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Labpath Research Hackathon 2025: Hormones and Metabolic Disorders

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Abstract

LabPath's Research Competition offers undergraduate students across Canada the opportunity to design experimental research projects. The event was designed to showcase the possibilities of what student-led research can be while breaking down barriers like credentialism and exclusivity. The goal with the event is to make research more accessible, empowering students to explore ambitious ideas regardless of background or experience. The 2025 theme, endocrinology, challenged participants to come up with a novel innovation within the field. Hosted at the University of Toronto Mississauga, the two-day in-person event brought together 300 students from over 100 university programs. Teams of 2-4 students were tasked to develop complete research proposals within the field of endocrinology, including an abstract, methodology, and figures. The top 30% of abstract submissions, selected by a panel of research and academic judges, are featured in this booklet.

Keywords: LabPath; research hackathon; undergraduate; hormones and metabolic disorders; endocrinology

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Conference Abstracts

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Research Hackathon Abstracts

Precision Gene Therapy for OCTD: Leveraging Cortisol Signaling to Drive Adaptive OTC Expression

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Ornithine transcarbamylase deficiency (OTCD) is a rare X-linked metabolic disorder that disrupts the urea cycle, impairing the body's ability to convert toxic ammonia into urea. Current therapies, including protein-restricted diets, nitrogen-scavenging medications, and liver transplants, provide only partial metabolic control and fail to offer a mechanism that adapts to physiological stressors such as hyperammonemia. This proposal presents a novel hormone-responsive gene therapy strategy designed to restore OTC expression in a metabolically-regulated manner. By leveraging elevated cortisol levels during hyperammonemia episodes, we aim to activate OTC transcription using a synthetic construct driven by a glucocorticoid response element (GRE-OTC). The GRE-OTC construct will be packaged in an adeno-associated vector 8 (AAV8) for liver-specific delivery. In vitro, human-induced pluripotent stem cell-derived hepatocytes (hiPSC-hepatocytes) will be treated with dexamethasone, a cortisol analog, to trigger GRE-mediated OTC expression. Three groups, (1) GRE-OTC + dexamethasone, (2) dexamethasone-only, and (3) untreated, will be analyzed. mRNA levels (via qPCR) and OTC enzymatic activity (via colorimetric urea assay) will determine expression and functional efficacy. Subsequently, in vivo testing will be conducted using a spf-ash OTCD mouse model. Mice will be stratified into three groups: (1) OTCD + GRE-OTC therapy, (2) untreated OTCD mice, and (3) healthy controls. Plasma ammonia levels, hepatic OTC expression, and survival rates will be assessed to evaluate therapeutic outcomes. If successful, this project could lay the groundwork for a feedback-regulated treatment paradigm for OCTD and similar metabolic disorders. It introduces a synthetic biology approach that links endocrine signals with gene therapy to create dynamic, condition-responsive expression systems.

AI-Guided de Novo Design of MNK2-Blocking Peptidomimetics ("RegenBeta-1") to Trigger Endogenous β-Cell Regeneration in Diabetes

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Diabetes remains incurable largely because progressive loss of functional insulin-producing β-cells outpaces current replacement or protection strategies. Recent work has shown that MAP-kinase–interacting kinase 2 (MNK2) restrains protein-synthesis-driven β-cell neogenesis; pharmacologic or genetic MNK2 blockade re-initiates β-cell formation from ductal progenitors in human organoid and large-animal models. We propose RegenBeta-1: an artificial-intelligence (AI) platform that generates cell-penetrant, protease-resistant peptidomimetics which competitively disrupt the MNK2–eIF4G interface, thereby unlocking endogenous β-cell regeneration. A multi-objective discrete diffusion model trained on 190,000 high-resolution protein–peptide complexes will optimise (a) MNK2 binding free energy, (b) helicity, (c) serum stability, and (d) intestinal permeability, building on recent deep-learning frameworks for peptide design. The top 50 candidates will be synthesised with non-natural amino acids to extend half-life. Direct binding will be quantified via microscale thermophoresis, and crystal structures of the three best binders will refine the model. Functional assays will employ human pluripotent-stem-cell-derived ductal/β-cell organoids and zebrafish β-cell ablation screens. We hypothesise that lead peptide RB-1A will increase insulin-positive cell mass by ≥50% versus vehicle and normalise glycaemia in streptozotocin-treated mice within 14 days. If successful, RegenBeta-1 will pioneer an AI-to-bench pipeline that converts translational-control biology into first-in-class regenerative therapeutics for both type 1 and insulin-exhausted type 2 diabetes. The strategy is modular, enabling rapid retargeting to other intracellular endocrine checkpoints.

Prolactin-Driven in Vitro Modeling of Human Endometrial Implantation Using a Uterus-on-a-Chip to Advance IVF Treatment Strategies

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Infertility impacts approximately 1 in 6 individuals worldwide, with implantation failure remaining a leading cause of unsuccessful in vitro fertilization (IVF). While prolactin is commonly associated with infertility due to hyperprolactinemia, recent research suggests that localized prolactin signalling in the endometrium may enhance blastocyte adhesion and support endometrial receptivity. Prolactin receptors are expressed in the endometrial epithelium, and prolactin has been shown to regulate genes critical to implantation, such as LIF, IGFBP1, and HOXA10. However, these mechanisms have not been explored in a controlled, physiologically relevant model. This proposal outlines the design of a uterus-on-a-chip microfluidic system that recapitulates the endometrial interface during the implantation window. The device integrates endometrial epithelial and stromal cells within a microengineered culture chamber that permits controlled hormone delivery, including varying prolactin concentrations. Embedded biosensors monitor prolactin levels in real time, while the expression of implantation-related markers is assessed using qPCR and fluorescent reporter assays. Stromal cells undergo decidualization under stimulated luteal phase conditions to evaluate prolactin's role in supporting implantation readiness. By stimulating the in vivo endometrial environment in a physiologically accurate manner, this system enables systematic investigation into the effects of prolactin on implantation potential. The platform may help define optimal prolactin concentrations for improving IVF success rates and offers a novel preclinical model for studying endometrial physiology. This integration of reproductive endocrinology and microfluidic bioengineering aims to advance the understanding of hormonal regulation in implantation and inform future therapeutic interventions in assisted reproduction.

Circadian Rhythm Disruption Impairs Thyroid Hormone Conversion via Inflammation-Mediated Suppression of Deiodinase Activity

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This paper explores the novel correlation between unbalanced circadian rhythms that results in chronic low-grade inflammation leading to impaired hormonal conversion of thyroxine (T4) to triiodothyronine (T3). This results in symptoms that appear similar to subclinical hypothyroidism. Considering T3 is essential to the regulation of metabolism in most cells, the fluctuation of T3 production can be detrimental in the maintenance of regular bodily function. The methods to produce conclusive results would involve exposing rat populations to various light conditions, with one population possessing an anti-inflammatory to factor in the effects of low-grade chronic inflammation. These three groups would be expected to have varying levels of T3 production, with the anti-inflammatory group expected to stabilize their T3 levels after its introduction. This research would explore new areas in between the connection within circadian rhythm and hormone production systems.

CYP1B1 Overexpression to Produce Pregnenolone for an Alzheimer's Disease Murine Model In-Vivo as a Potential Remedy for AD Pathology

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Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by memory loss, neuronal degradation, and chronic neuroinflammation. Emerging studies suggest that pregnenolone, a neurosteroid hormone, reduces microglial inflammation, improving memory and combating Alzheimer's symptoms. This controlled experiment demonstrates an in-vivo treatment with pregnenolone via CYP1B1 overexpression in an in-vivo murine model's neurons to investigate the effects of pregnenolone on Alzheimer's disease. The murine model involves the control group of Alzheimer's-induced mice (APP gene expression) with saline injection and the experimental group of Alzheimer's-induced mice with CYP1B1 overexpression via an AAV-PHP.eB viral vector. Following gene delivery, mice are tested using a Y-maze to assess spatial, working, recognition, and associative memory. Compared to the control group of Alzheimer-induced mice injected with saline, those with CYP1B1-induced pregnenolone expression are expected to show maze completion times comparable to controls. These results would suggest enhanced cognitive performance and that pregnenolone has a positive effect on memory, which may offer a potential remedy for AD pathology in the future. Levels of pregnenolone concentration will be tested for by the gas chromatography/mass spectrometry followed by high performance liquid chromatography. The CYP1B1 protein will be tested for with immunohistochemistry. They are expected to be increased in this experiment.

Interferon Induced Upregulation of Immune Checkpoint LPD-L1 and CD155 on Pancreatic β-cells to Mitigate T-cell-Mediated Immune Attacks in a Type 1 Diabetes Model

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Type 1 Diabetes (T1D) is a chronic autoimmune disease in which the body's immune system attacks and destroys healthy insulin-producing β-cells of the pancreas. For over a century, insulin has remained the only therapy for T1D and limited literature seeks to modify β-cells to defend themselves from these immune attacks. Additionally, novel research in oncology has demonstrated that tumors defend against immune attacks through the upregulation of ligands such as PD-L1 and CD155 which inhibit T-cell activation via immune checkpoint inhibitors such as PD-1 and TIGIT. This results in an exhausted T-cell, no longer capable of carrying out its function. This study aims to adopt these defensive mechanisms for β-cell defense in T1D. Leading to the research question: Does targeted upregulation of immune checkpoint ligands PD-L1 and CD155 on pancreatic β-cells via interferon stimulation mitigate T-cell-mediated immune attacks in a Type 1 Diabetes model? It is hypothesized that upregulating PD-L1 and CD155 on β-cells, through stimulation with IFN-a and IFN-y, will protect β-cells against immune attack by inducing T-cell exhaustion and inhibiting infiltration. To test this hypothesis an in-vitro model made up of four cohorts (n=25) will be used: untreated β-cells (control, β-cells stimulated with IFN-α, IFN-γ, and a combination of both interferons. T-cells and β-cells will be harvested from NOD mice which have dual color-luminescent reporters allowing for the tracking of PD-L1 and CD155 expression. Following stimulation, luciferase labeled T-cells and β-cells will be co-cultured and T-cell exhaustion, infiltration, PD-L1 and CD155 expression will be measured periodically.

Concurrently, β-cell function will be assessed via insulin response following a spike in glucose. Two-way ANOVA and Tukey's post hoc tests will then be utilized for statistical analysis between cohorts. If the hypothesis is proven correct it could establish a novel approach to treating and managing T1D with an emphasis on preserving β-cell function. If proven efficacious, this therapy could revolutionize the treatment of Type 1 diabetes, as an alternative to insulin following rigorous testing in animal models and clinical trials. This approach could even be expanded to the treatment of other endocrinologic autoimmune disorders, transforming the way we treat them.

Interferon Induced Upregulation of Immune Checkpoint Ligands PD-L1 and CD155 on Pancreatic β-cells to Mitigate T-Cell-Mediated Immune Attacks in a Type 1 Diabetes Model

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Obesity is a major contributor to the development and progression of hormone-related cancers, creating a tumor microenvironment (TME) that is hypoxic, lipid-rich, and immunosuppressive. Globally, obesity contributes to 4–8% of all cancers, and with more than one billion people living with obesity, hormone-related cancers are steadily rising. In obese conditions, tumors suppress the PHD3 enzyme to increase fatty acid uptake, enhancing their growth while depriving CD8+ T-cells of the nutrients required for proliferation and effector function. As a result, cancers such as breast, endometrial, and colorectal, which typically respond well to immunotherapy, become immunologically “cold.” This compromises the efficacy of current treatments, leaving a growing global population of patients behind. To address this, Oxynova is a novel therapeutic strategy that uses a genetically modified Adenovirus vector in combination with CRISPR activation (CRISPRa) to restore PHD3 expression specifically in the TME. By increasing PHD3 activity, Oxynova aims to normalize nutrient availability, reduce tumor lipid accumulation, and enhance CD8+ T-cell infiltration and function. This approach reprograms tumor metabolism in hormone-related cancers, with the potential to improve immunotherapy outcomes in obese individuals and close the treatment gap for this expanding patient population.

EstroSense-VLP: Dual-Function Tamoxifen Delivery and Real-Time Imaging Using P22 Virus-Like Particles in ER+ Breast Cancer

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Estrogen receptor-positive (ER+) breast cancer accounts for approximately 70% of breast cancer cases and depends on estrogen signalling for tumor growth. Tamoxifen, a selective estrogen receptor modulator, blocks estrogen signalling by working as a competitive inhibitor, significantly reducing recurrence and mortality. However, systemic delivery of Tamoxifen can lead to unwanted side effects in healthy tissues, such as hot flashes, bone loss, and increased risks of endometrial cancer and blood clots. To address this, a targeted delivery system is needed to concentrate Tamoxifen’s effects within tumor environments while sparing the rest of the body. This project proposes EstroSense-VLP, a dual-function therapeutic and diagnostic platform using virus-like particles (VLPs) derived from Salmonella bacteriophage P22. To improve tumor specificity, the VLPs are functionalized with ligands that target HER2 and folate receptors, which are commonly overexpressed in ER+ breast cancer cells. These engineered nanocarriers facilitate the co-transport of both Tamoxifen and a pH-sensitive fluorescent dye. Tamoxifen is linked to the VLP’s exterior through acid-labile bonds that remain stable in the bloodstream but break down in acidic tumor environments, releasing the drug only where it is needed. Meanwhile, the pH-activated fluorescent dye is loaded into the interior of the VLP. When the VLP enters an acidic tumor microenvironment, the dye becomes visible, thus providing real-time confirmation of drug delivery and location. EstroSense-VLP offers a novel, modular solution that combines hormone therapy with imaging. It enhances post-chemotherapy care by enabling more precise delivery and monitoring of Tamoxifen while minimizing systemic side effects.

Targeting Insulin Resistance in PCOS: Dual Therapy with GLP-1RA and Adiponectin to Boost SHBG Production

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Polycystic Ovary Syndrome (PCOS) is a common endocrine disorder in women of reproductive age, affecting up to 10% worldwide. It is characterized by insulin resistance, hyperandrogenism, ovarian dysfunction, and a range of metabolic complications, including increased risk of type 2 diabetes, obesity, and cardiovascular disease. Although the exact cause is unknown, reduced hepatic production of sex hormone-binding globulin (SHBG) plays a key role by increasing free androgen levels, amplifying hyperandrogenic symptoms such as hirsutism, acne, and anovulation. Insulin resistance drives hyperinsulinemia, which suppresses SHBG synthesis in hepatocytes and promotes hepatic steatosis, further impairing liver function. Adiponectin–an insulin-sensitizing hormone produced in adipocytes–is integral to maintaining metabolic homeostasis and modulating hepatic SHBG production via the adiponectin-AMP-activated protein kinase (AMPK)-hepatocyte nuclear factor 4 alpha (HNF4α) signalling pathway. However, PCOS patients often exhibit decreased circulating adiponectin levels, which aggravates insulin resistance and hormonal imbalance. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are incretin-based therapeutics widely used in managing type 2 diabetes and obesity, known to improve insulin sensitivity and increase adiponectin secretion, suggesting potential therapeutic benefits in PCOS beyond glycemic control. This proposal examines the novel combined use of glucagon-like peptide-1 receptor agonists (GLP-1RAs) and adiponectin to synergistically increase hepatic SHBG production and reduce free androgen levels in PCOS. GLP-1RAs improve insulin sensitivity and upregulate adiponectin, both influencing SHBG expression. Using HepG2 hepatocytes, cells were treated with GLP-1RA, adiponectin, or both, measuring HNF4α and SHBG expression and free testosterone levels. It is hypothesized that combined treatment will enhance SHBG production more than single treatments and cause a greater decrease in free androgen levels. This study aims to establish a dual-therapy approach targeting the root metabolic dysfunctions of PCOS, providing groundwork for future liver-targeted delivery systems to improve treatment efficacy in endocrine and metabolic disorders.

Using Gut Microbiome and Hormonal Profiles to Predict Effective Therapies for Insulin-Resistant PCOS: A Machine Learning Approach

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Polycystic Ovarian Syndrome (PCOS) is a prevalent endocrine disorder, affecting up to 26% of reproductive-aged women, with insulin resistance (IR) playing a critical role in its pathogenesis. Current treatments, such as Metformin and GLP-1 receptor agonists, are quite general, often overlooking the complex biological diversity of PCOS patients, including distinct gastrointestinal microbiome compositions. Recent studies show associations between these gut microbial compositions and insulin signaling, particularly through short-chain fatty acid (SCFA) production, which modulates GLP-1 secretion and glucose level regulation by insulin. However, due to variation in gut microbiome profiles, especially among PCOS patients, there is a clear need for more personalized strategies. We propose a novel precision medicine approach that leverages artificial intelligence to prescribe personalized synbiotic therapies based on gastrointestinal microbiome and hormonal profiles. Using fecal sampling and shotgun metagenomic sequencing from PCOS patients, we will develop a machine learning model to develop precise synbiotics for specific gut bacteria compositions. Germ-free mice will be colonized with these microbiomes via fecal transplant and assigned to matched-pair trials comparing personalized synbiotics versus a general Diabetes medication, Metformin. Key outcome measures will include fasting insulin, glucose levels, butyrate levels, and GLP-1 expression. Through the incorporation of microbiome composition alongside targeted synbiotic formulations, our model aims to treat insulin disorders more effectively than current generalized therapies. This project has the potential to redefine PCOS treatment by reducing reliance on one-size-fits-all treatments and providing a scalable framework for treating other insulin-resistance conditions. This project and its implications seek to shift clinical practice toward technology-enhanced precision medicine in endocrinology treatment methods.

Neural-Network-Enabled Forecast of Alzheimer's Disease Integrating Prescient Neuroendocrine and Bioenergetic Blood Signatures

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Often referred to as "Type III diabetes," Alzheimer's disease (AD) is increasingly recognized as a condition rooted in endocrine and metabolic dysfunction, not purely late-stage neuropathology. While decades of research have focused on amyloid-β plaques and tau tangles, interventions that effectively target and reduce these hallmarks have largely failed to halt cognitive decline, suggesting that these features may be consequences rather than causes. This has prompted a critical shift toward upstream mechanisms, including hormonal imbalance, insulin resistance, and mitochondrial dysfunction, which may underlie AD's earliest stages. Our project aims to develop a predictive model for AD based on these endocrine and metabolic signals. We hypothesize that midlife disruptions, such as reduced estrogen post-menopause, impaired glucose metabolism, altered lipid profiles, and accelerated epigenetic aging, can serve as early biomarkers for progression to mild cognitive impairment (MCI) or AD. We will train a neural network using large-scale, longitudinal datasets (ADNI, NACC, ONDRI), integrating the following blood-based predictors for AD development: estradiol, FSH, LH, testosterone, lactate-to-pyruvate ratio, fasting insulin and glucose, targeted lipoproteins, and PhenoAge epigenetic age acceleration. Model performance will be assessed via ROC curve analyses, using area under the curve (AUC), sensitivity, specificity, positive predictive value, and negative predictive value as metrics of model efficacy. We will then validate the model in a five-year prospective study of 500 cognitively normal adults aged 45-70, using cognitive (MMSE, MoCA) and neuropsychiatric (MBI-C, NPI-Q) outcomes. By emphasizing the neuroendocrine-metabolic axis, this project reframes AD as a systemic disorder that begins long before plaques appear. If validated, our biomarker panel could support early, minimally invasive risk screening in at-risk populations, like perimenopausal women, those with AD risk genes, or individuals with insulin resistance. Such a tool would enable biology-guided interventions targeting metabolism and hormone regulation, offering a more proactive and personalized approach to AD prevention.

Utilizing Serum Thyroglobulin as a Functional Biomarker to Differentiate Struma Ovarii-Induced Hyperthyroidism from Primary Thyroid Gland Disorders: A Diagnostic Approach for Early Teratoma Detection and Growth Monitoring

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Struma ovarii is a rare ovarian teratoma composed primarily of thyroid tissue, which can become hormonally active and result in conditions that mimic primary hyperthyroidism. This overlap complicates diagnosis and may delay appropriate treatment. Traditional thyroid function tests and imaging often fail to distinguish between hypothyroidism arising from the thyroid gland and that caused by ectopic thyroid tissue. This study proposes the use of serum thyroglobulin (Tg), a protein secreted exclusively by thyroid follicular cells, as a functional biomarker to differentiate between these causes. We hypothesize that elevated serum Tg levels and suppressed TSH can indicate ectopic thyroid function originating from struma ovarii if there is Tg expression in ovarian tissue. To investigate this, a preclinical study using female Sprague-Dawley rats was designed with three groups: Group A (primary thyroid-induced hyperthyroidism via T4 injections), Group B (struma ovarii model), and Group C (healthy control). Hormonal profiles (T3, T4, TSH) and serum Tg levels will be monitored over 12 weeks, with histological analysis and Tg localization performed post-mortem. We expect that both Group A and B will display high T3/T4 and low TSH levels; however, Tg in Group A will originate from the thyroid gland, whereas Group B will show ectopic Tg expression in ovarian tissues. Group C is hypothesized to have normal hormone and Tg levels. The ability to trace Tg to its source may enable early, noninvasive detection of functional ovarian teratomas and better distinguish them from traditional thyroid disorders such as hyperthyroidism. This approach could significantly improve diagnostic precision, enable earlier intervention, and reduce unnecessary thyroid-targeted treatments in patients with struma ovarii-induced hyperthyroidism.

Multi-Air Pollutant Index and Machine Learning Model to Predict Thyroid Cancer Incidence

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Using publicly available county-level data from the U.S. Environmental Protection Agency (EPA) and State Cancer Profiles, we evaluated whether a novel, weighted "Cancer Risk Score," combining six air pollutants (PM2.5, PM10, O3, CO, SO2, NO2) via pollutant-specific weights (wi) derived from their Pearson r with thyroid cancer incidence and normalized through a sigmoid, can predict age-adjusted thyroid cancer rates. We hypothesized that this composite score would outperform any single pollutant metric and that integrating it into a hybrid random forest model alongside raw pollutant values would further enhance predictive accuracy. We calculated county-year pollutant averages (2008-2021), derived weights from univariable correlations between each standardized pollutant and 2017-2021 incidence rates, computed the Cancer Risk Score for each county, and then (1) assessed linear correlations (Pearson's r) for the score versus incidence and each pollutant versus incidence, and (2) trained a random forest regressor (80/20 train/test split) using both the score and raw pollutant features. Preliminary analyses indicate that the Cancer Risk Score yields a higher |r| than any individual pollutant (comparing max |r|), and the hybrid model achieves out-of-sample r and RMSE significantly better than a basic model, demonstrating the value of a weighted, normalized composite index for environmental risk mapping.

Investigating the Causal Mechanism of Rotavirus-Induced Type 1 Diabetes in Humanized Rat Models

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Type 1 diabetes (T1D) is an autoimmune disease that has consistently been correlated with rotavirus (RV) infection, but the exact mechanism of which remains unclear. There are three evidence-supported mechanisms: molecular mimicry between RV antigens and pancreatic β-cell proteins, bystander activation through RV-induced inflammation of accessory organs, and immune dysregulation of the gut by RV-induced permeability/microbiota changes. This study plans to systematically investigate these mechanisms in rodent models to further the understanding of RV-induced T1D. This study compares wild-type (WT) mice, RV-induced T1D (RV_T1D) mice, and non-obese diabetic (NOD) mice, which develop T1D spontaneously. Each of these groups will be assayed and compared through three different mechanisms of immune response (viral peptides for molecular mimicry, viral genome presence for bystander activation, and gut microbiota presence for gut dysregulation). These data will suggest the primary mechanism of RV-induced T1D and will support more specific medication/treatments for RV-infected patients. Overall, this study will clarify the effects of RV on T1D, but the generalizability to humans requires further research.

Development of PolyGone: A Textile Treatment for Endocrine Disrupting Chemicals Through Per- and Polyfluoroalkyl Substance Adsorption

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Clothing is central to daily life for every individual, yet the fabrics we wear often serve as an overlooked source of chemical exposure. To achieve qualities such as water-, stain-, and wrinkle-resistance, many textiles are treated with chemical finishes that come at the expense of human health. These finishes often contain endocrine-disrupting chemicals (EDCs), which interfere with hormonal pathways that are essential for the body. These pathways can include fertility, metabolism, immune response, cardiovascular health, neurological development and growth, and countless more. EDCs of particular concern are poly- and perfluoroalkyl substances (PFAS), widely used for performance textiles. Their persistent risk of dermal absorption highlights an urgent need for a consumer-accessible solution to reduce exposure. PolyGone is a single-use dryer sheet engineered to encapsulate PFAS during the drying cycle. The sheet features a layered structure: an outer cellulose mesh that promotes EDC desorption from fabrics; a middle nanocomposite layer of graphene oxide with β- and γ-cyclodextrins to capture both short- and long-chain PFAS through size-selective adsorption; and finally, a solvatochromic dye that provides a visible saturation indicator to determine when disposal is required. To evaluate its efficacy, PolyGone sheets will be used as a laundry additive for PFAS-treated fabrics under controlled conditions and compared against modified prototypes and

untreated controls. Post-drying analysis using liquid chromatography–mass spectrometry (LC-MS/MS) will quantify PFAS levels in tested fabrics, with success defined as ≥70% PFAS removal, along with a measurable colour change in the dye. By effectively removing these EDCs from textiles, PolyGone sheets offer a practical intervention to mitigate endocrine disruption risks linked to everyday fabric use. This technology also holds potential for broader applications in removing other harmful EDCs from textiles, further advancing public health protection.

The Effect of Vaginally Administered Melatonin on Luteinizing Hormone Levels in a Mouse Model of Polycystic Ovary Syndrome

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Polycystic ovary syndrome (PCOS) is a prevalent chronic endocrine disorder affecting women of reproductive age. A hallmark of PCOS is elevated levels of luteinizing hormone (LH), which disrupts ovulation and promotes excess androgen production. Studies suggest that the hormone melatonin may influence LH levels, and that women with PCOS exhibit lower follicular fluid melatonin than serum melatonin. While oral melatonin has been studied, vaginal melatonin delivery has not been explored, leading to the research question: Can vaginal administration of melatonin through a biodegradable insert reduce serum LH levels in a letrozole-induced mouse model of PCOS? This study proposes a preclinical, 21-day experiment using a letrozole-induced PCOS mouse model to evaluate whether vaginal melatonin administration can reduce serum LH levels. Female C57BL/6 mice (6 to 8 weeks old) will be divided into three groups (n = 10 per group): Group A (healthy controls receiving a vehicle insert), Group B (PCOS-induced with a vehicle insert), and Group C (PCOS-induced with a melatonin-loaded vaginal insert). The insert is composed of chitosan, a safe, biodegradable, and mucoadhesive polymer designed to release 10 mg/kg/day of melatonin gradually over 6 to 8 hours, mimicking natural nocturnal hormone release. Serum LH levels will be measured through ELISA on day 21. It is hypothesized that Group C will show significantly reduced LH levels compared to untreated PCOS controls (Group B), suggesting that localized melatonin delivery can help restore hormonal balance by decreasing elevated LH. This study introduces a novel, non-invasive, circadian-aligned hormone regulation method using biodegradable vaginal inserts. This method offers a therapeutic approach for PCOS that targets reproductive hormone balance with minimal expected side effects.

PathPatch: Real-Time Biosensor for Monitoring mTOR as a Derivative of Estrogen Signaling

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Endometriosis is a chronic, estrogen-dependent inflammatory disorder characterized through ectopic growth outside the uterus. Current treatments rely on surgery and are often costly and invasive that focus on symptoms rather than addressing drivers of disease. There is an urgent need for preventative and accessible monitoring tools to support early intervention in estrogen-driven conditions like endometriosis. Recent studies implicate the dysregulation of the PI3K/PTEN/AKT/mTOR signalling pathway in the pathogenesis of endometriosis. Hyperactivation of the mTORC1 components (e.g. mTOR) promotes cell proliferation and the eventual development of endometriosis. The proposed innovation, PathPatch, a wearable microneedle-based biosensor that monitors mTOR pathway activity by detecting phosphorylation through the downstream protein 4E-BP1 at Thr37 and 46. To achieve this, we will engineer a minimally invasive microneedle, coated with the phospho-specific antibodies (Phospho-4E-BP1) which will selectively bind to phosphorylated 4E-BP1 in interstitial fluid. The biochemical process will be transduced into an electrical signal which is then read by an electrochemical sensor. The PathPatch, connected to an external reader, displays to the users the mTOR activity status and provides insight into underlying estrogenic hormonal fluctuations. The PathPatch offers a convenient and patient-friendly solution for managing hormone-sensitive conditions. If the user detects elevated mTOR activity, PathPatch advises to consult a healthcare provider. Early action preventative measures include hormone-balancing interventions such as rapamycin to restore hormonal equilibrium.

Development of a Microneedle Biosensor for Continuous Monitoring of Sex Hormones in Hormone Replacement Therapy

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Hormone replacement therapy (HRT) is widely used in postmenopausal care and gender-affirming treatment, where accurate monitoring of sex hormones such as estrogens, progesterone, and testosterone is critical. Currently, clinical protocols rely on infrequent blood tests which may not effectively capture natural hormonal fluctuations caused by external factors like circadian rhythms, stress and medication interactions, increasing the risk of under- or overdosing, potentially leading to side effects such as blood clots, metabolic disturbances, or reduced treatment efficacy. This paper introduces a novel wearable biosensor patch that enables continuous hormone monitoring through minimally-invasive microneedles coated with molecularly imprinted polymers (MIPs). These synthetic receptors selectively bind to targeted sex hormones in the interstitial fluid resulting in a signal transduced by an integrated organic electrochemical transistor (OECT) into an electrical signal. This results in a signal transmitted via Bluetooth to a smartphone application for accurate, constant tracking. To evaluate the device's specificity and accuracy, an experimental protocol will test the biosensor against four sample types, including control and interference conditions. A successful outcome would show selective detection of only the target hormone in mixed or pure samples. If validated, this system would offer a powerful alternative to conventional blood testing by enabling continuous, non-invasive hormone tracking. It has the potential to improve the safety and effectiveness of HRT through individualized dosing adjustments, reduce side effects, and enhance patient outcomes. In the future, the platform may be expanded to monitor additional hormones and integrated into broader personalized health technologies.

The Development, Implementation and Evaluation of a Cortisol Solubilization Converting Device

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Cushing's syndrome is a rare yet dangerous endocrine disorder characterised by chronic exposure to elevated cortisol levels, often resulting in serious metabolic, cardiovascular, and cognitive complications. Despite its low prevalence, estimated at approximately 60 cases per million annually, its symptoms often overlap with obesity and stress-related conditions, leading to frequent misdiagnosis and delayed treatment. Current therapeutic options, such as transsphenoidal surgery, adrenalectomy, and radiotherapy, are effective but invasive, costly, and associated with long-term risks and recovery periods. CortiClear is a minimally invasive device designed to address these limitations by facilitating the selective solubilization of cortisol. The device is implanted in the hepatic portal vein, where it captures cortisol right outside the adrenal gland, where it is produced by using a binding layer of SULT2A1, an enzyme which detects cortisol and attracts cortisol. It then converts cortisol to its water-soluble, inactive form: cortisol sulfate through an enzymatic layer containing immobilised SULT2A1. This solubilization reduces the metabolic burden on the liver and allows the kidneys to excrete excess cortisol more efficiently. A pilot study involving 125 participants demonstrated a 52.9% average reduction in circulating cortisol over six weeks, indicating promising efficacy. Although limitations remain regarding population diversity and real-world testing, the device presents a novel, hormone-selective approach to treating Cushing's syndrome. Its success may also pave the way for broader applications in hormone regulation and endocrine health.

Closed-Loop Auricular Vagus Nerve Stimulation with Integrated Biosensing for Adaptive Hypothalamic Control of Hepatic Glucose Output, and Public Health Data Enhancement

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Type 2 diabetes, a chronic disease, has surged to record levels in recent decades, as a result of sedentary lifestyles, calorie-dense diets, and an aging population, it is one of today's most widespread and urgent health challenges. This paper introduces CARL (computer automated regulatory limb), a novel device integrating non-invasive transcutaneous auricular vagus nerve

stimulation (taVNS) therapy together with heart rate variability (HRV) and continuous glucose monitoring (CGM) to create a closed loop system. This will combat insulin resistance and impaired glucose tolerance (IGT), and thus, irregular hepatic glucose production (HGP) which are core drivers of type 2 diabetes. The paper furthers these impacts to a public health setting, leveraging data collected through biosensors to create publicly accessible data correlating vagus nerve stimulation with hepatic glucose output. Beyond pancreatic defects, central insulin signaling in the hypothalamus modulates HGP via autonomic efferents, including the vagus nerve. With novel taVNS therapy in a physiologically-informed closed loop, we aim to test its efficacy in restoring hypothalamic control of HGP by reducing adipose-hepatic inflammation through the cholinergic anti-inflammatory pathway, thus improving overall physiological insulin sensitivity. Isolated taVNS therapy has shown promise in prediabetes but mixed acute metabolic effects in healthy adults, underscoring the need for a comprehensive closed-loop design tailored to an individual's unique metabolic state. The expected outcome is proof that taVNS, tuned by both autonomic and glucose feedback, suppresses HGP, improves insulin sensitivity, and down-modulates inflammatory cytokines. The resultant impact would then be to establish an effective therapeutic blueprint to combat insulin resistance.

Development and Evaluation of Adaptive Music Therapy for Cortisol Modulation Using Bayesian Optimization

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Harmonix is a novel mobile application that elevates music therapy into precision endocrinology by combining Bayesian optimization with cortisol feedback from a collaborating wearable biosensor provider. Music interventions have been shown to reduce cortisol by 10–13% and improve mood and anxiety across diverse populations. In Harmonix, users complete ten rapid "music preference" trials, selecting between 10-second clips. After each choice, the app ingests the latest available cortisol measure via the MatrixBiotech secure API, updates its Gaussian Process model over acoustic-feature space (tempo, spectral flux, harmonic complexity), and recommends a list of songs. The paper further explains the feasible integration of Harmonix in existing biomarker tracking devices and software such as MatrixBiotech.

Improving Early Polycystic Ovary Syndrome (PCOS) Detection Through Rapid Urine-Based LH and FSH Ratio Screening via Lateral Flow Assay

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This paper introduces a novel all-in-one device, PCOSense, designed with a focus on affordability, ease-of-use and accuracy to support early screening for Polycystic Ovary Syndrome (PCOS). Despite being a common endocrine disorder affecting women of reproductive age, with 1.4 million affected in Canada alone, PCOS remains highly underdiagnosed. Delayed diagnosis may contribute to complications (i.e. risk of infertility and type diabetes), making timely lifestyle interventions crucial for symptom management and improving quality of life. PCOSense targets this diagnostic gap through a rapid, urine-based self-screening tool that simultaneously measures luteinizing hormone (LH) and follicle-stimulating hormone (FSH), two hormones often imbalanced in PCOS. The PCOSense integrates two lateral flow immunoassay strips into a single, user-friendly format that visually indicates LH/FSH ratios through colourimetric output. The goal is to assess whether PCOSense can differentiate PCOS-associated hormonal profiles (e.g. LH/FSH $\geq$2.0) from those within a healthy range (<1.5). To evaluate its potential, a preclinical experiment using an androgen-induced mouse model, which mimics hormonal disruptions in humans with PCOS. Urine samples from both PCOS-model and control mice will be analyzed using the device, and results will be validated against blood tests. Statistical analysis, including a paired t-test, will be conducted with a significance level of $p < 0.05$ to assess the screening accuracy of the device. All procedures involving animals will be reviewed and approved by an Institutional Animal Care and Use Committee (IACUC) to ensure ethical compliance. This foundational study will determine the validity of PCOSense as a low-cost, accessible first-line screening tool, designed to support early problem recognition and motivate timely medical consultation for individuals at risk of PCOS.

Quantum Biosensing of Hormones Using Nitrogen-Vacancy Centers in Nanodiamonds for Real-Time Monitoring

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Accurate and continuous monitoring of endocrine hormones is essential for the effective management of a range of chronic conditions, including reproductive hormone imbalances, metabolic syndromes, and cardiovascular diseases. These hormones often fluctuate rapidly in response to physiological changes, stress, medication, or environmental factors, making real-time monitoring critical for timely clinical decisions. Current diagnostic methods for hormone monitoring are primarily based on intermittent blood draws and centralized laboratory tests which are limited. They are invasive, slow, and provide only snapshot measurements, which delays feedback and hinders the personalization of treatment. These conventional approaches lack the resolution needed to capture changing hormonal changes, reducing their utility in dynamic or point-of-care clinical settings. Despite the clear clinical need, real-time biosensors capable of tracking hormone levels in biological fluids remain largely undeveloped. Major technical challenges persist, particularly in achieving the necessary sensitivity, selectivity, and stability in complex biological environments. Hormones typically exist in low concentrations and often share similar chemical structures, complicating efforts to detect them selectively without interference. This study explores whether nitrogen-vacancy (NV) centers in nanodiamonds can be leveraged to overcome these limitations through quantum biosensing. NV centers are atomic-scale defects in nanodiamond structures that exhibit spin-dependent optical properties, allowing for highly sensitive detection of local magnetic and electric field changes, as well as temperature variations. When functionalized with hormone-specific ligands, NV centers can report the presence and concentration of target hormones through changes in their spin state, detectable via optically detected magnetic resonance or fluorescence spectroscopy. The goal is to enable continuous endocrine level monitoring to advance personalized disease management and therapy optimization.

Integrative Selective Glucocorticoid Receptor Modulation for Long-Term Health Outcomes: Cortisol Profiling for Predominantly Inattentive ADHD and Respiratory Tract Infection Risk

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Predominantly Inattentive Attention-Deficit/Hyperactivity Disorder (PI-ADHD) is a neurodevelopmental condition linked to dysfunction of the hypothalamic–pituitary–adrenal (HPA) axis, resulting in chronic hypocortisolism and impaired inflammatory activity. This biological profile may contribute to attentional deficits but also to increased susceptibility to comorbid respiratory tract infections (RTIs) due to a lessened immune response (reduced inflammation). The study investigates the therapeutic potential of oral-dose Selective Glucocorticoid Receptor Modulators (SGRMs) in improving attention and immune function in children with PI-ADHD. SGRMs work to regulate the activity of glucocorticoid receptors (GRs). A randomized, double-blind, placebo-controlled trial will be conducted with 40 participants aged 7–18, diagnosed according to DSM-5 criteria. Participants will be randomly assigned to receive either 150–300 mg of Relacorilant (low-impact SGRM) or placebo daily for eight weeks. ADHD symptoms will be assessed with Conners' rating scales and computerized attention tasks. Immune function will be evaluated through inflammatory markers (TNF-α, IL-6), and RTI frequency will be tracked via caregiver diaries. Salivary cortisol will be re-measured at the midpoint and post-treatment to monitor HPA axis modulation. The target of SGRMs is the glucocorticoid receptors (GRs) to stabilize cortisol levels and demonstrate cortisol effects on enhancing attentional performance and reducing RTI risk (immune response). The paper examines the potential of SGRMs as a precision medicine alternative to traditional biologically-stressing stimulants, addressing the cognitive and immunological dimensions of PI-ADHD. This research may shed light on novel neuroendocrine pathways underlying ADHD and inform future treatment strategies that integrate hormonal and behavioural health. This study seeks to establish a neuroendocrine-based therapeutic strategy that addresses both attention deficits and immune vulnerability in PI-ADHD.

Classification of Estrogen-Receptor Endocrine Disrupting Chemicals Using Logistic Regression and SHAP

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Endocrine disrupting chemicals (EDCs) are substances that affect the human endocrine system by interfering with hormonal function. Among these, estrogen receptor (ER) EDCs are one of the most common types and showcase a significant concern as they directly interact with receptors ERα and ERβ. These compounds either bind to an estrogen ligand-binding domain or alter the estrogen-regulating genes, leading to significant disruptions in critical biological functions. This study hypothesizes that chemicals with high structural similarity to already known ER EDCs are also likely to exhibit similar effects to ER EDCs. The structural similarity between chemicals was investigated using a logistic regression machine learning model, which classifies chemical compounds as ER EDC or non-ER EDC. SMILES from the Endocrine Disruptor Knowledge Base and PubChem were numerically represented using Morgan fingerprints. These fingerprints were used to train a logistic regression model, setting 20% of the data aside for training, and achieving an overall accuracy of 93% in its ability to distinguish ER EDCs from non-ER EDCS. SHAP (SHapley Additive exPlanations) was retroactively applied to the model to identify which specific characteristics of a compound make it more likely to be classified as an ER EDC. Some limitations to the model created include the potential mislabeling of non-EDCs during the random selection of compounds from PubChem. Additionally, the loss of critical 3D molecular information during the conversion from SMILES to Morgan fingerprint could add to the inaccuracy of the model. However, some of the future applications of this model include integrating it into real-time environmental monitoring tools, particularly in regions with high chemical compositions to identify ER EDC. These tools could identify both known and unknown ER EDCs, helping protect public health. This study highlights the potential of structural similarity modeling and interpretable machine learning to predict the classification of untested chemical compounds.

Utilization of Supervised Machine Learning for the Treatment of Psychological Comorbidities in Hashimoto's Thyroiditis Patients

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Hashimoto's thyroiditis is an autoimmune condition and the leading cause of hypothyroidism in Canada, affecting approximately 10% of the population. While standard levothyroxine therapy aims to restore thyroid hormone levels, over 40% of patients continue to experience psychological comorbidities, despite normalized biochemical markers. This disconnect reveals a gap where conventional models often exclude psychological ramifications and fail to provide holistic health care. This approach addresses this gap through a supervised machine learning model, consisting of a Random Forest model trained on national primary care databases. This includes free thyroid hormones (T3 and T4), thyroid-stimulating hormone (TSH), cortisol, and patient-reported mental health validations. The model is designed to predict patients at an increased risk of psychological distress to support a medical practitioner's approach to treatment. A correlation heatmap generated from simulated datasets (MMASH and PLCO) revealed strong associations between key biomarkers of Hashimoto's thyroiditis and symptoms of psychological comorbidity (e.g., TSH and depression, $r \approx 0.95$; HRV and emotional distress, $r \approx -0.97$), informing feature selection and feedback design. This patient-informed system integrates real-time hormone monitoring with self-reported psychological data, creating a dynamic, closed-loop feedback framework. It supports clinicians in providing more holistic treatments to Hashimoto's thyroiditis by considering the relationship with psychological comorbidities that are commonly overlooked. Outcome evaluation will involve standardized mental health validation tools (e.g., BDI-II, GAD-7, SF-36), changes in biomarkers, and clinician observations to assess improvements over traditional care. Integrated mental health care bridges a longstanding divide between mental and endocrine health by utilizing chemical biomarkers and psychological reports to offer holistic treatment. The proposed model represents a scalable and adaptive strategy to evaluate the implications of hormone replacement and accompanying mental health outcomes in patients with Hashimoto's thyroiditis.

Utilization of Chaperone Assisted Stem Cells in Treatment of Congenital Hypothyroidism brought on by Thyroid Dysgenesis

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Congenital hypopituitarism represents a rare global health burden, affecting approximately 1 in 4000 newborns and contributing to a range of physiological and developmental complications. Common genetic causes include mutations in the PROP1 or POU1F1 genes, both of which act as regulators of pituitary development. This proposal presents a novel therapeutic strategy that employs chaperone-assisted induced pluripotent stem cells (iPSCs) to restore pituitary function in cases of pituitary developmental failure. Patient-derived iPSCs possess the ability to differentiate into any cell type, including those of the pituitary lineage. In this approach, iPSCs are co-transplanted with supportive chaperone cells that facilitate integration within the hypothalamic microenvironment and promote vascularization, neuroendocrine signaling, and hormone responsiveness. Therapeutic efficacy will be assessed by evaluating the expression of PROP1 and POU1F1, along with the detection of essential pituitary hormones, following treatment. The strategy will be tested through both ex vivo and in vivo experiments. Initially, iPSCs will be co-cultured in a brain slice model using pituitary-removed mouse tissue to assess preliminary gene expression. Subsequently, chaperone-assisted iPSCs will be transplanted into a PROP1 and POU1F1 deficient mouse model. Restoration of gene expression and detection of pituitary-derived hormones in the bloodstream will serve as outcome measures. A positive correlation between the intervention and hormonal restoration would support the therapeutic potential of this approach for treating congenital hypopituitarism and its associated endocrine deficiencies.

Conflicts of Interest
The authors declare that they have no conflict of interests.

Authors' Contributions
AL: Co-founder of LabPath, served as a planning committee for the conference, drafted the conference abstract booklet, and gave final approval of the version to be published.
KM: Co-founder of LabPath, served as a planning committee for the conference, drafted the conference abstract booklet, and gave final approval of the version to be published.

Acknowledgements
We would like to thank the LabPath executive team for their contribution to the successful competition of the first LabPath research hackathon. Special thanks to our esteemed panel of judges for evaluating all abstract and manuscripts. Judges consisted of graduate students, medical students, postdoctoral fellows, and doctors: Alex Pham, Laura Wang, Dr. Edwige Martin-Valiente, Dr. Qiujiang Du, Kristen Kyone, Dr. Amish Parikh, Teagan Haggerty, Dr. Victoria Lorant, Shania Sheth, Siddharth Seth, and Rohaan Syan. Finally, we would like to thank all hackathon delegates for their attendance to our event.

Funding
Funded by the RAYI Microgrant supported by The Canadian Center for Community Advancement (3CA) & Canada Service Corps; The Ingenious+ grant supported by the Rideau Hall Foundation; The CanConnect microgrant supported by GLOCAL.

Article Information
Managing Editor: Jeremy Y. Ng
Article Dates: Received Nov 01 25; Published Dec 01 25

Citation
Please cite this article as follows:
Lumb A, Mendapara K. Labpath Research Hackathon 2025: Hormones and metabolic disorders. URNCST Journal. 2025 Dec 01: 9(11). https://urncst.com/index.php/urncst/article/view/998
DOI Link: https://doi.org/10.26685/urncst.998