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Prematurity, Socioeconomic Status, And Childhood Asthma: A Canadian Cohort Study

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Abstract

Background: Preterm birth (PTB) and socioeconomic status (SES) have both been associated with childhood asthma in high income countries. Yet, these factors have been studied minimally outside of an exposure or confounded relationship, and amongst the Canadian population.

Purpose: The purpose of this study was determine the associations of childhood asthma, PTB, and SES in a nationally representative dataset of Canada.

Methods: Merging household interview data from Cycles 2 to 5 of the Canadian Health Measures Survey, this study analyzed data from weighted respondents in early (3 to 5 years, n = 1,096,609) and middle (6 to 11 years, n = 2,112,059) childhood. Prematurity was determined based on a gestation of less than 37 weeks at birth. SES was measured by highest household education level, and household values of total income adjusted based on household size. Bivariate, stepwise logistic regression, and logit decomposition were performed using STATA software. Intersectionality guided the study methodologically.

Results: PTB ($p < 0.1$), highest household education ($p < 0.05$), and total household income ($p < 0.1$) were significantly associated with childhood asthma until entered into a multivariable regression. Instead, middle childhood age was found to be the most significant predictor of childhood asthma. Female sex, upper adjusted household income, and presence of siblings were protective of childhood asthma onset. The relationship of PTB with childhood asthma was found to also be partially mediated by SES, but this indirect effect of SES was relatively small (education 0.6%, income 3.1%).

Conclusion: These findings acknowledge the interaction between biological and social influences of childhood asthma, and are meaningful to forthcoming studies of asthma within the Canadian context. Implications of results direct healthcare systems to adopt multipronged approaches that reach beyond the confines of healthcare to diminish the onset of childhood asthma. Study strengths include a large sample size, robust analysis,

and use of the theoretical lens of intersectionality to locate findings within society. Additional study is required to confirm and expand on this study's findings.

Keywords: Asthma, childhood, logistic regression, prematurity, socioeconomic status.

Lay Summary

Childhood asthma and asthma-like symptoms have been strongly associated with both PTB and SES, but these three factors have been infrequently studied together. Being concepts within different healthcare models, biological and social, PTB and SES are often recognized in the literature as having separate causal pathways. This study sought to address the literature divide between PTB and SES using the theoretical lens of intersectionality, and by statistically testing each variable's relationship to asthma, and one another within the Canadian population.

Co-Authorship Statement

Crystal McLeod conducted this thesis work under the supervision of Dr. Richard Booth and Dr. P. Tryphonopoulos, who will co-author any resulting publications from this work.

Dedication

This thesis is dedicated in the memory of René T.H. Laënnec, whose works continue to spark a love of both pulmonology and research within me.

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[Disclaimer: The research analysis and results are based on data from Statistics Canada and the opinions expressed herein do not represent the views of Statistics Canada.]

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Chapter 1 Introduction

Literature investigating the links between asthma and causative factors is expansive. To date, researchers have studied a variety of associations with childhood and adulthood asthma, ranging from enterovirus to specific personality traits, to uncover causal mechanisms of this chronic disease (Cheng et al., 2018; Coelho, de Souza-Machado, & Souza-Machado, 2017; Ding, Ji, & Bao, 2015; Wang et al., 2018). The leading conclusion from this field of research is that the development of asthma, in any stage of life, is influenced by a complex combination of biological and social factors (Beasley, Semprini, & Mitchell, 2015; Canadian Institute for Health Information, 2018; Cheng et al., 2018; Cook & Saglani, 2016; Ding et al., 2015; Fouzas & Brand, 2013; Watts, 2012; World Health Organization, 2017). Though the exact combination of causative factors that lead to asthma onset is unknown, the high prevalence, adverse outcomes, elevated costs and lack of a definitive cure of asthma has continued to motivate significant research activity in this clinical domain (Ehteshami-Afshar, Fitzgerald, Doyle-Waters, & Sadatsafavi, 2016; Watts, 2012; World Health Organization, 2017).

1.1 Background

Children experience a heavy burden of respiratory disease, with asthma being one of the most pervasive chronic respiratory diseases of childhood worldwide (Beasley et al., 2015; Global Initiative For Asthma, 2018; Samoliński, Fronczak, Włodarczyk, & Bousquet, 2012; Sennhauser, Braun-fahrla, & Wildhaber, 2005; Simpson & Sheikh, 2010; Watts, 2012). Asthma tends to be a lifelong disease and will present in varying degrees of severity, but the vast majority of asthma symptoms begin in childhood (Beasley et al., 2015; Ducharme et al., 2015; Nunes, Pereira, & Morais-Almeida, 2017). Children, both in early and middle childhood, can experience significant disability, absenteeism from school, lost caregiver work days, reduced quality of life for caregiver and child, and premature death as a result of asthma (Cerdan, Alpert, Moonie, Cyrkiel, & Rue, 2012; Cook & Saglani, 2016; Gershon, Guan, Wang, & To, 2010; Nunes et al., 2017; Ramratnam, Bacharier, & Guilbert, 2019; Sadatsafavi et al., 2010; Sennhauser et al., 2005; Woodgate, 2009). The experience of asthma can also vary greatly throughout

childhood. For children under five years of age, there are often greater diagnostic challenges, and more hospitalization and emergency visits related to asthma than other age groups (Canadian Institute for Health Information, 2018; Ducharme et al., 2015; Global Initiative For Asthma, 2018; Hasegawa, Tsugawa, Brown, & Camargo, 2013; Ismaila, Sayani, Marin, & Su, 2013). While children 6 to 11 years tend to experience a larger incidence of asthma and tend to use more medication to control their asthma symptoms (Jang et al., 2013; Karlstad, Nafstad, Tverdal, Skurtveit, & Furu, 2010). Long term, uncontrolled childhood asthma can be as significant of a risk factor for reduced lung function and the development of adult chronic obstructive pulmonary disease (COPD) as smoking in adulthood (Cook & Saglani, 2016; Ramratnam et al., 2019; Svanes et al., 2010).

1.1.1 Overview of asthma

Asthma is a chronic inflammatory disease characterized by acute exacerbations of dyspnea and bronchospasm (Global Initiative For Asthma, 2018; Nunes et al., 2017). Presentations of asthma can be quite diverse in both timing and symptomology (Global Initiative For Asthma, 2018; Ramratnam et al., 2019). However, classic manifestations of childhood asthma include wheezing, shortness of breath, chest tightness, varied expiratory breath, and cough exacerbated by specific environmental triggers (Asher & Pearce, 2014; Colicino, Munblit, Minelli, Custovic, & Cullinan, 2019; Ducharme et al., 2015; Global Initiative For Asthma, 2018; Tesse, Borrelli, Mongelli, Mastrorilli, & Cardinale, 2018). Depending on a child's developmental stage, dyspnea and shortness of breath may be verbalized in terms of feeling heavy, scared, or not being able to engage in play activities (Pieper et al., 2018; Woodgate, 2009). Physiologically, asthma is also varied in pathology and etiology (Global Initiative For Asthma, 2018; Ramratnam et al., 2019). To date, several groupings or *phenotypes* of asthma have been identified, which share clinical, demographic and pathologic traits, but these phenotypes do not predict treatment responses (Global Initiative For Asthma, 2018). Common childhood phenotypes of asthma include wheezing, allergic (atopic), non-allergic (non-atopic), late-onset, and fixed airflow limitation (Global Initiative For Asthma, 2018, 2019; Wang et al., 2018). Confirming a diagnosis of asthma can be difficult when considering this

clinical variability, so emphasis is placed on the child's pattern of respiratory symptoms and the documentation of these patterns by caregivers (Asher & Pearce, 2014; Global Initiative For Asthma, 2018). Once a detailed history of the child's respiratory symptoms is obtained, testing in the form of spirometry, exercise challenges, exhaled nitric oxide, allergy testing, and bronchodilator reversibility testing may be used to confirm the diagnosis (Global Initiative For Asthma, 2018).

Managing childhood asthma is a delicate balance of prevention and treating acute exacerbations (Cook & Saglani, 2016; Nunes et al., 2017). Children and their families are frequently instructed by healthcare professionals to avoid environmental triggers, such as air pollutants, infection, or allergens, and control co-morbidities to prevent asthma symptoms (Beasley et al., 2015; Cook & Saglani, 2016; Global Initiative For Asthma, 2018; Nunes et al., 2017). Therapies like bronchial thermoplasty, vaccinations, stress management, exercise, dietary changes, vitamin D, fish oil, and allergen immunotherapy should be considered relevant in reducing the frequency of asthma symptoms (Beasley et al., 2015; Global Initiative For Asthma, 2018; Hon, Fung, Leung, Leung, & Ng, 2015; Ramratnam et al., 2019). Anti-inflammatory medications, for which inhaled corticosteroids or leukotriene receptor antagonists are most popular, are also considered instrumental in reducing the frequency of acute exacerbations of asthma globally (Global Initiative For Asthma, 2018; Lougheed et al., 2012; Nunes et al., 2017; Pacheco-Galván, 2010; Ramratnam et al., 2019). Active exacerbations of asthma are best managed with rescue bronchodilators, such as long and short-acting beta agonists via metered dose inhalers, to reverse airway obstruction (Global Initiative For Asthma, 2018; Lougheed et al., 2012; Nunes et al., 2017; Pacheco-Galván, 2010; Pollock et al., 2017; Sullivan et al., 2018; Tesse et al., 2018). Due to ethical limitations in the research of pediatric asthma treatment and the labile nature of children's asthma symptoms, there may be a need for frequent stepwise or cyclical assessment, review, and alterations to treatment plans as a child grows (Backlund et al., 2006; Canadian Institute for Health Information, 2018; Global Initiative For Asthma, 2018; Lougheed et al., 2012; Tesse et al., 2018). Emergency care and hospitalization may be required if an asthma exacerbation is especially severe; non-responsive to prescribed treatment; access to outpatient resources

are limited; or, a child exhibits reduced treatment compliance (Global Initiative For Asthma, 2019; Nunes et al., 2017; Sullivan et al., 2018).

Beyond rising incidence and the difficult experiences of patients and families with childhood asthma, the burden of this disease is further delineated in terms of economic cost (Achakulwisut, Brauer, Hystad, & Anenberg, 2019; Global Initiative For Asthma, 2019; Puig-Junoy & Pascual-Argenté, 2017; Sennhauser et al., 2005). The cost of childhood asthma can be determined by combining direct and indirect costs of disease prevention, diagnosis, and treatment (Bahadori et al., 2009; Bedouch, Marra, Fitzgerald, Lynd, & Sadatsafavi, 2012; Nunes et al., 2017; Sennhauser et al., 2005). Direct costs refer to utilization of healthcare services, products, and resources to reduce the effects of childhood asthma (Bahadori et al., 2009; Cisternas et al., 2003; Nunes et al., 2017). While indirect costs comprise of financial losses related to disability, reduced quality of life, and early mortality from childhood asthma (Bahadori et al., 2009; Cisternas et al., 2003; Nunes et al., 2017). Indirect losses are represented less in the literature and great variations in cost exist between countries, but globally childhood asthma incurs distinct additional expenses for individual families, communities, and nations (Bahadori et al., 2009; Beyhun, Çilingiroğlu, & Şekerel, 2007; Cisternas et al., 2003; Ehteshami-Afshar et al., 2016; Nunes et al., 2017; Puig-Junoy & Pascual-Argenté, 2017; Sadatsafavi et al., 2014). In comparison to other childhood illnesses, asthma tends to report higher costs of medication, emergency care, and lost time at work for caregivers (Bahadori et al., 2009; Davies, Paton, Beaton, Young, & Lenney, 2008; Grupp-Phelan, Lozano, & Fishman, 2001; Radhakrishnan, Dell, Guttmann, Salimah, & To, 2018). Generally, asthma costs are considered substantial, and international/national organizations continue to seek methods of streamlining asthma management (Bahadori et al., 2009; Ismaila et al., 2019; Nunes et al., 2017; Sullivan et al., 2017). Comprehensive prevention and early intervention programs are noted as particularly cost-effective strategies in childhood, as the majority of childhood asthma costs are incurred from treatment of acute exacerbations (Bahadori et al., 2009; Beyhun et al., 2007; Canadian Institute for Health Information, 2018; Davies et al., 2008; Ducharme et al., 2015; Global Initiative For Asthma, 2019; Nunes et al., 2017; Radhakrishnan et al., 2018; Sadatsafavi et al., 2010; Sennhauser et al., 2005;

Visitsunthorn, Durongpisitkul, Uoonpan, Jirapongsananuruk, & Vichyanond, 2005; Wang, Zhong, & Wheeler, 2005).

1.1.2 Childhood asthma in Canada

Though the burden of asthma appears to be increasing internationally, distribution of this chronic respiratory disease varies across countries (Asher & Pearce, 2014; H. Cheng et al., 2018; Ding et al., 2015; Global Initiative For Asthma, 2019; Nunes et al., 2017). Currently, high income, Westernized countries experience the greatest rates of asthma and as such, asthma has been deemed a significant public health problem in Europe, Canada, and the United States (Asher & Pearce, 2014; Beasley et al., 2015; Gershon et al., 2010; Global Initiative For Asthma, 2019; Nunes et al., 2017; Samoliński et al., 2012; Stewart, Mitchell, Pearce, Strachan, & Weiland, 2001). This disproportionate occurrence of asthma is considered more likely to be the result of lifestyle and environmental factors than wealth, as there is only a weak association between gross national product and asthma globally (Stewart et al., 2001).

In Canada, 3.8 million people or 10.8% of the population have been diagnosed with asthma (Government of Canada, 2018). This proportion has been growing, increasing by 67% or from 2.1 million (6.5%) to 3.8 million (10.8%), since 2000 (Gershon et al., 2010; Government of Canada, 2018; Masoli, Fabian, Holt, & Beasley, 2004). Fortunately, mortality rates have remained stable during this expansion in asthma incidence, with Canada ranking the third lowest worldwide at approximately 1.6 fatalities per 100,000 asthmatics or 250 total asthma-related deaths annually (Asthma Canada, n.d.; Masoli et al., 2004). At a national prevalence of 15% or 850,000 individuals, a 62% greater proportion of Canadian children and youth are living with asthma than adults (Asthma Canada, n.d.; Canadian Institute for Health Information, 2018; Government of Canada, 2018). Of this 15% of Canadian children diagnosed with asthma, approximately 30% report ongoing and frequent acute exacerbations (Masoli et al., 2004).

Examining Canadian children with asthma, males are more disproportionately affected by childhood asthma until puberty, experiencing a 18% versus 13% national incidence, and have more hospitalization and emergency visits in childhood related to

asthma than females (Government of Canada, 2018; Rosychuk et al., 2018). During acute illness, Canadian children are hospitalized and visit emergency departments more frequently for asthma than youth and adults, and account for approximately 16.8% of the direct medical costs associated with asthma in Canada (Bedouch et al., 2012; Canadian Institute for Health Information, 2018). Furthermore, a recent study of pediatric asthma incidence indicates that the burden of asthma is still increasing among Canadian children. Comparing high-income nations and major cities globally, the study ranked Canada first, and Toronto second for the greatest number new diagnoses of childhood asthma related to environmental pollution (Achakulwisut et al., 2016). Increasing asthma incidence and disease burden, though not a new trend in Canada, is concerning as this disease is already very common among children, and heightens the urgency to better understand the factors that cause childhood asthma. (Achakulwisut et al., 2016; Gershon et al., 2010; Sadatsafavi et al., 2014).

1.1.3 Determinants of childhood asthma

With as many as half of all people with asthma experiencing onset of symptoms in childhood, comprehending the casual mechanisms behind the onset of childhood asthma could significantly reduce the burden of this disease among children and throughout other lifetime stages (Beasley et al., 2015; Ducharme et al., 2015; Holt & Sly, 2007; Simpson & Sheikh, 2010; Visitsunthorn et al., 2005; Xu et al., 2016). To date, no single or combination of predictors has been attributed to the prevalence of childhood asthma and resulted in an intervention to prevent the disease (Beasley et al., 2015; Global Initiative For Asthma, 2018, 2019; Smit et al., 2015). In some studies, even the prominence of asthma predictors have been found to fluctuate based on temporal trends, geography, and sociocultural context (Beasley et al., 2015; De Verdier et al., 2017; Hasegawa et al., 2013). A many number of factors, including age, sex, ethnicity, family history, birth outcomes, allergies, and persistence of respiratory symptoms, have been examined exhaustively for predictive value (Biagini Myers et al., 2018; Castro-Rodríguez, Holberg, Wright, & Martinez, 2000; Colicino et al., 2019; Ducharme et al., 2015; Fouzas & Brand, 2013; Leonardi et al., 1984; Savenije et al., 2012; Smit et al., 2015). Creating an accurate model to predict the onset of childhood asthma is widely

considered a complex and difficult undertaking (Fouzas & Brand, 2013). Thus, more study is required to identify and verify the full extent of risk factors that predict childhood asthma (Bao et al., 2017; Colicino et al., 2019; Smit et al., 2015).

Past study of the determinants of childhood asthma have been driven by several theories, which seek to explain asthma onset and prevalence. A broadly accepted and long standing theory is the *hygiene hypothesis* (Ball et al., 2000; Beasley et al., 2015; Liu, 2015; National Asthma Education and Prevention Program, 2007). According to the hygiene hypothesis, a lack of exposure to other children (e.g., siblings, early enrollment in child care, etc.); certain infections; household pets; and rural settings, paired with frequent antibiotic use increases a child's risk of asthma (Ball et al., 2000; National Asthma Education and Prevention Program, 2007). Biologically, introduction to the aforementioned exposures is believed to downregulate innate immunity factors responsible for the expression of allergic diseases and asthma (Ball et al., 2000; National Asthma Education and Prevention Program, 2007). Failure to explain the links of obesity, poverty, and urban environments to asthma are notable shortcomings of the hygiene hypothesis (Litonjua, 2008; Liu, 2015). Dietary hypotheses, first popularized by Barker and colleagues, have also been suggested to clarify the causation of childhood asthma (Dover, 2009; Litonjua, 2008; Tantisira & Weiss, 2001). These hypotheses tend to link reduced intake of dietary antioxidants (e.g., vitamin A, D, and E) and minerals (e.g., Magnesium, Selenium, Zinc) during pregnancy and in early years of life with childhood asthma by alteration of lung function and airway responsiveness (Beasley et al., 2015; Litonjua, 2008; Stocks & Sonnappa, 2013). Research has supported this theory, uncovering a great dearth of associations between nutrients and whole foods to asthma (Beasley et al., 2015; Litonjua, 2008). Today, foods like breastmilk, fish oils, fruits, and vegetables are considered protective of childhood asthma, while fast foods, salt, and trans-fatty acids are linked to asthma onset (Beasley et al., 2015). However, the link between dietary factors and childhood asthma may be more tenable for maternal diet preconception and in utero, versus early life diet (Beasley et al., 2015; Dover, 2009; Litonjua, 2008).

Reviews of the literature on childhood asthma risk factors clearly display this chronic respiratory disease is precipitated by a combination of biological and social factors (Bao et al., 2017; Colicino et al., 2019; Rodriguez-Martinez, Sossa-Briceno, & Castro-Rodriguez, 2017; Smit et al., 2015; Strina, Barreto, Cooper, & Rodrigues, 2014). The relationship between biological and social factors, both how they interact and influence one another in the development of childhood asthma, is largely unknown (Bao et al., 2017; Global Initiative For Asthma, 2019; Smit et al., 2015). With the relationships between predictors of childhood asthma not fully known, Figure 1 offers a summary of well-known biological and social risk factors under the premise that there is interaction between them. As researchers hypothesize that predictors have excursive and intricate associations, this study will be exclusively studying the interaction of preterm birth (PTB) and socioeconomic status (SES) with the intention of contributing the wider research narrative of childhood asthma prediction.

1.2 Purpose and Rationale

This study sought to identify the relationship between childhood asthma, and PTB and SES in Canada. This relationship was studied by examining PTB, SES, sociodemographics, and confounding variables for a predictive association with childhood asthma. Then the relationship of PTB, SES, and childhood asthma was further statistically modelled for significance and mediation.

Given that a vast array of factors contribute to childhood asthma, this study could be valuable in clarifying and prioritizing factors within the Canadian context (Ding et al., 2015). Mediation analysis especially, which has been used to study other childhood illnesses, may improve understanding of how predictive factors of childhood asthma modify one another (Beauregard, Drews-Botsch, Sales, Flanders, & Kramer, 2018; Joseph et al., 2018; Richards, Chapple-Mcgruder, Williams, & Kramer, 2015; Wai et al., 2018). Results from these analyses may inform prevention and awareness of childhood asthma onset.

1.3 Theoretical Perspective

Given the multidimensional nature of childhood asthma, this study has adopted the conceptual framework of intersectionality to better apprise the illness across many facets simultaneously. Intersectionality specifically seeks to better understand inequality and marginalization by simultaneously examining the many social categories that an individual belongs to, and how those categories interact (Bauer, 2014; Crenshaw, 1989). The social categories examined by intersectionality can vary widely, but often include sex, gender, ethnicity, socioeconomic status, educational background, culture, and other positions of social status (Hancock, 2007; Hankivsky, 2012). At the level of population health, social categories can also expand to include policy and institutional structures (Bauer, 2014; Green, Evans, & Subramanian, 2017). Viewing a person through the many layers and contexts in which they live is complex, but essential under the conceptual framework of intersectionality to explore health and wellness (Hankivsky, 2012; Mccall, 2005).

Since inception, intersectionality has been primarily utilized in qualitative studies and alongside the theory of feminism (Green et al., 2017; Hancock, 2007). Applying intersectionality as an overarching framework for quantitative studies is a relatively new approach (Bauer, 2014; Bauer & Scheim, 2019a; Hancock, 2007; Scheim & Bauer, 2019; Wemrell, Mulinari, & Merlo, 2017). Yet, popularity for this framework has been increasing in the sectors of public health (Agénor, Krieger, Austin, Haneuse, & Gottlieb, 2014; Bowleg, 2012; Mulinari, Bredstrom, & Merlo, 2015; Springer, Hankivsky, & Bates, 2012), epidemiology (Bauer & Scheim, 2019a, 2019b; Sun, Crooks, Kemnitz, & Westergaard, 2018; Wemrell et al., 2017), sociology of health (Gkiouleka, Huijts, Beck, & Bambra, 2018; Hinze, Lin, & Andersson, 2012; Sen & Iyer, 2012; Seng, Lopez, Sperlich, Hamama, & Meldrum, 2012; Veenstra, 2011; Warner & Brown, 2012), and psychology (Bowleg & Bauer, 2016; Stirratt et al., 2008). From this early research, intersectionality has been perceived as valuable to quantitative studies by offering more precise and relevant results with less bias (Bauer, 2014). As well, intersectionality can complement other theoretical frameworks when used in the same study, and generates unique opportunities for triangulation of results across both qualitative and quantitative

studies (Bauer, 2014; Green et al., 2017). Overall, the effects of institutional powers, structures, and policies upon populations can be examined at a new depth when a study is directed by intersectionality (Bauer, 2014).

In this study, intersectionality was drawn upon in forming research questions, selecting variables, and during statistical modelling. Examining logistic regression and mediation among variables, the study's statistical functions, and analytical strategies were aimed to be reflective of intersectionality (Bauer, 2014; Bauer & Scheim, 2019a; Hinze et al., 2012; Longman Marcellin, Bauer, & Scheim, 2013; Veenstra, 2011). Mediation modelling especially goes beyond the assumption of multiple regression that all variables equally influence health, and searches to uncover the strength, direction, and underlying influences of variables (Bauer, 2014; Bauer & Scheim, 2019a). Intersectionality has also guided the perspective in which results have been analyzed and this paper has been written. Discussing the origin, exclusions, contexts, and composition of variables, as representatives of social categories, is integral to the principles of intersectionality and creating meaningful implications for populations experiencing inequality (Green et al., 2017; Hancock, 2007; Hankivsky, 2012).

1.4 Significance

Biomedical tradition can dictate that health is the responsibility of the individual and at times, may overlook the view that health is a collective issue (Kagan, Smith, & Chinn, 2014; Matwick & Woodgate, 2016; Rogers & Kelly, 2011; Yanicki, Kushner, & Reutter, 2015). In turn, this belief can lead nurses, healthcare facilities, and healthcare structures to omit the impact of social inequalities among patients (Buettner-Schmidt & Lobo, 2011; Kagan et al., 2014; Wilmot, 2012). However, comprehending and engaging with population health to promote social justice and health equity is a core value of the nursing profession (Buettner-Schmidt & Lobo, 2011; Canadian Nurses Association, 2010, 2013; Wilmot, 2012; Yanicki et al., 2015). Assisting nurses to overcome the ideals of biomedical health, value population health, and reduce the ambiguity of achieving health equity in clinical settings are central implications of this study. Exclusively analyzing the health of children, nurses working in the field of pediatrics will particularly find the results of this research applicable to his or her role.

This study is also significant to the field of childhood asthma in using the theoretical lens of intersectionality, and pursing joint analysis of PTB and SES. No theoretically-based research, as known by the author, has been located that examines how both PTB and SES are associated with, and predictive of, childhood asthma. Further, little known research has been found that examines how prematurity and childhood asthma are mediated by SES. The results of this study could inform future interventions to prevent childhood asthma and enhance the quality of life for children with asthma.

1.5 Summary

Childhood asthma is a complex, heterogenous chronic disease. Academic literature has linked numerous biological and social contributors to the causation of asthma, but to date no combination of factors can fully account for the onset of this disease. PTB and SES are among these contributors, with each significant associations to childhood asthma across multiple studies globally. Although, examining how these two factors are associated with one another and childhood asthma in Canada may provide more causational insights, and healthcare strategies to help reduce the onset, prevalence, and burden of this widespread disease nationwide.

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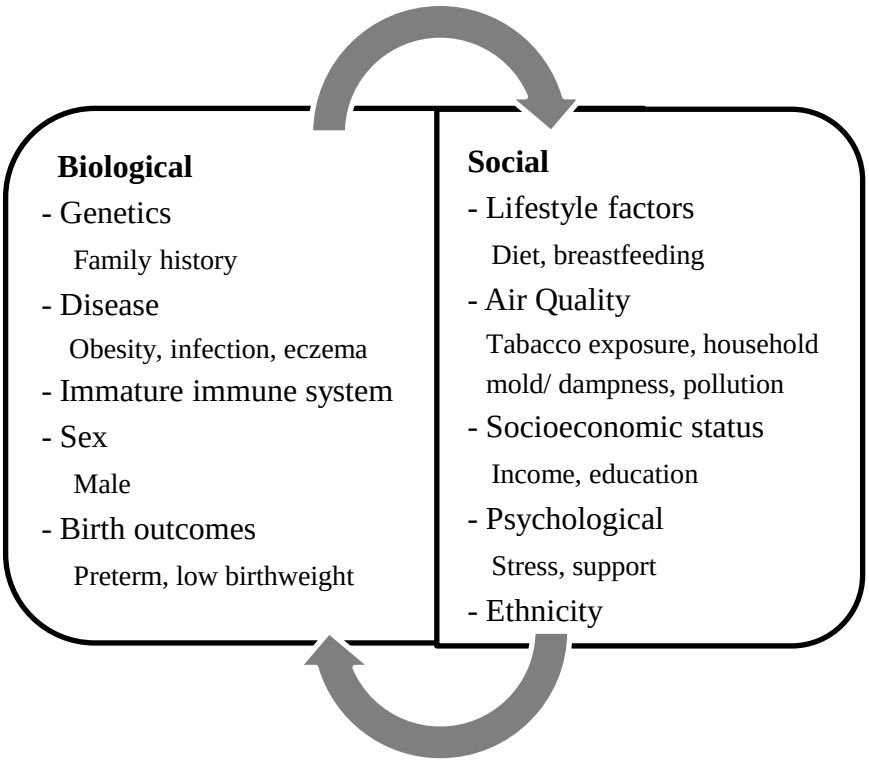

Figure 1: Biological and social determinants of childhood asthma

(Bao et al., 2017; Beasley et al., 2015; Biagini Myers et al., 2018; Colicino et al., 2019; Fouzas & Brand, 2013; Global Initiative For Asthma, 2019; Holt & Sly, 2007; Kashanian et al., 2017; Rodriguez-Martinez et al., 2017; Smit et al., 2015; Strina et al., 2014; Xu et al., 2016)

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Defined as chronic inflammation of the airways and airflow obstruction, asthma impacts approximately 11% of children globally, and approximately 15% of Canadian children report current asthma symptoms (Asher & Pearce, 2014; Canadian Institute for Health Information, 2018; Government of Canada, 2018; National Institues of Health, 1992; The National Asthma Control Task Force, 2000; To et al., 2006). Asthma creates a significant burden on the Canadian healthcare system, utilizing over half a billion healthcare dollars annually to provide medications, treatments, and hospitalization to children with asthma (Asher & Pearce, 2014; Chang, Chang, Grady, & Torzillo, 2009). For children and their families, symptoms and complications of asthma, like prolongment of respiratory infections and increased risk of respiratory disease in adulthood, can cause significant morbidity and risk of early mortality (Asher & Pearce, 2014; Chang et al., 2009). Researchers suggest that an understanding of childhood asthma causation will subsequently improve treatment and prevent incidence of childhood asthma, which in turn will result in lower healthcare costs and ongoing illness for affected children (Asher & Pearce, 2014).

Past research has demonstrated that the prevalence of childhood asthma and asthma-like symptoms are strongly associated with: (a) PTB (Leung, Lam, Leung, & Schooling, 2016); and, (b) SES (Hafkamp-de Groen et al., 2013). While past research has provided insights into the potential causal mechanisms of childhood asthma, more in-depth analysis related to the dynamic relationship between asthma, PTB, and SES have not been conducted. Most commonly, PTB is presented as a biological factor, resulting from genetics or pathology (Manuck et al., 2015), and SES is considered the result of inequity in social systems (Galobardes et al., 2007). Being concepts within different healthcare models (i.e., biological and social, respectively) PTB and SES are often recognized in the literature as having separate causational pathways. This study seeks to address the literature divide between PTB and SES by statistically testing each variable's relationship to childhood asthma, and one another.

2.1 Literature Review

Like other respiratory illnesses, asthma is considered multifactorial in causation (Asher & Pearce, 2014; Strina et al., 2014). Numerous biological and social factors, including genetics, gender, allergies, other respiratory diseases (i.e., Bronchopulmonary dysplasia), tobacco smoke exposure, maternal breastfeeding, and obesity, have been identified as risk factors of asthma (Asher & Pearce, 2014; Bao et al., 2017; Islam, Keller, Aschner, Hartert, & Moore, 2015; Lawson et al., 2017; Rodriguez-Martinez et al., 2017; Rosas-Salazar et al., 2015; Soto-Ramírez et al., 2012; Zugna et al., 2015). The selection of this study's variables arose from the fact that PTB and SES have already well-founded relationships with childhood asthma, but have been rarely analyzed outside of an exposure or confounded relationship. Considering Canadian context, studies that examine asthma's association with both PTB and SES among Canadian children appear to be lacking too (Crighton, Wilson, & Senècal, 2010; Crockett, Brownell, Heaman, Ruth, & Prior, 2017; Lethbridge & Phipps, 2005; Lin et al., 2004; Seguin, Nilkiema, Gauvin, Zunzunegui, & Xu, 2007; Seguin et al., 2005; Shah et al., 2018; Tu, Perreault, Séguin, & Gauvin, 2011). Potential implications from this study, which seeks to further the collective understanding of asthma causation, could assist global researchers in comprehending the larger picture of asthma causation, and aide Canadian stakeholders who are taking strides to diminish childhood asthma incidence (Canadian Paediatric Society, 2018; Seguin et al., 2005).

PTB is defined by the World Health Organization as birth before completing 37 weeks or 259 days of gestation, as determined by last menstrual period or obstetrical ultrasound (Trønnes, Wilcox, Terje Lie, Markestad, & Moster, 2013; World Health Organization, 2012). Globally and in Canada, approximately 10% and 8% of all births are preterm (Blencowe et al., 2012, Shah et al., 2018; Statistics Canada, 2016; The National Asthma Control Task Force, 2000; To et al., 2006). This definition of PTB was adopted to reflect the significant risk factor of being born at or earlier than 36 weeks and 6 days poses to neonatal morbidity and mortality (Harijan & Boyle, 2012). PTB also carries health risks into late infancy, childhood and adulthood, which are being studied with renewed interest as advances in neonatal care produce greater preterm survivorship

(Gibson & Doyle, 2014; Hack et al., 2005; Hille et al., 2001; Kramer, 2011; Vollsæter Røksund, Eide, Markestad, & Halvorsen, 2013). Most notably, children born preterm often experience greater rates of hospitalization, infection, alterations in growth, learning disabilities, reduced lung function, and respiratory diseases like asthma (Annesi-Maesano, Moreau, & Strachan, 2001; Crump, Sundquist, Winkleby, & Sundquist, 2011; Harijan & Boyle, 2012; Jaakkola et al., 2006; Mackay, Smith, Dobbie, Cooper, & Pell, 2013; Miller et al., 2016; Moster, Lie, & Markestad, 2008; Paranjothy et al., 2013; Vollsæter et al., 2013). For childhood asthma, in particular, meta-analyses estimate children born preterm have as high as four times greater risk of developing asthma in early and middle childhood than children born at term (Jaakkola et al., 2006; Sonnenschein-van der Voort et al., 2014).

The pathway from prematurity to childhood asthma, though considered a biological process, remains largely unidentified. Interruption of fetal lung development from PTB is theorized by some researchers to diminish lung development or alter lung structure in childhood, and subsequently leave preterm born children with heightened risk for asthma onset (Consortium on Safe Labor et al., 2010; Leung et al., 2016; Zhang et al., 2018). While other experts suggest PTB is not the direct culprit, but rather antenatal microbial, genetic, and immune factors that underlie PTB later precipitate childhood asthma onset (Burke et al., 2012; de Marco, Pattaro, Locatelli, Svanes, & the ECRHS Study Group, 2004; Ding et al., 2015; Holt & Sly, 2007; Sindičić Dessardo et al., 2014) Cesarean sections and low birth weight, each well-established risk factors for childhood asthma in their own right, frequently accompany PTB and may inadvertently contribute to the overall association (Boyle & Reddy, 2012; Stocks, & Sonnappa, 2013; Thavagnanam, Fleming, Bromley, Shields, & Cardwell, 2008).

SES can be difficult to characterize but is often considered an organizational system to stratify individuals based on the positions and resources they hold within society (Cheng & Goodman, 2015; Galobardes et al., 2007). Terms like social class and social stratification are also commonly interpreted as synonymous with SES, despite having different theoretical backdrops (Cheng & Goodman, 2015). Initial theoretical interest in the role of SES upon health disparity and inequality was displayed by Marmot

and Wilkinson (1999), and by Krieger (2001). Today, SES is viewed as a complex, multi-dimensional representation of hierarchy, power, and access to resources that influences health by numerous explanatory pathways (Braveman et al., 2005; Galobardes et al., 2007). Disparities in well-being and chronic health conditions among children is generally accepted globally, Canada included, in part to be the effects of material deprivation secondary to low SES (Simons, Dell, Moineddin, & To, 2019; Zuijdwijk, Cuerden, & Mahmud, 2013).

As a substantiated risk factor of childhood asthma, SES is believed by researchers to be an umbrella concept that draws association through a subset of sociodemographic characteristics tied to low education and income households (Cerdan, Alpert, Moonie, Cyrkiel, & Rue, 2012; Ding et al., 2015; Watts, 2012). Such characteristics include reduced access to healthcare, health literacy, well-maintained living accommodations, adequate nutrition, safe living conditions, and clean air (Belfort, Cohen, Rhein, & McCormick, 2015; Ding et al., 2015; Hawlader et al., 2013; Mangini, Hayward, Dong, & Forman, 2015; Sullivan et al., 2019; Taylor-Robinson, Pearce, Whitehead, Smyth, & Law, 2015; Watts, 2012; Wing, Gjelsvik, Nocera, & McQuaid, 2015). Lack of clean air is often associated with childhood asthma and SES by increased prevalence of maternal smoking with low maternal education levels, higher rates of outdoors environmental pollution in low-income neighborhoods, and a greater presence of mold or moisture in low-income homes (Buteau et al., 2018; Harju et al., 2015; Ramratnam, Bacharier, & Guilbert, 2019; Simons et al., 2019; Strickland et al., 2014; Vo, Bair-Merritt, Camargo, Eisenberg, & Long, 2017). The exception in the link between low SES, air quality, and childhood asthma is seen among children living on farms in rural and remote settings, who often experience improved air quality and a lower incidence of childhood asthma (Brokamp et al., 2016; Harju et al., 2015; Karner, Eisinger, & Niemeier, 2011). Psychosocial stress induced from the challenges of living in poverty is also associated with childhood asthma, but the biological pathways involved are less well understood than other factors linked to SES (Hafkamp-de Groen et al., 2012; Sullivan et al., 2019; Wing et al., 2015; Wright et al., 2010).

The influence of SES upon childhood asthma may too be altered based on timing, severity, and fluctuation of SES. For instance, an Australian study revealed that low SES seems to play a greater role in asthma onset in early childhood, as compared to older age groups, and the risk of asthma decreases when a child moves to higher levels of SES (Kozyrskyj, Kendall, Jacoby, Sly, & Zubrick, 2010). Two additional studies, examining prenatal, perinatal, and postnatal SES factors upon childhood asthma, found parental education levels had the greatest influence upon onset of childhood asthma during the first year of life (Gong et al., 2014; Hafkamp-de Groen et al., 2012). Even the simple act of moving neighborhoods in childhood can alter SES exposures and, in turn, the risk of asthma onset (Brokamp et al., 2016). Studies examining the differences in SES throughout a child's life trajectory, and incidence of asthma will be crucial in forming accurate associations in the future (Brokamp et al., 2016; Simons et al., 2019).

Internationally, the relationship between PTB and SES to childhood asthma has been substantiated and continues to grow in academic literature (Dombkowski, Leung, & Gurney, 2008). Specific to Canada, several past studies have examined PTB and SES as an exposure or confounder with childhood asthma in Ontario, Manitoba, and Quebec (Kozyrskyj et al., 2008; Martel et al., 2009; Seguin et al., 2007; Simons, Dell, Moineddin, & To, 2018; Simons et al., 2019; Simons, To, Moineddin, Stieb, & Dell, 2014). Notable results include PTB (Simons et al., 2019; Simons et al., 2014) and SES (Kozyrskyj et al., 2008; Martel et al., 2009; Seguin et al., 2007; Simons et al., 2018; Simons et al., 2019) each being associated with the onset of childhood asthma, and one population based study concluded that SES was a greater predictor of childhood asthma than PTB (Simons et al., 2019). Considering these associations, the aim of this present study was to continue to affirm the predictive value of these factors in a representative population dataset of Canada. As well, this study sought to further analyze SES for mediation in the relationship between PTB and childhood asthma. Examining SES for mediation has rarely been performed in this topic area in the past (Hafkamp-de Groen et al., 2012; Taylor-Robinson et al., 2015; Wai et al., 2018), and to the best of the author's knowledge, has not been conducted within a Canadian context (Campbell & Seabrook, 2016; Seguin et al., 2005). SES has been found to mediate the onset of other childhood respiratory diseases, like infection and intermittent wheezing, via prenatal smoke

exposure, pollution, breastfeeding, and PTB (Hafkamp-de Groen et al., 2012; Shankardass et al., 2009; Violato, Petrou, & Gray, 2009). Thus, there is theoretical support to test for mediation of PTB and childhood asthma by SES. Low SES is hypothesized to elicit or expedite asthma onset among children born preterm by exacerbating their biological predisposition to the disease, causing a pathological 'double jeopardy' or an additive interaction (Richards, Chapple-McGruder, Williams, & Kramer, 2015; Seguin et al., 2007; Wai et al., 2018).

2.2 Theoretical Framework

Intersectionality, a theoretical lens with fairly recent applications in quantitative studies, was employed as the overarching theoretical framework of this study (Bauer, 2014; Bowleg & Bauer, 2016; Hancock, 2007; Scheim & Bauer, 2019; Wemrell et al., 2017). Quantitative intersectionality does not concentrate on the experiences and narratives of diverse people, but instead captures a broader story of social categories and inequality (Bauer, 2014; Bowleg & Bauer, 2016; Covarrubias, 2011). Examining population patterns and intersections of race, gender, social status, culture, and privilege across time are hallmark characteristics of quantitative intersectionality (Covarrubias, 2011; Hankivsky et al., 2017, 2010). Although social concepts do not have to be the central focus of quantitative intersectional studies. Biological topics, especially of health and disease, are often found at the centre of intersectional studies seeking to understand health inequity, find new ways to promote social justice for health, and avoid isolated biological reductionism of participants (Bauer & Scheim, 2019b; Hankivsky et al., 2017; Sun et al., 2018; VanderWeele & Knol, 2014; Wemrell et al., 2017). As well, identifying associations of disease prevalence and causation, as seen in this study, is a familiar pursuit of quantitative intersectionality (Axelsson Fisk et al., 2018; Bauer, 2014; Bauer & Scheim, 2019b; Kelly, 2009; Richman & Zucker, 2020; VanderWeele & Knol, 2014). Complexity and seemingly biological dominance of a disease's etiology should not deter use of intersectionality, as this methodology can leverage new treatment options that have previously been discounted or are completely undiscovered (VanderWeele & Knol, 2014).

Designed by Statistics Canada, the Canadian Health Measures Survey (CHMS) was a survey designed to capture information regarding Canadians' health and lifestyle habits. The CHMS was not designed or conceptualized to encompass distinct intersectional dimensions or related intersectional assumptions (Bowleg, 2008; Hankivsky et al., 2017). However, quantitative intersectionality has been incorporated into this study through the conception of research questions, selection of statistical modelling, and interpretation of results. Specifically, predictors of childhood asthma were conceived, analyzed, and interpreted within both multi-dimensional and sociohistorical contexts to reflect intersectionality (Bauer, 2014; Bowleg, 2008; Green et al., 2017; Hancock, 2007; Hankivsky et al., 2017). The statistical functions of logistic regression and mediation analysis were selected as they exceed the assumption that all variables are equidistant and delineate associations more closely to the principles of intersectionality (Axelsson Fisk et al., 2018; Bauer, 2014; Hankivsky et al., 2017; Hinze et al., 2012; Longman Marcellin et al., 2013; Seng et al., 2012; Veenstra, 2011; Weitkunat & Wildner, 2002). Discussion of study results, again drawing on conceptual aspects of intersectionality, endeavored to observe results at a deeper level than comparing demographic and social group intersections, and uncover systemic sources of unequal power for Canadian children (Bauer, 2014; Bowleg, 2008). The principle tenets of intersectionality, interdependence and multi-dimensionality, have also been used throughout this manuscript to reframe past research of childhood asthma (Bowleg, 2008, 2012). Finally, intersectionality aides the central rationale of this study by providing a structure to integrate biological and social factors influencing childhood asthma (Bauer, 2014; Evans & Erickson, 2019; Hankivsky et al., 2017; Kelly, 2009; Sun et al., 2018).

2.3 Research Questions

This study addressed three principal research questions:

a. What are the associations of asthma, PTB, and SES in childhood?

b. What are the major predictors, biological and social, of childhood asthma in Canada?

c. Is the relationship of PTB with childhood asthma mediated by SES?

2.4 Methods

2.4.1 Study design

In this study, data from Cycle 2 (2009-2011), Cycle 3 (2012-2013), Cycle 4 (2014-2015), and Cycle 5 (2016-2017) of the CHMS were pooled into one dataset. Launched in 2007, CHMS is an ongoing, national cross-sectional survey where respondents were asked to complete both a household interview and mobile clinic visit (Statistics Canada, 2018a). Through household interviews, CHMS collected information related to health outcomes, medical history, current health status, lifestyle, and environmental characteristics. The second part of the CHMS, mobile clinic visits, obtained direct physical health measures like blood pressure, height, weight, physical fitness, and blood samples were obtained. The target population of the CHMS was respondents 3 to 79 years living in private dwellings across Canada's ten provinces. Canada's three territories, reserves or settlements of Indigenous peoples, institutionalized residents, and full-time members of the Canadian Forces were purposely excluded from data collection. As such, the multi-stage sampling process utilized by the CHMS to select respondents, which considers population size, geographic region, household dwelling, and age-group, generated a sample representative of 96% of all Canadians (Thielman, Harrington, Rosella, & Manson, 2018). Additional information about CHMS data collection and sampling procedures are published elsewhere (Statistics Canada, 2018a, 2018b).

Only results from the household interview of the CHMS were required to inform the variables of this study. The CHMS household interview is unique to other data sets, as both current health information and retrospective birth information was collected among children three to 11 years of age, allowing associations to be made between current health and birth outcomes. As well, the CHMS dataset allows researchers to project childhood asthma and other childhood illnesses at the level of the Canadian population being a nationally representative survey in sampling and frequency weighting.

2.4.2 Ethics

Data collection of the CHMS were reviewed and approved by the Health Canada Research Ethics Board to ensure that internationally recognized ethical standards for human research were met and maintained (Statistics Canada, 2018d). Confidentiality and disclosure of results during data collection and analysis was authorized by the Statistics Act.

Western University Office of Human Research Ethics, on behalf of Western's Research Ethics Boards, does not require board review for secondary data analysis (Research Western, n.d.).

2.4.3 Location of research

Data analysis was conducted at the Research Data Centre (RDC) of Western University, London, Ontario, Canada.

2.4.4 Variables

This study examines fourteen variables derived from seventeen original variables of the RDC CHMS master file. As no public use file exists for the CHMS, Appendix A provides a detailed overview of how data was presented and abstracted from the master file. After recoding, this study's focal variables were as follows:

- childhood asthma as the dependent variable;
- prematurity, highest household education and total household income as independent variables;
- age, living arrangement, racial group, and sex as sociodemographics;
- birthweight, body mass index (BMI), exclusive breastfeeding, maternal smoking in pregnancy, and neonatal unit admission as cofounders.

All variables in this study, obtained in the household interview, were self-reported by an adult and assisted by the child when applicable. Answers relating to asthma diagnosis, BMI, education, household size, and income reflected respondent's situation at

the time of the household interview. Personal interviews are considered a popular data collection method to draw meaningful conclusions on a variety of topics from respondents (Bowling, 2005; van de Mortel, 2008). Self-reported answers from face-to-face interviews tend to be more complete, but can be influenced by social desirability and interviewer bias with greater frequency than answers from self-administered survey modes (Bowling, 2005; van de Mortel, 2008).

Childhood asthma. Presence of childhood asthma, this study's outcome of interest, was measured based on the question, "Do you have asthma?" Respondents could answer either "yes", "no", or "don't know". Measuring asthma prevalence by self-report is cornerstone in population-based studies like the CHMS, as this allows for a larger number of respondents to be reached at a lower cost and in less time (Asher & Pearce, 2014). Clinical respiratory testing (spirometry) and assessment of asthma-like symptoms (wheezing), which are both alternative strategies to measuring asthma prevalence, were performed as a part of the CHMS mobile clinic visits (Statistics Canada, n.d.-a). However, these variables were excluded from this study as spirometry was not performed on children less than six years of age, an age group that often provides unreliable results, and the specificity of asthma-like symptoms for the indication of asthma is lower than self-reported asthma prevalence (Global Initiative For Asthma, 2018; Lukrafka et al., 2010; Pekkanen & Pearce, 1999; Statistics Canada, n.d.-a; Yang, To, Foty, Stieb, & Dell, 2011).

Prematurity. Gestational age at birth was measured by the CHMS based on caregiver reports of the number of days before or after the due date the child was born. Then these survey results were recoded into three categories of prematurity: early preterm (gestation 33 weeks, 6 days or less), late preterm (gestation 34 to 36 weeks, 6 days), and term (gestation 37 to 42 weeks; Catt, Chadha, Tang, Palmquist, & Lange, 2016). Defining PTB under clinical standards was an intentional approach to create results relevant to patients and healthcare professionals.

Socioeconomics. SES, or economic factors that influence an individual's position in society, has been predominantly measured by education, income, and occupation

(Duncan, Daly, McDonough, & Williams, 2002; Galobardes et al., 2007). Since children are in progress of completing their education and do not generate income, household education and income are frequently used measures of SES among children (Currie, Elton, Todd, & Platt, 1997; Currie & Goodman, 2010; Fotso & Kuate-Defo, 2005; Whitaker & Orzol, 2006). Household education can assist in predicting a child's future transition from a caregiver's SES to their own, and household income best indicates the material resources available to a child (Duncan et al., 2002; Galobardes et al., 2007). Additionally, household education and income have each been significantly associated with childhood asthma in past studies (Abe, Shapiro-Mendoza, Hall, & Satten, 2010; Goyal, Fiks, & Lorch, 2011; Hsu et al., 2015). Household education was measured by Statistics Canada (n.d.-a) as the highest level of education between household members. Statistics Canada (n.d.-a) measured household income by asking parents to estimate the total income received by all household members and from all sources before taxes in one calendar year (Statistics Canada, n.d.-a). To better reflect the income accessible to individuals within a household, income was adjusted based on household income by dividing total household income by the square root of the household size (Duncan, Daly, Mcdonough, & Williams, 2002; Humphries & van Doorslaer, 2000; Rodd, Feely, Dart, Sharma, & McGavock, 2018). Occupation was excluded as a measure of SES in this study due to the inability to link caregiver's occupation to children in the CHMS (Polasnka et al., 2015).

Sociodemographics. Age, living arrangement, racial group, and sex comprised the demographics of this study. Primarily, these demographics were used to adjust the association between dependent and independent variables, which better represents real-world effects (Kellar & Kelvin, 2013).

As children with asthma in early and middle childhood were the central focus of this research, respondents greater than twelve years of age were excluded from analysis, and remaining respondents were stratified (3 to 5 years and 6 to 11 years; Beauregard, Drews-botsch, Sales, Flanders, & Kramer, 2018). Age is an important consideration, as risk factors and etiology of asthma is believed to vary between early and middle childhood (Global Initiative For Asthma, 2018). These two age groups also follow the

CHMS approach to sampling stratification and data collection (Statistics Canada, 2018a). For example, data on birthweight and prematurity was not collected among respondents 12 years of age or greater (Statistics Canada, n.d.-a). Cycle 1 of the CHMS did not capture children age 3 to 5 years, and hence, could not be pooled for this study (Statistics Canada, 2010). The CHMS variable of respondent's living arrangement was asked across all age groups, and hence not all answers are applicable to children. To remove these inapplicable answers and better delineate the separate effects parents and siblings have upon childhood asthma, this variable of living arrangement was recoded into two new variables.

Racial group and sex were also categorical CHMS variables. Race in Cycles 3 to 5 of the CHMS was more expansively categorized than in Cycle 2 (Statistics Canada, n.d.-b). To pursue variable consistency and allow accurate pooling of data, Cycles 3 to 5 coding of racial groups was collapsed to match the five categories of Cycle 2. Sex was a binary coding of male and female only.

Confounders. Birthweight, body mass index (BMI), exclusive breastfeeding for three months, maternal smoking in pregnancy, and neonatal unit admission were selected as confounders due to their previously established associations with childhood asthma (Jaakkola & Gissler, 2004; Lydersen, 2015).

Birthweight was answered by CHMS respondents in grams or pounds and ounces. This variable, using Health Canada's *Birthweight for gestational age in completed weeks* guideline, was recoded into the categories of small for gestational for age (SGA, $< 10^{th}$ percentile), appropriate for gestational age (AGA, 10^{th}-90^{th} percentile), and large for gestational age (LGA, $> 90^{th}$ percentile; Health Canada, 2004; Kramer et al., 2001; Synnes et al., 2017). Gestational birthweight in Canada only accounts for differences in sex, and does not currently account for ethnicity (Chavkin, Wainstock, Sheiner, Sergienko, & Walfisch, 2019). SGA and LGA are considered significant risk factors for fetal and newborn morbidity, and SGA has been linked as a risk factor of childhood asthma (Brooks, Byrd, Weitzman, Auinger, & McBride, 2001; Carter, Woolcott, Liu, & Kuhle, 2019; Chavkin et al., 2019; Lowe, Kotecha, Watkins, & Kotecha, 2018).

In past research, a link between elevated BMI, especially obesity, and childhood asthma has been verified (Longo et al., 2017). Although classifying BMI among children is difficult, as children undergo a number of physiological and normal changes in weight as they grow (Sweeting, 2007; World Health Organization, 2019). Typical methods to classify BMI among children are as follows: Centers for Disease Control and Prevention (CDC), which uses age and sex specific percentiles for cut points; the International Obesity Task Force (IOTF), which maintains adult cut points are suitable for children; and the World Health Organization (WHO), which relies on standard deviations of global growth standards for cut points (Cole, Bellizzi, Flegal, & Dietz WH, 2000; Grummer-Strawn, Reinold, & Krebs, 2010; Kuczmarski et al., 2000; Monasta, Lobstein, Cole, Vignerov, & Cattaneo, 2011; Shields & Tremblay, 2010). Since 2004, Statistics Canada has primarily measured BMI among children using the IOTF classification, but the CHMS offers BMI variables classified with CDC, IOTF, and WHO guidelines (Roberts, Shields, de Groh, Aziz, & Gilbert, 2012; Statistics Canada, n.d.-b, 2010).The BMI variable using CDC classification was selected for this study due to consistency across Cycles (Statistics Canada, n.d.-b, 2010). CDC classification is considered an accurate measure and conservative estimate of childhood obesity at the population level (Reilly, Kelly, & Wilson, 2010; Roberts et al., 2012; Shields & Tremblay, 2010). Appendix B displays the percentiles used by the CDC to categorize childhood obesity.

Exclusive breastfeeding for three months, neonatal admission, and smoking in pregnancy were measured by the questions, “For how long was [Respondent Name] fed only breast milk?”; “Was [Respondent Name] admitted to a special neonatal unit or an intensive care unit immediately following birth, before [he/she] left the hospital?”; and “Did [you/she] smoke during [your/her] pregnancy with [Respondent Name]?” The answers provided to these questions were dichotomous, coded as yes or no.

2.4.5 Statistical Analysis

Previous to statistical analysis, G*Power 3.1 was used to conduct a power analysis (Faul, Erdfelder, Lang, & Buchner, 2007; Lydersen, 2015). The analysis determined only 308 respondents are required, under a 0.05 alpha and 0.8 power, to achieve statistically significant results in this study (Faul et al., 2007; NCSS, n.d.). An

estimated 1580 respondents aged 3 to 11 years took part in Cycle 4 alone (Statistics Canada, n.d.-a). However, pooling Cycles 2 to 5 of the CHMS was deemed necessary to improve the study's precision and representation of asthma and PTB rates (Statistics Canada, 2018c; Zugna et al., 2015). With approximately 10% of Canadian children experiencing asthma symptoms and 8% of Canadian births being preterm, a larger sample size is required to accurately analyze asthma and PTB (Asher & Pearce, 2014; Shah et al., 2018; Statistics Canada, 2016; The National Asthma Control Task Force, 2000; To et al., 2006). Pooling Cycles additionally ensured missing data would not impede study power.

Cleaning the data, respondents were excluded if they were born outside of 20 to 42 weeks, their reported birth weight was implausible, or had no response to the variable of asthma (Beauregard et al., 2018). Other variables with missing data, including birthweight, BMI, breastfeeding, gestation, household education, living arrangement, neonatal admission, cultural or racial group, and smoking in pregnancy, were handled by multiple imputation (Lydersen, 2015). Multiple imputation by chained equations (MICE) was considered an appropriate technique to replace missing data based on *missing-value patterns* tables derived from the dataset and the multilevel nature of missing data (Pedersen et al., 2017; Resche-Rigon & White, 2018; UCLA Institue for Digitial Resarch & Education, n.d.-b). Attempts to identify auxiliary variables were made through correlation, but none of the variables met the 0.4 threshold of association with one another (Pedersen et al., 2017; UCLA Institue for Digitial Resarch & Education, n.d.-b). To avoid estimate bias and accommodate the substantial number of variables missing data, childhood asthma as the outcome of interest was used as the sole auxiliary variable among imputed variables (Hardt, Herke, & Leonhart, 2012; Pedersen et al., 2017). Using chained multiple imputation, the continuous variables of birthweight and gestation were imputed by regression (STATA Press Publication, n.d.-d). After imputation, these continuous variables were recoded into their categorical forms, as discussed in Appendix A. Categorical and binary variables were imputed with multinomial, ordinal, and binary logistic regression specifications (Resche-Rigon & White, 2018; STATA Press Publication, n.d.-c, n.d.-b). For reproducibility, datasets were imputed fifteen times with a reseed number of 1025, and Rubin's rules were applied to pool results over the imputed data sets (Rezvan, Lee, & Simpson, 2015; Rubin, 1987).

Descriptive statistics in the form of univariate frequencies, including the mean, median, and standard deviation (SD) were obtained for all continuous variables. Relative frequencies, mode, and percentages were analyzed as appropriate. As only categorical variables were included in multivariate analysis, Pearson or chi-square of independence was used to test to determine bivariate associations between variables (McHugh, 2013). A variable's association with asthma, as determined during this bivariate analysis, later guided model building for logistic regression and mediation analysis (Ranganathan, Pramesh, & Aggarwal, 2017; Rosas-salazar et al., 2014; Yamakawa et al., 2015). Specifically, variables with a bivariate significance with childhood asthma of $p < 0.1$, and a significance level of $p < 0.005$ with other variables was entered into multivariable regression models (Antwi et al., 2017; Joseph et al., 2018; Ranganathan et al., 2017; Rosas-Salazar et al., 2014; UCLA Institue for Digitial Resarch & Education, n.d.-b; Yamakawa et al., 2015).

To evaluate research question 2, stepwise logistic regression was performed. Directed by the binary nature of the outcome (i.e., presence or absence of asthma or asthma-like symptoms) and the aim to analyze two independent variables, logistic regression was determined a priori to be the most appropriate method to identify major predictors of childhood asthma (Kellar & Kelvin, 2013; Schumacher, Robner, & Vach, 1996). Furthermore, *backward stepwise* regression was selected to establish the importance of variables in relation to predicting childhood asthma through sequenced models (Lewis, 2007; Ranganathan et al., 2017). Initially, all variables significantly associated with childhood asthma in bivariate analysis were placed into the regression model (Ranganathan et al., 2017). In subsequent models, variables that did not contribute to the predictive power of the model, as evidenced by non-significance, were excluded (Antwi et al., 2017; Lewis, 2007). With a dichotomous outcome, odds ratios (ORs) was the measure of effect used to analyze and compare all predictors and models (UCLA Institue for Digitial Resarch & Education, n.d.-a).

Question 3 was last addressed through logit three-way decomposition mediation analysis (Baron & Kenny, 1986; Buis, 2010; Gunzler, Chen, Wu, & Zhang, 2013; Hayes, 2009). Structural equation modelling was considered to conduct mediation analysis, but

due to the nature of this study's data and limitations of structural modelling with a binary outcome, an alternate method was pursued (Buis, 2010; Hicks & Tingley, 2011; Kohler, Karlson, & Holm, 2011). Figure 2 represents the conceptualized path diagram or relationship being explored between PTB, SES, and childhood asthma with SES as a mediator. Despite academic pushback on simple and full mediation for more complex and partial mediation (Hayes, 2009; Naimi, Schnitzer, Moodie, & Bodnar, 2016; Richiardi, Bellocco, & Zugna, 2013; Valeri & Vanderweele, 2013; Vanderweele, 2013), this study has embraced the methods of Baron and Kenney (1986) with enhancements from Breen, Karlson, & Holm (2013); Buis (2010); and Zhao, Lynch, and Chen (2010). Using the Zhao et al. (2010) decision tree to establish appropriate mediation types, the variables of childhood asthma, PTB, and SES met the guidelines for Baron and Kenney's (1986) mediation method (significant direct effect in the same direction). This type of mediation analysis is also supported by theoretical interest a priori, and the Sobel test for significance has been replaced with modern bootstrapping methods (Baron & Kenny, 1986; Rucker, Preacher, Tormala, & Petty, 2011; Zhao et al., 2010). However, simple mediation was conducted under the premise that SES is not the sole mediator of PTB and asthma (Valeri & Vanderweele, 2013). Due to the fact that asthma remains a dichotomous variable, a logit three-way decomposition method was utilized to calculate direct, indirect, and total effects of mediation for SES upon PTB and childhood asthma (Breen et al., 2013; Buis, 2010; Imai, Keele, & Tingley, 2010; Stata Press Publication, 2019; Valeri & Vanderweele, 2013).

Several tests were used to determine the validity and reliability of results. Sensitivity and specificity, both probabilities concerned with accuracy, were measured to understand logistic model detection (Genders et al., 2012). The fit of multivariable logistic models was examined using McFadden's R^2 (Pseudo R^2), the Hosmer-Lemeshow goodness of fit test, and the Akaike information criterion (AIC) and the Bayesian information criterion (BIC, Archer & Lemeshow, 2006; Burnham & Anderson, 2004; Kellar & Kelvin, 2013; McFadden, 1974; Williams, 2015). For models of logistic regression and mediation, bootstrapping of 500 replications was conducted alongside the multivariate analysis (A Stata Press Publication, 2013; Buis, 2010; STATA, n.d.-d). Bootstrapping is a statistical resampling technique that allows researchers to further

examine the accuracy of nonparametric data (Chernick, 2008). Bootstrapping generates standard errors that indicate how precisely and reliably the sample of study participants estimates the wider population (Chernick, 2008; McHugh, 2008). Additionally, bootstrapping can be valuable when data is weighted, as seen with the CHMS. Weighting can falsely lower the variance of results, so utilizing the bootstrap weights provided by Statistics Canada further ensured the internal validity of results (Antwi et al., 2017; Buis, 2010; Imai et al., 2010; Statistics Canada, 2018c).

Statistical analysis was conducted using STATA, version 14.0 (STATA 14.0, 2017). The TRIPOD checklist was used to analyze and report results of this study (Collins, Reitsma, Altman, & Moons, 2014). Finally, RDC staff assisted during statistical analysis and vetted final outcomes to ensure acceptable procedures were applied, and the confidentiality of respondents was maintained.

2.5 Results

Each individual Cycle of the CHMS recruited approximately 5,700 unweighted respondents (Cycle 2 = 6395, Cycle 3 = 5785, Cycle 4 = 5794, Cycle 5 = 6361), with children 18 years and under accounting for an estimated 2,200 respondents per a cycle (Ata, 2014; Khanam, 2017; Mudryj, Riediger, & Bombak, 2019; Statistics Canada, 2018b; Thielman et al., 2018). For Cycles 2, 3, 4, and 5, combined household interviews and mobile clinic response rates were 55.5%, 51.7%, 53.7%, and 45.8% respectively (Ata, 2014; Mudryj et al., 2019; Statistics Canada, 2019; Thielman et al., 2018). The overall response rate for all four Cycles was 51.7%.

Of the children studied by the CHMS, those aged 3 to 11 years represented 9.3 % (n = 3,208,668) of the total respondents. Approximately 9.3% (297,483) of children reported as being asthmatic, of which 57.7% (174,792) experienced an asthma exacerbation in the last year. Examining PTB among respondents, 6.8% (216,842) were preterm with 4.7% (148,979) being late preterm and 2.1% (67,864) were early preterm. Lower middle income (35.7%) and achievement of post-secondary graduation (84.5%) were the largest categories of household SES. Most respondents were White (68.0%), unattached living with others (51.9%), male (51.0%), and the mean age was 7.0 years.

Fetal and infant outcomes portray generally good early life health among respondents, with approximately three quarters of children being unexposed to smoke in pregnancy (88.4%), having an AGA birthweight (73.4%), and not requiring admission at birth (87.0%). Exclusive breastfeeding for the first three months of life occurred only slightly more among respondents than other methods of infant feeding (54.6% versus 45.4%). Analysis of BMI categories presented 57.4% (1,842,105) of respondents as normal weight, 31.1% (997,290) as overweight or obese, and 11.5% (369,273) as underweight. Creating a visual of this study's independent variables, Figure 3 and Figure 4 further compares the trends between prematurity and indicators of SES in early and middle childhood.

In cleaning this study's data, a total of 119,023 weighted respondents were subject to complete case analysis, with 14,461 removed for postdates gestation of greater than 42 weeks, 99,562 excluded for multiple gestation (Lung, Shu, Chiang, & Lin, 2009), and approximately 5,000 removed for missing asthma values (unable to release actual number due to small size of unweighted cell count). Differences among variables with missing values and imputed values were noted. For bivariate analysis, the variables of birthweight, BMI, and household education showed a marked decrease in significance after imputation. However, premature gestation became significant after imputation of missing values. There was no significant difference in the results of logistic and mediation analysis between data with missing values and imputed values.

2.5.1 Characteristics of children with asthma

Among the 297,483 children with asthma in this analysis, most children were aged 6 to 11 years (75%, mean age of 7.4 years), male (61.6%), and nearly three-quarters of the children identified as White (n=214,940, 72.3%). The majority of children with asthma identified as being unattached and living with others (59.4%) followed by living in a two-parent household with siblings (31.6%). Compared with children who did not report having asthma, children with asthma had slightly lower mean gestational age (39.0 weeks versus 38.9 weeks). Additionally, the percentage of premature born children was greater among those who reported having asthma (early preterm 2.6%, late-term 7.4%) than those without (early preterm 2.1%, late preterm 4.4%).

The modes of highest household education (post-secondary graduation), and total adjusted household income (low-middle income) remain unchanged among children with and without asthma. Yet, a greater percent of asthmatic children live in a low income household (16.3% versus 11.3%), and a larger percentage of children without asthma live in a upper income household (9.2% versus 5.2%). Mean household income was higher among children without asthma ($48,912) compared to those with asthma ($42,689) by $6,223. Household make up also varied with a greater percentage of children with asthma reporting household living arrangements of "unattached" (59.4% versus 51.1%) and single parent (8.9% versus 7.8%) households than children without asthma. Household size held at 4.3 people between both children with and without asthma. Table 1 offers further comparison of study variables among children with and without asthma.

2.5.2 Predictors of childhood asthma from logistic modeling

Weighted bivariate analysis revealed a significant association ($p < 0.1$) between all variables and childhood asthma, with the exception of BMI, birthweight, and cultural or racial group (See Table 2 for variable's Pearson p-values). Among other variables, several other significant ($p < 0.005$) relationships emerged in bivariate analysis. Premature gestation was significantly associated with admission to a neonatal unit ($p < 0.0001$) and birthweight ($p < 0.0001$). Highest household education was significantly associated with exclusive breastfeeding for three months ($p < 0.0001$), household living arrangements ($p < 0.0001$, presence of siblings and parents), smoking during pregnancy ($p < 0.0001$), and total adjusted household income ($p < 0.0001$). Total adjusted household income was significantly associated with exclusive breastfeeding for three months ($p < 0.005$), household living arrangements ($p < 0.0001$, presence of parents and siblings), cultural or racial group ($p < 0.0001$), and smoking during pregnancy ($p < 0.0001$). From bivariate analysis, a total of nine variables were deemed significantly associated with childhood asthma, and were considered appropriate for stepwise logistic modelling.

Three backwards stepwise logistic regression models were constructed to reveal major predictors of childhood asthma in Canada. All three models were tested for fit using the McFadden or Pseudo R^2, and the Hosmer-Lemeshow test of fit (Archer & Lemeshow, 2006; Kellar & Kelvin, 2013). Resulting measures of fit included low

McFadden R^2 values (Model 1 = 0.8, Model 2 = 0.6, and Model 3 = 0.4), and significant Hosmer-Lemeshow tests ($p < 0.0001$), each indicating poor fit across the three models. However, using the AIC and the BIC between models revealed smaller values with subsequent regressions (Model 2 = 3956, 4018; Model 3 = 3744, 3778). Such findings indicate increasing parsimony in this study's final model (Burnham & Anderson, 2004). Table 3 summarizes the ORs of all study variables entered into the first logistic model.

Within the final logistic model, onset of childhood asthma was most significantly associated with middle childhood age (6 to 11 years). Children in middle childhood were specifically 1.7 (95% CI, 1.1- 2.5) times more likely to experience asthma onset than children in early childhood (3 to 5 years). While female sex (OR 0.62; 95% CI, 0.44-0.90), upper adjusted household income (OR 0.43; 95% CI, 0.19-0.96), and presence of siblings (OR 0.70; 95% CI, 0.50-0.99) were significantly protective of childhood asthma onset at odds ratios less than one (Szumilas, 2010). As such, female respondents and high-income respondents living with siblings were 62% and 43% less likely to experience childhood asthma onset, respectively. The overall rate of correct classification in this model was estimated to be 91.2%, with 100% of the normal weight group correctly classified (specificity).

2.5.3 SES as a mediator in logit decomposition

Evaluating the null hypothesis that SES does not act as a full mediator of the association between PTB and childhood asthma, two three-way decomposition logit models were created (Buis, 2010; Erikson, Goldthorpe, Jackson, Yaish, & Cox, 2005). Two decomposition models were required as highest household education, and total adjusted household income were modelled alternatively as observed mediators. Forming a latent variable of SES to concurrently analyze education and income as mediators posed challenges to model convergence. Model simplification, as recommended by STATA, was thus conducted to make the model orthogonal, independent, and more likely to converge (STATA Press Publication, n.d.-a). Deriving indirect and direct effects, predicted and counterfactual proportions/odds, and standard errors, the mediation between highest household education or total adjusted household income, and PTB and childhood asthma was calculated (Buis, 2010; Erikson et al., 2005; Hicks & Tingley,

2011). Unlike other methods of mediation analysis among binary outcome datasets, Buis' (2010) *ldecomp* command did not require a comparison of logit and probit mediation models (Breen et al., 2013; Imai et al., 2010; Karlson, Holm, & Breen, 2012). Findings from the two logit decomposition models were interpreted as direct, indirect, and totals effect. Direct effects representing the association between PTB and childhood asthma, indirect effects accounted for the mediation of SES upon PTB and childhood asthma, and total effects the sum of direct and indirect effects (Buis, 2010). No standardization of variance occurred prior to modelling, as these calculations may not impact performance (Rijnhart, Twisk, Eekhout, & Heymans, 2019). Recognizing the limitations of nonlinear mediation analysis, no comparisons were made between indicators of PTB and SES through ratios of effect (Breen et al., 2013; Rijnhart et al., 2019).

Resulting decomposition models revealed that PTB and childhood asthma appears to be partially mediated by the SES indicators of education and income. Using the *products of coefficient* decomposition method, the null hypothesis can be rejected as both household education and income make up a component of the total effect of PTB upon childhood asthma. For highest household education, the indirect effect was found to be 0.6% (0.002/0.479 x 100%, unweighted 0.2%) of the total effect. The indirect effect of total adjusted household income was higher at 3.1% (0.015/0.479 x 100%, unweighted 1.2%) of the total effect. Although the magnitude of these indirect effects are small and may be less sensitive in the presence of other mediators, there was a slightly greater odds of childhood asthma onset among children born preterm based on their SES (Breen et al., 2013; Buis, 2010). Specifically, children born at term had a 0.1% and 0.6% higher odds of getting asthma with the same household education and income as preterm children (indirect effect). Additionally, preterm children had a 1.62 times higher odds childhood asthma onset than children born term when measures of SES were held constant (direct effect). Table 4 and Table 5 further summarize the results of three-way logit decomposition analysis conducted between PTB, SES, and childhood asthma.

From mediation analysis, predicted and counterfactual proportions of the logit model found 9% (unweighted 8.5%) of children born term, and 13.7% (unweighted 13.2%) of children born preterm will develop asthma. If children born term had the same

household education and income levels as children born preterm than 9.1% (unweighted 8.6%) would develop asthma. Therefore, it is deduced that for every one child born term and preterm that does not develop asthma, there are 0.10 (unweighted 0.09) term and 0.2 (unweighted 0.15) preterm children that do develop asthma. These statistics further reinforce that the direct effect of prematurity is stronger than indirect effects of SES, as represented by household education and income, upon childhood asthma. Complete predicted and counterfactual proportions are provided in Table 6.

2.6 Discussion

This study examined the relationships between PTB, SES, and childhood asthma in a nationally representative sample of Canadian children. The specific purpose of this study was to understand the empirical associations of PTB and SES with childhood asthma, as both predictors and SES as a mediator. Bivariate analysis revealed premature gestation, and the SES measures of education and income were independently associated with childhood asthma. However, the strength of these associations waned with exposure to confounding variables. Small, yet, statistically significant protectors of childhood asthma were instead found to be female sex, upper adjusted household income, and presence of siblings. The age of children, six to eleven years of age or in middle childhood, was a significant predictor of childhood asthma onset. Yet, this finding could reflect the challenges of diagnosing asthma in early childhood over the sudden onset of disease between six to eleven years of age (GINA, 2018). Children in early childhood often present with transient respiratory wheezes and are only diagnosed when symptoms progress into older ages (Karlstad, Nafstad, Tverdal, Skurtveit, & Furu, 2010).

Fit of logistic models, as indicated by post-estimation testing, was lacking in this study. Fit can be a helpful determinant of model utility and strength of future associations between variables, but is not definitive a measure of model accuracy (McGill University Faculty of Medicine, n.d.). Caution should be exercised in the application of these models considering their poor fit, but variables entered into this study's model were supported by *a priori* reasoning and the theoretical lens of intersectionality. Such a statistical strategy upholds the aim and methodology of this study to determine which variables are

important to the onset of childhood asthma, and prevents inclusion of extraneous values (Burnham & Anderson, 2004; McGill University Faculty of Medicine, n.d).

A series of Dutch studies previously examined how socioeconomic factors relate to asthma onset in early and middle childhood (Hafkamp-de Groen et al., 2012; Hafkamp-de Groen et al., 2013; Ruijsbroek et al., 2011). All studies found childhood asthma had a significant predictive relationship with the SES measures of maternal education and household income. PTB, as examined by Hafkamp-de Groen et al. (2012) and Hafkamp-de Groen et al. (2013), acted as covariate and significantly contributed to logistic modelling. In comparison, the results outlined in this manuscript differ notably by finding neither the SES measure of education or PTB as major predictors of childhood asthma. Differences in education may be attributable to how this variable was measured. Highest parental education is hypothesized to be less closely related to asthma and other atopic illnesses than maternal education, due to the substantial influence mothers have in childrearing (Hafkamp-de Groen et al., 2012; Prickett & Augustine, 2016; Ruijsbroek et al., 2011; Spencer, 2005; Weber & Haidinger, 2010). A gradual decline in the effect of parental education upon disease onset as a child ages may be another explanation for this study's results. Numerous studies, examining asthma and other childhood illnesses, show a stronger impact from parental education in early childhood than older age categories (Gong et al., 2014; Hafkamp-de Groen et al., 2012; Hafkamp-de Groen et al., 2013; Lajunen, Kaprio, Rose, Pulkkinen, & Silventoinen, 2012). PTB has been found insignificant in several other studies pertaining to childhood asthma (Annesi-Maesano, Moreau, Strachan, 2001; Katz, Pocock, & Strachan, 2003; Villamor, Iliadou, & Cnattingius, 2009). In part, these past results have been accounted for by the challenges of correctly diagnosing early childhood asthma, which may dilute possible associations (Liu et al., 2014). Since participants in early childhood were examined in this study, diagnostic clinical error could have reduced the influence of PTB in a similar manner.

As for mediation analysis, this study's results indicated there was a small indirect effect from SES upon the relationship between PTB and childhood asthma (education 0.6%, income 3.1%). Although these differences seem unsubstantial, this study is the first known analysis to attempt mediation upon the relationship of PTB and SES with

childhood asthma among a representative sample of the Canadian population. This study could serve as a useful reference point to plan and conduct future studies examining SES, birth outcomes, and childhood asthma in Canada. The associations of PTB and SES to childhood asthma are overall likely to continue to be of interest to researchers, as these two factors represent the growing conceptualization of how biological and social elements interact to influence asthma. Combined analysis of biological and social influences hold promise in reducing health inequalities, and increasing the relevance of disease prevention interventions to specific populations (Smit et al., 2015; Watts, 2012; World Health Organization, 2017).

Furthermore, the results of this mediation analysis are consistent with what has been reported in previous research about birth outcomes and childhood asthma being mediated by SES (Hafkamp-de Groen et al., 2012; Panico, Stuart, Bartley, & Kelly, 2014; Ruijsbroek et al., 2011; Taylor-Robinson et al., 2016; Wai et al., 2018). However, unlike these past studies, which used another variable like smoking, race, and housing conditions as proxies for SES, in this study SES was measured more directly (Panico et al., 2014; Ruijsbroek et al., 2011; Taylor-Robinson et al., 2016; Wai et al., 2018). It is conceivable that other SES proxy variables may affect the relationship between PTB and childhood asthma, but such associations were not observed in this study. Perhaps, differences in the magnitude of SES as a mediator may be accounted for in this study by the homogeneity of household education and income variables, which appeared to better represent higher SES households (Martel et al., 2009).

Certain aspects of this mediation analysis were unique to the data provided from the CHMS and thus, directed how statistical analysis was conducted. For instance, during mediation, highest household education and total adjusted household income were treated as separate indicators of SES in relation to childhood asthma. Such a statistical choice, though optimal to the merging of data, may have prevented a cumulative effect from being studied. A recent study conducted in Toronto, Canada, made use of the Material Deprivation Index to study SES and childhood asthma (Simons et al., 2019). The Material Deprivation Index accounts for not only household education and income, but single parenthood, government transfers, unemployment, and homes in need of major

repairs, as well as parental income (Collins, Kim, Grineski, & Clark-Reyna, 2014; Simons et al., 2019). This study's researchers found children living in neighborhoods of high deprivation carried a greater odds (OR 1.03; 95% CI, 1.02-1.05) of requiring a healthcare visit for childhood asthma over a one-year span. The Material Deprivation Index may serve as a more accurate criteria to represent SES, and the full scope of social disparities that can accompany SES in Canada over just household education and income (Simons et al., 2019; Midodzi, Rowe, Majaesic, Saunders, & Senthilselvan, 2010; Zuijdwijk et al., 2013). The results of this study support a SES index or cumulative SES measure in a way by displaying household education and income effect childhood asthma in a similar way. Understanding disparity of SES at a greater, multidimensional level could contribute to the narrative of quantitative intersectionality in furute research.

Strengths of this study include the large sample size, robust analysis, and use of a theoretical lens to locate findings within society. As well, utilizing the CHMS dataset had several important strengths for this study. First, the CHMS provided multiple variables reflective of determinants that have demonstrated association in the past with childhood asthma, allowing disentanglement of a wide variety effects within the Canadian population (Martel et al., 2009). CHMS data was collected prospectively and independently of the outcome under study. Finally, the standardized interviews organized and delivered by the CHMS added objectivity and reliability to the dataset (Boynton & Greenhalgh, 2004).

2.7 Limitations

This study encountered both analytical and methodological limitations. Primarily, the study's conclusions were constrained by the use of self-reported outcomes and a cross-sectional design. Self-reported outcomes are often subject to response bias from social desirability, modelling, transference, memory, and rapport with interviewers (Paulhus & Vazire, 2005). Uncorroborated by physical assessments and measures, self-reported results should be viewed and interpreted with caution (X. Liu et al., 2014; Paulhus & Vazire, 2005). Meanwhile, the CHMS cross-sectional design limited conclusions of causality to association (Niruban, 2014). For example, this study was unable to examine theories of reverse causality that place PTB as a mediator of the

association between preconception SES (Chen et al., 2017; Delpisheh, Brabin, & Brabin, 2006; Harju et al., 2015) and childhood asthma, or the variable trajectories of SES and asthma in childhood without retrospective or longitudinal data (Beasley et al., 2015; Gong, Brew, Sjolander, & Almqvist, 2017; Jaakkola & Gissler, 2004; Leung et al., 2016; Nasreen, Wilk, Mullowney, & Karp, 2019; Schyllert, Andersson, Lindberg, Rönmark, & Hedman, 2018). Other limitations, like missing data, sample weighting, and data constraints, created statistical adversity and may have strained results. Readers are encouraged to consider these limitations when interpreting and utilizing the results of this study.

Of the seventeen CHMS variables examined, thirteen variables were missing data, in greatly varying amounts (0.14% - 53%), as a result of design and item nonresponse (age, household income, household size, and sex were the only variables with no missing data, Statistics Canada, n.d.-b). Missingness of data appeared overall random from monotone patterns, and imputation was conducted to replace most missing values, but this study still remains vulnerable to bias due to the volume of missing data (de Leeuw et al., 2003; UCLA Institue for Digitial Resarch & Education, n.d.-b). Interview answers of "Not stated", "Don't know", "Refusal" and "Valid skip" were not viewed as meaningful and recoded as missing (de Leeuw, Hox, & Huisman, 2003; Statistics Canada, n.d.-b). Although for the variables of racial group and living arrangement, responses of "Other" were maintained as distinct categories. However, the author cannot exclude that missing CHMS data arose from social desirability, recall bias, respondents being unable to identify with the answers provided, or errors in data processing on the part of interviewers (de Leeuw et al., 2003). As a secondary analysis, comprehensive understanding and correction of missing data through pre/posts-tests and follow-ups with respondents or interviewers was not possible (de Leeuw et al., 2003).

Practical and theoretical challenges existed in weighting this study's variables. During data analysis, the design weights provided by Statistics Canada for the collective Cycles of 2 to 5 were unable to be located by the author. In consulting with RDC analysts, the weights were confirmed as absent, and a request to generate these weights was placed directly with Statistics Canada. With time, weights were received and applied

to all descriptive and inferential tests as per the CHMS Data User Guide (Statistics Canada, 2018b). More generally, weighting data in complex statistical models like logistic regression and logit decomposition models can be cause for concern. Weighting data, even in linear regression, can add calculations, change data poststratification, and create ambiguity within results (Bollen, Tueller, & Oberski, 2013; Gelman, 2007). To observe and account for weighting issues, unweighted and weighted data was compared for changes in variance, trimming of weights was minimized, and the weighted nature of the dataset was taken into account when conducting MICE (Bollen et al., 2013; Young & Johnson, n.d.).

During design and execution of this study, data constraints were noted that prevented and reduced certain lines of inquiry. Foremost, this dataset was designed primarily for nationally representative statistics rather than exclusive research of Canadian children, which might have led to missing variables, values, and data considerations (Harju et al., 2015). Examining asthma in late childhood and youth was not pursued as the variables of birthweight, breastfeeding, neonatal admission, and prematurity were only collected among respondents aged three to eleven years in Cycles 2 to 5 of the CHMS (Statistics Canada, n.d.-b; Statistics Canada, 2018a) . Being able to add respondents 12 to 18 years of age to this analysis could have expanded this study's sample by 2,826,537 weighted respondents, and improved the representativeness of childhood asthma in the Canadian population (Schyllert et al., 2018; Sharma, 2017). Moreover, survey responses from biological mothers, who are often considered the most accurate proxy-reporter of birth outcomes and early childhood research, were not received for all respondents (Beauregard et al., 2018; Jaakkola & Gissler, 2004, X. Liu et al., 2014). In this analysis, about 2.4% (n = 79,607) of respondents were identified as having non-biological caregivers act as proxy reporters, and the exact number of reports that were obtained from biological mothers was not measured in the CHMS (Statistics Canada, n.d.-b). Thus, the accuracy and credibility of all variables pertaining to birth outcomes and early childhood health could reasonably be questioned. The representation of respondent's living arrangements (presence of parents and siblings) may too have been lowered in this study with 52% identifying as "unattached individual living with others" and no further clarity provided as to household make up. As for alternate lines of inquiry,

no examination of urban and rural settings, which have well-established associations with childhood asthma, could be performed due to the regional data collection methods used by the CHMS (Marfortt et al., 2018; Statistics Canada, 2018b).

Finally, this study only considered household survey data, and variables of clinical assessment and diagnostics were precluded. These exclusions create challenges in delineating the full effect of confounding variables, like current presence of a wheeze or recurrent respiratory infections, on this study's observed association (Colicino et al., 2019; Statistics Canada, n.d.-a). Yet, inclusion of clinical variables linked with childhood asthma would have likely expanded this study beyond the resources of the author. The CHMS Data User Guide asks researchers to limit the degrees of freedom used during analysis, as the variance from regional sampling is already great, and adding more variance with additional variables may have created increased inaccuracy in results (Statistics Canada, 2018b).

2.8 Implications and Recommendations

This study identified sex, age, adjusted household income, and presence of siblings as predictors of childhood asthma, reinforcing past findings that asthma is caused by a combination of biological and social factors (Cheng & Goodman, 2015). As such, approaches to preventing asthma should be multipronged and reach beyond the confines of healthcare. Greater effort must be made to understand and diminish social inequity associated with SES, which by itself is a complex concept intersecting a wide range of other social categories (Williams, Priest, & Anderson, 2016). Generic interventions targeting SES alone will likely not be enough to create change, and using the principles of intersectionality, interventions that cross multiple societal levels of inequity should be given priority (Steegers, Barker, Steegers-Theunissen, & Williams, 2016; Williams et al., 2016). Data collection and analysis, being that multi-level interventions are new to healthcare, should continue to play a significant role in refining and standardizing interventions before becoming population-based (Gottlieb, Tobey, Cantor, Hessler, & Adler, 2016).

Results of this study also have unique implications for SES, coming from a developed country with publicly funded healthcare, implying economic access to medical care may not be as significant a SES contributor to childhood asthma as previously thought (Gold & Wright, 2005; Seguin et al., 2005; Simons et al., 2019). In fact, a population study from Norway, another country with a universal healthcare system theorized families of preterm children could potentially reduce their long-term indirect costs associated with asthma by prolonged exposure to the healthcare system and health teaching at birth (Trønnes et al., 2013). Other means of reducing the impact of SES upon children must be considered to effectively eliminate this risk factor for childhood asthma. Currently, expanding preconception and antenatal services to women living in poverty and economically disadvantaged areas is being considered in the United States and Europe to reduce various health inequalities common in childhood (Steegers et al., 2016).

2.9 Conclusion

With major predictors of childhood asthma found to be gender, age, income, and presence of siblings, biological and social factors appear to both play an important role in the onset of this chronic disease. Further large-scale epidemiological studies are required to establish true associations between PTB, SES, and childhood asthma. This study only provides a glimpse into the biological and social associations of childhood asthma, and found preliminary evidence related to how these factors interact by mediation.

2.10 Summary

In this large, population-based, nationally representative survey, PTB and SES were analyzed for association with childhood asthma. Canadian children of high-income households experienced a protective effect from childhood asthma. Canadian children born preterm and in low education households do experience some association with childhood asthma, but the significance of these relationships were greatly reduced in the presence of other exposures. Evidence for partial mediation by SES upon PTB and childhood asthma was upheld, but this association was also small in magnitude.

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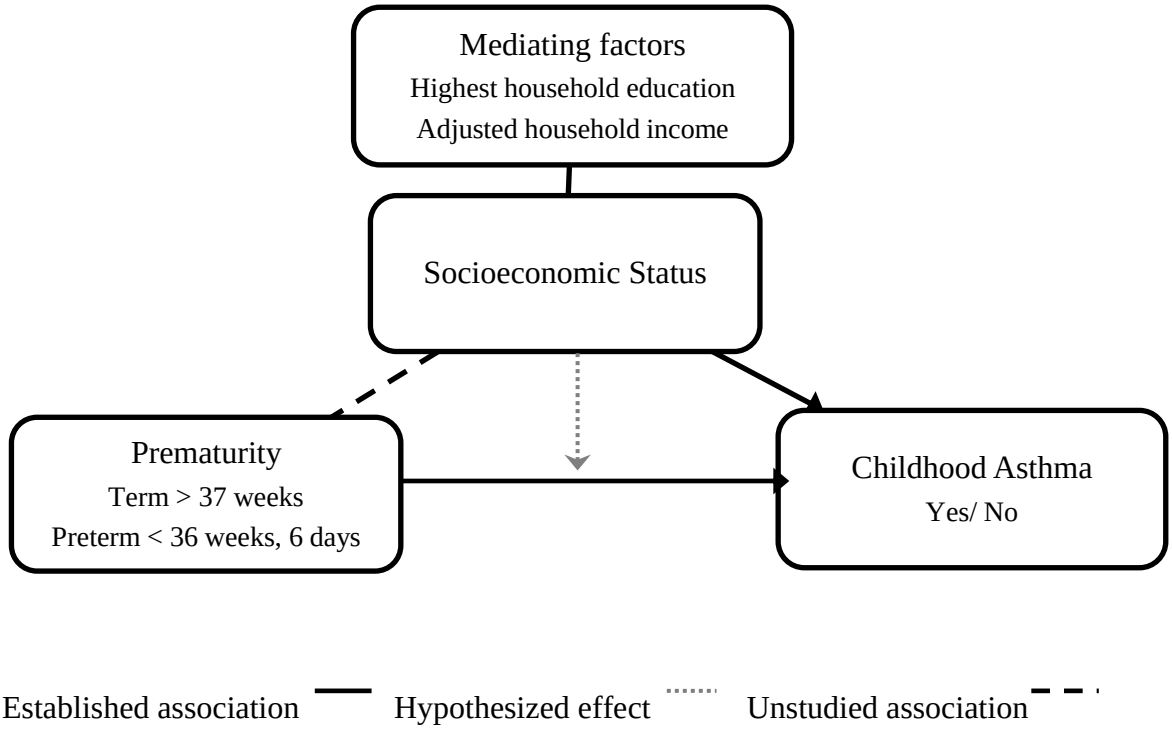
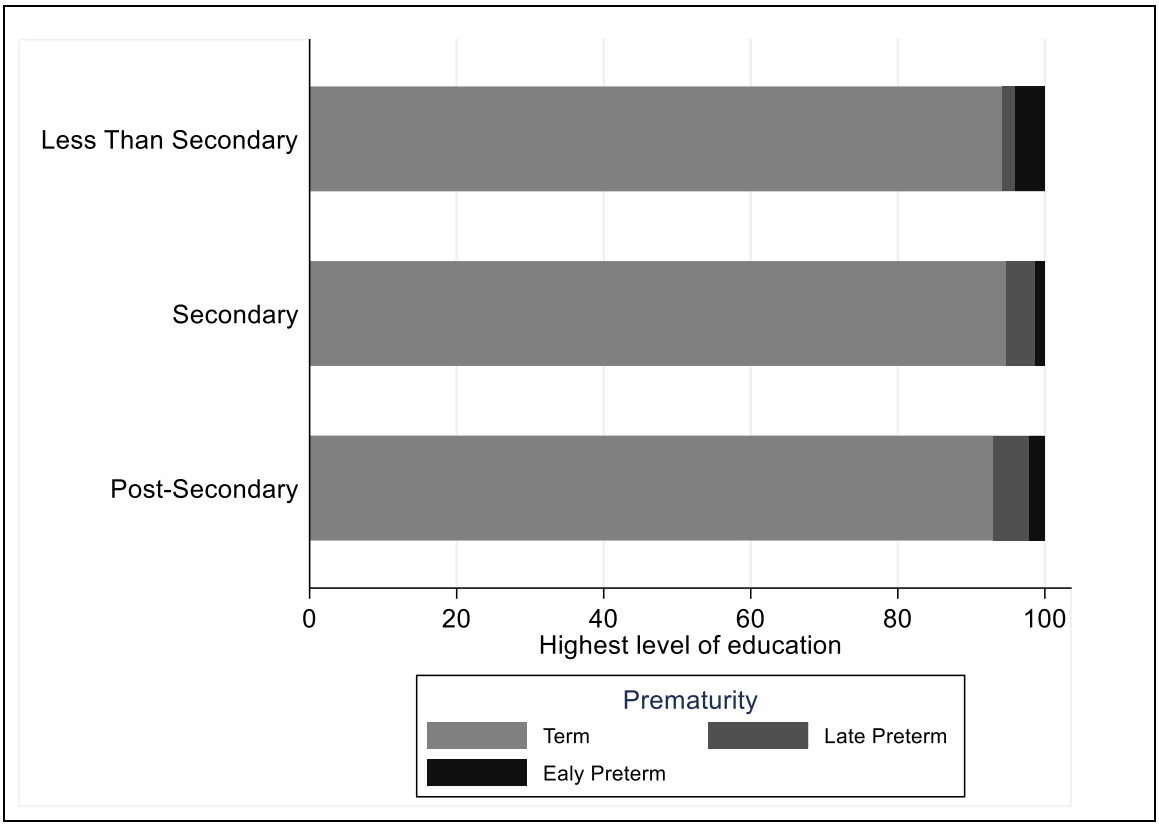


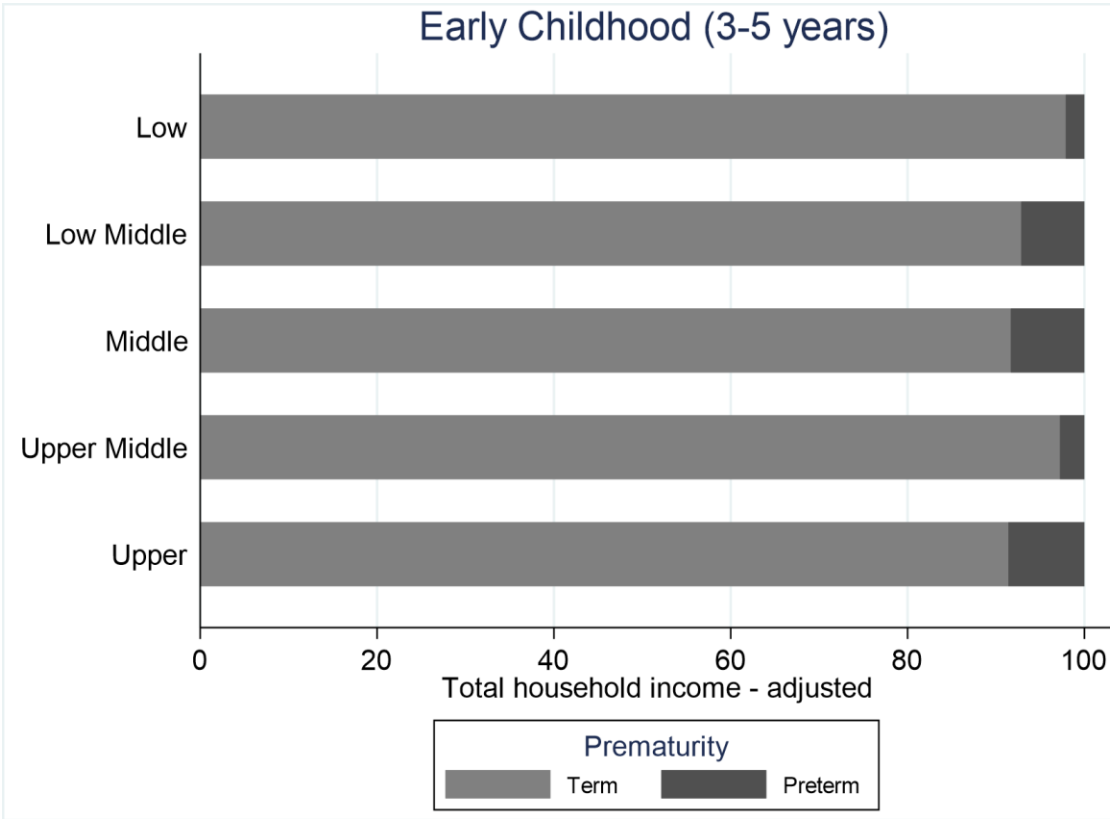
Figure 2: Conceptual framework for relationships between PTB, SES, and childhood asthma

Figure 3: Trends between prematurity and highest household education among all respondents



Note: Unable to release data divided among age groups, as minimum cell counts not met.

Figure 4: Trends between prematurity and total adjusted household income by age group



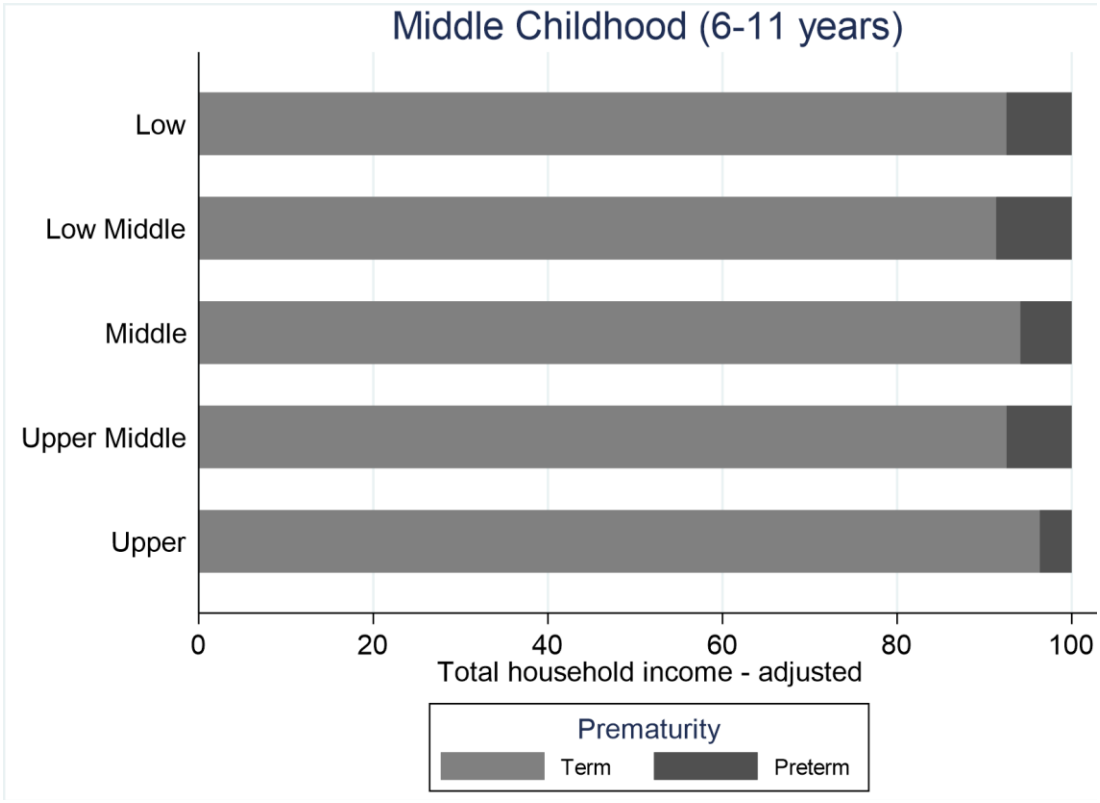


Table 1: Characteristics of age groups with and without asthma

Characteristic	Mean (standard deviation) or frequency (percentage)			
	Early childhood		Middle childhood	
	Asthma	No asthma	Asthma	No asthma
Weighted respondents - Estimated persons in Canadian population	1096609 74429 (6.8)	1022180 (93.2)	2112059 223054 (10.6)	1889005 (89.4)
Age, mean	4.0 (0.8)	4.0 (0.8)	8.5 (1.6)	8.6 (1.7)
Sex, %				
Male	43124 (57.9)	518145 (50.7)	140074 (62.8)	937343 (49.6)
Female	31305 (42.1)	504035 (49.3)	82979 (37.2)	951661 (50.4)
Ethnicity, %				
White	**	684608 (67)	165381 (74.1)	1283869 (68)
Black	**	35794 (3.5)	12400 (5.6)	93339 (4.9)
Hispanic	**	189762 (18.5)	29917 (13.4)	345132 (18.3)
Multicultural & Asian *	**	112016 (11.0)	15356 (6.9)	166664 (8.8)
Parents, %				
Unattached	50200 (67.4)	532799 (52.1)	126632 (56.8)	954554 (50.5)
One-parent	**	80872 (7.9)	23912 (10.7)	147022 (7.8)
Two-parent	**	408509 (40.0)	72510 (32.5)	787430 (41.7)
Siblings, %				
Unattached	50200 (67.4)	532799 (52.1)	126632 (56.8)	954,554 (50.5)
No	4939 (6.6)	85030 (8.3)	15673 (7.0)	100948 (5.4)
Yes	19290 (26.0)	404351 (39.6)	80749 (36.2)	833504 (44.1)
Gestation at birth (weeks), mean	39.2 (1.9)	39 (2.1)	38.8 (2.7)	39 (2.4)
Gestation at birth (term), %				
Term	69881 (93.9)	958683 (93.8)	197896 (88.7)	1765366 (93.5)
Late preterm	**	43800 (4.3)	18712 (8.4)	83228 (4.4)
Early preterm	**	19697 (1.9)	6446 (2.9)	40411 (2.1)
Total preterm	4550 (6.1)	63497 (6.2)	25158 (11.3)	123639 (6.5)
Highest household education, %				
Less than secondary school	**	46162 (4.5)	6165 (2.8)	64316 (3.4)
Secondary school graduation	**	112158 (11.0)	24300 (10.9)	201975 (10.7)
Post-secondary graduation	**	863859 (84.5)	192589 (86.3)	1622715 (85.9)
Household size, mean	4.0 (1.3)	4.3 (1.4)	4.4 (1.4)	4.4 (1.3)
Adjusted household income, mean	42127 (29288)	46970 (31399)	42876 (25819)	49963 (37307)
Adjusted household income, %				
Low	12812 (17.2)	127879 (12.5)	35743 (16.0)	201491 (10.7)
Low-middle	28245 (37.9)	374311 (36.6)	76731 (34.4)	665012 (35.2)
Middle	19492 (26.2)	269792 (26.4)	74763 (33.5)	557124 (29.5)
Upper middle	7996 (10.8)	172212 (16.9)	25198 (11.3)	276917 (14.7)
Upper	5884 (7.9)	77985 (7.6)	10619 (4.8)	188462 (9.9)

Characteristic	Mean (standard deviation) or frequency (percentage)			
	Early childhood		Middle childhood	
	Asthma	No asthma	Asthma	No asthma
Body Mass Index, %				
Underweight	4879 (6.6)	139050 (13.6)	27356 (12.3)	197988 (10.5)
Normal	45550 (61.2)	544294 (53.3)	121874 (54.6)	1130387 (59.8)
Overweight	7028 (9.4)	118909 (11.6)	30622 (13.7)	253597 (13.4)
Obese	16972 (22.8)	219927 (21.5)	43202 (19.4)	307034 (16.3)
Birthweight, mean	3467 (683.7)	3343.5 (606.2)	3330.2 (680.6)	3362.0 (589.5)
Birthweight, %				
SGA	10409 (14.0)	154393 (15.1)	30979 (13.9)	256579 (13.6)
AGA	49666 (66.7)	738057 (72.2)	167525 (75.1)	1398244 (74.0)
LGA	14355 (19.3)	129729 (12.7)	24550 (11.0)	234183 (12.4)
Neonatal unit admission, %				
No	62243 (83.6)	904928 (88.5)	178560 (80.0)	1644775 (87.1)
Yes	12187 (16.4)	117251 (11.5)	44494 (20.0)	244230 (12.9)
Breastfed for at least 3 months, %				
No	31094 (41.8)	441757 (43.2)	112555 (50.5)	871887 (46.2)
Yes	43336 (58.2)	580423 (56.8)	110499 (49.5)	1017119 (53.8)
Smoke exposure in pregnancy, %				
No	64082 (86.1)	923046 (90.3)	183611 (82.3)	1666940 (88.2)
Yes	10347 (13.9)	99134 (9.7)	39443 (17.7)	222066 (11.8)

*Multicultural and Asian values combined due to minimum cell counts not being met.

** Results not able to be released due to minimum cell counts not being met.

Table 2: Potential predictors of childhood asthma identified by bivariate analysis

Block	Predictor	P-value	
		Complete case analysis (n = 2,014,154)	Multiple imputation (n = 3,208,668)
Demographics	Age	0.015	0.015
	Sex	0.013	0.013
	Ethnicity	0.723	0.533
	Parents	0.031	0.049
	Siblings	0.027	0.043
	Gestation at birth	0.224	0.082
SES	Highest household education	0.005	0.009
	Household income	0.178	0.178
	Adjusted household income	0.087	0.087
Confounders	Body mass index	0.410	0.923
	Gestational birthweight	0.358	0.952
	Neonatal unit admission	0.032	0.035
	Breastfed for at least 3 months	0.242	0.301
	Smoke exposure in pregnancy	0.055	0.055

Table 3: Potential predictors of childhood asthma identified by stepwise multivariable logistic regression

Block	Predictor	OR (95% CI)	
		Complete case analysis (n = 2,014,154)	Multiple imputation. (n = 3,208,668)
Demographics	Age		
	Early childhood	(Reference)	(Reference)
	Middle childhood	1.71 (1.68, 1.73)	1.68 (1.67, 1.70)
	Sex		
	Male	(Reference)	(Reference)
	Female	0.79 (0.78, 0.80)	0.64 (0.63, 0.64)
	Ethnicity		
	White	(Reference)	(Reference)
	Black	0.80 (0.78, 0.83)	0.93 (0.92, 0.95)
	Asian	0.82 (0.78,0.86)	0.57 (0.55, 0.59)
	Hispanic	0.80 (0.78, 0.81)	0.79 (0.78, 0.80)
	Multicultural	0.70 (0.68, 0.71)	1.08 (1.07, 1.10)
	Parents		
	Unattached	(Reference)	(Reference)
	One-parent	1.00 (0.98, 1.02)	0.87 (0.49, 1.54)
	Two-parent	0.64 (0.63, 0.65)	0.72 (0.48, 1.07)
	Siblings		
	Unattached	(Reference)	(Reference)
	No	1.64 (1.61, 1.68)	1.08 (1.06, 1.10)
	Yes	omitted	0.71 (0.71, 0.72)
Gestation	Age at birth		
	Term	(Reference)	(Reference)
	Preterm	1.00 (0.97, 1.01)	1.25 (1.23, 1.27)

Block	Predictor	OR (95% CI)	
		Complete case analysis (n = 2,014,154)	Multiple imputation. (n = 3,208,668)
SES	Highest household education		
	Less than secondary school	(Reference)	(Reference)
	Secondary school graduation	1.27 (1.21, 1.32)	1.29 (1.26, 132)
	Post-secondary graduation - other	4.27 (4.04, 4.50)	5.54 (5.36, 5.72)
	Post-secondary graduation	1.58 (1.51, 1.64)	1.81 (1.76, 1.85)
	Household income		
	Low	(Reference)	(Reference)
	Low middle	0.62 (0.60, 0.64)	0.60 (0.59, 0.61)
	Middle	0.46 (0.44, 0.48)	0.77 (0.75, 0.79)
	Upper middle	0.55 (0.52, 0.58)	0.91 (0.88, 0.94)
	Upper	1.05 (1.00, 1.10)	1.43 (1.39, 1.48)
	Adjusted household income		
	Low	(Reference)	(Reference)
	Low middle	1.36 (1.32, 1.41)	0600 (0.59, 0.61)
	Middle	1.42 (1.36, 1.48)	0.44 (0.43, 0.46)
	Upper middle	0.63 (0.60, 0.66)	0.25 (0.25, 0.26)
	Upper	0.43 (0.41, 0.45)	0.20 (0.20, 0.21)
Confounders	Body mass index		
	Under	(Reference)	(Reference)
	Normal	0.75 (0.74, 0.77)	1.03 (1.02, 1.04)
	Over	1.04 (1.02, 1.07)	1.04 (1.03, 1.06)
	Obese	1.07 (1.05, 1.09)	1.15 (1.14, 1.17)
	Gestational birthweight		
	SGA	(Reference)	(Reference)
	AGA	0.66 (0.65, 0.67)	1.01 (1.00, 1.02)
	LGA	0.58 (0.56, 0.59)	0.98 (0.97, 1.00)

Block	Predictor	OR (95% CI)	
		Complete case analysis (n = 2,014,154)	Multiple imputation. (n = 3,208,668)
	Neonatal unit admission		
	No	(Reference)	(Reference)
	Yes	2.01 (1.98, 2.03)	1.41 (1.39, 1.42)
	Breastfed for at least 3 months		
	No	(Reference)	(Reference)
	Yes	1.03 (1.02, 1.04)	1.01 (1.00, 1.02)
	Smoke exposure in pregnancy		
	No	(Reference)	(Reference)
	Yes	1.44 (1.41, 1.46)	1.53 (1.51, 1.55)

Table 4: Decomposition of total effect of PTB on childhood asthma into direct and indirect effects via education

Term/ preterm gestation status origin	Association					
	Method 1			Method 2		
	Coefficient	P-value	95% CI	Coefficient	P-value	95% CI
Total effect	0.488	0.152	0.190, 0.787	-	-	-
Direct effect	0.487	0.152	0.189, 0.786	0.487	0.152	0.189, 0.786
Indirect effect (household education – highest level)	0.0009	0.003	-0.005, 0.007	0.0009	0.003	-0.005, 0.007
Relative effect (indirect through education/ total)	0.001	0.009	-0.017, 0.021	0.001	0.009	-0.017, 0.021

Notes: P-values represent bootstrap standard error (Uggen & Shannon, 2014)

Table 5: Decomposition of total effect of PTB on childhood asthma into direct and indirect effects via income

Term/ preterm gestation status origin	Association					
	Method 1			Method 2		
	Coefficient	P-value	95% CI	Coefficient	P-value	95% CI
Total effect	0.488	0.153	0.187, 0.790	-	-	-
Direct effect	0.483	0.154	0.181, 0.785	0.483	0.154	0.181, 0.785
Indirect effect (total household income-adjusted)	0.005	0.006	-0.005, 0.176	0.005	0.006	-0.005, 0.177
Relative effect (indirect through income/ total)	0.012	0.024	-0.036, 060	0.012	0.024	-0.036, 0.060

Notes: P-values represent bootstrap standard errors (Uggen & Shannon, 2014)

Table 6: Predicted and counterfactual proportions

Distribution	Association			
	Household education – highest level		Total household income – adjusted	
	Term	Preterm	Term	Preterm
Term	0.0895	0.1370	0.0895	0.1350
Preterm	0.0897	0.1370	0.0907	0.1370

Notes: The presented results are percentages. Rows indicate the indirect effect, and columns show the direct effect (Liu, Wagner, Sonnenberg, Wu, & Trautwein, 2014).

Chapter 3 Implications

This study explored the associations between childhood asthma, PTB, and SES in Canada. Predictors of childhood asthma were identified as male sex, low income, presence of siblings, and middle childhood age (6 to 11 years). Plus, logit decomposition revealed partial mediation of the relationship between PTB and asthma by SES. Expounding on the results of chapter two, implications and recommendations for findings are discussed in this chapter. Application of the study's results are expressly discussed in terms of practice and policy, professional education, governance of healthcare systems, theory, and future research.

3.1 Implications for Practice and Policy

Deriving clinical relevance from population-based research can be difficult, and especially so for childhood asthma (Fouzas & Brand, 2013; Gardner & Altman, 1986; Smith et al., 2011). Conferring the risk of childhood asthma at the individual level may be inconsequential if the effects of predictors, this study included, is only observed among population-based groups (Ege et al., 2011; Haughney et al., 2008; Martin et al., 2011; Moffatt et al., 2010; Torgerson et al., 2012; Wi et al., 2017). Statistical significance does not denote clinical relevance either (Gardner & Altman, 1986; Rijnhart et al., 2019). The magnitude of significant associations, total number of associations, and the power of sample sizes that fuel these associations should ultimately apprise the clinical utility of a study's findings (Fouzas & Brand, 2013; Rijnhart et al., 2019; Smith et al., 2011). Reviewing this study's associations, no immediate intervention or change in clinical practice or policy can be recommended. Additional research will be required to alter preventative programming and management of childhood asthma. Though, returning to this study's initial research questions, which were set to explore the clinically compelling issue of causation from both a biological and social standpoint, a broader applicability of these findings emerges at the bedside (Smith et al., 2011).

Ideally, disease causation provides practical direction to healthcare professionals who strive to prevent and manage illness. In reality, the source of disease is often interpreted and merged into clinical practice under the direction of complex philosophical

perspectives or theories (Krieger, 2001). Thus, the components of disease causation are not disembodied notions, but rather triggers to deeper conversations of worldviews, accountability, deprivation, and agency (Krieger, 2001). The longstanding and mainstream theory of disease causation has been the biological model of health (Wade & Halligan, 2004). The biological model dictates that disease arises from an abnormality in the body (ranging from cellular to multi-organ dysfunction), and is the responsibility of the individual (Buettner-Schmidt & Lobo, 2011; Kagan et al., 2014; Krieger, 2001; Matwick & Woodgate, 2016; Rogers & Kelly, 2011; Wade & Halligan, 2004; Wilmot, 2012; Yanicki et al., 2015). The social model of health, a newer theoretical model, offers the explanation that disease is provoked by actual or perceived societal disadvantage, and should be accounted for by all members of a population as a collective (Frankish et al., 2007; Krieger, 2001; Marmot et al., 2020; Wade & Halligan, 2004). Although the biological and social models of health appear to oppose one another and have conflicted in the past, research and healthcare leaders advocate to have these models accepted as one (Frankish et al., 2007; Krieger, 2001; Van De Velde, Eijkelkamp, Peersman, & Vriendt, 2016; Wade & Halligan, 2004). Considered holistically, these two models can expand clinical knowledge and reciprocate into one another to provide a richer understanding of health than either model alone (Collins, 2004; Krieger, 2001; Marmot et al., 2020; Robertson, 1998; Van De Velde et al., 2016; Wade & Halligan, 2004).

This study sought to examine and confirmed both biological and social influences upon childhood asthma. As such, the results of this study contribute to the general, widespread discourse that disease is a product of both biological and social models (Marmot et al., 2020; Wade & Halligan, 2004). Yet, delving deeper, this study also contributes to the unique and ongoing philosophical shift in understanding of health from the bedside. The uptake of a balanced vision of biological and social health, specifically of the social model, has been slow among individual healthcare professionals, facilities, and policy makers (Bury, 2001; Frankish et al., 2007; Van De Velde et al., 2016). This delayed comprehension is particularly concerning among healthcare professionals, as improved understanding of disease processes can improve communication, cultural competence, and the facilitation of resources between healthcare professionals and patients (Bury, 2001; Koffman et al., 2015; Van De Velde et al., 2016). Examining the

major predictors of childhood asthma from this study, a mix of biological and social factors can reveal a more intricate picture of holistic causation to those in the clinical setting. The simplicity of a biological or social health model alone may even be pointedly challenged among healthcare professionals who care for children with asthma, and in turn, alter how they view and react to the illness clinically. Again, this study does not answer what multifaceted steps should be taken to prevent childhood asthma, but directs healthcare professionals to the fact that such multimodal interventions will be needed to address biological and social influences of causation in the future (Frankish et al., 2007).

3.2 Implications for Education

This study reinforces what is already known of childhood asthma causation, and supports educators of healthcare professional's asserting the complexity of asthma causation. Though this study identified several predictors of childhood asthma, for educational purposes these predictors should not be viewed as exclusive. Instead, predictors located in this study should only serve as a starting point for educating healthcare professionals of the biological and social influences upon childhood asthma (Lara et al., 2002). For Canadian educators, this study will hopefully renew interest in the topic of asthma and offer new energy to exploring contextual theories of causation in the classroom setting. However, the primary focus of childhood asthma education for healthcare professionals should continue to be of disease management and treatment, which has greater clinical usability (Global Initiative For Asthma, 2019; Lara et al., 2002).

3.3 Implications for Healthcare Systems and Government

Population-based research has become an increasingly popular means, especially in Canada, for governments and healthcare systems to target and coordinate services (Cohen et al., 2014; Sikka, Morath, & Leape, 2015; Stine, Chokshi, & Gourevitch, 2013; Yan, Kwan, Tan, Thumboo, & Low, 2018). Acknowledging trends in population health can anticipate resources, prevent disease, and prioritize health needs (Department of Health & Social Care, 2015; Lynn, Straube, Bell, Jencks, & Kambic, 2007; Yan et al., 2018). Lowered healthcare expenditures and resource depletion can also be lucrative

motivators for healthcare systems and governments to pursue planning and services based on shifts in population health (Lynn et al., 2007; Neudorf, 2012; Suter, Oelke, Adair, & Armitage, 2009). Managing the growing burden of chronic disease is currently a major focus of population health and research (Canadian Institute for Health Information, 2011; GBD 2016 Disease and Injury Incidence and Prevalence Collaborators, 2017; Yan et al., 2018).

Childhood asthma, as a growing contributor of chronic disease burden, creates a unique disparity within population health (Ding et al., 2015; Samoliński et al., 2012). Conforming healthcare models to tackle risk factors and support best practice care of childhood asthma may advantage healthcare systems and governments striving to prevent and manage this disease (Samoliński et al., 2012; Yan et al., 2018). Results of this study could inform how risk factors of childhood asthma are addressed by strengthening the knowledge of population-based predictors. Healthcare systems and governments of Canada may find this study particularly useful, as incidence and causation of childhood asthma changes geographically (Brozek et al., 2016; Ding et al., 2015; Global Initiative For Asthma, 2019; Ma et al., 2009; Malik, Kumar, & Frieri, 2012; Marfortt et al., 2018; Samoliński et al., 2012; Valet et al., 2016). For example, high household income was a significant protector of childhood asthma and prematurity among respondents of this study and in other Canadian studies (Carter et al., 2019; Simons, Dell, Moineddin, & To, 2018; Simons, Dell, Moineddin, & To, 2019). This finding could indicate to Canadian healthcare systems and governments that intensifying programs to prevent asthma among low-income children could improve national asthma rates (Beasley et al., 2015: Harju et al., 2015; Ramratnam, Bacharier, & Guilbert, 2019, 2019).

3.4 Implications for Theory

Use of quantitative intersectionality appears to be gaining steady popularity and has expanded in application to a wide variety of academic disciplines (Bauer, 2014; Bowleg, 2012; Evans & Erickson, 2019; Lizotte, Mahendran, Churchill, & Bauer, 2019; Moradi & Grzanka, 2017; Sun et al., 2018). Although, the growth of this methodological approach has not occurred without confusion and tension over quantitative conceptualization and mathematical application of intersectionality (Bauer, 2014; Bowleg

& Bauer, 2016; Hankivsky et al., 2010; Moradi & Grzanka, 2017; Nash, 2008; Richman & Zucker, 2020). As such, this study contributes to the growing body of quantitative intersectionality by reinforcing and exemplifying the theoretical framework's utility in terms of health research. Specifically, this study is likely to hold theoretical value among other asthma researchers, being the seminal research application of quantitative intersectionality to childhood asthma, and Canadian researchers, who appear to be aptly latching onto this methodology (Hankivsky & Christoffersen, 2008; Hankivsky et al., 2010; Sun et al., 2018).

Future studies of childhood asthma employing this methodology have a substantial opportunity to build on the intersectional work of this study by rectifying said issues. As a secondary analysis, this study did not address design concerns or achieve set data collection standards associated with intersectionality. Experts of quantitative intersectionality postulate that employing the characteristics of intersectionality throughout design of study questions and data collection protocols will produce more meaningful measurements of intersectionality (Bowleg, 2008; Cole, 2009; Kelly, 2009; Richman & Zucker, 2020). Design features that forthcoming studies may find advantageous to the study of intersectionality include variables measuring institutional discrimination and respondent's perceptions of intersectionality, mathematical stratification of variables, and multi-level analysis (Bowleg, 2008; Evans, 2019; Evans & Erickson, 2019; Hankivsky et al., 2010; Jones, Johnston, & Manley, 2016; Lizotte et al., 2019; Merlo, 2018; Scheim & Bauer, 2019; Sun et al., 2018). SES, the primary social category of interest in this study, may also reveal a greater intersectional effect and capture more relevance to the identities of children with asthma in future research if measured in several more dimensions (Bauer & Scheim, 2019b, 2019a; Bowleg, 2012; Evans & Erickson, 2019; Jackson & Vanderweele, 2019; Sun et al., 2018). For example, accumulated wealth, cost of living, caregiver's occupation, and joint disparity of SES with other social categories like race may reveal new influencers, and an overall greater magnitude of effect upon childhood asthma (Duncan et al., 2002; Galobardes et al., 2007; Jackson & Vanderweele, 2019).

Matching statistical functions to the principles of intersectionality has posed a longstanding issue for quantitative intersectionality (Bauer & Scheim, 2019a, 2019b; Bowleg, 2008, 2012; Evans, 2019; Evans & Erickson, 2019; Lizotte et al., 2019; Richman & Zucker, 2020; Scheim & Bauer, 2019). Currently, a wide array of additive, multiplicative, mediating, and moderating statistical interaction models are being recommended to conduct quantitative intersectionality in the field of population health (Axelsson Fisk et al., 2018; Bauer, 2014; Evans, 2019; Lizotte et al., 2019; Richman & Zucker, 2020). Facilitating consensus on terminology, validity of functions, and interpretation of results among researchers in the field of childhood asthma may be required to advance the methodological development of quantitative intersectionality in this topic area (Bauer, 2014; Evans, 2019; Kelly, 2009; Lizotte et al., 2019; Richman & Zucker, 2020). Prospective studies, in the meantime, should offer explicit reasoning towards the selection of mathematical functions to study quantitative intersectionality, and support their choices with recent literature. Extrapolating on the functions used in this study, researchers should look towards the work of Bauer and Schiem (Bauer & Scheim, 2019a, 2019b; Scheim & Bauer, 2019) to derive more detailed intersectional models using mediation analysis.

3.5 Recommendations for Future Research

A prominent disadvantage of this study, which could be rectified in future research, is the inability to accumulate and compare SES predictor's interactions during mediation. Effect in nonlinear logit decomposition, though a reliable calculator of effect for an individual variable, changes scales in error distribution and variance across models (Karlson et al., 2012; Rijnhart et al., 2019). Hence, adding or comparing variables in two different nonlinear decomposition models, such as highest household education and total adjusted household income, is difficult and cannot be assumed similar to linear models (Breen et al., 2013; Rijnhart et al., 2019). Alternative methods to rescale models exist, but the reliability and the rate of inferential error from these methods remains under investigation (Karlson et al., 2012; Kohler, Karlson, & Holm, 2011). Other dichotomous outcome models can be impacted by unobserved heterogeneity as well, which presents more unique challenges in interpreting the results of nonlinear mediation analysis

(Rijnhart et al., 2019). These issues could be avoided and results optimized with future study designs of nonbinary outcomes and mediators of childhood asthma.

Obtaining prenatal measures of SES, a reflection of the family's socioeconomic status prior to a child's birth, would also enrich the results of future research examining the interaction of predictors of childhood asthma (Beasley et al., 2015; Beauregard et al., 2018). Past studies of childhood health have revealed actual and substitute variables of preconception SES (i.e., prenatal maternal stress and smoking in pregnancy) are meaningfully associated with childhood asthma (Hafkamp-de Groen et al., 2012; Jaakkola & Gissler, 2004; Lee et al., 2016; Lupien, King, Meaney, & McEwen, 2000; Sternthal, Coull, Chiu, Cohen, & Wright, 2011; Wright, 2007), and prematurity (Currie & Goodman, 2010; Joseph, Liston, Dodds, Dahlgren, & Allen, 2007; Pickett, Ahern, Selvin, & Abrams, 2002; Thompson, Irgens, Rasmussen, & Daltveit, 2006). As such, there is support to examine prematurity as a mediator of preconception SES and childhood asthma, and preconception SES as a modifier of childhood SES (Sternthal et al., 2011). Unfortunately for this present study, no preconception measurements of SES were available in the CHMS dataset.

3.6 Conclusion

Childhood asthma, though not well understood, is clearly linked in this research and in other studies to biological and social factors. More scholarship is required to expand and comprehend on this link, but one may hypothesize societal inequality is an emerging area of interest in this subject area. As well, quantitative intersectionality could aide future research of childhood asthma by allowing new aspects of social inequality to be studied statistically and cumulatively. With CHMS cycles ongoing, the dataset used in this study should be considered as a growing source of knowledge on childhood asthma in Canada, and be reviewed in the future for new paths of social association.

For clinical practice and the education of healthcare professionals, this study supports the continued shift from a biological to biopsychosocial model of asthma onset. Healthcare professionals receiving this research should view the findings not as directly applicable to the clinical setting, but rather question how they perceive disease onset and

if these findings evolve that perception. Furthermore, recognizing multimodal interventions will be needed to address the many biological and social influences of childhood asthma causation in the future, healthcare systems and governments should continue to endorse population-based research like this study. Trends of childhood asthma onset observed in nationally representative datasets may offer focused direction for the prevention and reduction of this complex disease.

Summary

This study provides a national depiction of childhood asthma's association with PTB and SES. Findings deepen the understanding of biological and social risk factors of childhood asthma development. Mediation analysis was particularly interesting, showing partial intercession of SES into the relationship of prematurity and asthma. These results underscore the need for diversified understanding of how childhood asthma occurs, and calls for the implementation of a health interventions to prevent childhood asthma incidence. Continued academic exploration of risk factors for childhood asthma will only provide a more complete understanding of this disease for clinicians, healthcare systems, and governments in the future.

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Appendices

Appendix A: Data abstraction of variables from the Canadian Health Measures Survey, household interview

Variable	Name	Value	Comments
Dependent variables			
ccc_11	Has asthma	Yes – 1 No – 2 Don't know – 7	Missing values (% unable to be released due to minimum cell counts not being met), complete case analysis
Demographics			
dhh_age	Age	003-080	Recoded into categories of: 3 to 5 years (early childhood) and 6 to 11 years (middle childhood) (Beauregard et al., 2018) No missing values
dhh_sex	Sex	Male – 1 Female - 2	No missing values
dhhdlvg	Living arrangement	Unattached individual living with others – 2 Child living with single parent – 6 Child living with single parent, siblings – 7 Child living with two parents - 8 Child living with two parents, siblings - 9 Other – 10 Not stated - 99	Recoded into categories of: 1 One or two-parent, unattached 2. Presence of siblings, unattached Codes 1, 3, 4, 5 not applicable responses for children Missing values (5%), multiple imputation
pgdcgt	Cultural or racial group	White – 1 Black – 2	Cycle 2 this variable is spm_22 Cycle 3-5 coded additional values (Korean, Japanese, Filipino,

Variable	Name	Value	Comments
		Asian – 3 Hispanic – 4 Multicultural – 5 Other – 6 Valid skip – 96 Not stated - 99	Chinese, South Asian, Southeast Asian, Arab, West Asian). Recoded to match Cycle 2 and merge. Missing values (12.5%), multiple imputation
Independent variables			
bir_21	Born before, on, or after due date	Before the due date – 1 After the due date – 2 On the due date – 3 Valid skip – 6 Don't know – 7	Recode into categories of : Early preterm (33 weeks, 6 days or less), late preterm (34 to 36, 6 days weeks), and term (37 to 42 weeks) (Catt et al., 2016) Complete case analysis greater than 42 weeks gestation
bir_22	Days before/ after due date	001-091 Valid skip - 996 Don't know - 997 Not stated - 999	bir_21 and bir_22 missing values (1.4%), multiple imputation
thid_14	Total household income - groups	Less than $5000 – 1 $5000 to less than $10,000 - 2 $10,000 to less than $15,000 – 3 $15,000 to less than $20,000 - 4 $20,000 to less than $30,000 – 5 $30,000 to less than $40,000 – 6	Recode into total household income into categories of: Low, low-middle, upper middle, and upper (adjusting for household size; Rodd et al., 2018) Household income adjusted by dividing total household income by the square root of the household size (Humphries & van Doorslaer, 2000) Cycle 2 variable name inc_21 -value is continuous ($)

Variable	Name	Value	Comments
		$40,000 to less than $50,000 – 7 $50,000 to less than $60,000 – 8 $60,000 to less than $70,000 - 9 $70,000 to less than $80,000 - 10 $80,000 to less than $90,000 - 11 $90,000 to less than $100,000 - 12 $100,000 to less than $150,000 - 13 More than $150,000 – 14	Cycle 5 variable name inc_hhld -value is continuous ($) All four cycles had partially (12.1%) and fully imputed (12%) household income values (Statistics Canada, 2018a).
dhhdhsz	Household size	01-15	dhhdhsz - number of persons 16 or 17 years old in household No missing values
edudh04	Highest level of education – household	Less than secondary school graduation – 1 Secondary school graduation – 2 Post-secondary graduation other - 3 Post-secondary graduation – 4 Not stated - 9	Code value 3 not available in Cycle 3 and 4 Missing values (3%), multiple imputation
Confounders			
bir_12	Birth weight -pounds	01-12	Recode into gestation and gender-specific quartiles unique to Canada

Variable	Name	Value	Comments
bir_13	-ounces	00-15	(Health Canada, 2004; Kramer et al., 2001).
bir_14	-grams	0940-5400 Valid skip – 96/9996 Not stated – 99/9999	Recode into three categories of: large for gestational age, appropriate for gestational age, and small for gestational age. Missing values (4%), multiple imputation
bir_24	Admitted to neonatal unit	Yes – 1 No – 2 Valid skip – 6 Don't know – 7 Not stated – 9	Missing values (0.4%), multiple imputation
brifeb6	Exclusive breastfeeding for 3 months	Yes – 1 No – 2 Valid skip – 6 Not stated - 9	Missing values (3%), multiple imputation
hwtdbmik	BMI norms	Under – 1 Normal – 2 Over – 3 Obese – 4 Valid skip – 6 Not stated -9	Only collected among 3 to 17 years old Missing values (37.2%), multiple imputation
prg_11	Smoked during pregnancy	Yes – 1 No – 2 Valid skip – 6 Don't know - 7	Missing values (2.6%), multiple imputation

*all values self-reported (Statistics Canada, n.d.-a)

Appendix B: CDC definition of childhood obesity

Weight status category	Percentile range
Underweight	Less than the 5th percentile
Normal or healthy weight	5th percentile to less than the 85th percentile
Overweight	85th to less than the 95th percentile
Obese	95th percentile or greater

(Centers for Disease Control and Prevention, 2018; Kuczmarski et al., 2000)

Curriculum Vitae

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