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Memo

To: CAAIF Membership
From: Andrea Waserman, Managing Director, CAAIF
Date: November 8, 2023
Re: **2023 CAAIF Research Grants and Fellowships**

BACKGROUND:
Thus far, together with our partners, CAAIF has distributed $832,215 this year with another $300,000 to be distributed by the end of the year totally $1,132,215 in research funds in 2023. Below is a list of all Awards that have been distributed thus far.

ALLERGIC AIRWAYS DISEASE INNOVATION GRANT

Awardee: Dr. Paul Forsythe, University of Alberta
Project Title: *Pulmonary Neuroendocrine Cells as targets for gene therapy in asthma*
Amount: $84,940
Lay Summary: This study focuses on specialized cells in the lung called Pulmonary Neuroendocrine Cells (PNEC). PNEC are found in the surface lining of the lung (epithelium) and are very rare (less than 1% of cells in the epithelium) but play an important role in detecting changes in the content of the air we breathe, allowing the lungs to respond and maintain healthy function. Recent studies, in mice, suggest that PNEC play a key role in the development of asthma and enhance the severity of asthma symptoms. Our preliminary work shows that, when exposed to allergens, human PNEC release Calcitonin Gene Related Peptide (CGRP), a neuropeptide known to enhance airway inflammation. By modifying CGRP gene expression in PNEC we may be able to control airway inflammation associated with asthma. Our study will determine if PNEC are suitable and meaningful targets for inhaled gene therapies. We will use stem cell derived human PNEC to develop lipid nanoparticle delivered gene therapy that can supress production of CGRP. We will then test the efficacy of PNEC targeted gene therapy in a preclinical model of asthma. If successful, this project will be the first stage in the development of an entirely new approach to treating asthma.

ALLERGIC AIRWAYS DISEASE INNOVATION GRANT

Awardee: Dr. Bruce Mazer, McGill University
Project Title: *Mitigation of Type 2 Asthmatic Inflammation using B-cell derived Extracellular Vesicles*
Amount: $84,400
Lay Summary: Extracellular vesicles (EVs) are produced by a wide variety of cells under many conditions, and their potential as intercellular communication molecules is an emerging area of study. There is paucity of work in understanding EVs in allergic diseases, how they contribute to the regulation of immune inflammation, and ultimately how EVs may

be harnessed to address a therapeutic need for severe allergic airways disease. We have found in a mouse model of asthma that B-lymphocytes, stimulated under conditions that produce IgE, release large amounts of EVs that that appear to inhibit important facets of allergic inflammation, including eosinophil growth and inflammation in the airways of allergic mice.

Our CAAIF funded project will address for the first time the production of EVs using human B-cells and demonstrate their effects on the key asthma-related inflammatory cell, the eosinophil. We will determine the cellular and molecular impact of B-cell-derived EVs on eosinophil growth, maturation and function. B-cell derived EVs have extreme promise as a source of nanoparticles that can be delivered to the airways by aerosol and can inhibit eosinophil growth and other facets of Th2 inflammation via the expression of key surface receptors and inhibitory genetic material.

ALLERGIC AIRWAYS DISEASE INNOVATION GRANT

Awardee: Dr. Channakeshava Sokke Umeshappa, Dalhousie University
Project Title: *Development of a novel, targeted, cell-based immunotherapeutic drug for asthma*
Amount: $85,000
Lay Summary: The overall objective of this proposal is to develop a cell-based immunotherapeutic drug of ground-breaking significance for severe asthma.

There are only a few specific drugs, unlike we have for bacterial infections, for asthma, perhaps because of the complex immune mechanisms involved. Severe asthma is treated generally by non-specific immunosuppressants such as steroids that suppress the whole immune system that is otherwise necessary for defending our body. Consequently, steroid therapy can adversely affect our immune system, increasing the risk of lung infections.

Asthma is caused by small particles present in the environment called allergens. Certain white blood cells respond to these allergens, become dangerous and damage airways. We will develop a cell-based anti-asthma treatment, which will kill these harmful white blood cells that respond to allergens, a long-sought-after goal in severe asthma therapy. We will test our drug's therapeutic efficacy and safety using a well-established animal model of asthma, which facilitates our subsequent clinical translation of this therapeutic platform.

The research will be pursued with a leading-edge interdisciplinary team of established scientists, clinicians, and trainees spanning the spectrum of synthetic biology, bioinformatics, and immunology.

By restoring normal lung functions in a disease-specific manner, without causing general immune suppression, our treatment should prevent asthma patients from getting chronic breathing problems and secondary infections, making Canada and the World a healthier place to live.

CAAIF RESEARCH FELLOWSHIP IN IMMUNODEFICIENCY SUPPORTED BY TAKEDA CANADA

Awardee: Dr. Vanessa Polito, McGill University
Project Title: *Phenotypic and genotypic characterization of hyper IgE immune dysregulation*
Amount: $50,000
Lay Summary: Immune dysregulation is a broad term that encompasses dysfunction of the immune system. This includes recurrent and severe infections, but also malignancy, lymphoproliferative disease, autoimmune disease, and allergic inflammation or atopy. Patients with severe atopy are perhaps the most underrecognized manifestation of potentially important immune dysregulation, which may be caused by an underlying inborn error of immunity (IEI). Some of the most well-known IEIs with severe atopy are the hyper-IgE syndromes, which manifest with eczema, significantly elevated IgE, and skin or pulmonary infections. In order to better understand the heterogeneous population of patients with high IgE, we need to better understand how to group these patients based on their clinical presentations, identify 'red flag' features that should prompt further investigation, and continue to identify genetic mutations that underlie these conditions. Our study aims to identify key phenotypes of patients with hyper IgE-like presentations, with or without infections, categorize their clinical and laboratory presentations, and identify key genetic mutations using Whole Exome Sequencing. Identifying genetic mutations will allow for better understanding of the mechanisms of these diseases and eventually contribute to precision medicine. Finally, we hope to elaborate on the bioethical issues that intersect genetics research and patients with immune dysregulation.

CASP-CAAIF RESEARCH GRANT IN HEREDITARY ANGIOEDEMA (HAE)

Awardee: Dr. Stephen Betschel, University of Toronto
Project Title*: Canadian physician hereditary angioedema management practice pattern survey*
Amount: $50,000
Lay Summary: Hereditary angioedema (HAE) is a rare but serious and potentially life-threatening condition due to random and unpredictable swellings involving extremities, face, abdomen, and the upper airway. This condition is associated with a significant impairment of quality of life. There have been significant advancements to prevent attacks and treat attacks when they occur. Current guidelines recommend an approach that aims to normalize patients' lives. The Canadian Physician Hereditary Angioedema Practice Pattern Survey is intended to better understand how physicians in Canada manage HAE patients and if guideline recommendations are followed. The goal of the study is to identify any potential care gaps so the future versions of guidelines can address barriers that might interfere with optimizing care for HAE patients.
This study will be conducted across Canada by reaching out to physicians to treat HAE through the Canadian Hereditary Angioedema Network (CHAEN). The study consists of a survey that physicians will complete, as well as a qualitative interview process, to better understand potential barriers that physicians may face in trying to optimize goals of HAE treatment. This will be the first study that combines both a survey and an interview format to assess physician practice patterns related to HAE patient care.

CAAIF-SEARCHLIGHT PHARMA RESEARCH GRANT IN UPPER AIRWAY ALLERGIC DISEASE

Awardee: Dr. Chris Carlsten, University of British Columbia
Project Title *Effects of common air pollutants on allergic rhinitis: A controlled human exposure study*

Amount: $50,000
Lay Summary: Allergic rhinitis (AR) affects 1 in 4 Canadians, with impacts on their well-being and productivity. It is closely linked to asthma, and both conditions are aggravated by air pollution, causing worsened symptoms and reduced quality of life. Traffic-related air pollution (TRAP) and wood smoke (WS) are major contributors to Canada's outdoor air pollution. Diesel exhaust (DE - model of TRAP) and WS are known to have adverse health effects, including respiratory and cardiovascular issues. However, no human studies have compared the harm caused by the two. Our research aims to address this gap, as we cannot assume that WS has the same effects as DE and must form guidance specific to WS.

We will conduct a randomized, double-blinded, crossover trial to examine how DE and WS exposure affects airway function in individuals. This study will look into the impacts of DE and WS on airway function, especially those with allergic rhinitis. Such data is essential amidst growing support, driven by economic and government initiatives, for transitioning diesel generators to wood pellet systems in rural, remote, and indigenous communities. The findings from this research could contribute to developing exacerbation prevention strategies, inform government policies, and shape treatment approaches for rhinitis.

CAAIF-CLA RESEARCH FELLOWSHIP

Awardee: Dr. Clarus Leung, University of British Columbia
Project Title*: Endo-phenotyping of Asthma and Chronic Obstructive Pulmonary Disease Overlap by Airway Inflammation and Structure*
Amount: $33,000
Lay Summary: Chronic obstructive pulmonary disease (COPD) and asthma are two different diseases that affect the airways. Around one-third of COPD and asthma patients have features of both asthma and COPD and thus are diagnostically labeled as asthma-COPD overlap (ACO). ACO patients experience worse symptoms and more serious respiratory attacks than those with COPD or asthma alone, but we do not know why. To address these questions, our study will investigate the underlying inflammatory mechanisms in the airways of patients with ACO. We will collect tissue samples and cells from the airways of volunteers with ACO using a technique called bronchoscopy and perform genomics on these samples. These data will enable us to identify the key airway features of ACO. We will also use this cohort to determine which features of ACO lead to a good therapeutic response from inhaled corticosteroids, a class of medications used in COPD and asthma. We will use high-resolution imaging techniques to investigate how inflammation relates to persistent changes in the structure of the airways and the lungs.

Our research will reveal the disease mechanisms of ACO so that we can better diagnose and prescribe the most effective therapies for ACO in clinical practice.

CAAIF-CLA ALLIED HEALTH RESEARCH GRANT

Awardee: Shirley Quach, The Hospital for Sick Children
Project Title: *Does the use of biologics in children with severe asthma improve health outcomes and quality of life? – A longitudinal cohort study*
Amount: $10,000
Lay Summary: Asthma is the most common chronic disease in childhood, with about 2 to 5% of children with severe asthma reporting poor quality of life. "Asthma biologics" new injectable medications, may be beneficial in certain groups of children with severe asthma, improving their overall lung health. But asthma biologics is estimated to cost at least $8,000 per patient each year, a heavy price shared amongst patients, their families, and the healthcare system. Past studies focused on the use of asthma biologics in adults, with less attention on children and adolescents.
Health data before and after asthma biologics will be collected, reviewed and compared in children with asthma, aged 12 to 18 years, from 2014 to 2022, from the Hospital of Sick Children's health database. We will use different statistical tests to see if there are any short- or long-term trends after different asthma biologics.
It is important for healthcare providers and policymakers to understand the cost and benefits of asthma biologics to help guide practices, optimizing their use in children with severe asthma. It will also highlight how asthma biologics impact children and their families, directing future education and research to support their lung health and management.

CAAIF-IMMUNODEFICIENCY CANADA RESEARCH FELLOW IN IMMUNODEFICIENCY
Awardee: Dr. Ori Scott, The Hospital for Sick Children
Project Title: *Elucidating pathophysiology of auto-inflammatory disease mechanisms in the inborn error of immunity STAT1 gain-of-function*
Amount: $20,000
Lay Summary: STAT1 is a gene that plays a critical role in the immune response against infections, and in particular against viruses. Because STAT1 is such an important gene, its activity is tightly regulated in most healthy people. STAT1 gain-of-function (GOF) is a genetic primary immunodeficiency, which is caused by increased activity of STAT1. The most common symptoms in patients with STAT1 GOF IS chronic fungal infections, which are typically not life threatening. However, some STAT1 GOF patients also develop severe symptoms, such as lethal viral infections, or severe autoimmunity/auto-inflammation. In this work, we will use a mouse model to study what causes the development of such severe disease manifestations in STAT1 GOF. Importantly, STAT1 activity can be disrupted even in people who do not have STAT1 GOF. For this reason, by researching STAT1 GOF, we can learn a lot about the development of severe infections, autoimmunity or inflammation even in people without STAT1 GOF.

GRADUATE STUDENT AWARDS IN ASTHMA

Awardee: Courtney Hoskinson, PhD Candidate, Western University
Project Title: *Data from the CHILD cohort study: functionally linking the early-life gut microbiome to health and disease*
Amount: $30,000
Lay Summary: Allergic diseases affect hundreds of millions of children worldwide and continue to increase in prevalence. Many risk factors for allergic diseases, such as

antibiotic usage, also influence microbes and their genes within the gut, which, together, are commonly known as the gut microbiome. Maturation of the gut microbiome usually occurs at the same time as the development of healthy immune tolerance. However, if microbiome maturation is abnormal, allergic sensitization can emerge in some children as a result.

My research combines school-age allergic diagnoses with early-life gut microbiome composition, functional capability, and metabolite concentrations for the quantification of a 'normally' maturing gut microbiome. This data primarily stems from CHILD (n=3,455), a large Canadian longitudinal study with robust information on participant environment and microbiome. To increase the strength and relevance of my findings, I will not only identify associations in CHILD, but I am also working with my colleagues and collaborators to validate our findings in other populations with clinical and microbiome data, such as the Copenhagen Prospective Study on Asthma in Childhood (COPSAC).

The aims of my research are to (1) identify unifying gut microbiome maturation signatures in asthma, allergic rhinitis, food allergy, and atopic dermatitis, collectively called the 'Allergic March', (2) functionally link antibiotic usage to the onset of specific allergic diseases using microbiome data, and (3) connect microbial-dependent influences on participant immune cell profiles to allergy. My investigation of the early-life gut microbiome will thus empower new predictive and preventive strategies to avoid allergic diseases.

GRADUATE STUDENT AWARDS IN ASTHMA

Awardee: Natasha Kunchur, PhD Candidate, Carleton University
Project Title: *Mapping airway remodelling in asthma using multimodal Raman-Second Harmonic Generation imaging and machine learning*
Amount: $30,000
Lay Summary: Asthma is a chronic inflammatory disease, impacting approximately 11% of the Canadian population. Inhaled allergens damage the tissue barrier lining the lungs, leading to the inflammation of airways and difficulties in breathing. To remodel impaired tissue, damaged airways trigger a complex cellular response, denoted by the excessive accumulation of extracellular matrix (ECM) proteins; particularly collagen I. Evidence suggests that this fibrotic response, known as subepithelial fibrosis (SF), contributes to tissue stiffening, airway blockage and an overall reduction in lung function.

With the goal of setting a new precedence for imaging and to better visualize ECM protein deposition at high resolution, our research applies a label-free multimodal imaging system embedded with the technologies of both Raman microspectroscopy and Second Harmonic Generation. This imaging system is the only of its kind in Canada, and is used to develop biochemical maps of tissues and cells while simultaneously detecting signals related to fibrillar collagen. Due to the complex nature of the data obtained, the development of novel approaches based on machine learning (ML) strategies to identify biomarkers associated with asthmatic airway remodelling is necessitated. Using ML, an automated classification pipeline will be developed to characterize spectral signatures unique to the basement membrane, epithelium and lamina propria of airways.

Insight into the fibrotic responses in asthma with an unprecedented level of spatial and biochemical specificity will drive the identification of biomarkers active in disruptive airway remodelling and support therapeutic development minimizing the formation of scar tissue observed through the excessive burden of SF.

GRADUATE STUDENT AWARDS IN ASTHMA

Awardee: Courtney Marshall, PhD Candidate, University of Manitoba
Project Title:*Sex as a biological variable in immunomodulation of airway inflammation by Innate Defence Regulator (IDR) peptides*
Amount: $30,000
Lay Summary: Asthma is the most common chronic respiratory disease affecting nearly 3 million Canadians including children. Around 15% patients do not respond to available steroid therapies and represent the major burden of asthma accounting for annual healthcare costs of $2B. Also, common steroid therapies can increase the risk of lung infections, which can make asthma worse. New therapies are urgently needed that can alleviate steroid-unresponsive disease without compromising the ability to resolve infections.

There is a clear sex bias in asthma, for example adult females experience greater disease severity and are more likely to develop steroid-resistance, compared to males. These sex-related differences are largely ignored during drug development. Effective development of new treatments must consider the differences in disease and response to therapy between females and males.

This study focuses on new molecules known as innate defence regulator (IDR) peptides, which can control both inflammation and infection. We have shown that IDR peptides improve breathing capacity in an animal model of asthma, and control cellular processes linked to steroid unresponsiveness. This project aims to develop IDR peptides as a new therapy for asthma, by examining the effects in both females and males concurrently. This research will directly support the development of a new IDR peptide-based therapy for asthma, by taking into consideration how the treatment affects females compared to males. It is entirely possible that we will need to develop sex-specific treatment protocols to provide the most efficient care for asthma sufferers.

GRADUATE STUDENT AWARDS IN ASTHMA

Awardee: Jo-Chiao Wang, PhD Candidate, University of Montreal
Project Title*: Basophilic oncostatin M fuels nociceptor neuron-induced asthma*
Amount: $30,000
Lay Summary: Despite affecting less than 10% of asthmatic patients, severe asthma accounts for 60% of the asthma healthcare cost due to the lack response to corticosteroid treatments. Recent advance of single-cell gene profiling reveals a population of airway sensory neurons expressing similar genes as neurons sensing skin itch. However, molecular and pharmacological characterizations of this population are insufficient. With real-time calcium imaging, we can visualize the calcium influx, a neuronal activation event, in response to different stimulants. We can thus cluster the neurons based on their reactivity to different drugs as well as evaluate neuron's sensitivity under normal and asthmatic condition.

We have thus far demonstrated that lysophosphatidic acid-responding neurons and serotonin-responding neurons are two distinct populations, which respectively represent jugular and nodose nociceptors in vagal sensory ganglia, from where the airway sensory neurons arise.

We also noticed that some jugular neurons express the receptor of oncostatin M (OSM), a cytokine associated with exaggerated itch sensation in atopic diseases. We then ask if OSM is expressed in asthmatic context and by which cells. With cell sorting and RT-qPCR

techniques, we identified lung basophils as the main cellular source of OSM under normal and asthmatic conditions. As it sensitizes itch neurons, OSM can also sensitize vagal sensory neurons, shown by our calcium imaging data.

Whether OSM affects asthmatic pathophysiology through sensitizing airway sensory neurons is yet to be determined. We hope this novel neuroimmunology pathway provides a new possibility in seeking alternatives to glucocorticoid treatment.

GRADUATE STUDENT AWARDS IN ASTHMA

Awardee: Anam Ara, MSc Candidate, University of Manotiba
Project Title:*DNA methylation changes induced by prenatal cannabis exposure associated with asthma in mice*
Amount: $15,000
Lay Summary: Since legalization, there has been an increase in the use of cannabis in Canada, but worryingly there are few clear rules for its use and safety during pregnancy. Exposure to inhaled particulates in early life from other sources, including wildfires, tobacco smoke, or pollution, have known health outcomes leading to risk of asthma. Early life cannabis exposure has been studied extensively in the brain, but no clear evidence is available for respiratory conditions like asthma. We plan to examine the molecular effects of prenatal and early life cannabis smoke on asthma development by examining epigenetics, or change in DNA function without change in DNA sequence. Specifically, this project will use DNA methylation (DNAm) as it is the best characterized.

We know prenatal exposure to some environmental pollutants including tobacco smoke causes changes in DNAm, but we do not yet know exactly how these changes cause lung problems like asthma. We will use a mouse model which will mimic our established tobacco exposure paradigm and expose pregnant mice to cannabis smoke. We will then examine their offspring's lung tissue to find DNAm changes at specific genes. By comparing these changes with to our recent findings on DNAm changes due to tobacco exposure, we can identify common patterns that could indicate potential novel genes and their mechanisms in causing asthma.

We hope that by identifying the particular mechanisms involved, in the future we can prevent or reverse the negative impacts of cannabis and tobacco smoke on the lungs that increase the risk of asthma.

GRADUATE STUDENT AWARDS IN ASTHMA

Awardee: Nandhitha Raguayaka, MSc Candidate, McMaster University
Project Title: *Quantitative Imaging to Understand the Early Manifestation and Therapeutic Relevance of Abnormal Airway Morphology and Function in Asthma*
Amount: $15,000
Lay Summary: Approximately 3.8 million Canadians are living with asthma, a chronic disease that impacts the airways and makes breathing difficult. For most people with asthma, symptoms can be adequately controlled, and normal lung function can be maintained with medications that are delivered by inhalation. Unfortunately, however, some people with asthma are considered to have uncontrolled or more severe disease as commonly prescribed inhaled medications do not improve their symptoms, lung function, or risk of an asthma attack. There might be several reasons for this. One might be that anatomical abnormalities in the structure of the airways originate early in life, which are not hallmark and treatable components of asthma. Alternatively, inhaled medicines may not be reaching the right areas in the lungs due to abnormalities in the structure of the airways.

Scientists have started to apply high-resolution medical imaging methods to study this. Using advanced magnetic resonance imaging and computed tomography techniques, we will investigate if abnormal features of airway structure: (1) are present, and explain reduced lung function, in early adulthood, (2) have unique temporal trajectories indicative of progressive or non-progressive disease in early adulthood, and (3) influence the effectiveness of first-line inhaled medications. Knowledge gained from this research will improve our understanding of airway disease in asthma and will be applied to improve how asthma is managed and how asthma medications are developed and delivered.

GRADUATE STUDENT AWARDS IN ASTHMA

Awardee: Michael Yoon, MSc Candidate, University of British Columbia
Project Title: *Investigating the interactions of air pollution and an anti-inflammatory asthma medication using scRNA sequencing*
Amount: $15,000
Lay Summary: With the high prevalence of global air pollution, asthmatic individuals are at increased risk of developing respiratory complications. Asthma exacerbations can cause inflammation of the airways leading to breathing difficulties, necessitating the need for prescription medications. Inhaled corticosteroids (ICS) are a common asthma medication that reduces inflammation, but previous studies show that these medications are less effective following exposure to air pollutants. Despite prior research, the exact mechanisms of how ICS treatment is disrupted remain elusive. A lack of understanding between ICS and air pollution can cause medical practitioners to incorrectly access their patient's condition, leading to under or overprescription of ICS and resultant adverse effects. Regardless of dosage, greater knowledge of ICS administration can aid in reducing treatment expenses by optimizing quality of care. For these reasons, the aim of this project is to understand how air pollution affects responses to ICS under well controlled conditions in human participants. More specifically, we can discern which cell-types and genes are directly involved in this process, with the hopes of identifying targets that may explain any decreased efficacy of ICS treatment following air pollution exposure.

Identification of gene targets can be determined using single-cell RNA sequencing technology, where we can find unique responses in individual cell-types and genes in ICS-treated participants exposed to air pollution or filtered air. Our research aims to guide improvements in ICS administration policies, ICS development or add-on medications to reduce the burden on healthcare services caused by air pollution.

EARLY CAREER RESEARCHER AWARD IN ASTHMA

Awardee: Dr. Cristina Longo, Université de Montréal
Project Title: *Treating Asthma by Integrating Learning Algorithms with Omics Research: Moving toward Automated High-Dimensional Endotyping in Children (TAILOR-MADE)*
Amount: $100,000 (Year two funding- to be distributed by year-end)
Lay Summary: Asthma is a common lung disease in children, which can cause severe breathing problems and affect their quality of life. When asthma symptoms happen, many kids will visit their doctor for a diagnosis and treatment. Doctors often treat asthma symptoms with medicines that you inhale. A major problem is that these medicines do not always work. This is because asthma has many different disease processes that need targeted treatments. Although we do not know much about these disease processes, we can understand more by collecting biological samples, like blood, breath, or saliva, and

analyzing them with new technologies. These samples contain millions of signals or 'biomarkers' that can help us discover new disease processes for childhood asthma. However, this large amount of data makes it challenging to find which biomarker might be important. We now have advanced computer tools, like machine learning, that can help us address this 'big data' problem. My research program uses machine learning to discover important biomarkers and different disease processes in children with asthma symptoms that can help doctors predict their response to treatment. This will help doctors identify which children will benefit from treatment or develop new medicines for those that will not benefit.

EARLY CAREER RESEARCHER AWARD IN ASTHMA

Awardee: Dr. Zihang Lu, Queen's University
Project Title: *Asthma phenotypes, risk factors and the implications for future management in Canadian children*
Amount: $99,875 (Year two funding)
Lay Summary: In preschool, defining asthma is challenging due to a lack of objective measures along with a heavy reliance on a non-specific symptom of wheeze and a diverse clinical course reflected by remission and relapse. Earlier research shows that asthma is likely caused by several different pathways that are influenced by genetic and environmental factors. We believe these disparate pathways reflect different types of asthma and we aim to identify these pathways in early life using objective methods. To do this, we will use two research data platforms, namely the CHILD Cohort Study and the Canadian Urban Environmental Health Research Consortium. The first aim is to define distinct asthma phenotypes by applying data-driven methods to asthma traits (e.g. wheeze, atopy, body mass index), and to determine whether these phenotypes are different in males and females. The second aim is to determine early-life risk genetic and environmental factors associated with these distinct phenotypes. This study will address several knowledge gaps in our understanding of early life asthma phenotypes and the influences of genetic and environmental exposures on these phenotypes. It will also promote future studies to understand the underlying disease mechanism and provide important evidence to develop disease prevention and management strategies.

Prepared by
Andrea Waserman
Managing Director, CAAIF
November 1, 2023