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MISA Case Competition 2024

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Abstract:
The MISA Case Competition is an undergraduate research conference organized by the Microbiology and Immunology Student Association (MISA) at Western University to promote innovation, collaboration, and scientific inquiry amongst students interested in microbiology and immunology. In 2023, MISA successfully ran our first case competition. This year, the theme for the MISA Case Competition 2024 was for our undergraduate teams to devise novel strategies and treatments to combat or prevent autoimmune disorders. Their approach could target any aspect of autoimmune diseases, whether it be through novel preventative initiatives, diagnostic techniques, or treatment strategies. Our conference includes oral presentations from the top six teams and a panel of judges composed of faculty members from the Department of Microbiology and Immunology and the Department of Biochemistry at Western University, with awards for the top three teams. Through the MISA Case Competition, we hope to provide an opportunity for students to conceive new ideas to advance the understanding and management of autoimmune diseases, improving patient outcomes and reducing the societal burden of these conditions.

Keywords: immunology; autoimmunity; undergraduate research; novel treatments; diagnostics; prevention

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Conference Abstracts

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Top 3 Oral Presentations

Transcription Factor Decoy Oligonucleotide Treatment Targeting Nf-κB in Inflammatory Bowel Disease

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Inflammatory bowel disease (IBD) is a chronic autoimmune inflammatory disease of the digestive tract. Current therapies for IBD yield a high proportion of primary nonresponse and are linked to significant adverse effects, underscoring the need for novel treatment approaches. A central contributor to inflammation in IBD is increased intestinal permeability via matrix metalloproteinase-9 (MMP-9) overexpression. Thus, this study aims to decrease MMP-9 levels by deactivating NF-kB, a transcription factor involved in its expression. Adeno-associated viruses will be used to deliver RNA transcription-factor decoy oligonucleotides (TFDON) that bind to and suppress NF-kB activity. The TFDON treatment will be administered to intestinal grafts extracted from CD34+ humanized mice reconstituted with human immune factors necessary to chemically induce IBD-characteristic intestinal inflammation using dextran sulfate sodium. To confirm successful TFDON transduction and oligonucleotide expression, live-cell single-molecule RNA imaging will be used. After treated and untreated grafts are placed in experimental and control mice, intestinal inflammation will be monitored weekly using P-selectin contrast-enhanced ultrasound. It is expected that the TFDON suppresses MMP-9 activity and intestinal permeability, thus reducing intestinal inflammation. Ultimately, this study offers a promising novel treatment that reduces IBD inflammation for patients who are averse to current therapies.

CRISPRi-Mediated Downregulation of TSHR to Reduce Hyperthyroidism Caused by Graves' Disease

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Graves' disease is an autoimmune disorder characterized by thyroid overstimulation by auto-immunogenic thyroid-stimulating immunoglobulins, leading to hyperthyroidism. Current treatments, like thyroidectomies, destroy the thyroid and may result in hypothyroidism. We propose CRISPRi-mediated thyroid-stimulating hormone receptor (TSHR) downregulation as a novel therapy that minimally disrupts thyroid function. To target TSHR, a lipid-based nanoparticle (LNP) delivery system coated with blocking human monoclonal anti-TSHR will be used. The anti-TSHR coat will target LNPs to thyroid cells by binding to TSHR, assisting in endocytosis of the LNP to release of CRISPRi DNA into the cells. In the thyroid cells, DNA is transcribed and translated by host enzymes. By using a gene-specific guideRNA co-delivered within the LNP, CRISPRi precisely binds to the TSHR gene, where CRISPRi transcriptional repressor domains suppress TSHR gene expression. This treatment has two-fold effects on downregulation of TSHR. First, activity of CRISPRi would cause translational inhibition of TSHR, decreasing its expression. Secondly, binding of anti-TSHR coated LNPs to TSHR would act as competitive antagonists to TSH. Together, these two mechanisms reduce TSHR activation, providing symptomatic short-term relief for Graves' disease.

Sort LNP Technology for Targeted Il-10 MRNA Delivery in Ulcerative Colitis: A Research Study

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Inflammatory bowel disease (IBD), encompassing ulcerative colitis (UC) and Crohn's disease (CD), is characterized by chronic inflammation of the gastrointestinal tract. UC, in particular, targets the colon and rectum with a continuous pattern of disease. The expression of anti-inflammatory IL-10 by gastrointestinal cells is essential for the modulation of these chronic inflammatory responses. Moreover, mutations in the genes encoding for IL-10 or its receptor, IL-10R, have shown to be associated with severe pediatric colitis. Previous studies have explored IL-10 mRNA as a treatment for UC; however, mRNA delivery and targeting of GI tissue has proven difficult. In our study, we suggest novel selective organ-targeting (SORT) lipid nanoparticle (LNP) technology as a means of overcoming these barriers. SORT LNP technology currently involves the modification of LNPs with SORT molecules that alter the surface of the LNP to drive targeted delivery of mRNA to the liver, lung, and spleen. In our study, we propose expanding this technology to drive targeted delivery of IL-10 mRNA to the GI tract to promote immunomodulation and remission in UC patients.

Top 4-15 Abstracts (No Particular Order)

Invitro Superantigen-Activated Regulatory T-Cells (Tregs): A Novel Approach to Combat Ankylosing Spondylitis

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Ankylosing Spondylitis (AS) presents as a chronic inflammatory disease affecting the axial spine, often characterized by lower back pain and stiffness and potentially affecting other joints and organs. While recent advancements in medication have improved treatment options, AS remains challenging to cure due to progressive inflammation. This study proposes investigating the efficacy of a novel therapeutic approach utilizing in vitro superantigen-activated regulatory T-cells (Tregs) to address AS. Through the activation of T cells via superantigens (sAgs), we can induce the production of sAg-Treg cells, which release immunosuppressive cytokines such as IL-10 and TGF-beta1, thus mitigating inflammation. Previous studies by Li et al. in rats with collagen-induced arthritis have shown that following injection with human Treg cells, the severity of the disease significantly decreased. They also noticed that the injected Treg cells increased the number of Tregs naturally present in the rats' bodies. By targeting cell surface markers unique to Tregs, such as CD4, CD25, and FoxP3, we can isolate them through methods such as affinity chromatography. This leads to the potential for a Treg vaccine that not only adds exogenous Tregs but also increases endogenous production. This research has the potential for a targeted immunomodulatory approach to alleviate AS symptoms.

Revolutionizing Autoimmune Disease Intervention: A Research Study

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Rheumatoid arthritis (RA) presents a formidable challenge, affecting millions and imposing a substantial economic burden. Our multi-disciplinary approach introduces a novel preventative initiative targeting key regulatory molecules in RA pathogenesis. We propose a personalized cytokine modulation strategy to intervene in early RA stages, aiming to normalize dysregulated levels of Tumor Necrosis Factor-alpha (TNF-α), Interleukin-1 (IL-1), and Chemokine (C-C motif) ligand 2 (CCL2). Simultaneously, our initiative includes a ground-breaking Matrix Metalloproteinase (MMP) inhibition strategy to halt extracellular matrix degradation and preserve joint integrity. Recognizing Toll-Like Receptors (TLRs) pivotal role in RA, we propose a novel TLR modulation approach, mitigating pathogen-associated molecular patterns (PAMPs) and danger-associated molecular patterns (DAMPs) recognition, curbing inflammation, and preventing RA progression. Our initiative focuses on harnessing Regulatory T Cells (Tregs) immunomodulatory potential, restoring immune tolerance. Emphasizing Janus Kinase (JAK) inhibitors integration, we target JAK-STAT signaling, aiming to reduce inflammation and intercept RA onset in genetically predisposed individuals. In conclusion, our preventative initiative holds promise for transforming RA prevention, ushering in a new era of personalized autoimmune disease intervention.

The Efficacy of Immunopathogenesis and Biomarking in Diagnosing and Treating Rheumatoid Arthritis: A Research Study

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Rheumatoid arthritis (RA) is an autoimmune disease characterised by chronic inflammation affecting joints and other body systems due to immune system attacks. Current treatments primarily rely on immunosuppression or corticosteroids, with methotrexate being a common option; however, it only significantly impacts about 70% of patients and often fails to induce remission. The limitations of these treatments underscore the need for diagnostic protocols focusing on immunopathogenic approaches rather than syndromic diagnosis. Incorporating biological and genetic markers to identify RA susceptibility could enhance diagnosis and treatment effectiveness, given the disease's heritability of 60%. As recent studies suggest, synovial lesions play a crucial role in RA pathology, with the synovial immune microenvironment differing significantly between RA patients and healthy individuals. Early identification and treatment are essential, as optimal management for inducing remission typically occur within 3-6 months. Focusing on the immunopathogenesis of RA may be done by analysing the immune response of already diagnosed patients to develop patterns for future diagnostic procedures. Understanding the autoimmune progression and genetic markers associated with RA could lead to earlier identification of potentially affected individuals and the implementation of preventive measures.

Bispecific Antibodies: A Precision Approach to Combat Multiple Sclerosis

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In addressing the challenge of combating Multiple Sclerosis (MS), a chronic autoimmune disease characterised by the immune system's attack on the central nervous system (CNS), a novel therapeutic approach emerges from integrating insights across immunology, bioengineering, and pharmacology. This strategy involves the development of bispecific antibodies engineered to precisely target and neutralise pathogenic T helper 1 (Th1) and T helper 17 (Th17) cells the blood-brain barrier (BBB) which is pivotal in MS pathogenesis and can prevent inflammatory infiltration. Leveraging the specificity of bispecific antibodies allows for dual-action: direct inhibition of the autoreactive T cells driving the autoimmune response and blockade of their CNS entry, thereby mitigating inflammation and subsequent neuronal damage. This approach is supported by current scientific literature indicating the central roles of Th1 and Th17 cells in MS and the therapeutic potential of modulating BBB dynamics to control neuroinflammation. By drawing on advances in antibody engineering and immunological understanding, this strategy promises a targeted, efficient intervention that minimises systemic immune suppression, reducing the risk of infections—a common drawback of existing MS treatments. This innovative solution exemplifies a multidisciplinary, evidence-based approach to autoimmunity, offering a blueprint for precision medicine in autoimmune disease management.

Evaluating the Therapeutic Potential of Efgartigimod and Amiloride Combined Therapy in Chronic Inflammatory Demyelinating Polyneuropathy

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Chronic inflammatory demyelinating polyneuropathy (CIDP) is characterized by symmetric limb weakness due to autoantibody-mediated demyelination. Current treatments include corticosteroids, intravenous or subcutaneous immunoglobulin, or plasma exchange to neutralize effects of autoantibodies. Efgartigimod, a novel therapeutic under clinical trials, utilizes monoclonal antibodies to mitigate immune-mediated neuronal damage by engaging Fc receptors on immune cells. Efgartigimod has shown efficacy in reducing IgG levels in myasthenia gravis patients. Amiloride, in phase 2 trials for multiple sclerosis, protects oligodendrocytes without modulating the immune system, complementing efgartigimod. We propose a combined therapy of efgartigimod and amiloride to reduce autoantibody-triggered phagocytosis of myelin while promoting oligodendrocyte-mediated myelin regeneration. Our hypothesis suggests that this approach will delay CIDP progression and facilitate neural communication repair through myelin regeneration. A randomized controlled trial on CIDP patients without complications will compare treatment to placebo, assessing symptom changes and adverse events. This study provides insights of future novel treatments with a possibility of neural network regeneration and antibody suppression with minimal harm, promising the rehabilitation of patients with CIDP and other autoimmune neurodegenerative diseases.

Advancing Crohn's Disease: Harnessing Ingestible Capsule Sampling Device for Patient-Specific Gut Microbiota Analysis

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Crohn's disease (CD) is an inflammatory autoimmune disease of the gastrointestinal tract lining characterized by dysregulated immune responses targeting self-antigens, ultimately leading to impaired interaction of the intestinal microbiota. Current diagnostic modalities (e.g. biochemical markers, endoscopy, radiologic findings) delay treatment due to their reliance on invasive procedures and symptom manifestations, indicating later disease stages. However, a non-invasive ingestible device within an enteric-coated capsule allows for early microbiome characterization in CD patients. The coating would dissolve at a pre-set pH, targeting multiple collection sites in the intestines and enabling the localization of skip lesions in CD. Active CD patients show increased *Proteobacteria*, presence of *Fusobacterium*, and decreased levels in *Clostridium* cluster IV of anaerobic bacteria. This CD interaction with the microbiota can serve as the diagnostic parameters for device collection. Intestinal peristalsis and pH gradients could influence the localization of samples, and stool contamination may affect gut microbiota profile analysis. Moreover, the device functions as a screening tool for high-risk patients as it can collect genetic material from the gastrointestinal tract. Overall, this device holds promising potential as a diagnostic and screening method for CD by capturing patient-specific gut microbiota thus improving management and outcomes for CD patients.

Developing Artificial Intelligence Models for Rheumatoid Arthritis Prediction and Diagnosis

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Rheumatoid arthritis (RA) is a chronic autoimmune disease affecting 18 million people worldwide. It is characterized by the immune system's attack on the synovial membrane within joints, leading to inflammation and deterioration of the joint tissue. Physical examinations and imaging techniques are key to diagnosing RA. Deep learning (DL), a subset of artificial intelligence (AI), can offer solutions in efficiently identifying patterns in imaging data to provide predictions supporting RA diagnosis. For example, AI has been shown to have 91% sensitivity in early breast cancer detection, compared to 74% sensitivity by radiologists. Therefore, a DL approach can be used in early diagnosis of RA for better health outcomes. By using knowledge from highly accurate, existing models predicting RA from X-ray images, a DL model can be developed to analyze various imaging data sources including magnetic resonance imaging and ultrasound images. The DL model can be further enhanced as a multi-modal AI model by using a database of anonymous, clinical RA data. Leveraging vast public database information will enable the development of cost-effective and reliable AI models for more efficient RA diagnostic solutions.

Breaking the Chains in Graves' Disease Using Anti-TSI Monoclonal Antibodies

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Graves' disease (GD) is a rare autoimmune disorder responsible for 60-80% of hyperthyroidism cases in the United States. GD significantly reduces patients' quality of life through symptoms like weight loss, tremors, and tachycardia. In GD, autoantibodies called thyroid-stimulating immunoglobulins (TSIs) bind to the thyrotropin receptor (TSHR) by mimicking thyroid-stimulating hormone (TSH). This hinders TSH from binding to the TSHR and causes the unregulated overproduction of thyroid hormone. Existing GD interventions focus on symptom management, overlooking the underlying autoimmune pathology. Alternatively, recent literature has proposed inactivating the TSHR to prevent TSI from binding; however, this often disrupts the natural TSH-TSHR interactions. Our novel approach involves developing a monoclonal antibody (mAB) to directly neutralize TSIs and reinstate regular TSH binding to normalize thyroid hormone levels. After collecting TSI-producing lymphocytes from GD patients, X-ray crystallography will identify a motif conserved among patients that is specific to the TSI variable heavy chain. Using this motif, ex-vivo generated mABs will neutralize TSIs in GD patients. Therefore, our intervention will restore thyroid hormone balance, offering a significant advancement in GD treatment without risking abnormal TSH-TSHR disruptions.

Revolutionizing Sjögren's Syndrome Treatment: A Microbiota-Based Therapeutic

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Sjögren's syndrome is an autoimmune disease characterized by the cellular attack of the moisture-producing glands, presenting with symptoms of dry mouth, eyes, and skin. There is a need for innovative approaches that target the underlying autoimmune mechanisms, beyond present-day replacement therapies. This proposal explores a non-invasive probiotic treatment to target the gut microbiome as evidence has linked Sjögren's syndrome with gut dysbiosis. Sjögren's syndrome patients are found to positively associate with an abundance of Enterobacter bacteria and negatively associate with Lachnospira, Roseburia, Bifidobacterium, Ruminococcus, Blautia, and Roseburia. A mouse model stimulating Sjögren's syndrome shows increased expression of proinflammatory cytokines and lower quantities of regulatory T cells (Tregs) which are cells critical in autoimmunity prevention. This suggests that gut bacteria serve both a protective effect in a healthy gut, and a pathogenic effect in a dysbiotic gut. Probiotics include live organisms and can modulate the functions T and B lymphocytes. Animal models show that certain probiotic strains, such as one 5-strain composition involving Bifidobacterium bifidum, can induce Treg expansion and downregulate proinflammatory cytokines. There is great therapeutic potential in probiotics as a natural treatment for Sjögren's syndrome.

A Novel Approach to Psoriasis Treatment Using RNA Interference Therapy: A Research Study

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Psoriasis is a chronic autoimmune disorder of the skin that causes inflamed, scaly patches. It is the result of a dysregulation of the immune system which leads to over proliferation of keratinocytes; the cells responsible for creating a protective barrier on the skin. The treatment options available today include "corticosteroids, phototherapy, systemic medications, and biological agents." A novel approach to treat this disorder would be to target the inflammatory cascade by using RNA interference therapy (RNAi). RNAi is a method of gene expression regulation. It works by silencing specific messenger RNA molecules, ultimately preventing translation of those proteins. By harnessing the mechanisms of this cellular process, TNF-alpha, interleukin-17, and interleukin-23: the major molecules involved in psoriasis, expression can be inhibited in patients. This targeted approach would also incorporate the use of small interfering RNA (siRNA) molecules to select for the pro-inflammatory molecules directly on the psoriatic lesions. siRNA has sparked the attention of researchers due to its "ability to inhibit specific genes in many genetic diseases." This reagent would work together with RNAi to result in the reduction of keratinocyte proliferation and subsequently skin inflammation.

Nanoparticle-Facilitated Treg Mimetics for Alopecia Areata: A Research Proposal

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Alopecia areata (AA) represents a paradigm of autoimmune pathology wherein autoreactive T lymphocytes orchestrate a targeted assault on hair follicles, resulting in non-scarring hair loss. The current therapeutic landscape, predominantly comprised of topical & intralesional corticosteroids and systemic immunosuppressants, suffer from non-specificity and systemic side effects. Therefore, we propose an innovative therapeutic strategy combining nanotechnology with regulatory T cell (Treg) mechanisms. Utilizing biodegradable nanoparticles targeted via ligands to receptors like Patched-1 (PTCH1) or Smoothened (SMO) within the Sonic Hedgehog (Shh) pathway, this method aims for precise delivery to the affected follicular regions. This aims for precise delivery to AA's inflammatory microenvironment. The nanoparticles encapsulate Treg mimetic peptides and siRNA molecules to specifically downregulate pro-inflammatory cytokines within the peri-follicular space, mitigating localized inflammation and restoring immune tolerance. This dual-action modality not only aims to mitigate localized inflammation but also to restore immune tolerance at the site of autoimmunity. Preliminary in vitro assays would test targeting and uptake by dermal papilla cells, with subsequent in vivo mouse model studies assessing reductions in lymphocytic infiltration and increases in hair regrowth. This localized immune modulator delivery system offers a novel, targeted therapeutic avenue for AA, enhancing efficacy and minimizing systemic toxicity.

Hacking Immunity: Building Tolerance in Addison's Disease

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Addison's disease is characterized by the autoimmune attack on an individual's adrenal glands. Specifically, the enzyme 21-hydroxylase (21OH), which affects the body's cortisol and aldosterone biosynthetic pathways, causing insufficient hormone production. Through a review of the literature, we suggest that modified dendritic cells (DC) could be used to induce immune tolerance. DCs play a critical role in T-cell development by presenting self-antigens to thymocytes in the thymic medulla. Thymocytes that react strongly with DCs are removed via clonal deletion or differentiated into regulatory T-cells. Regulatory T-cells suppress an autoimmune response. Previously, DCs have been altered to display self-antigens associated with autoimmune reactions. The resulting negative selection has been used to achieve tolerance for the autoimmune reaction in myocarditis and transplants in mice. By modifying DCs to present two epitopes of 21OH which are strongly associated with autoreactivity in Addison's, LLNATIAEV and ARLELFVVL, we propose that we can induce tolerance.

Conflicts of Interest

The authors declare that they have no conflict of interests.

Authors' Contributions

JV: Served as a planning committee for the conference, drafted the conference abstract booklet, and gave final approval of the version to be published.
DD: Served as a planning committee for the conference, drafted the conference abstract booklet, and gave final approval of the version to be published.
HA: Served as a planning committee for the conference, drafted the conference abstract booklet, and gave final approval of the version to be published.
SK: Provided support in the planning and execution of the conference and gave final approval of the version to be published.

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