

HLixCJUR | Special Edition

Centre for Heart Lung Innovation Research Day 2025



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UBC and St. Paul's Hospital

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CANADIAN JOURNAL *of* UNDERGRADUATE RESEARCH



HLIxCJUR: Centre for Heart Lung Innovation Research Day 2025

Special Edition

January 2026

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This issue is published on the traditional, ancestral, and unceded territory of the Coast Salish Nations, including x^wməθk^wəyəm (Musqueam), S_kwxwú7mesh (Squamish), and səliiwətał (Tsleil-Waututh).

For inquiries about the Trainee Association at the Centre for Heart Lung Innovation, please address correspondence to trainees@hli.ubc.ca. For inquiries about the Canadian Journal of Undergraduate Research, please contact cjur.uro@gmail.com.

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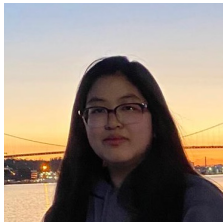
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The Trainee Association at the Centre for Heart Lung Innovation (TAHLI)

The Centre for Heart Lung Innovation (HLI) is the largest translational research centre within Providence Health Care's St. Paul's Hospital, a teaching hospital of The University of British Columbia.

The Trainee Association at HLI (TAHLI)'s vision is to foster the next generation of world-leading cardiopulmonary scientists. Our mission is to enrich the academic experience of HLI trainees by cultivating a collaborative training environment that promotes education, professional development, and career advancement. TAHLI organizes a variety of professional development and mentorship initiatives throughout the year, including an annual HLI Research Day.

Held each summer, the HLI Research Day highlights the exceptional research conducted across the centre, with a special emphasis on the work of our trainees, from summer students to postdoctoral fellows. The program features oral presentations, poster sessions, and knowledge translation activities, with top presentations recognized through awards and prizes. This year, 26 undergraduate students and 28 graduate students presented their work to an audience of 140 attendees.

This event would not have been possible without the invaluable support of our community. We would like to give our sincere thanks to our generous sponsors, including Merck, AstraZeneca, St. Paul's Foundation, and Providence Health Research. We would also like to thank the HLI investigators, staff, and trainees whose behind-the-scenes efforts made this day a success.



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Understanding Viral Protease-Mediated Complement Dysregulation in CVB3-Induced Myocarditis

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Myocarditis, inflammation of the heart muscle, accounts for up to 20% of deaths among young adults, with higher prevalence in males. Viral infections, such as coxsackievirus B3 (CVB3), are the leading cause. Viral myocarditis can induce immune-mediated myocardium damage. The complement system, a key innate immune response, removes pathogens through inflammation, opsonization, and membrane attack complex (MAC) formation. Complement component 3 (C3) drives early complement activation, while regulator CD55 prevents excessive complement-mediated activation. Prior studies showed that viral proteases can cleave C3 to evade immunity, and that CVB3 protease 3C cleaves host factors to disrupt immune defenses. However, the impact of CVB3 on complement regulation remains underexplored. We hypothesize that CVB3 downregulates CD55 through cleavage by viral protease 3C. Complement activation was assessed by immunohistochemical staining of MAC in human myocarditis cardiac tissue and controls (n=3 per group). CD55 levels were quantified in sham and infected mouse cardiomyocytes and tissue by immunoblotting. Putative 3C cleavage sites were predicted in CD55 via bioinformatics. In-vitro cleavage assays were performed using 3C with CD55 decreased in mouse cardiomyocytes 12 hours post-infection and in cardiac tissue 7 days post-infection. Cleavage assays showed that CD55 and C3 are cleaved by 3C. These findings suggest that CVB3 activates complement while suppressing regulators via 3C cleavage, potentially driving inflammation and immune-mediated damage. Future studies will confirm cleavage sites and downstream consequences.

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Real-World Effectiveness of Biologics in Patients with Severe Asthma: A Single-Center Registry Study at St. Paul's Hospital, Vancouver, BC

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Biologic treatments improve clinical outcomes in asthma by targeting pathways involved in the immune response, leading to long-term disease control. While clinical trials have demonstrated the efficacy of biologics, real-world data remain limited. This study aims to evaluate the real-world effectiveness of biologics in patients with severe asthma using data from a single-centre registry at St. Paul's Hospital in Vancouver, BC. We hypothesize that patients on biologics will show greater improvements in lung function, measured by forced expiratory volume in one second (FEV1), and reductions in airway inflammation, measured by blood eosinophil counts (BEC), compared to those not receiving biologics. We reviewed the medical records of patients treated with omalizumab (anti-IgE), mepolizumab (anti-IL-5), and benralizumab (anti-IL-5 receptor). Variables were assessed at baseline and at 1, 3, and 5 years after biologic initiation. Among 196 patients (mean age 60.9 years; mean BMI 28.9; 43% male; 97% non-smokers), 39.2% received mepolizumab, 40.3% omalizumab, and 20.4% benralizumab. A control group of 50 patients (mean age 58.1 years; mean BMI 28.8; 38% male; 95.9% non-smokers) was included for comparison. Over the five-year follow-up period, FEV1 improved significantly in patients treated with mepolizumab and benralizumab, with no significant changes observed in the control or omalizumab groups. Changes in BEC showed significant decreasing trends over time in all three biologic treatment groups. These findings highlight the long-term effectiveness of biologics and provide valuable single-centre, real-world evidence from St. Paul's Hospital to support their continued use in asthma management.

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Black Carbon Airway Macrophages in Cannabis Smokers: Relation to Worse Respiratory Symptoms and Lung Function

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The growing popularity of cannabis smoking in an era of legalization has raised concerns about long-term respiratory health effects. This study aimed to evaluate whether cannabis smoking is associated with increased black carbon (BC) accumulation in airway macrophages (AM) and whether this relates to respiratory symptoms. We hypothesized that black carbon (BC) accumulation in airway macrophages (AM) is positively associated with cannabis joint year (JY) exposure and worse respiratory symptoms. Bronchoalveolar lavage (BAL) samples, which primarily contain AM, were collected from cannabis smokers and non-smoking controls during research bronchoscopies. Participants with active asthma, COPD, bronchiectasis, or lung cancer were excluded. Saline was instilled in the right middle lobe or lingula, from which cell pellets from BAL samples were processed into cytopsin slides, stained, digitally imaged, and analyzed to quantify average BC area (μm^2) and average BC percentage per macrophage. BC content was compared against cannabis joint years (JY), St. George's Respiratory Questionnaire (SGRQ), and COPD Assessment Test (CAT) scores. The study included 41 cannabis smokers (mean age 33.6 ± 12.5 years, 22M/19F) and 9 controls (mean age 34.2 ± 10.7 years, 3M/6F). The mean JY among cannabis smokers was 19.2. Preliminary analysis found no significant correlation between BC content and joint year exposure, SGRQ, or CAT scores. These findings suggest BC accumulation in AM may not be as prominent in cannabis smokers as reported in tobacco smokers. However, it should be noted that because our cohort was young with relatively low exposure, further longitudinal studies with higher exposure populations are needed to clarify the relationship between cannabis smoking, AM BC content, and respiratory health.

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Uncovering the Role of Membrane-Associated Viral Proteins in Inducing Mitochondrial Damage in Viral Myocarditis

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Myocarditis, characterized by inflammatory infiltration of the myocardium, is a leading cause of sudden death in young people, with approximately 1.5 million cases annually. Viral infection is the most prevalent infectious cause, with coxsackievirus B3 (CVB3) commonly used as a preclinical model to study viral myocarditis. Our preliminary data revealed mitochondrial damage in CVB3-induced myocarditis. Mitochondria play a crucial role in various biological functions, and impaired mitochondria lead to decreased energy production, elevated reactive oxygen species (ROS) levels, and activation of mitophagy, a process of recycling damaged mitochondria. However, the precise mechanism of virus-induced mitochondrial damage remains unclear. Given the double-membrane structure of mitochondria, membrane-associated viral proteins are hypothesized to interact directly with mitochondrial membranes. This study aims to investigate how CVB3 disrupts mitochondrial integrity and how mitochondrial dysfunction contributes to viral propagation. Specifically, we will examine the roles of membrane-associated viral proteins 3A-3D in mitochondrial damage and evaluate the potential of MitoQ, a mitochondrial ROS-targeted antioxidant, to mitigate viral propagation. Our investigation confirmed the localization of 3A-3D to the mitochondrial membrane during CVB3 infection. These findings uncovered a synergistic impairment of mitochondrial function due to the overexpression of 3A-3D, leading to loss of mitochondrial network and consequent mitochondrial dysfunction, including diminished mitochondrial potential and mislocalization of mitochondrial DNA. Additionally, ROS inhibition with MitoQ impaired viral propagation, suggesting that mitochondria-derived ROS benefits CVB3 infection. In conclusion, CVB3 membrane-associated viral proteins 3A-3D localize to mitochondria and disrupt mitochondrial functions. Dysregulation of ROS can contribute to viral propagation.

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