherapy and why. The aim of this review is to provide a comprehensive analysis of the history, types, approved therapies, clinical trials, advantages, and challenges, as well as the future of cancer immunotherapy. *This research project is proposed as part of our Senior Biology Seminar capstone course. This project is proposed as part of our Senior Biology Seminar capstone course.*

43) THE COLLAPSE OF CORAL REEFS: HOW VARIOUS STRESSORS, CORAL SURVIVABILITY, AND MANAGEMENT EFFECTS CORAL REEF ECOSYSTEMS

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Human activity has caused the rapid degradation of coral reefs through several factors leading to severe damage in both the corals themselves and the ecosystems they are a part of. Eutrophication, acidification, and surface water temperatures are the main stressors that lead to the coral bleaching phenomenon and eventually contribute to

ecosystem collapse. Past studies identified these stressors to be the main contributors to coral reef ecosystem degradation and all can be attributed to human activity. These studies focus on the coral's response to these stressors and recent research has been conducted to determine which reefs are most at risk. These findings comparatively analyze past bleaching events with newly constructed models to determine patterns in location as well as coral species differences in response to stressors like those listed. In this review, species of corals at risk are determined. Regions where corals are more likely to survive future bleaching events are also identified. The regions that corals could not survive bleaching indicated environmental implications regarding ecosystem health. Current trends in American Samoa and the Great Barrier Reef are being used to understand the ability of different reefs to support species. This research conflicts based on the region depending on local species and current conditions; however, it has been found that the loss of corals will lead to a chain reaction event collapsing the reef ecosystem. Lastly, there are several management techniques that have been employed in American Samoa and the Great Barrier Reef to mitigate this situation. This review summarized the short-term results for local management and future risk reduction and explored the potential long-term effects. *This research project is proposed as a part of our Senior Biology Seminar capstone course.*

44) META-ANALYSIS ON THE MOLECULAR MECHANISMS UNDERLYING COVID-19 AUGMENTATION OF EPILEPSY

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The coronavirus, SARS-CoV-2 is a virus which has led to a worldwide pandemic of respiratory illness. It is spread through respiratory droplets and occurs from close contact with an infected person. Its symptoms include fever, cough, shortness of breath, etc. Some people infected with the virus, about 1 in 6, will have complications, some of which are life threatening, and can damage organs through inflammatory proteins. Additionally, epilepsy is a chronic neurological disorder which leads to spontaneous and recurrent seizures. Each seizure differs in the location in the brain, how it spreads, how much of the brain is affected, and how long the seizure lasts. We hypothesized that seizures and epilepsy can be augmented in patients with complications from the coronavirus. We have conducted meta-analysis on molecules affected by epilepsy and COVID-19 using Ingenuity Pathway Analysis (IPA) and QIAGEN Knowledge Base. These molecules were identified from the QIAGEN pathway explorer, then studied more thoroughly through the Core Analysis and Bio profiler. Our analysis suggests that in patients affected by COVID-19, the G-protein coupled protein receptors play an important role in the immune response, specifically C5aR1 and C5aR2. These two GCPRs act as complement receptors and modulate inflammatory responses. Complement-mediated inflammation acts through these two proteins and triggers downstream pathways to affect its target molecules, IL-6, IL1β, and IFNG which are cytokines, specifically interleukins and interferons. An overproduction of cytokines may lead to a cytokine storm, making seizures more frequent. This shows that C5aR1 and C5aR2 may lead to the production of pro-inflammatory cytokines IL-6, IL1β, and IFNG, enhancing the excitability of neurons and increasing seizure susceptibility. *This project is proposed as part of our Senior Biology Seminar capstone course.*

45) THE EFFECTS OF CLIMATE CHANGE ON WILDLIFE CONSERVATION

Serina Johnson
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Climate change poses a threat to wildlife conservation. Climate change can be from natural causes or from human activities that increase the rate of climate change. The natural causes of climate change are the Earth's orbit or the carbon dioxide content in the atmosphere. The rate of climate change is increasing which causes the Earth's temperature to increase. The increased temperature causes glaciers to melt which causes rising sea levels. Climate change has affected wildlife conservation and ecosystems. Some effects include shifts in species distributions, reduction in population size, extinction of isolated species and populations, direct loss of habitat due to the rising sea level, increase fire frequency, direct warming of habitats, increased spread of invasive species, and many more. In this study, modelled species data, climate data, and protected area data were reviewed to assess the climate change impacts on species. The data has predicted that climate change is already affecting wildlife. The articles then used this data to come up with adaptation strategies to combat the climate change. These strategies were then divided into four categories to make it easier to identify the strategies. The four categories are land and water management, direct species management, monitoring and planning, and law and policy. These strategies were implemented to protect wildlife conservation and ensure species will not become extinct. *This project is proposed as part of our Senior Biology Seminar capstone course.*

46) NETWORK META-ANALYSIS OF COVID-19 MODULATION OF PSEN1 LEADING TO ALZHEIMER'S DISEASE

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COVID-19 (Coronavirus Disease 2019) is a highly contagious respiratory disease that is caused by SARS-CoV-2 virus. Many know of this disease to cause primarily respiratory problems. However, the virus can cause a potential cytokine storm, which in turn causes damage to cells and organs. SARS-CoV-2 virus causes an autoimmune response within a patient's body; therefore, it can influence certain molecules to cause harm within one's body. One molecule is Tumor Necrosis Factor (TNF), which can cause inflammation in the body. Alzheimer's disease(AD) is a progressive disease that causes destruction of brain cell connections leading to cell death and the subsequent memory loss and disruption of other mental functions. TNF has been found to be related to COVID-19, AD and PSEN1 gene using QIAGEN IPA knowledge base. Further, the networking molecules to COVID-19, AD, and PASE1 were analyzed. Our studies have found that persistent illness with COVID-19 has elevated TNF and the build-up of inflammation, leading to apoptosis and cell death. The resulting factors caused by a cytokine build-up can potentially mutate PSEN1. Mutation of PSEN1 could enhance onset and progression of AD. COVID-19 possible enhancement of AD onset and progression shall be a serious clinical challenge following COVID-19 pandemic. *This project is proposed as part of our Senior Biology Seminar capstone course.*

47) ANALYSIS ON TYPE 2 DIABETES MELLITUS AUGMENTATION OF COVID-19 PATHOLOGIES

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The purpose of this study is to identify novel molecular connections that link Type 2 Diabetes Mellitus and COVID-19 in order to reveal why diabetic patients are predisposed to severe cases of COVID-19. In this study, I identified genes and proteins of interest that are involved in diabetes and COVID-19 by using Qiagen's Ingenuity Pathway Analysis (IPA) genomic software. The IPA software consists of the cutting-edge bioinformatics tools and QIAGEN Knowledge Base. It can identify important targets such as genes or proteins, interconnect their importance to diabetes and COVID-19, and predict their likely effects in organisms. IPA analysis was completed by inputting terms such as "non-type 1 diabetes mellitus", "COVID-19", and relevant molecule names into IPA's search tool. These were then inputted into IPA's Pathway Designer tool and connected using the Build and Connect tools. The Grow tool was used to find more genes relevant to both conditions. Unhelpful findings were reduced by filtering out items that did not yield viable connections. The IPA results demonstrated that the gene ACE2 and relevant inflammatory molecules like IL-6 could significantly increase the severity of COVID-19 in diabetic patients. *This project is proposed as part of our Senior Biology Seminar capstone course.*

48) NETWORK META-ANALYSIS OF DEHYDRATION EFFECT ON PATHOPHYSIOLOGY OF MYASTHENIA GRAVIS THROUGH ANALYSIS OF INTERLEUKIN 4

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Dehydration affects approximately between 17-28% of the population. Various studies were scrutinized on the effects of dehydration on volunteers varying from sample sizes of 21 to over 1000. Based on these premises, we have to hypothesized if dehydration leading to a decreased Mucociliary clearance, air capacity and strength of immune response will exacerbate, not only, the severity of COVID-19 but Myasthenia Gravis as well. Although, no clear-cut way to detect the presence of dehydration, speculation lies in that dehydration begins when the serum osmolality concentration becomes lower than 295 mOsm/kg. Below this point indicates a physiological alteration that is responsible for a significant reduction in forced vital capacity (300 ± 190 ml, $p = 0.001$) and concomitant increases in residual volume (260 ± 180 ml, $p = 0.001$) and functional residual capacity (260 ± 250 ml, $p = 0.011$). This study was conducted on athletes suffering from dehydration. Generally, the results indicate dehydration facilitates a reduction in function of respiratory system. Bringing forward the question of susceptibility to increased severity from COVID-19 and from the famous COVID-19 cytokine storm. The primary component responsible for pathogen removal through mucociliary clearance (MCC) is mucus which is hindered during dehydration. Therefore, from dehydration to a decreased function of MCC, this leads to theorize whether a possible strong correlation of there being a higher contraction rate of pathogenic material during a dehydrated state. As well as the implications of how the increased inflammatory mediators, specifically interleukin 4, could substantialize the hypothesis. Creating more inflammatory mediators to attack the neuromuscular junctions affected in Myasthenia Gravis. The tie lies in using interleukin 4 (IL-4) as a locus and then investigating the implications of an increase in concentration of IL-4 through an analysis of the JAK-STAT pathway. Which correlates to Myasthenia Gravis through STAT6. A big picture analysis based on the information accrued leaves the theory that it is possible increased concentration of IL-4, decreased vital, residual and forced capacity from the lungs and decreased MCC function would attribute to an increased severity of COVID-19 symptoms as well as progress the severity of Myasthenia Gravis.

49) PATHWAY ANALYSIS OF THE MECHANISMS UNDERLYING SMOKER'S ENHANCED SUSCEPTIBILITY TO COVID-19

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SARS-CoV-2 (COVID-19) is a coronavirus that directly affects the respiratory system and causes a variety of symptoms and can cause severe illness. Those who smoke are aware of the risks, which include: cancer, heart disease, stroke, lung disease, diabetes, and chronic obstructive pulmonary disease. When smoking, Carbon Monoxide (CO), a toxic chemical, is inhaled and passes through the lungs into the blood. We hypothesized that smoking, and therefore an increased intake of the chemical CO, would increase the susceptibility to infection by COVID-19. We identified the molecules affected by CO and COVID-19 by using the QIAGEN Knowledge Base and published literature, respectively. We used the QIAGEN Ingenuity Pathway Analysis (IPA) to connect molecules and find if an intake of CO affects the susceptibility of COVID-19. Using the QIAGEN Knowledge Base, we identified that an influx of CO leads to inflammation. We used IPA for a pathway analysis of CO to inflammation and noted all of the molecules involved in the pathway and identifies which were a common factor. We noted that cytokine IL-6 was involved in every pathway. We found that the ACE2 receptor is the receptor for COVID-19 and that smoking increases cytokine IL-6 while down regulating ACE2 receptor by inhibiting superoxide dismutase 2 (SOD2). Our research using QIAGEN IPA shows that individuals who smoke are putting themselves at a greater risk of getting COVID-19 since smoking allows the passage of CO into the blood which increases plasma level of cytokine IL-6 and inhibits SOD2, which is the protein that counteracts the oxidative stress caused by COVID-19 by neutralizing oxidants. This inhibition of SOD2 leads to a downregulation of the ACE2 receptor and this leads to increased susceptibility of COVID-19. *This project is proposed as part of our Senior Biology Seminar capstone course.*

50) META-ANALYSIS OF COVID-19 MODULATION OF INTERMEDIARY MOLECULES AUGMENTING THE OCCURRENCE OF SEIZURES

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Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a novel coronavirus that causes mild to severe respiratory disease called coronavirus disease 2019 (COVID-19) in humans and has caused a global pandemic in 2020. COVID-19 modulates varying acute conditions such as fever, respiratory distress syndrome, and in some cases neurological conditions. There have been isolated cases of COVID-19 patients that have experienced seizures following their diagnosis, but there is not much research yet on why seizures are triggered by COVID-19. Our hypothesis was that COVID-19 causes changes in expression of a number of genes that may augment seizures in COVID-19 patients. This study utilized an *in-silico* approach to examine possible relationships between COVID-19 infection and the trigger of seizures. Molecules affected by COVID-19 were identified using the QIAGEN Knowledge Base. Molecules that augment seizure activity were identified using the BioProfiler Tool, and those molecules subjected to the Core Analysis Tool in IPA to find which molecules were modulated by COVID-19. These molecules were used to formulate a pathway that may explain the influence COVID-19 has on the augmentation of seizures through intermediary molecules. Our analysis suggests that COVID-19 infection may affect the activity of cytokines in the brain, causing inflammatory responses such as fevers, which often precede seizures. Two cytokines that were found to have connections to both COVID-19 and seizures were IL6 and IL-1β. IL-6 was found to be a molecule triggered downstream as part of the IL-10 signaling pathway. In addition, our finding indicated that the transcription regulator FOS may increase the activity of seizures when downregulated. Our findings using the QIAGEN Knowledge Base indicate that COVID-19 may modulate cytokines and transcriptase regulators to augment seizures. This is an important finding because individuals who have epilepsy could be at higher risk when infected with COVID-19 to have recurring seizures. *This project is proposed as part of our Senior Biology Seminar capstone course.*

51) ANALYSIS ON HOW COVID-19 AFFECTS PATIENTS WITH VALVULAR REGURGITATION

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COVID-19 poses a risk of death to people and a high infection rate which is ravaging the world in a pandemic. Considering there are many people with heart disorders, this project is aiming to assess the risk COVID-19 poses to patients with Valvular Regurgitation (VR) by comparing the two diseases and seeing what molecules both affect and attempting to understand if COVID-19 can increase VR symptomology, increasing death rate. This project also aims to see what drugs would be effective in reducing symptoms of both diseases as well. I have been using IPA Qiagen database to compare both disease and associated molecules to find the molecules of interest as well as the graphs. I will be using the MAP tool to check how increasing or decreasing a specific drug affects the diseases as well. I have found that there 22 molecules that are associated with VR and COVID-19 which are: 5-hydroxytryptamine, AGTR1, aspirin, CACNA1C, CACNA1D, CACNA1S, CACNB2, cyclooxygenase, PDE3A, PDE4A, PDE4B, PDE4C, PDE4D, PDE7A, PDE7B, PDE8A, PTGS1, PTGS2, rivaroxaban, valsartan, voltage-gated calcium channel. I have mainly found that aspirin (by bonding to cyclooxygenase and preventing binding with PTGS1/2) and rivaroxaban (as anticoagulant/blood thinner) specifically is useful in reducing symptomatology as seen from utilizing MAP. My findings thus far conclude that COVID-19 will have more risk on VR patients and some good drug to combat these in conjunction are aspirin and rivaroxaban. *This project is proposed as part of our Senior Biology Seminar capstone course.*

52) THE IMPACT OF THE GUT MICROBIOTA ON DEPRESSION

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Depression is a mental disorder that causes a constant feeling of loss of interest and sadness, which causes termination of normal activities in those impacted by depression. Recent studies have shown that the gut microbiota can have a serious impact on mental disorders due to the communication of the gut and the brain. The use of probiotics has showed very promising results for the treatment of anxiety and depression. In a trial using a combination probiotic which contained *Lactobacillus helveticus* and *Bifidobacterium longum*, it was found that these specific probiotics can be used to help treat those with depression. Probiotics secrete signaling molecules throughout different pathways in order to apply their effects. Mesaoudi et al. reported that when *Bifidobacterium longum* is used in addition to *L. helveticus* in probiotic sticks, depressive-like symptoms were reduced in healthy individuals. *L. helveticus* also improves cognitive impairments, such as memory and concentration, in chronic-stressed rats. This shows that probiotics may in fact have very similar affects as antidepressants do on individuals with symptoms of depression. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

53) SEXUAL DIMORPHISMS IN AUTISM SPECTRUM DISORDER: A COMPREHENSIVE REVIEW OF THE COMPONENTS ASSOCIATED WITH MALE PREVALENCE IN ASD

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Autism Spectrum Disorder (ASD) is a developmental disorder characterized by social and behavioral challenges. In the United States, 1 in 54 children are diagnosed with autism and the majority of them are males. The male prevalence in autism is estimated to be a 3:1 or 2:1 ratio when compared to the diagnosis of females. Despite numerous research studies, the causes and risk factors of ASD are still unknown as autism is defined by its heterogeneity. This review has explored the recent findings regarding various biological differences between males and females that are potentially contributing to the male prevalence in ASD. One of these biological differences being male risk factors which define the vulnerability of males to be diagnosed with ASD and other neurological disorders and female protective effects which cause females to fall short of the diagnostic threshold for ASD. Sex hormones and gender-related differences, specifically fetal testosterone, and androgens, demonstrated an increase in "masculinization" and a risk for diagnosis of ASD in males. Neural masculinization and its effect on males and females with ASD are described by the Extreme Male Brain (EMB) theory. The criteria tested for the EMB theory conclude a shift toward masculinization among those females who were diagnosed with ASD and hypermasculinity for individuals with ASD. Finally, brain volumes were measured and proven to be larger in males, especially those with autism. These sexual dimorphisms suggest the biological differences that are likely contributing to the prevalence of males in ASD diagnoses. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

54) MOLECULAR NETWORKING BETWEEN GUILLAIN-BARRE SYNDROME AND COVID-19

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Guillain Barre Syndrome (GBS) is a rare medical disorder in which the body's immune system mistakenly launches an attack on the peripheral nerves. The attack is mainly targeted at myelin sheaths and when they are damaged, nerves are not able to conduct electrical impulses normally. As more damage is done to the nerves, the person will develop a difficulty with walking, an increase in fatigue, as well as a difficulty in breathing. Most people get paralyzed even after consistent therapy and end up not fully recovering. GBS can even cause paralysis of the muscles that control one's own breathing. COVID-19 is a respiratory and infectious disease caused by SARS-CoV-2. Symptoms may appear 2-14 days after exposure to the virus and common early phase symptoms include coughing, fever, and body aches. Even though GBS is pretty rare, it seems that more studies are being done on this disease because of covid. Some doctors have noticed a trend between patients that had both GBS at one point and covid at another. They are particularly concerned about GBS recurrences that they have noticed in patients after they test positive for covid. This has happened to some as early as 7-10 days after testing positive. I chose to relate this syndrome to covid because I wanted to find any similarities in genes or molecules that may tie these two together. Molecules affected by COVID-19 were identified from published literature using QIAGEN Knowledge Base, as well as molecules that affected GBS. I also used Ingenuity Pathway Analysis (IPA) to compare which molecules were responsible for increasing or decreasing the chances of getting both diseases. The analysis performed showed that there are similarities in molecules between covid and Guillain Barre. For example, I was able to find that molecules C3 is one of the main molecules that affects GBS, and meanwhile in covid, the same molecule C3 is responsible for affecting infection by coronavirus. Molecules C4A/C4B are molecules that affect GBS, and that same molecule is responsible for affecting infection by covid. Using IPA tools, I was also able to find similarities in genes that affected GBS and also affected infection by coronavirus.

These genes included FCGR1B, FCGR2A, FCGR2B, FCGR2C, and FCGR3A/FCGR3B. Based on my studies on these 2 diseases, I found there were many molecules that were similar in both. This is very interesting because it shows that even though there is still little information on a tie between the two, there may be some sort of networking of molecules connecting these two diseases. *This project is proposed as part of our Senior Biology Seminar capstone course.*

55) ANALYSIS OF THE EFFECTS OF ADDERALL ON COVID-19 PATHOLOGY

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The coronavirus disease 2019 (COVID-19) is a worldwide pandemic that is caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). A common symptom of COVID-19 is cytokine storms. The drug Adderall is used to treat common disorders such as Attention Deficit Disorder. When a short analysis of the drug was done using QIAGEN Ingenuity Pathway Analysis (IPA) it was found that an increased level of Adderall caused an activation in viral infection. This led to the hypothesis that Adderall increases the pathology of COVID-19. Using IPA, I found 9 molecules that showed a relation between Adderall (amphetamine in IPA) and COVID-19. Of these 9 molecules, only one of them was a cytokine, interleukin 10 (IL-10). IL-10 is also known as human cytokine synthesis inhibitory factor (CSIF), is an anti-inflammatory cytokine. In humans, interleukin 10 is encoded by the IL-10 gene and is primarily produced by monocytes and, to a lesser extent, lymphocytes. IL-10 downregulates the expression of Th1 cytokines, MHC class II antigens, and co-stimulatory molecules on macrophages. It also enhances B cell survival, proliferation, and antibody production. Using the QIAGEN literature database I was able to find a study from ScienceDirect conveyed that IL10 is a predictor for how severe the infection of COVID-19 will be. The study showed that the IL-10 levels are much higher in the critical group of patients than the moderate and severe groups. A study from Taylor & Francis showed that amphetamine increases the quantity of IL-10 in lab rats. While the affect that Adderall has on the pathology of COVID-19 was not found, however, it was found that Adderall can make the infection of COVID-19 much worse through raising the levels of IL-10. My findings show that Adderall can augment COVID-19 pathologies. *This project is proposed as part of our Senior Biology Seminar capstone course.*

56) CREATINE MONOHYDRATE, THE MOST POPULAR SUPPLEMENT USED BY ATHLETES, INTENDED USE AND EFFECTS

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Creatine monohydrate is arguably one of the most used supplements by athletes in recent years because of its ability to help improve athletic performance and recovery. Creatine is a naturally occurring substance in the body, mostly found in skeletal muscles, which plays a role in maintaining energy homeostasis, especially in cells that require a high amount of energy to function. Creatine's role in energy homeostasis is to act as a way of buffering ATP levels in tissues that have a high, but fluctuating level of energy demands such as the skeletal muscles in your body. Since these tissues in your body require such a high amount of energy so rapidly, creatine is necessary for these tissues to function optimally. In this review, the creatine usage and its effects for both athletes and non-athletes are addressed. Improved muscle recovery as well as improved cognitive function have been observed from multiple studies. Potential therapeutic application for certain diseases such as Parkinson's has also been explored. Studies of the effects of creatine monohydrate on non-athletes should be continued to understand the benefit and health risks of this supplement on the general public. Further research assessing long-term effects and potential adverse effects is also required to monitor supplement safety. *This project is proposed as part of our Senior Biology Seminar capstone course.*

57) T-LYMPHOCYTE EXHAUSTION IN COVID-19 AND STAGE I-II NON-SMALL CELL LUNG CANCER: A META-ANALYSIS

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With the development of severe acute respiratory syndrome coronavirus 2 (SARS-CoV2) as a novel respiratory virus in 2019, research has been aimed at understanding molecular targets that can be exploited for the development of novel therapeutics. Though emerging evidence demonstrates the pleiotropic effects of SARS-CoV2 within the human body, the primary mode of infection occurs via a respiratory-dependent pathway that targets the angiotensin converting enzyme 2 (ACE2) receptor. Among patients affected with severe cases of SARS-CoV2, the mechanism of respiratory distress and tissue damage is postulated to occur via cytokine storms, which can give way to T-lymphocyte exhaustion. Exhaustion of T-lymphocytes is also a phenomenon exhibited in certain cancers, yet current studies are lacking on the potential correlative effects between SARS-CoV2 and lung cancer. As such, the current analysis focuses on the molecular mechanisms of severe SARS-CoV2 [coronavirus disease 2019 (COVID-19)] infection and its effects on the lungs to determine potential correlations between severe COVID-19 and lung cancer risk. In the following meta-analysis, the QIAGEN database was implemented for core analysis of molecules associated with severe COVID-19 and for core analysis of molecules associated with stage I-II non-small cell lung cancer (NSCLC). Review of the canonical pathway for T-lymphocyte exhaustion revealed several molecules from both datasets that were implicated in this pathway. Using an *in-silico* method, it was determined that several biomarkers for severe COVID-19 upregulate the NSCLC biomarkers KRAS, PDCD1, and CD274 within the T-lymphocyte exhaustion pathway. This correlation warrants further analysis to determine whether such connections indicate increased risk of developing NSCLC as a result of severe COVID-19 infection. Furthermore, this data may provide context for analysis of immune checkpoint inhibitor efficacy in NSCLC patients who previously suffered from severe COVID-19. *This project is proposed as part of our Senior Biology Seminar capstone course.*

58) EXAMINING MECHANISM UNDERLYING PERSISTENT COVID-19 PNEUMONIA ENHANCING RISK OF LUNG CANCER

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The coronavirus disease 2019 (COVID-19) pandemic is a crisis that has impacted millions of people worldwide. COVID-19 is triggered by SARS-CoV-2 infection and leads to respiratory tract infections. Many hospitalized COVID-19 patients develop Pneumonia and other respiratory issues. The Pneumonia initiated by COVID-19 is harmful due to the potential spread of infection to smaller sections of the Lung, affecting the overall functionality of the lung. Lung cancer is a specific type of cancer that originates in the lung but metastasizes to other parts of the body through the bloodstream. This type of cancer proliferates in the bronchi, bronchioles, and alveoli affecting lung functionality. Examination of the common symptoms of COVID-19, including widespread Pneumonia, suggests possible relations between COVID-19 Pneumonia and Lung cancer which we sought to study in this report. We hypothesized that a prolonged exposure of SARS-CoV-2 pneumonia can stimulate cell proliferation in the lungs potentially leading to lung cancer. To begin, molecular relations between COVID-19, Pneumonia, and Lung Cancer were identified by the QIAGEN Knowledge Base (QKB). Then, utilizing the database different molecules were analyzed and signaling pathways were examined in relation to different diseases. Our analysis suggests that the long exposure of COVID-19 pneumonia infection may augment the effects of lung functionality. While researching the gene GATA3- a transcription regulator and gene TTF1+ a

transcription factor resulted to be expressed in Squamous Cell Lung Carcinoma and Adenocarcinoma of the lung. From the analysis obtained the viral protein downregulates gene TTF1 which affects the function of the gene. Our studies utilizing QKB and IPA tools showcase that there is a possibility of Lung Cancer being initiated from the inflammation, damage, and targeted infection of COVID-19 Pneumonia. Our research advises that limited exposure to virus infection and rapid recovery can lead to minimal lung damage decreasing the chances of Lung Cancer. *This project is proposed as part of our Senior Biology Seminar capstone course.*

59) PATHWAY ANALYSIS OF THE MECHANISMS INVOLVED IN THE SUSCEPTIBILITY OF COVID-19 FOR INDIVIDUALS WHO SMOKE

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SARS-COV-2 (COVID-19), a corona virus that affects the respiratory system. Individuals who smoke are already causing damage to their lungs from the increased intake of the toxic chemical, Carbon monoxide (CO). We hypothesized that smoking (an increased intake of the chemical CO) would increase the susceptibility to augment COVID-19 pathologies. Using QIAGEN Knowledge Base, we identified the molecules affected by Carbon monoxide (CO) and or COVID-19 we used the QIAGEN Coronavirus Network Explorer. We took the overlapping molecules between both of the CO and COVID-19 and used the QIAGEN Ingenuity Pathway Analysis (IPA) to connect the different molecules to find if an intake of CO affects the susceptibility of COVID-19. Using the QIAGEN knowledge base, we identified that an influx of Carbon monoxide leads to multiple inflammatory diseases to the respiratory system whether that is alveolitis, hypoxia, and chronic inflammation to lung tissue. We then used IPA for a pathway analysis of CO to each of the three inflammatory diseases and COVID-19. We noted all of the molecules involved in the pathways and highlighted the ones that occurred throughout. We noted that every canonical pathway included the molecule inflammatory cytokine IL-6. We also found that COVID-19's receptor is the ACE 2 receptor. Using IPA, we connected IL-6 with ACE 2 and found all pathways connected at SOD2, protein that regulates the activity of the ACE 2 receptor. After additional research we found that the intake of Carbon monoxide not only increases IL-6 production but also inhibits SOD2 activity, causing downregulation of ACE 2. Our research using QIAGEN IPA showed that individuals who choose to smoke are putting themselves at a greater risk of getting COVID-19 since smoking directly increases the amount of cytokine IL-6 and inhibiting SOD2 leading to a downregulation of ACE 2 receptor and increased susceptibility to COVID-19. *This project is proposed as part of our Senior Biology Seminar capstone course.*

60) META-ANALYSIS OF COVID-19 IN DEVELOPMENT OF NEURODEGENERATIVE DISEASE

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Covid-19 is an infectious disease also known as severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). Patients infected with Covid-19 have expressed issue with concentrating, headaches, and memory issues. Neurodegenerative diseases occur when nerve cells are damaged, lose their function, and eventually die. The risk of developing neurodegenerative disease increase with age. Additionally, a person's genes and environment contribute to the risk of disease development. We hypothesize that Covid-19 increases the risk of developing neurodegenerative disease. To study neurodegeneration, we hose to study one of the most common

neurodegenerative diseases, Alzheimer's. We also examine the P2X7 receptor (P2X7R), an ATP-dependent receptor expressed on the central nervous system (CNS) with several mechanisms associated with multiple neurodegenerative diseases. Molecules affecting Covid-19 and Alzheimer's disease were identified using the QIAGEN database. These molecules are found to be produced by P2X7R. IPA pathway analysis is used to develop and analyze a pathway demonstrating the relationships between the molecules of interest. Our study shows that a patient infected with Covid-19 enhances levels of extracellular ATP which triggers activation of P2X7R. P2X7R enhances production and activity of cytokines like IL-1β, IL-18, and TNF-α, transcription factors such as JNK, MEK, and NFκB. P2X7R activation causes an efflux of K^+ and influx of Ca^{2+}. Overexpression of P2X7R induces apoptosis of cells via the activation of caspase pathway as well as neuronal damage via cytokines. Additionally, specific activity of this receptor induces the cleavage of Amyloid Precursor Protein (APP) generating Aβ, a factor that plays in production of amyloid plaques which is commonly found in Alzheimer's by disrupting the function of neuron. Using IPA, our findings show that patients infected with Covid-19 may increase susceptibility in developing neurodegenerative disorders including Alzheimer's disease. *This project is proposed as part of our Senior Biology Seminar capstone course.*

61) THE COVID-19 CYTOKINE STORM AND THE ASSOCIATION OF THIS CYTOKINE STORM WITH BROAD SPECTRUM ACUTE CORONARY SYNDROMES

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The Cytokine storm that is associated with Covid-19 is believed to cause the heart damage. This study is to investigate the cytokines that are known to be upregulated by Covid-19 infection, and to look for pathways that will result in increased risk of developing an Acute Coronary Syndrome (ACS) due to Covid-19 infection. For this investigation, the IPA QIAGEN database was used to collect the data of the pathways. The primary method for collecting the data was utilizing the ability to form pathways in the IPA program. The first step to building this pathway was done by acquiring both the Node for Acute Coronary Syndrome, and Covid-19. After these markers are acquired you can run a pathway explorer between the two diseases with it going from covid to ACS. Once you create the pathway make sure to add shortest pathways +1 to expand the effects of the previously selected cytokine node type. Once this is done you can go through a molecule activity predictor where you can see the results and take note that you can also acquire information through the information provided by IPA about the pathway by double clicking on it. From this we can see there are 6 principle cytokines that get upregulated by Covid-19 which are IL-18, IL-6, C5, IFNG,TNF, CSF3.The upregulation of these 6 cytokines is responsible for the activation of many other molecules that are responsible for causing or increasing the chances of expressing many different types of ACSs in humans. The research that was conducted pointed to IL-18, IL-6, TNF, IFNG upregulation leading to cascading effects in the human body that lead to increased risk of an individual developing an ACS. *This project is proposed as part of our Senior Biology Seminar capstone course.*

62) GENOME EDITING AND DE-EXTINCTINCTION: THE APPLICATION OF CRISPR-CAS9 IN CONTEMPORARY SPECIES TO RESTORE EXTINCT GENOMES AND THE ECOLOGICAL IMPLICATIONS

Samantha Russo
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The world is experiencing its sixth mass extinction event known as the Holocene extinction. As a result, the biodiversity loss is devastating ecosystems and leaving important ecological niches empty. De-extinction efforts are working to recreate new versions of extinct species from their contemporary counterparts to increase biodiversity and fill ecological niches. At the forefront of these efforts are the revival of woolly mammoths and passenger pigeons from Asian elephants and band-tailed pigeons, respectively. Sequencing of the preserved genomes of extinct species and the living model species is used to identify the molecular differences between the genomes to establish sequences to target. Many of the target genes specify for phenotypic differences that affect the species' ability to survive in their original environment and ecological niche. Of the 1,642 protein-coding genes that differed between mammoths and elephants, most are associated with the mammoth's adaptation to extremely cold climates. One such change is the TRPV3 gene, encoding for a temperature-sensitive transient receptor potential channel that mediates hair growth and thermal sensation. Once targets are identified, CRISPR-Cas9 can be used to edit the target genes in the germline cells of extant species to resemble more closely that of their ancestors. Following gene editing, selective breeding of the model species is used to create offspring with similar genomes to their ancestor. The effort to revive mammoths is still in the stages of CRISPR-Cas9 gene editing, while passenger pigeon efforts have reached the stage of selective breeding. If successful, these de-extinction efforts will create faithful replicas of extinct species to satisfy the ecological niche they once held. However, de-extinction efforts should only focus on restoring species that our environment is equipped to handle, as the reintroduction of extinct species into functional ecosystems may lead to competition between the revived species and current members of the system. *This research project is proposed as a part of our Senior Biology Seminar capstone course.*

63) THE ROLE OF SEROTONIN DYSFUNCTION IN COVID-19 AND PARKINSON'S DISEASE

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Along with various respiratory symptoms, COVID-19 causes the release of cytokines leading to inflammation throughout the body. Parkinson's disease is a neurodegenerative disorder characterized by tremors, stiffness, and impaired balance and coordination. The presence of Lewy bodies, causing degeneration of dopamine in the striatum is a hallmark of PD. I hypothesized that there could be a connection between COVID-19 and Parkinson's disease, and possibly between the exposure to COVID-19 and the risk for Parkinson's disease. Molecules affected by COVID-19 exposure and Parkinson's disease were identified using QIAGEN knowledge base. A core analysis was run on the list of common molecules between the two diseases using QIAGEN Ingenuity Pathway Analysis (IPA). The core analysis of the common known molecules between COVID-19 and Parkinson's disease listed serotonin (5-HT) receptor signaling and serotonin degradation as top canonical pathways. Growing evidence suggests that dopamine is not the only neurotransmitter altered in PD, but that serotonin may play a role as well. The serotonergic system is involved in regulating several cognitive and psychological processes, such as mood, emotion, sleep, and appetite. Alteration to this system can play a role in the motor and nonmotor disturbances seen in PD. Reduced binding of the 5-HT transporter (SERT) was seen in the forebrains of PD patients in a postmortem study. An in vivo study also showed reduced binding in PD patients, with more loss observed as the stages advanced. The role of 5-HT is also being studied in COVID-19, and it has been suggested that serotonin may be able to reduce the inflammatory response. Reduced 5-HT levels in the hypothalamus coincided with hypotension, fever, and increased cytokine levels in severe and mild systemic inflammation. There are also similarities related to gene co-expression, co-regulation, and function between ACE2 and Dopa Decarboxylase (DDC), the enzyme that catalyzes the biosynthesis of dopamine, serotonin, and

histamine. Defective expression of ACE2 induced by COVID-19 might be paralleled by a DDC dysfunction. Infections that lead to cytokine storms may also contribute to reduced serotonin and melatonin activity. Our *in silico* study using IPA show that decreased serotonin levels were observed in PD and COVID-19 patients, indicating that this neurotransmitter is likely involved in both diseases. *This project is proposed as part of our Senior Biology Seminar capstone course.*

64) NETWORK META ANALYSIS OF COVID-19 MODULATION OF ACE2 LEADING TO PARKINSON'S DISEASE

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The fever inducing, inflammatory properties of COVID-19 sparks interest in a connection between the respiratory disease and Parkinson's disease. We are hypothesizing that the nature of inflammation that comes about through COVID-19 entering the body through the ACE2 receptor can potentially set of a chain reaction which can render humans more susceptible to neurological damage leading to Parkinson's Disease. Using the QIAGEN Knowledge Base and its "Canonical Pathway of Parkinson's Signaling", an expansive set of molecules connecting COVID-19 to Parkinson's disease was able to be observed. IPA's "path designer" and specific build tools such as "grow", "connect", and "path explorer" were able to be utilized to narrow down the connections of the molecules to allow some concrete observations to be made. A connection between the ACE2 receptor, which allows for the entry of COVID-19 into the body, bradykinin, and neurotensin was able to be formed. QIAGEN Knowledge Base was able to be utilized to observe that that protein bradykinin promotes inflammation and the neuropeptide, neurotensin has a strong connection to our body's dopamine systems. Parkinson's disease is caused by abnormal protein deposits that clump together in our brains called alpha synuclein, causing damage to the cells that produce dopamine. Using an IPA's "pathway analysis", a strong connection between alpha synuclein and Tubulin polymerization promoting protein family member 3 and D-aminoacyl-tRNA deacylase 2 were found. TPPP3 is a protein that plays a role in tubulin binding and DTD2 is an enzyme the works with protein binding. Damage to these cells from inflammation can lead to gait and movement problems connected to Parkinson's. Ultimately, the inflammation caused by COVID-19 can reach the brain, potentially causing an influx of alpha synuclein to build, damaging the cells that produce dopamine, leading to Parkinson's disease. *This project is proposed as part of our Senior Biology Seminar capstone course.*

65) META-ANALYSIS OF THE MECHANISMS UNDERLYING NICOTINE MODULATION OF COVID-19 PATHOLOGIES

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SARS-CoV-2 is the virus that causes the pathologies of COVID-19 which directly affects the respiratory system. Nicotine is a highly addictive substance with many negative connotations, especially with its connections to cancers including, but not limited, to lung cancer. However, nicotine has been also reported to have therapeutic values, particularly due to its ant-inflammatory properties. These include the positive effects of exposure to nicotine on the outcome of COVID-19. Angiotensin converting enzyme 2 (ACE2) is the principle receptor molecule for SARS-CoV-2. It has been identified as a potential target for COVID-19 treatment. Additionally, smoking tobacco or use of e-cigarette products has been shown to upregulate ACE2 in the lower respiratory tract by receptor mediated endocytosis as well as upregulation of neuroinflammation signaling pathways. Moreover, the protective mechanisms of nicotine in COVID-19 may include an anti-inflammatory effect, reducing the risk

of a cytokine storm in COVID-19**.** Nicotine use inhibits production of pro-inflammatory cytokines such as TNF, IL-1, and IL-6, without inhibiting anti-inflammatory cytokines such as IL-10, thereby possibly protecting against the cytokine-storm syndrome which is responsible for the pathophysiology of severe COVID-19. Tobacco, e-cigarette and smoking have been reported to modulate expression of ACE2 expression in multiple organs. With these premises, we have hypothesized that nicotine may interfere SARS-CoV-2 binding to ACE2 receptor and may provide efficient prevention and/or treatment for COVID-19. To substantiate our hypothesis, we have reviewed and analyzed existing literature in the context of nicotine as a drug/chemical and COVID-19. Molecules affected by nicotine were identified by using IPA's Pathway feature to identify and organize molecules and biological relationships, the "Build," "Grow," and "Connect" tools were used to obtain information about the molecules we were interested in. Molecules affected by COVID-19 were identified from these tools and literature including, but not limited to, ACE2, NF-kB, and a7nAChR. The molecules affected by both COVID-19 and nicotine were uploaded into IPA for Core Analysis to find the connection between the different molecules to analyze whether or not nicotine possesses protective or modulatory mechanisms against COVID-19 pathogenesis. Additionally, smoking tobacco or e-cigarette products (along with other nicotinic agents) has been shown to upregulate ACE2 in the lower respiratory tract by receptor mediated endocytosis as well as upregulation of neuroinflammation signaling pathways. The research completed using QIAGEN's KB and IPA software showed that individuals who are using nicotinic agents are potentially protecting themselves from COVID-19 due to the modulation properties that nicotine has on ACE2. Although there is research that shows the carbon monoxide leads to inflammatory diseases such as alveolitis, the modulative properties that nicotine has on ACE2 may promote adverse effects by decreasing COVID-19 severity as nicotine and COVID-19 compete for the ACE2 receptor. *This project is proposed as part of our Senior Biology Seminar capstone course.*

66) THE IMPLICATIONS OF RHEUMATOID ARTHRITUS AND THE CONNECTS TO OTHER MALIGNACNIES

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Rheumatoid arthritis (RA) is an inflammatory autoimmune disease characterized by chronic pain and swelling typically at the joints. Autoimmune diseases occur when your own immune system attacks healthy tissues and cells in your body causing chronic inflammation among other problems. Despite many research studies, the pathogenesis of RA is not fully understood, so there is no cure only mechanisms to help relieve the pain. Recent studies have shown the primary trigger of RA is due to the defective Treg cells in the synovial fluid which therefore cannot suppress pro-inflammatory cytokine production. The lack of regulation in this process results in the increase of TNF alpha, the primary cytokine responsible for inflammation throughout the body. In the present study we conducted using Qiagen's IPA software, we were able to identify a set of genes related to the susceptibility of RA including CD244, PAD14, SYNGR1, SLC22A4, PTPN22, COG6, IRF5, EOMES, RAD51B, UBASH3A, ETS1, LBH, and CIITA. By optimizing the tools of IPA, we were able to link different malignancies, such as melanoma, prostate cancer, and breast/colorectal cancer to those susceptible genes of RA creating 3 pathways. The results from the IPA pathways support the various published research studies suggesting there is a relationship that shows patients diagnosed with RA have a higher susceptibility towards melanoma and breast cancer. We were able to find additional research suggesting there is also a connection between RA and an increase for prostate cancer. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

67) PATHWAY ANALYSIS OF COVID-19 MODULATION OF ACE2 LEADING TO ALZHEIMER'S DISEASE VIA OVERLAPPING MOLECULES

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Coronavirus 2019 (COVID-19) is a worldwide pandemic that has been a crisis since March 2020. The disease caused by the virus SARS-CoV2 (severe acute respiratory syndrome coronavirus-2. The expression of the angiotensin-converting-enzyme 2 (ACE2) is upregulated in Alzheimer's disease (AD) brain and ACE2 protein expression is found to be higher in Alzheimer's brain. Furthermore, the SARS-CoV-2 entry receptor, also ACE2, is highly expressed in nasal goblet and ciliated cells, maintaining the premise that SARS-CoV-2 is likely entering the human brain by the olfactory nerves. Our hypothesis is that if ACE2 is internalized and there is a viral infection of COVID-19 then it can thereby render a patient more likely to develop AD. For this study, an *in silico* approach was utilized in order to examine the possible relationships and connections between COVID-19, ACE2, and AD. The molecules that were found to be affected by COVID-19 and AD were identified through the QIAGEN Knowledge Base. The analysis suggests that there is a connection between COVID-19, ACE2, and AD. SARS-CoV-2 infects the human by binding to ACE2, and in an Alzheimer's brain, there is a high expression of ACE2. Some of the molecules found to be in correlation with COVID-19, ACE2, and AD are ACE2, APP (amyloid precursor protein), AGT (angiotensin), cytokines such as IL6, C5, TNF, and IL1β. These molecules were also found in the published literature, as well as within the core analysis. All of these molecules (and more – in total 5500 molecules) are overlapping (some more so than others) were analyzed using the QIAGEN Ingenuity Pathway Analysis (IPA). The *in silico* study done for this research utilized IPA to make evident that COVID-19 modulation of ACE2 leading to AD. The findings suggest that the data determines a link in the COVID-19 and ACE2 leading to Alzheimer's disease. *This research project is proposed as part of our Senior Biology Seminar capstone course.*

68) CHRONIC OPIOID USE MODULATES IMMUNE FUNCTION LEADING TO INCREASED RISK OF SEVERE COVID-19

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The coronavirus disease 2019 (COVID-19) pandemic is a worldwide crisis caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). This pandemic has impacted the lives of everyone, especially those with mental health or substance abuse problems. The COVID-19 pandemic has further fueled deep-rooted substance use problems within the United States such as the Opioid Crisis, which began in the late 1990s with the overprescribing of opioid pain-relief drugs that were argued to be "non-addictive". The focus of this study was to demonstrate the relationship between chronic opioid use signaling pathways and the subsequent altered immune system response seen in chronic opioid users. It was predicted that these patients will present an increased risk of developing severe COVID-19. This study utilizes *in silico* approach to examine possible relationships between opioid abuse (with a focus on morphine-containing drugs such as heroin) and more severe COVID-19 prognoses. Molecules affected by morphine use were identified via analysis with QIAGEN Knowledge Base (QKB) using the Core Analysis and BioProfiler tools. Molecules affected by COVID-19 were identified from various published literature on COVID-19 immunology, QIAGEN Coronavirus Network Explorer, and the Core Analysis and BioProfiler tools. These common molecules were then analyzed by QIAGEN Ingenuity Pathway Analysis (IPA).

The analysis suggests that opioid abuse may augment the inflammatory immune response to COVID-19, resulting in an upregulation of proinflammatory cytokines that are linked to severe disease and death in COVID-19 patients. Opioids may enhance the activity of key proinflammatory cytokines such as IL-1β, IL-2, IL-7, and TNF-α leading to an increased inflammatory response to SARS-CoV-2 and subsequent tissue/organ damage due to cytokine toxicity. These molecules were mapped to signaling pathways predicted to associate with SARS-CoV-2 proteins, including the TLR4 and IL-6 pathways. The *in silico* studies using IPA demonstrate that chronic opioid use may augment SARS-CoV-2 induced inflammatory immune response by altering the activity of key inflammatory mediators. Our findings emphasize the importance of focusing attention on the Opioid Crisis in America, which has become exacerbated by the COVID-19 pandemic and is putting millions struggling with addiction at an increased risk of developing severe disease. *This project is proposed as part of our Senior Biology Seminar capstone course.*

69) POTENTIAL THERAPEUTIC TREATMENTS USING BEE VENOM

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Bee venom has been utilized as a natural remedy for centuries in locations such as the Middle East. Recent studies have found that bee venom therapy (BVT) can be used to successfully treat many common disorders because of its active compounds and biologically significant components. There are eighteen pharmacologically active compounds found in bee venom, including peptides like melittin and phospholipase A2 that enable it to be a potentially effective therapeutic treatment. These components have cytotoxic, antitumor, apoptotic, immunomodulatory, anti-inflammatory, and analgesic effects. The most prominent diseases that bee venom therapy has been used to treat include arthritis, cancer, Parkinson's Disease, Immune Thrombocytopenia, statin-induced myopathy, and several other disorders including depression and multiple sclerosis. While BVT is often noted as safe for humans when utilized properly, potential adverse effects and safety concerns should be studied and addressed. These side effects include anaphylaxis and death. People who participate in BVT under the supervision of a healthcare provider are advised to take a test sting before using this treatment and they are encouraged to carry a personal kit that includes a dose of epinephrine and antihistamine medication. The possibility of refining bee venom to rid of any dangerous components could limit these side effects. Other challenges of BVT include non-specific-cytotoxicity, degradation, and hemolytic activity, especially regarding the treatment of cancer cells. More research needs to be conducted in order to improve BVT and determine its full scope of prospective therapeutic uses. *This project is proposed as part of our Senior Biology Seminar capstone course.*

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