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SciNapse 2024-2025 Undergraduate Science Case Competition: The Gut Microbiome

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Abstract
The SciNapse Undergraduate Science Case Competition (USCC) offers undergraduates the chance to craft an innovative research proposal. In this competition, a case study is provided, and students conduct comprehensive literature reviews—including scholarly publications, reports, and studies—to identify and connect crucial elements, which then form the basis of a supporting hypothesis. They also design a methodology to assess the validity of their hypothesis. This year's case focused on the intricate and often overlooked realm of the gut microbiome, exploring its significant effects on human health, disease, and wider ecological systems. In teams of 1-4, undergraduate students engaged with the challenge by crafting innovative research proposals aimed to catalyze breakthroughs and deepen our understanding of the intricate gut microbiome. In total, the 2024-2025 USCC attracted 626 undergraduate students from 14 universities across North America. The top 10% of written submissions in each division are highlighted in this abstract booklet. You may find more information on the annual SciNapse USCC on our website at https://scinapsescience.com.

Keywords: SciNapse USCC; undergraduate research; science case competition; gut microbiome

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Conference Abstracts

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Lower Division

Gut-Microbiome-on-a-Chip as a Personalized Patient Model to Predict Fecal Microbiota Transplant Compatibility for Vancomycin-Resistant Enterococci: An Original Approach

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Background: Antimicrobial resistance, specifically vancomycin-resistant enterococci (VRE), has recently emerged as a leading threat to public health due to its resistance of over 80% to vancomycin antibiotics. Without proper treatment, VRE in the gut can infect the bloodstream and may become life-threatening. Fecal microbiota transplant (FMT) has recently become promising in decolonizing VRE, however it still varies in success. The goal of this proposal is to investigate the use of gut-microbiome-on-a-chip (GMoC) to develop a personalized patient model that can predict FMT compatibility between recipient and donor *in vitro* without placing the recipient at risk of adverse side events.
Methods: A GMoC model is developed presenting a personalized model replicating the gut microbiome of the recipient partaking in the FMT process. Patient intestinal epithelial cells, endothelial cells, and fecal samples will be cultured to develop the gut and microbiome within the GMoC. A real-time polymerase chain reaction technique will be carried out pre and post-FMT to compare the quantification of VRE *vanA* and *vanB* genes on the GMoC. The decrease of VRE by 63% after FMT will demonstrate successful compatibility between FMT recipient and donor.
Conclusion: By leveraging the ability of GMoC to accurately recapitulate the human gut in an *in vitro* model, FMT procedures can be tested for success without placing the FMT recipient at risk of adverse side events. This original approach to precision medicine highlights the possibilities of GMoC and other microfluidic *in vitro* cell culture systems to improve the success of various health procedures.

Lactobacillus acidophilus as a Probiotic Through the Measure of Lactic Acid Effects on *Salmonella* Kentucky: A Research Proposal

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Introduction: *Salmonella*, a bacterium generally found in the intestines of humans and animals are known to cause symptoms that negatively impact the wellbeing of the individual. A specific serotype of *Salmonella, S. Kentucky*, is known to cause diarrhea, hindered growth, and even death in chickens. It has been identified in chickens worldwide and has shown antimicrobial resistance towards antibiotics. The methods in which the probiotic, *Lactobacillus acidophilus*, kills *Salmonella* should be investigated because it can bring forth improvements in chicken feed while fighting antibiotic resistance.
Methods: The effects of lactic acid on *S. Kentucky* are tested using a Kirby-Bauer Disk Diffusion Antimicrobial Susceptibility Test across 1%, 2%, 3% and 4% concentrated lactic acid solutions. Positive and negative control groups are done with 70% ethanol and no treatment respectively. The diameter of the Zone of Inhibition (ZOI) would be measured to

determine the efficiency of the lactic acid treatment as an antimicrobial. The values would then be graphed as ZOI (mm) vs lactic acid concentration (%) and the statistical significance would be determined.
Conclusion: The mechanism that *L. acidophilus* uses to inhibit the growth of *Salmonella* can provide insight on its influence in dietary products, such as chicken feed. By understanding the role of lactic acid as a by-product, it raises interest in new types of supplements for agricultural use.

Combination Therapy of Reduced-Dose Fecal Filtrate Transfer With Human Milk Oligosaccharides Supplementation to Prevent Necrotizing Enterocolitis in Premature Infants: A Research Protocol
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Necrotizing enterocolitis (NEC) is an inflammatory disease affecting the gut that disproportionately affects premature infants due to an underdeveloped gut microbiota (GM). A current treatment, fecal microbiota transfer (FMT), poses safety concerns. However, a recent study explored a novel treatment, fecal filtrate transfer (FFT), as a potential treatment for NEC. Despite showing promising results, the beneficial impacts were accompanied with side effects. Meanwhile, human milk oligosaccharides (HMOs) are effective in preventing NEC symptoms, but remain limited in availability. This study proposes a procedure to explore utilizing reduced-dose FFT and synthetic HMOs in combination as a safer treatment to prevent NEC. It is hypothesized that the combination therapy will have additive effects, while also reducing FFT-associated side effects. This study will use a piglet model to stimulate human gastrointestinal physiology and compare four groups: control, FFT-only, HMOs-only, and combined FFT-HMOs. FFT will be prepared by isolating virus-like particles (VLPs) from maternal porcine feces, and use of two HMO supplementations. The supplemented HMOs will be 2'-Fucosyllactose (2'-FL) and 6'-Sialyllactose (6'-SL). Histological grading of intestinal tissue will assess NEC severity, and quantitative reverse transcription polymerase chain reaction (RT-qPCR) will measure pro-inflammatory cytokines IL-1β and TNF-α, evaluating immune responses to NEC. This protocol aims to develop a novel therapy for NEC, addressing limitations of current treatments. The results will validate FFT for treating NEC in premature infants and guide future research on combining FFT with synthetic HMOs or human donor milk.

Exploring the Effects of Maternal Immune Factors and Gut Microbiota on Neonatal Peyer's Patch Development
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The neonatal immune system is constantly surrounded by new antigens and must learn to balance defense against threats with tolerance for the gut microbiome. Since improper neonatal immune development is associated with conditions like asthma, allergies, and chronic bowel inflammation later in life, understanding factors that influence immune development is crucial to preventing these diseases. Intestinal immunity is managed by the Peyer's patches (PPs) of the small intestine, where microfold cells (M cells) sample antigens from the intestinal lumen to stimulate B cells to produce secretory immunoglobulin A (sIgA), which protects the body from potential pathogens. Neonatal PP development is encouraged by maternal immune factors in breast milk and the presence of bacterial genera *Bifidobacterium*, *Lactobacillus*, and *Staphylococcus* in the gut microbiome, but these interactions have not been thoroughly researched. Therefore, we propose an in vitro study to determine the effects of breast milk and each genus of bacteria on neonatal PP development. Microfluidic devices will permit interactions between PPs, M cells, and cultures of *Bifidobacterium infantis*, *Lactobacillus salivariu*s, or *Staphylococcus epidermidis* in the presence of breast milk or a formula control. PP development will be measured by increased sIgA production and M cell maturation markers, which will be compared between experimental groups. It is predicted that samples involving breast milk will have the greatest immune development, and that *Bifidobacteria* will induce more development than *Lactobacilli* and *Staphylococci*. The results of this study will present evidence that breastfeeding and probiotics can improve neonatal immune development to help prevent inflammatory disease.

Microplastics and Mental Health: Investigating Microplastic-induced Gut Dysbiosis, Neurotransmitter Disruption, and Gut-brain Axis Dysregulation and its Impact in Depression

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The gut microbiome plays an essential role in producing neurotransmitters, such as serotonin, gamma-aminobutyric acid (GABA), and dopamine, that interact with the brain via the gut-brain axis. These neurotransmitters influence mental health, and disruptions to their production have been associated with depression. Microplastics, a ubiquitous environmental pollutant, may disrupt this axis by affecting the gut microbiome's neurotransmitter synthesis pathways, yet this potential mechanism remains underexplored. Microplastics may interfere with gut microbial communities through mechanisms such as increased oxidative stress, inflammation, or the disruption of microbial cell membranes. This research investigates the effects of microplastic exposure on neurotransmitter synthesis and mental health outcomes by disrupting microbial gene expression and metabolic pathways. We hypothesize that microplastics interfere with the microbial production of key neurotransmitters, leading to reduced levels of serotonin, GABA, and dopamine. To test this hypothesis, we combine in vitro microbial cultures and in vivo mouse models. Specifically, we will use *Lactobacillus rhamnosus* and *Bifidobacterium longum* - two bacteria known to be vital to neurotransmitter synthesis. Additionally, we will expose a cohort of C57BL/6 mice to both clean and sorbed microplastics to evaluate gut-brain axis disruptions and behavioural changes associated with depression. Behavioural assessments will include the forced swim test for depression and the sucrose preference test for anhedonia. These assessments will be couples with biochemical analyses of neurotransmitter levels and gut microbial diversity. This interdisciplinary approach aims to uncover a novel environmental risk factor for mental health disorders, providing insight that could inform public health policies and preventive strategies to mitigate microplastic exposure.

Investigating the Effects of Fecal Microbiota Transplantation Enriched with Probiotic Strains and Microalgae in App/ps1 Mice, a Promising Novel Solution for Alzheimer's Disease

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Alzheimer's Disease (AD) is a progressive neurodegenerative disorder marked by amyloid-beta (Aβ) plaque accumulation, driven by the activity of BACE1 and γ-secretase. Recent findings underscore the gut-brain axis as a key modulator of AD pathophysiology, where alterations in gut microbiota impact neuroinflammation and oxidative stress. This study investigates the combined therapeutic potential of fecal microbiota transplantation (FMT), enriched with anti-inflammatory *Bifidobacterium* and *Lactobacillus* strains, and antioxidant-rich microalgae (*Chlorella* and *Spirulina*), in mitigating AD pathology. Using aged APP/PS1 mice, a well-established AD model, it is hypothesized that this treatment will decrease BACE1 and γ-secretase activity, reduce Aβ plaque formation, and lower reactive oxygen species (ROS) levels in the brain. The study employs Western Blotting, Sandwich ELISA, and ROS fluorescent markers to quantify changes in enzymatic activity, Aβ production, and oxidative stress markers, respectively. By addressing Aβ accumulation, neuroinflammation, and oxidative damage simultaneously, the intervention targets multiple aspects of AD pathophysiology. The inclusion of *Chlorella* and *Spirulina* enhances antioxidant defenses, while FMT rebalances gut microbiota, influencing metabolite production to modulate enzyme activity. This novel combination therapy seeks to advance current treatments for AD by leveraging the gut-brain axis to address systemic contributors to the disease, offering a potentially transformative novel strategy for combating neurodegenerative disorders.

Engineering *E. coli* Nissle 1917 for Polystyrene Degradation to Address Microplastic-Induced Dysbiosis in the Human Gut

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The global increase in plastic production has led to significant microplastic (MP) contamination, posing risks to natural environments, organisms and human health. Recent evidence suggests that MP accumulation disrupts the human gut microbiome, inducing dysbiosis. Polystyrene (PS), a common MP, is of particular concern due to its ability to allow opportunistic bacteria such as *Escherichia coli* to facilitate biofilm growth. To address this issue, we propose the use of genetically engineered *E. coli* Nissle 1917 (EcN) expressing the styrene-degrading catabolic pathway identified in *Pseudomonas spp*., as a probiotic to catabolically degrade PS and reverse dysbiosis. This study employs the gut-on-a-chip

(GoC) model to simulate the human gut environment to evaluate the efficiency of the engineered probiotic in reducing PS levels and regulating MP-induced dysbiosis. This experiment aims to offer a potential solution to the gut microbiota health impacts of MP-contamination and the advancement of human intestinal health and environmental regulations.

Targeting Helicobacter Hepaticus: A Dual-approach Strategy for Biofilm Disruption and Microbiota Preservation
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Helicobacter hepaticus is a Gram-negative pathogen implicated in chronic intestinal inflammation, colitis, and hepatic carcinogenesis. It is present in patients with chronic liver disease as well as the intestinal epithelium and crypts, particularly in the cecum. Its ability to form biofilms and induce microbiota dysbiosis presents significant challenges to eradication. Current antibiotic treatments often result in incomplete clearance and microbiota disruption, leading to recurrence. We propose a dual-targeted therapeutic approach combining host-mimicking peptides to inhibit bacterial adhesion and biofilm formation, with nanoparticle drug delivery for enhanced precision and biofilm penetration. This integrated strategy aims to mitigate inflammation, restore microbiota balance, and provide a scalable treatment for biofilm-mediated infections. A comprehensive experimental plan, including in vitro studies, murine models, and translational applications, is outlined to evaluate efficacy and safety. This proposal addresses an unmet clinical need, offering potential implications for managing other biofilm-associated pathogens.

Oh Crap!: Investigating the Joint Effects of Fecal Microbiota Transplant and Diet on Alzheimer's Severity
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Alzheimer's disease is characterized by abnormal protein buildup in the brain, causing a decline in memory and other cognitive functions. The underlying mechanisms of Alzheimer's disease has long been unknown. Emerging evidence suggests the gut-brain axis impacts the progression of Alzheimer's disease (AD). Results from previous studies suggests diet alters gut health. Gut microbiota health affects the production of amyloid-beta (Aβ) peptides, a mark of Alzheimer's disease (AD) pathology. Dysbiosis may cause cognitive decline or AD. Dysbiosis can result from a high-fat, high-sugar, low-fiber diet, like the Western Diet (WD). This experimental study will feed healthy mice three diets: an "ideal" diet with standard lab nutrition, an "average" diet with moderate fat and salt, and a "poor" diet with high fat and salt. After 16 weeks of diet manipulation, fecal microbiota from each diet group will be transplanted into 5xFAD model mice via colonoscopy. Y-maze memory tests will assess cognitive spatial memory ability. MRI imaging will visualize Aβ plaque deposition in the hippocampus, an area responsible for spatial memory. Gut microbiome composition analysis of donor and recipient mice will be achieved via 16S rRNA sequencing.This study aims to test the role of the gut-brain axis in AD development and test the effectiveness of FMT coupled with a healthy diet.

Investigating the Role of Cannabis in Regulating Gut Microbe Dysbiosis in Systemic Lupus Erythematosus (SLE) Patients Through the Endocannabinoid System in a Murine Model
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Systemic Lupus Erythematosus changes gut microbiome composition, leading to negative impacts including gut permeability and resulting inflammation, worsening the disease's symptoms for human patients. The gut microbiome plays a critical role in regulating immune responses in lupus patients and restoring its balance offers a novel therapeutic approach for the management of lupus symptoms. Exogenous endocannabinoids, ingested from cannabis, including CBD, have previously demonstrated anti-inflammatory effects and an ability to regulate gut microbiome compositions positively using the ECS. This study applies a novel connection between lupus-induced gut microbiome dysbiosis and cannabis' therapeutic use. This study hypothesizes that orally administering CBD through cannabis in lupus-infected mice will restore gut microbe homeostasis and raise beneficial bacterial populations, reducing gut permeability, and decreasing inflammation. This would be tested using a murine model with MRL/Ipr female mice that develop lupus. CBD (20 mg/kg/day) or a placebo would be administered in their diet for 12 weeks and fecal samples would be collected after 3, 6, 9, and 12 weeks for 16sRNA sequencing to assess the states of gut microbiomes. Inflammation markers (including IL-6, TNF-α) and endocannabinoid

system activity would also be analyzed using techniques including flow cytometry and immunohistochemistry. It is anticipated that the use of CBD will restore the gut microbiome homeostasis in lupus-infected mice and thus reduce inflammation and improve gut permeability. This study could implicate the potential of CBD, an often-stigmatized cannabinoid, as a novel therapeutic treatment for gut microbiome disruptions in autoimmune diseases, offering an alternative to conventional treatments.

Upper Division

Bacterial Trojan Horse: Reprogramming Regulatory T Cells via an Oncotropic Probiotic to Launch an Inside-out Attack on Colorectal Cancer

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Colorectal cancer (CRC) is the third most common cancer and the second leading cause of cancer mortality. Unfortunately, the application of immunotherapy is limited by CRC's immunosuppressive tumour microenvironment (TME). Regulatory T cells (Tregs) are a subset of helper T cells that help prevent autoimmunity through their immunosuppressive activity. In CRC, tumour-infiltrating Tregs inhibit anti-tumour immunity. The Treg phenotype is characterized by expression of the forkhead box protein P3 (FOXP3) transcription factor; loss of FOXP3 expression corresponds to loss of immunosuppressive activity. Thus, FOXP3 destabilization effectively reprograms Tregs into pro-inflammatory effector T cells (Teffs), enhancing anti-tumour immunity. Glucocorticoid-induced tumour necrosis factor receptor (GITR) is a cell surface receptor constitutively expressed in Tregs. GITR activation destabilizes FOXP3 expression, decreasing Treg immunosuppressive function. Several Treg-reprogramming drugs (e.g., monoclonal antibodies (mAbs)) have been developed but are limited by low efficacy and autoimmunity due to a lack of tumour specificity. Excitingly, several bacterial strains have demonstrated unique tumour-targeting abilities. Recently, attenuated oncotropic bacteria (e.g., *Escherichia coli* Nissle 1917 (EcN)) equipped with quorum-regulated lysis circuits have been used to selectively deliver genetically-encoded therapeutic proteins to the TME. Here, we propose using an oncotropic EcN probiotic to deliver agonistic anti-GITR mAbs to the CRC TME, reprogramming tumour-infiltrating Tregs into Teffs to enhance anti-tumour immunity, analogous to a "Trojan horse". To our knowledge, oncotropic bacteria have not yet been used to reprogram tumour-infiltrating Tregs.

Genetically-modified *E. coli* Nissle 1917 Provides Potential Long-term Weight Loss and Fat Content Decrease in a Mouse Obesity Model

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Obesity is a chronic, pressing problem that disproportionately affects low-income populations, who tend to purchase foods with higher fat and sugar content. Such diets lead to obesity and negatively impact the gut microbiome, which has further negative influence on the development and worsening of obesity. Genetically-modified bacteria are a recent proposed solution; notably, modified *E. coli* Nissle 1917 (EcN) has been shown to have promising anti-obesity effects reducing mice body weight and fat content. However, the transience in its effects makes it not viable in the long-term and not accessible for low-income populations. This study aims to create and test a strain of EcN that can remain in the gut for longer durations. An existing anti-obesity EcN strain (EcN-GM) will be engineered to overexpress F1C fimbriae, proteins required for EcN to adhere to surfaces through forming biofilm. This strain will be tested in vitro to confirm F1C was successfully added and extra biofilm is produced. It will then be tested in vivo against EcN-GM using C57BL/6J mice to evaluate its ability to decrease subjects' body weight and fat content, and its colonization duration in the gut microbiome compared to that of EcN-GM. Overall, this study aims to demonstrate that a novel strain of EcN possesses not only anti-obesity properties, but long-lasting effects on the gut — pointing towards a potential solution to the accessibility problem of obesity treatments.

Investigating the Effect of *Clostridium butyricum* on Mdx Mouse Models to Promote Bone Health in the Progression of Glucocorticoid-induced Osteoporosis in the Context of Duchenne's Muscular Dystrophy

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Duchenne's muscular dystrophy (DMD) is a fatal genetic disorder caused by mutations in X-linked genes encoding dystrophin. DMD has an early childhood onset with an incidence of one in 5000 males. The loss of dystrophin protein expression leads to adverse outcomes such as skeletal muscle wasting, loss of ambulation, risk of fractures, and wheelchair reliance by age thirteen. Apart from the multidisciplinary and therapeutic approaches to managing DMD, a cure does not exist. Although glucocorticoid treatment is currently the most common form of therapy, glucocorticoid-induced osteoporosis (GCOP) is a severe side effect of long-term use. Co-administration of bisphosphonates aims to prevent and treat GCOP, by inhibiting osteoclasts to prevent bone resorption, however, this mechanism is coupled with a decrease in bone formation. Recent studies have identified metabolites in the human gut that play an important role in bone homeostasis. This regulatory effect is the Gut-Bone axis, where short-chain fatty acids (SCFA) like butyrate play a vital role. Butyrate has been proven to decrease bone resorption while stimulating bone growth by promoting Wnt signalling in osteoblasts. *Clostridium butyricum* in the gut microbiome is a key player in forming large amounts of butyrate. This study will use WT and glucocorticoid-treated Mdx mice to assess the effects of *C. butyricum* on GCOP. The proposed protocol would be the first experiment to test the efficacy of *C. butyricum* in reducing osteoporotic bone resorption, while increasing bone formation, and may offer a promising alternative to bisphosphonates in the treatment of GCOP in DMD patients.

Investigating the Effects of Variable Δ^9-tetrahydrocannabinol (THC) and Cannabidiol (CBD) Exposure on the Gut Microbiome of a Pregnant Rat Model and Their Offspring

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The endocannabinoid system (ECS) is implicated in the homeostatic functions of the enteric nervous system (ENS), and thus bidirectional communication with the central nervous system (CNS) via the gut-brain axis. New research suggests the role of the ECS in regulating gut microbiome composition and functioning. Since the legalization of cannabis in Canada in 2018, approximately 10% of pregnant persons reported using cannabis to alleviate pregnancy symptoms. However, the effects of cannabis use on gut microbiome composition during pregnancy and its long-term impact on offspring remain poorly understood. Given increasing Δ^9-tetrahydrocannabinol (THC) content compared to other cannabinoids such as cannabidiol (CBD) in cannabis products, the present study aims to explore pregnancy symptom relief and microbiome composition changes in Sprague-Dawley rats as a result of whole-body smoke exposure to THC-prevalent, THC=CBD, CBD-prevalent blends, compared to a control group (n=15 per group). Additionally, one female and one male offspring from each litter will be included in the study. Total gastrointestinal (GI) transit time will indicate GI motility through interactions and shifts in microbiome patterns, an indirect indication of pregnancy symptom changes. Shotgun metagenomics sequencing will assess changes in the composition and metabolic function of microbial communities. Additionally, gas chromatography–mass spectroscopy will quantify altered microbiota composition by analyzing their products, which are short-chain fatty acids. The findings from this study can begin to inform safer cannabis formulations and offer autonomy to pregnant persons seeking evidence-based advice on relieving pregnancy symptoms while prioritizing the long-term health of themselves and their children.

***Fusobacterium nucleatum* Enhances Combined Anti-PD-1 and Anti-CTLA-4 Immune Checkpoint Blockade Efficacy in Microsatellite Stable Colorectal Cancer**

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Colorectal cancer (CRC) is a major cause of cancer-related deaths worldwide, with microsatellite stable (MSS) CRC accounting for 85% of cases. Despite its prevalence, MSS CRC remains resistant to therapies, particularly immune checkpoint blockade (ICB) treatments like anti-PD-1 and anti-CTLA-4. Recent studies have suggested *Fusobacterium nucleatum* (*Fn*), a gut microbiota commonly enriched in the CRC microbiome, can modulate immune responses and enhance the effectiveness of ICB therapies. Specifically, *Fn*'s butyric acid production, alleviates immune cell exhaustion and improves efficacy of anti-PD-1 therapies. However, the role of *Fn* in influencing ICB response beyond anti-PD-1 pathways, particularly its potential synergistic effect with anti-CTLA-4 treatment by fecal microbiota transplant (FMT) in MSS CRC, remains unclear. As such, we seek to understand whether *Fn* can enhance the efficacy of combined anti-PD-1 and anti-

CTLA-4 therapies and can serve as a predictive biomarker for selecting MSS CRC patients most likely to benefit from combination therapy. Our results may reveal that *Fn*, through its production of metabolites, can increase the effectiveness of combined anti-PD-1 and anti-CTLA-4 therapies, mediated by FMT. These findings ultimately provide new insights into how the gut microbiome influences tumor immunology and present potential strategies to improve ICB treatments in regards to treating MSS CRC.

Investigating the Role of Lipoglycanated *Ruminococcus blautia gnavus* in Autoimmune Flare Responses in Lupus-prone Mice

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Previous literature has demonstrated a correlation between the increased presence of the bacterial strain *Ruminococcus blautia gnavus* (*R. gnavus*) in the gut microbiome and lupus nephritis flare-ups, yet the mechanisms by which this bacterium may influence these flares have not been determined, nor has causality been established. It is hypothesized that the lipoglycanated *R. gnavus* strain will elicit stronger autoimmune flare-up responses than the regular strain, as evidenced by higher antinuclear antibody (ANA) levels and increased kidney immune complex deposition in treated mice, particularly in lupus-prone (NZM2328) mice. Understanding the connection between the presence of lipoglycanated *R. gnavus* and the severity of lupus nephritis markers could inform future research on treatment options involving modulation of the gut microbiota, offering novel therapeutic avenues for managing lupus nephritis.

Neutralization of *Candida albicans*-secreted Candidalysin via Nanobody-lactobacilli Biohybrids in Colorectal Cancer Pdx Mice

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Colorectal cancer (CRC) ranks among the most common cancers globally, with cases projected to rise significantly in the coming decades. Current treatments for advanced CRC are limited, underscoring the need for innovative therapies. Recent studies reveal that *Candida albicans* (*C. albicans*) promotes CRC progression through candidalysin, a peptide toxin that causes epithelial damage and inflammation. Conventional antifungal treatments are problematic due to resistance and systemic side effects, creating a need for alternative approaches. This research explores a novel biohybrid therapeutic: *Lactobacillus paracasei* (*L. paracasei*) engineered to express anti-candidalysin nanobodies on its surface. This dual-action strategy aims to neutralise candidalysin toxicity while leveraging the probiotic's intrinsic anti-cancer effects. In vitro, human colorectal epithelial cells (CECs) are exposed to three conditions: the biohybrid (treatment), no treatment (negative control), and *L. paracasei* (vehicle control). This setup aims to evaluate the biohybrid's effectiveness in reducing epithelial damage and inflammation in CECs exposed to candidalysin. In vivo, patient-derived xenograft (PDX) mouse models transplanted with CRC tumours will be treated with the biohybrid, non-engineered *L. paracasei* (vehicle control), or no treatment. Tumour progression will be monitored via magnetic resonance imaging and bioluminescence imaging. This approach aims to establish a microbiome-based therapy for CRC, offering a safer, targeted alternative to traditional antifungals. Findings from this study could pave the way for new, sustainable therapeutic strategies to improve CRC patient outcomes.

Conflicts of Interest

The authors declare that they have no conflict of interest.

Authors' Contributions

JGK: Co-President of SciNapse and Co-Chair of the USCC planning committee, assisted authors with their abstract submissions, drafted the conference abstract booklet, and gave final approval of the version to be published.
CK: Co-President of SciNapse and Co-Chair of the USCC planning committee, assisted authors with their abstract submissions, drafted the conference abstract booklet, and gave final approval of the version to be published.
MD: President of the Undergraduate Research Initiative, served on the planning committee for the USCC, drafted the conference abstract booklet, and gave final approval of the version to be published.

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