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2023-2024 SSGSA STEM Sustainability Case Competition: Global Health

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Abstract:
The SSGSA STEM Sustainability Case Competition is an annual research case competition hosted by undergraduate students from the STEM Students Guelph Support Association (SSGSA). The mission of this competition is to provide University of Guelph undergraduate students with an opportunity to develop their own research proposal while gaining valuable experience in innovative thinking and critical research analysis. Each year students, in teams of up to three are paired with an experienced mentor to develop and present a novel research proposal aligning with the competition's theme. During the competition, students are taught fundamental principles outlining three lab techniques which they could write about in their proposal. The theme of this year's competition was Global Health, and competitors learned about Enzyme Linked Immunosorbent assay (ELISA), Flow Cytometry, and Radio Carbon Dating. In the 2023-2024 SSGSA STEM Sustainability Case Competition over 175 participants submitted abstracts for judgment, and we present the Top 19 winning submissions to be read by you in our competition abstract booklet. We hope you enjoy reading this year's best abstract submissions and encourage you to participate in the growing SSGSA community as we strive to encourage interest in novel scientific research fields surrounding STEM.

Keywords: STEM sustainability case competition; SSGSA; abstract submissions; global health; ELISA; flow cytometry; radio carbon dating; undergraduate; STEM; University of Guelph

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Conference Abstracts

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SSGSA STEM Sustainability Case Competition Abstracts

Regeneration of Neuron Synapses in Mice Induced With β-Amyloid Toxicity: A Novel Approach to Alzheimer's Treatment

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Alzheimer's disease is the leading cause of dementia affecting more than 55 million people worldwide. Alzheimer's is characterized by the accumulation of hyperphosphorylated, insoluble tau protein, as well as insoluble β-amyloid peptide aggregates which result in the impaired functioning/structure of synapses. The progression of Alzheimer's can lead to the complete dysfunction of neurons. Current treatments to remove this plaque buildup include aducanumab (Aduhelm™), which is an immunotherapy designed to remove β-amyloid. Spinal injury research found that the connection between axons can be regenerated through the chemoattraction of axons to their natural target through the injection of netrin-1. We propose a similar approach to testing this method through the transgenic mouse model in which two groups are induced with β-amyloid toxicity resulting in Alzheimer's pathologies. We will give one group netrin-1 and aducanumab and the other just aducanumab. We would test the synaptosomes of the mice through flow cytometry to determine if the axons of the neurons reconnected. The desired outcome would result in a larger number of viable synaptosomal particles being found in the mice that were given both the chemoattractor and aducanumab compared to the control group. If successful, our approach could be combined with the current treatment of Alzheimer's to allow for the reversal of dead synapses in addition to preventing the buildup of plaque. This will act to reverse symptoms of the disease in addition to slowing the progression.

Bioremediation of Plastic Pollution in Estuaries: Genetic Engineering of *Ideonella sakaiensis* for Improved Polyethylene Terephthalate Degradation

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On a global scale, millions of metric tons of plastics are annually manufactured from petroleum hydrocarbons. Polyethylene terephthalate (PET) makes up most of these plastics and increases the natural degradation timeline by polluting essential aquatic ecosystems like estuaries. The bacteria *Ideonella sakaiensis* can degrade PET using two of its enzymes to hydrolyze the plastic into usable energy and nutrients. While PET can take 16-48 years to biodegrade in soil, the enzymatic action of *I. sakaiensis* can reduce the biodegradation process to just 6 months, serving as a solution to the long-lasting nature of plastics in the natural environment. However, one limitation of bioremediation using *I. sakaiensis* is the restrictive environments that can support the growth of the bacteria. By genetically engineering *I. saikaiensis* with *Shewanella oneidensis*, a bacterium with the ability to thrive in hypoxic environments; we can expand the habitat niche of *I. sakaiensis* to aquatic environments. We aim to

test this by using water samples collected from the St. Lawrence estuary. Trials will be conducted on the separate water samples; one sample will be used as the control, while the other samples will be manipulated using different concentrations of the genetically modified *I. sakaiensis*. PET concentrations of each sample will be measured weekly over a six-month period using NMR spectroscopy, a cost-effective and precise method for quantifying and qualifying PET granules. We expect to find that PET concentrations will be significantly lower in samples treated with modified *I. sakaiensis* compared to the control sample. The results of this study provide a novel approach to the bioremediation of plastic pollution in aquatic environments. If successful, subsequent studies may delve into the potential application of *I. sakaiensis* in plastic-polluted aquatic environments, offering a sustainable approach to pollution reduction without inducing further environmental harm.

Lactate Inhibition to Mitigate the Progression of Neuron Degeneration in Parkinson's Disease

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Parkinson's disease is the second most common neurodegenerative disease in the United States and is estimated to affect 6 million people worldwide. Parkison's disease may be characterized by a deficiency of dopaminergic neurons exhibited in the substantia nigra of the brain. The accumulation of lactate, a by-product of glycolysis under anaerobic conditions, has been shown to increase the apoptosis of dopaminergic neurons. However, the result of lactate inhibition on the progression of Parkinson's disease has yet to be studied. 3-dihydroxy-6-methyl-7-(phenylmethyl)-4-propylnaphthalene-1-carboxylic acid (FX11) is a known selective inhibitor of lactate dehydrogenase A, an enzyme that aids in the production of lactate. This study looks to investigate FX11's effectiveness in reducing the progression of Parkinson's disease. Leveraging a murine model, C57BL6 mice will be treated with 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), a neurotoxin that elucidates Parkinson-like symptoms, by intraperitoneal injection for 5 days. Following MPTP treatment the mice will either be left untreated, establishing a control group, or be treated with FX11 (2 mg/kg) by oral gavage for a further 4 weeks. A double-antibody sandwich enzyme-linked immunosorbent assay (ELISA) will be performed on autopsied matter from the substantia nigra of murine subjects. We anticipate that treatment with FX11 will reduce the rate of neural degradation in mice with Parkinson's-like symptoms, by inhibiting the production of lactate. Inhibiting lactate production will elicit a decrease in neural apoptosis thus mitigating neuron degradation and the progression of Parkinson's disease. Further research should be performed to investigate the feasibility of lactate inhibition as a novel therapeutic approach to Parkinson's disease.

Comparative Autoantibody Response: Developing a Mouse Model for Predicting SARS-Cov-2-Linked Type 1 Diabetes

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Officially recognized as a pandemic in March 2020, SARS-CoV-2 presents an ongoing concern for global health due to its proposed role in the development of autoimmune diseases. SARS-CoV-2 is hypothesized to be linked to the onset of type 1 diabetes as a result of cytokine storms, which ultimately dysregulate autoantibody production. Existing mouse models, including those modified with humanized ACE2 receptors for viral entry, do not accurately mimic the cytokine cascade or support in vivo viral replication; thus, their utility in studying SARS-CoV-2-induced type 1 diabetes is limited. Humanized CD147 (hCD147) SARS-CoV-2 receptors in mice have demonstrated better mimicry of the human cytokine storm, but its use has not been verified in studies of COVID-linked type 1 diabetes. This study aims to validate the hCD147 mouse model using current human SARS-CoV-2 infection data. Using a hCD147 mouse model (n=30), half the mice will be intranasally infected with Omicron SARS-CoV-2, while the other half will act as a negative control. The primary self-antigen related to type 1 diabetes in both humans and mice is Glutamic Acid Decarboxylase (GAD65). Indirect ELISA will be performed against the GAD65 antigen using plasma collected 4 weeks post infection from infected and non-infected SARS-CoV-2 hCD147 mice as well as a sample of humans diagnosed with type 1 diabetes post-SARS-CoV-2 infection. Secondary enzyme-labelled antibodies will be introduced to selectively bind anti-GAD65 to quantify the autoimmune response. It is expected that the hCD147 mouse model will mount a similar SARS-CoV-2 cytokine response to humans. Similar levels of anti-GAD65 autoantibodies are anticipated in comparative ELISA titres from hCD147 mice and post-SARS-CoV-2 diabetic humans. With the potential to incorporate subsequent glucose monitoring steps in the future, the hCD147 mouse model may allow for preventative research on SARS-CoV-2-linked type 1 diabetes with broader applications in other COVID-linked autoimmune diseases.

Enzyme-Linked Immunosorbent Assay and Reverse Transcription Polymerase Chain Reaction Investigated as Detection Methods for Avian Influenza

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Avian influenza (AI) is a zoonotic disease transmitted through birds and caused by Influenza Virus A, a negative-strand RNA virus with subtypes H5 and H7 presenting as highly pathogenic to birds. Strain H5N1 poses a risk to humans with a mortality rate of over 50%. Bird symptoms include head swelling and respiratory issues. It can be transmitted to humans through air or food, causing severe complications like organ failure. AI remains a major contributor to epidemics worldwide, also causing significant annual financial losses in the poultry sector. The high variability of the virus makes it difficult to create a vaccine with a long-lasting effect; hence, diagnosis is crucial. The diagnostic standard for AI is virus isolation (VI) in avian egg or cell cultures. Once cultured, virus identification occurs using techniques such as immunoassays or antigen detection via immunofluorescence. VI is time-consuming and expensive, taking 1 to 2 weeks to receive detectable results. RT-PCR removes the need for the VI step and offers cost efficiency, scalability, and a quicker diagnosis. This method quickly detects AI genetic material, targeting specific segments such as the matrix gene and providing results in just 3-5 days. Moreover, enzyme-linked immunosorbent assay (ELISA) is a useful diagnostic tool for identifying the presence of antigens and antibodies. It demonstrates positive results in detecting H5 and H7 AI subtypes with high specificity and sensitivity. Additionally, it produces quicker results than those obtained from the haemagglutination inhibition test, typically used for serological AI diagnostics. Results are highly reproducible with Pearson correlation coefficients between 0.96-0.98. Through these considerations, RT-PCR and ELISA techniques can be developed together as a diagnostic method of AI in poultry to allow for a quicker, larger scale, more specific, and inexpensive detection to prevent outbreaks, human and avian health issues, and financial losses globally.

Utilization of Phenotype-Driven Mouse Models with ELISA to Study Neurological Dysfunction from Mitochondrial Encephalomyopathy, Lactic Acidosis, and Stroke-Like Episodes (MELAS)

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MELAS is a rare mitochondrial inherited genetic disorder where a mutation in the MT-TL1 gene causes a mistranslation of the amino acid leucine (due to mutated $tRNA^{Leu(UUR)}$), inhibiting oxidative phosphorylation. MELAS, although rare, is the most common mitochondrial disease, with an estimated incidence of 1 in 4000. Symptoms include cognitive decline, epilepsy, myopathy and diabetes, with no cure, and diagnosis ranges from ages 2 to 40. Due to the inability to transfect mammalian mitochondrial DNA (mtDNA), there are few mouse models to study MELAS. However, the discovery of a phenotype-driven approach to mouse models presents a streamlined way to develop these models and study the effect of mutated mtDNA. We will replicate the methods to breed female mice carrying a mutation in the polymerase gamma (PolyA) gene to allow for a different mutation in the gene sequence, specifically $tRNA^{Leu(UUR)}$. Here we propose using ELISA to identify neurofilament light (NfL) in these mouse models to compare it to human studies. NfL has recently been discovered as a novel biomarker for neurological dysfunction in MELAS patients, which can serve as future study for the treatment of neurological dysfunction. With these mice, we can use mazes in a controlled environment to further understand the mechanisms behind the neurological dysfunction of MELAS and watch for other behavioural and physiologic changes in the mice. In summary, we will employ specific methodologies to validate the mouse model and its expression of NfL, allowing for examination of the neurological dysfunction characteristics of MELAS, which will be of great importance for studies of disease pathophysiology, and preclinical treatment trials.

Assessment of the Immune Response to Phage Treatment of Tuberculosis in Humanized Mice via Flow Cytometry

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Tuberculosis (TB), caused by the intracellular bacterial pathogen *Mycobacterium tuberculosis*, is the second leading infectious cause of mortality worldwide, causing an estimated 1.3 million deaths in 2022. In addition to inaccessibility of treatment, the evolution of multidrug-resistant TB is a major contributor to the high prevalence and mortality of the disease. Mounting evidence supports the use of bacteriophages in treating other *M. tuberculosis* infections, and recent in vitro studies have supported their applicability to TB. In the first in vivo study of this technique, Yang *et al.* show that phage DS6A both lowers *M. tuberculosis* bacterial load in NSG-SGM3 humanized mice and lyses *M. tuberculosis* strain H37Rv inside human

macrophages. Following this early evidence of the efficacy of phage treatment, we aim to address the question of safety by assessing the immune response to the phages in the same NSG-SGM3 mouse line. We will reproduce the Yang *et al.* treatment group structure–negative control, *M. tuberculosis*-infected, *M. tuberculosis*-infected and treated with DS6A–but add a fourth group receiving the phage treatment but not infected with *M. tuberculosis*. This facilitates assessment of the immune response to the phages both separately and alongside concurrent *M. tuberculosis* infection of the host. After confirming phage retention in the mice using ELISA specific to phage-induced antibodies, we will perform flow cytometric analysis of immune cells at regular intervals over the course of the treatment. By quantifying the presence of both myeloid and lymphoid immune cells we would be able to observe the magnitude and profile of immune activation and hence understand the potential for collateral damage phage treatment, and the resultant immune response may pose. Progress such as this towards phage therapy against TB is of great significance because of the potential phages hold for treatment of multidrug-resistant TB with significantly less risk itself for development of resistance.

Comparative Analysis of ELISA and Flow Cytometry Versus MRI and CT Scans in the Early Detection of Lung Cancer

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Lung cancer is the primary cause of mortality among all cancer-related deaths. The current lung cancer diagnosis includes screening high-risk individuals based on their history of tobacco use. Diagnostic techniques include the use of MRI and CT scans for individuals already showing symptoms of lung cancer. The development of lung cancer initiated through carcinogen inhalation leads to DNA adduct formation and subsequent mutations if the cells are not repaired or do not undergo apoptosis. These mutations, specifically in oncogenes and tumor suppressor genes, further develop into lung cancer. Moreover, genetic susceptibility contributes significantly, with smoking further enhancing lung cancer risk. Flow cytometry, high in sensitivity and specificity, combined with machine learning, accurately classifies small nodules of cancer samples at early stages. ELISA detects specific biomarkers crucial in lung cancer progression including the overproduction of epidermal growth factor receptors, *KRAS* mutations, and PD-L1 expression. We propose to recruit high-risk individuals, from a diverse participant pool, to undergo MRI and CT scans, while simultaneously collecting blood and sputum samples for ELISA and flow cytometry. Participants will be grouped based on their test results recorded. Testing will be repeated in follow-up appointments over 2-5 years and changes to lung health status will be monitored. Integrating flow cytometry and ELISA in lung cancer diagnosis is anticipated to result in the efficient identification of early-stage lung cancer and will enable early initiation of treatment crucial for improving patient outcomes. The use of these technologies is expected to lead to increased survival rates and less invasive treatment options. This study would provide comparative data on the effectiveness of these diagnostic methods in the early detection of lung cancer, potentially influencing future screening guidelines and practices. Limitations to this study include long-term participant commitment and the potential for false positive/negative test results.

Curcumin and Genistein as Dietary Inclusions to Inhibit Estrogen-Dependent Cancer Proliferation in Breast Cancer Mice Model Induced by Atrazine, an Estrogenic Pesticide

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Estrogen-dependent cancers (EDCs), such as breast, ovarian, and uterine cancer are some of the most common cancers affecting women globally. Exposure to pesticides mimicking estrogen, such as atrazine, causes women working in agriculture to be more susceptible to malignant cancer growth. Micromolar concentrations of curcumin and genistein, natural compounds within turmeric and soybeans respectively, have shown to inhibit estrogen activity, and consequently the growth of breast cancer cells in vitro. Both curcumin and genistein mimic 3β-Adiol, an agonist which competes with estrogen to bind to estrogen receptor beta (ER-β) in EDC cells. These compounds block extracellular estrogen migration, preventing its metabolism by cancer cells in blood. Increased ER-β binding activity also counteracts ER-α expression, a key promotor of hyperproliferation. Combined inhibition of estrogen-signaling pathways and a greater ER-β/ER-α ratio slow metastasis in most EDC pathogenesis. This study will investigate curcumin and genistein's individual and combined effect on ER-β/ER-α levels in mice with breast cancer induced by atrazine. Mature female mice are injected with 200mg/kg of atrazine everyday for two weeks. The control group continues with a regular diet, whilst treatment groups switch to a curcumin, genistein, or combined diet. At the end of two weeks, enzyme linked immunosorbent assay (ELISA) will be used to measure autoantibody concentrations in blood, specifically ER-β/ER-α expression. Tumour volume and characteristics will be visualized using

microCT and compared. Mice individually treated with curcumin or genistein are expected to have a higher ER-β/ER-α ratio than the control group, while treatment with both compounds is expected to have the largest ratio. Since there is limited research exploring dietary changes on EDC progression in vivo, evidence of antiproliferation in breast cancer mice-models is a basis for human clinical trials to study their efficacy on other EDCs. Further research paves the way for accessible and novel lifestyle preventions for EDCs in agriculturally-employed women internationally.

ELISA-Based Analysis of Probiotic Influence on Cortisol Regulation in the Gut-Brain Axis and its Implication for Mental Health Improvement

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The gut-brain axis (GBA) is a bidirectional communication system that is critical in modulating physiological and psychological well-being. Anxiety and acute stress, two of the most prevalent mental illnesses in today's society, have been linked to gut inflammation, emphasizing the GBA's pivotal role. Cortisol, a key stress hormone, regulates physiological processes and mediates mental health. The introduction of live microorganisms or probiotics, often administered through supplements, may regulate cortisol's association, creating a potential therapeutic avenue by fostering a balanced microbial environment in the gut. Nurturing a healthy gut via consistent probiotic supplementation has the potential to alleviate cortisol's impact, consequently improving microbial balance, and promoting better mental health through the GBA. In a 12-week experiment, 40 male participants aged 20-25 with elevated cortisol levels will be randomly split into 2 equal groups. The experimental group will receive probiotic supplements, while the control group will receive a placebo. Cortisol levels will be measured at the beginning and the end of the experiment using a Cortisol ELISA kit with blood samples being used as the medium for analysis. The methodology of the test consists of a reaction between the added cortisol antibody and the cortisol antigen present in the blood. Following incubation, the addition of tetramethylbenzidine (TMB) substrate displays a signal, detected on a 450 nm plate. Once detected, cortisol levels are determined by assessing intensity in relation to a standard curve. After a 12-week period, cortisol levels are expected to decrease significantly among participants in the experimental group, consequently reducing gut inflammation and offering potential therapeutic benefits for mental disorders. The findings of this study provide a potential avenue for holistic treatments in mental health through modulation of the GBA. Maintaining regular intake of probiotic supplements may serve to regulate cortisol levels thus, diminishing its contribution to chronic stress and anxiety.

A Novel Zinc (II) Complex for the Treatment of Malaria-Related Bacterial Infections

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Malaria continues to be among the deadliest diseases in the world, affecting nearly 500 million people every year. Of those affected, children represent those with the highest mortality rate. Malaria in children will often co-present with the invasive bacteria *Staphylococcus aureus* (SA) and *Escherichia coli* (EC), both of which can have devastating effects on a developing immune system. While a great deal of attention has been paid to treating malaria, an effective treatment targeting both SA and EC has yet to see significant progress. The improved efficacy of antibiotics when used cooperatively has been well-documented, but slow uptake among practitioners makes the need for a demonstratable use apparent. This study will examine a bidentate zinc (II) complex of Gemifloxacin and Azithromycin for the simultaneous treatment of SA and EC post-malaria infection. Synthesis will be performed in liquid ammonia; bidentate coordination through the pyridine and carboxylate oxygens of Gemifloxacin, followed by coordination with the desoamine of Azithromycin will yield the desired complex. Successful synthesis will be evaluated via characterization with both ^{1}H and ^{13}C NMR in $CDCl_3$. Individual strains of SA, EC, and a combination (CT) will be treated with injections of the free ligands and zinc complex individually. The major histocompatibility complex class I chain related molecules of the ligands and complex will be determined using serial dilution in a 96-well plate and measurement of the absorbance on a UV-vis spectrophotometer. It is expected that the zinc complex will demonstrate markedly greater potency against both the individual and combination strains than the free ligands alone. The results of this study will show the clear benefits of a single treatment for two malaria-related bacterial infections and should serve as the basis for further in vivo studies. Adoption of a single, multi-target antibacterial can reduce the need for complex treatment plans and improve treatment uptake among at-risk populations.

Testing Drugs for Inhibition of MMP14 Phosphorylation Involved in Tumor Metastasis Using Cell-Based ELISA: A Faster Approach to Drug Discovery

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Tumor metastasis is the leading cause of cancer deaths. Metastasis involves tumor cell migration to other parts of the body through the formation of invadopodia, which are protrusions of the plasma membrane. One of the proteins localized at invadopodia, the membrane type 1 metalloproteinase (MT1-MMP; MMP14) is involved in cell migration. Phosphorylation of MMP14 is known to increase recruitment and recycling of MMP14 to invadopodia, resulting in enhanced cell motility. Inhibition of MMP14 phosphorylation offers a strategy to block metastatic spread of tumor cells allowing time for clinical interventions to treat the primary tumor. However, the detection of MMP14 phosphorylation has been challenging due to the low abundance and the challenges of phosphatase degrading phosphoproteins in lysed cells. Therefore, we propose to use a cell-based ELISA as a more sensitive assay to quantify intact MMP14 phosphorylation to quantify the efficacy of a mutant non-phosphorizable MMP14 in reducing phosphorylation of MMP14. The cell-based ELISA allows simultaneous detection of phosphoproteins and total protein levels simultaneously without cell lysis, allowing phospho-protein levels to be accurately assessed. We hypothesize that the MDA-MB-231 breast cancer cells treated with mutated (Y573F) MMP14 will have a detectable reduction in phosphorylation as the non-phosphorizable MMP14 is internalized and recycled at the site of invadopodium in a competitive inhibitory manner. Further validation to assess the outcome of MMP14 mutant treatment will be performed by using a gel degradation assay to measure the surface area of fluorescent gelatin degraded by the tumor cell. Cell-based ELISA is predicted to be a faster and more accurate alternative to traditional western blots when testing the efficacy of potential kinase inhibitor drugs used to block tumor metastasis as well as expedite research in addressing cancer as a global health concern.

Precision Therapy in Pancreatic Ductal Adenocarcinoma: A Ph-Responsive Nanotreatment

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Pancreatic ductal adenocarcinoma is the fourth leading cause of cancer-related deaths worldwide. It is often diagnosed in its late stages, resulting in ineffective treatments. Surgery is typically reserved for patients diagnosed in the early stages of infection, while chemotherapy, such as gemcitabine, is often used for late-stage cancer patients who have a favorable prognosis. However, chemotherapy lacks precision and does not effectively penetrate the stroma - a key barrier in cancer treatment. Precision medicine may rectify the drawbacks of conventional cancer therapeutics by tailoring treatment to the individual disease. This study aims to investigate the effectiveness of a pH responsive polymer against gemcitabine as a treatment for late-stage pancreatic cancer. A pH-responsive polymer that activates in the acidic tumor microenvironment will be constructed for improved gemcitabine delivery as previously described. A control group [CON] of late-stage pancreatic patients (n=63) receiving 1,000 mg/m^2 weekly of gemcitabine for 7 weeks, with one week of no treatment followed by three treatments weekly for 28 days, will be compared to a group of late-stage pancreatic patients (n=63) treated with the novel pH-responsive drug delivery system [TRT]. Urine (630 µL) will be collected prior to treatment (t = 0), and at week 7, 9, 11 and 13. Samples will be analyzed using nuclear magnetic resonance spectroscopy for metabolites of amino acid metabolism (ie. threonine, tryptophan and aminobutyrate), sugars (ie. glucose and fructose) and the urea cycle, previously linked to pancreatic cancer. An MRI will be taken at weeks 0, 6 and 12 to measure tumor size in response to treatment. It is expected that the markers of pancreatic cancer will exhibit a greater decrease to the initial values in TRT compared to CON, and tumors will decrease more for TRT than CON. This study serves as the foundation for nanomaterial to make a paradigm shift in cancer treatment and biotherapeutics.

Treatment of Endometriosis Using Lithospermum ruderale to Decrease Estrogen and Inflammatory Cytokines, Measured Through ELISA

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Endometriosis is a chronic condition associated with implantation and growth of endometrium-like tissue outside of the uterus with increased estrogen levels. Excess tissue causes symptoms including severe pelvic pain which can interfere with daily activities. Inflammatory cytokines are produced from endometriotic tissue which increases inflammation and neuropathic pain. These cytokines include interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF- α),

which are elevated in peripheral blood. Common treatments include surgical excision of lesions and hormonal-based medications. Oral contraceptive pills (OCPs) are often the first treatment, including estrogen and progestin combined or progestin-only medications. Secondary treatment involves GnRH to suppress estrogen levels. OCPs may not relieve all pain and can have undesirable side effects. *Lithospermum ruderale* was historically used as a means of oral contraception. A previous study demonstrated the effectiveness of *Lithospermum ruderale* in reducing estrogen levels in mice through action on the pituitary gland. This study aims to investigate the effectiveness of *Lithospermum ruderale* as an alternative treatment for endometriosis symptoms. Mice with induced endometriosis will be divided into three groups: one group following a diet of 15% sucrose placebo (C; n: 20), another group following a diet of 15% *Lithospermum ruderale* powder (E1; n: 20), and a third group receiving 1mg/kg of Dienogest powder incorporated in the diet (E2; n: 20). Treatments will be administered over a four-week period, with blood draws every four days. Estrogen is assayed using the Mouse Estrogen ELISA diet. Inflammatory cytokine levels of IL-1β, IL-6, and TNF-α will be assayed using the Mouse Cytokine Th1 Panel. We anticipate that *Lithospermum ruderale* will reduce measured estrogen and inflammatory cytokine levels. This provides a basis for further studies to investigate the effectiveness of *Lithospermum ruderale* as an alternative treatment for endometriosis.

Eliminating Grade IV Glioblastoma Multiforme (GBM) Using FAK Inhibitors and Immunotherapeutic Engineered RNA-Encoded Viral Structure

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The prognosis for Grade IV glioblastoma multiforme (GBM) includes a one-year survival rate, with the tumor comprising two-thirds of all astrocytomas and predominantly infiltrating the frontal and temporal lobes of the brain. Factors of GBM include hypercellularity, nuclear atypia, endovascular hyperplasia, and specific to GBM, necrosis of neurological tissue cells. The neurological evaluation of the patient involves identifying a neoplastic intrinsic lesion, glioblastoma. Screening involves systematic physical evaluation, mental status tests, cranial nerve tests, motor examination, sensory examination, reflex examination, and coordination and gait testing. After identifying elements for the aforementioned assessments, problems with levels of consciousness, orientation, afferent pupillary function and motor impairment, the patient must undergo a contrast-enhanced magnetic resonance imaging (MRI), or brain computed tomography (CT) brain scan. The technological assessments will detect in detail the parenchymal implication of the tumour using the edema-encompassing masses shown as a result of the imaging. The imaging utilises advanced sequences involving diffusion and perfusion to account for identifying the type of lesion. The first line of treatment is typically a surgical attempt at biopsy and tumour excision, accompanied with radiation therapy, however inoperable tumours are common and complete resolution is rare. This study will analyse the impact of immunotherapy-based virally encapsulated treatment coupled with Focal Adhesion Kinase (FAK) Inhibitors. It involves the engineering of an RNA-encoded viral structure that targets a ligand on a GBM4, with the lab-grown adaptation to an individual's genes via immunotherapeutic implication from a biopsy. The cells will be administered to the feeding vessels of the tumour, following the initial treatment of FAK inhibitors, used to suppress any further tumour growth. Expected findings include the cessation of tumour growth, in addition to non-invasive, yet most efficient method of eradicating inoperable tumours and preventing the commonality of recurrence. It will act as a springboard for future, immunotherapeutic virally targeted hybrids as a more preferable, patient-friendly treatment.

Nothing Can Last Forever: Understanding the Enzyme Dynamics of Breaking F-C Bonds to Improve the Bioremediation of PFAS

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Perfluoroalkyl and polyfluoroalkyl substances (PFAS) are synthetic compounds used for their stability and hydrophobic properties due to their F-C bonds. The stability of F-C bonds contributes to their persistence as pollutants and bioaccumulants. More than 98% of United States citizens have these chemicals in their bloodstreams. Nicknamed “forever chemicals,” some of the global long-term health effects of PFAS are cancer development, reproductive harm, thyroid disease, and hormonal disturbances. The recent discovery of bioaccumulation in fetuses and breast milk is of critical concern due to PFAS's adverse effects on development. Bioremediation is a novel strategy proposed to remove PFAS from the environment. This technique uses microorganisms to metabolize compounds organically. Enzymes can carry out the cleavage of the F-C bond through a defluorination process. This study aims to determine the conformational dynamics of PFA bioremediation enzymes with nuclear magnetic resonance (NMR). This study focuses on the enzymatic dynamics of benzoyl-coenzyme A

reductase (BCR) derived from *Thauera aromatica* bacteria. NMR will be used to determine changes in the enzymatic structure of BCR following each step of the previously characterized anaerobic degradation pathway of the PFAS 2-fluorobenzoate. BCR will be characterized based on dynamic processes in the 0.3 to 5000 milisecond time range using three NMR methods: EXchange SpectroscopY (EXSY) to analyze the ligand binding and release, Lineshape analysis to analyze affinity and conformational changes in the bound state, and Carr-Purcell Meiboom-Gill Relaxation Dispersion (CPMG RD) to analyze secondary structure changes. These methods combined will provide a complete catalytic cycle for the degradation of 2-fluorobenzoate by BCR. Clarifying the mechanism of the F-C bond defluorination allows for the design of novel enzymes to breakdown PFAS. Bioremediation can be improved by designing novel enzymes by increasing the clearance rate and the range of chemicals that can be cleared.

Application of Flow Cytometry and Fluorescent in Situ Hybridization (FISH) to Investigate the Effects of Antidepressants on Gut Microbiome Diversity

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Many students pursuing higher education in North America experience symptoms of mental illness. The majority of medication for mental illnesses target the brain; however, the gut microbiome is shown to play a crucial role in mental health status. Antidepressants exhibit antimicrobial properties, decreasing bacterial richness in the gut. Flow cytometry, combined with fluorescent in situ hybridization (FISH), allows for visualization of antidepressant-induced changes on gut microbiome composition. A longitudinal study will be conducted over a year observing university students (n=40) experiencing mental health problems. Participants will be screened for autoimmune/immune disorders and given a dietary outline. Stool samples will be collected each month to determine correlation between relative abundances of gut bacteria and antidepressants. Twenty individuals selected for the control group will not be taking antidepressants. The comparison group will have started on selective serotonin reuptake inhibitors (SSRIs) within 1-6 months of the study. Stool samples will be stored at -70°C, transferred over to a dry ice sample, then labeled using FISH. FISH employs an anti-16S rRNA oligonucleotide probe that tags bacteria, such as *Streptococcus*, *Enterococcus* and *Escherichia coli*, which are responsible for serotonin, dopamine and norepinephrine production in the gut. The analyte is subsequently passed through a buffer in the flow cytometer, analyzing these bacteria based on the sample's fluorescence level. SSRI individuals will have a predicted greater decrease in fluorescence within each flow cytometry cycle, in comparison to those without medication. Over time, it is expected that SSRI individuals will have a significant decline in bacteria diversity, and a decrease in species abundances, particularly in the aforementioned bacteria. Considering gut bacteria are responsible for 95 percent of serotonin production, and antidepressants reduce gut microbiome diversity and the essential hormone-producing microbes, exploring alternative treatments that support gut health may provide a better long-term solution to treating mental illnesses.

Utilizing ELISA for the Detection and Quantification of Microplastics in the Circulatory System

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Microplastics, small plastic particles with diameters less than 5 mm, have been associated with numerous health effects, including endocrine disruption, inflammation, oxidative stress, and genotoxicity. The proposed enzyme-linked immunosorbent assay (ELISA) development aims to utilize its sensitivity and specificity to quantify the presence of microplastics within the circulatory system. This approach could significantly enhance our ability to assess the extent of human exposure to microplastics and their links to various health conditions. The assay's design would involve the creation of a sandwich ELISA, leveraging antibody-antigen interactions specific to plastic additives such as phthalates or bisphenol A (BPA), which are pervasive in human tissues. In vertebrates, including mice, exposure to microplastics has altered the expression of genes related to mucin (mucosal barrier), inflammatory markers like tumour necrosis factor-alpha (TNF-α), interferon-gamma (IFN-γ), and interleukin-6 (IL-6), among other immune responses. Another potential antibody is the enzyme glutathione S-transferase, which is elevated in organisms exposed to excessive microplastics. The ELISA would be configured to identify and quantify microplastic-associated antigens, with the feasibility of employing a dual-target bridging ELISA format to simultaneously detect multiple microplastic markers, akin to methods used for bispecific antibodies. The assay would include antibodies against the microplastic polymer itself or its additives, which could be detected in the blood due to their role in oxidative stress and inflammatory responses. To create a specific ELISA method, antibodies would be synthesized against these markers. These would capture the antigens from blood samples, and a secondary antibody linked to an enzyme would bind to the complex. Upon adding a substrate, the enzyme would catalyze a colour change, providing a

measurable signal indicative of the antigen concentration. Expected findings include a reliable correlation between antigen concentration and microplastic exposure, offering a new avenue for non-invasive monitoring and epidemiological research into the health effects of microplastics.

Exploring Chimeric Nanoparticles to Enhance AAV Efficacy in Huntington's Disease Gene Therapy

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Huntington's Disease arises from an autosomal dominant neurodegenerative condition triggered by the expansion of CAG repeats. This expansion causes an increased polyglutamine sequence within the mutated protein, consequently leading to degeneration in the striatum and cerebral cortex of the brain. One promising avenue under study involves utilizing the adeno-associated viral (AAV) vector for gene therapy. However, challenges arise with AAV usage, notably concerning immunogenicity and the decreased production of the wild-type Huntingtin (wtHTT) protein. To address these problems, we suggest using core-shell viral/non-viral chimeric nanoparticles (ChNPs) surrounded by a polyketide (PK) shell, for the co-delivery of the wtHTT gene and siRNA. The purpose of this method would be to silence transcription of the mutant Huntingtin (mHTT) protein while introducing the wild-type for transcription. The PK shell encompassing the AAV serves the purpose of minimizing the potential for an immune response. This protective coating becomes essential as multiple administrations are necessary for comprehensive silencing and transcription. This study aims to analyze mHTT and wtHTT levels in vitro pre- and post-introduction of ChNPs using ELISA. We hypothesize that employing ChNPs is expected to elevate the quantity of wtHTT protein while concurrently reducing the expression of mHTT. This anticipated effect should manifest as a noticeable disparity in concentration within the ELISA results. Our primary goal is to enhance the efficiency of AAVs in gene therapy by tackling their significant limitations. Huntington's disease serves as an ideal model to investigate the potential of ChNPs due to the coexistence of both wild-type and mutant proteins within cells. Genetic modifications influencing the concentration shift will play a pivotal role in determining the experiment's efficacy.

Conflicts of Interest

The authors declare that they have no conflicts of interest. These authors include: Michael Hamilton, Grace Basso, Meryam Tawfik, Sanya Sareen, Amelia Rilling, Massimo Maiuri, and Sukhjot Pooni.

Authors' Contributions

MH: Founder of the SSGSA, Co-Head Author of Authorship Committee, drafted the SSGSA Global Health competition case package, peer-reviewed the abstract submissions and ensured that they adhered to correct formatting standards, and gave final approval of the version to be published.
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MM: Peer-reviewed the abstract submissions and ensured that they adhered to correct formatting standards

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