SMALL AREA RATE VARIATION IN CARDIOVASCULAR DISEASE PREVALENCE IN NOVA SCOTIA, CANADA

by

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Submitted in partial fulfilment of the requirements
for the degree of Master of Science

at

Dalhousie University
Halifax, Nova Scotia
July 2017

DEDICATION

To the many individuals who have been impacted by cardiovascular disease, and those who work vigorously to improve cardiovascular health outcomes.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

This study assessed small area rate variation (SARV) in cardiovascular disease (CVD) prevalence in Nova Scotia, Canada. It described how estimates varied according to population demographics and estimated the contribution of area socioeconomic status (SES) to between-area variation. Applying multilevel logistic regression to healthcare utilization databases, pronounced spatial variation was observed across forward sortation areas (FSAs) in the prevalence of ischemic heart disease, congestive heart failure, and hypertension. Pockets of FSAs were identified with above-average and below-average prevalence estimates. Age-sex standardization resulted in noticeably different estimates in many areas. Stratified comparisons between relatively older and younger age groups revealed SARV that generally followed variation in the overall population, although younger age groups typically had greater variation in prevalence. Statistical associations were observed between area-level variables and variation in prevalence. This study contributes to the knowledge base on CVD and has policy implications for a disease that significantly impacts Nova Scotia.

LIST OF ABBREVIATIONS USED

AMI	Acute Myocardial Infarction
BMI	Body Mass Index
CABG	Coronary Artery Bypass Graft
CAD	Coronary Artery Disease
CCDSS	Canadian Chronic Disease Surveillance System
CCHS	Canadian Community Health Survey
CCI	Canadian Classification of Health Interventions
CHD	Coronary Heart Disease
CHF	Congestive Heart Failure
CHHS-AP	Canadian Heart Health Strategy and Action Plan
CI	Confidence Interval
CIHI	Canadian Institute for Health Information
CRI	Credible Interval
CVD	Cardiovascular Disease
CVHNS	Cardiovascular Health Nova Scotia
DAD	Discharge Abstract Database
DHA	District Health Authority
EBIC	Economic Burden of Illness in Canada
EBLUP	Empirical Best Linear Unbiased Predictor
FSA	Forward Sortation Area
GIS	Geographic Information Systems
ICD	International Statistical Classification of Diseases and Related Health Problems
ICONS	Improving Cardiovascular Outcomes in Nova Scotia
IHD	Ischemic Heart Disease
IOR	Interval Odds Ratio
MAUP	Modifiable Areal Unit Problem
MED	MSI Physician's Billings
MOR	Median Odds Ratio
MSI	Medical Services Insurance
MSSU	Maritime SPOR SUPPORT Unit
NPV	Negative Predictive Value
NS-PHICI	Nova Scotia Primary and Integrated Health Care Innovations Network
OLS	Ordinary Least Squares
OR	Odds Ratio
PCI	Percutaneous Coronary Intervention
PHAC	Public Health Agency of Canada
PPV	Positive Predictive Value

PYLL	Potential Years of Life Lost
SARV	Small Area Rate Variation
SES	Socioeconomic Status
TIA	Transient Ischemic Attack
VPC	Variance Partition Coefficient

ACKNOWLEDGEMENTS

I would like to give thanks to the many individuals whose guidance and assistance have supported me throughout my Master's degree. First and foremost, I owe my sincerest gratitude to my supervisor Dr. George Kephart. George's enthusiasm for research and teaching in research methods were as pleasurable to witness as they were infectious. Moreover, his mentorship and encouragement have made this thesis a possibility and deserve significant credit in helping to propel this work throughout its various stages of completion. I would like to thank my supervisory committee of Dr. Mikiko Terashima and Dr. Iqbal Bata. Mikiko and Iqbal provided insightful comments and suggestions from health planning and clinical perspectives throughout the work that helped shape the thesis into its final form, for which I am extremely grateful. A special thanks to Mikiko and Dr. Pantelis Andreou for their assistance in the analysis phase of my thesis, volunteering their expertise and making noteworthy contributions to the end product of my results.

I would like to thank Health Data Nova Scotia and the Nova Scotia Department of Health and Wellness for granting me access to its population-based, healthcare utilization databases that enabled the completion of this work. I would also like to thank the faculty and staff in the Department of Community Health and Epidemiology for their support throughout my studies, with a special thank you to Dr. Kathy MacPherson, Tina Bowdridge, Kasia Wolkowicz, and Jodie Lawrence for all of their technical support.

I would like to thank my family and friends for all of their support throughout my studies. This is especially meant for my dad Józef and my partner Monica, whose encouragement and love were unwavering throughout this journey. Finally, to my late mother Joanna, who always encouraged me academically, and whose memory remains alive today through the way in which I strive to live and conduct my life. Thank you all.

CHAPTER 1. INTRODUCTION

Cardiovascular disease (CVD) is one of Canada's most pressing health challenges. CVD and its component conditions are responsible for nearly 30% of all Canadian deaths each year, with 1.6 million Canadians living with heart disease or the effects of stroke. [1] This manifests into vast economic consequences, with CVD estimated to cost the Canadian economy $12.3 billion per year. [2,3] However, some regions of Canada are worse off than others, with CVD and its risk factors displaying a general east-west gradient. [1] Nova Scotia—the context of this study—is a comparatively older and sicker province that is disproportionately affected by higher CVD mortality rates and higher prevalence of modifiable risk factors. [4-8] As CVD is largely preventable through risk factor modification and management, there is an enormous opportunity for improvement. Yet many Nova Scotians remain at risk, and if changes are to be broadly successful, then health policy and public health efforts must focus on upstream interventions. [9,10]

Where individuals live matters to their health. However, empirical evidence from specific epidemiological investigations that consider CVD in the context of place is relatively lacking. [9,10] Yet, not all areas are equal, and if CVD prevention on a population level is to be successful, then research must identify those areas that experience higher burden. Small area rate variation (SARV) in the prevalence of CVD can identify the magnitude of the problem, but more importantly, its spatial distribution. Moreover, as areas can have above average rates for different reasons (e.g., high rates due to demographics or due to the broader socioeconomic status [SES] of areas), SARV also generates knowledge on local CVD needs. This can then be utilized to match health care resources to these needs by informing remedial actions and targeted interventions. [11] This method of addressing the burden of CVD through a spatial perspective can be promoted by rooting research in spatial epidemiology, a conceptual framework with an overall goal of spatializing health research. With disease mapping as one of its chief types of spatial inquiry, spatial epidemiology ultimately promotes the production of thematic maps that steer end users and policy makers to areas of interest. [12-14]

As place is inherently a geographic concept, research into the analysis of variation in CVD prevalence requires context-specific examinations. Within Nova Scotia, recent

efforts have led to the development of a Health Atlas, which has visualized differences in the distribution of some CVDs according to small areas.[15] The present thesis capitalized on these developments by not only documenting area variation in CVD prevalence, but also by quantifying and describing the performance of areas relative to this variation. Moreover, this study described how CVD prevalence varied according to compositional (i.e., demographics) and contextual (i.e., socioeconomic properties of areas) characteristics of the areas themselves, thus identifying potential avenues for evidence-based planning and resource allocation. In doing so, the long-term outlook of this study was to complement recent evidence by providing some direction for diminishing inequalities in CVD outcomes, with an ultimate goal of contributing to lessening the total burden of this disease in Nova Scotia.

CHAPTER 2. BACKGROUND

2.1 CVD

CVD is a chronic, non-communicable disease of the circulatory system comprised of several diseases existing along a pathophysiological continuum [16] which broadly includes: ischemic heart disease (IHD), also known as coronary artery disease (CAD) or coronary heart disease (CHD)— [6] circulation problems to the heart muscle that can result in an acute myocardial infarction (AMI), also known as a heart attack; congestive heart failure (CHF), wherein the heart is weakened and pumps inadequately to meet the body's needs, often resulting from an AMI; rheumatic heart disease, wherein a bacterial infection results in rheumatic fever that can precipitate into damaged heart valves; congenital heart disease, whereby a birth defect affects the structure of the heart; cerebrovascular disease, which involves circulatory problems in the brain and as group includes strokes, transient ischemic attacks (TIAs), and stroke sequelae; and peripheral vascular disease, or circulatory disorders outside of the heart and brain (in the extremities). [17-19]

2.1.1 CVD Epidemiology in Canada and Nova Scotia

Within Canada, CVD-related health outcomes exhibit a general east-west gradient. The highest rates of CVD are generally found in Atlantic Canada, followed by lower rates in Central Canada and the Prairie Provinces and the lowest rates in Western Canada (with mixed data for the territories). [1,20-24] Table 2.1 compares the CVD-specific mortality between Nova Scotia, Atlantic Canada, and Canada overall. Based on average mortality rates between 2010-12, the highest rates were observed in Newfoundland and Prince Edward Island, followed by lower rates in Nova Scotia and New Brunswick, which were still significantly higher than the Canadian average. [7] When measuring the potential years of life lost (PYLL), which gives more weight to deaths that occur at an earlier age and is a measure of premature mortality, [25] the same trends are observed, with CVD being a higher principle cause of early death in Nova Scotia and Atlantic Canada than in the rest of Canada (Table 2.1). [7]

CVD has a high disease burden in Nova Scotia. In 2014, 8.2% of adults in Nova Scotia self-reported having CVD. [26] Similarly, the age-standardized prevalence of IHD in Nova Scotia was 8.8% based on surveillance data up to March 2016, which was the

second highest prevalence estimate in Canada after New Brunswick, and above the Canadian average of 8.1%. [27] Conversely, in the same surveillance data, the age-standardized prevalence of CHF in Nova Scotia was 3.3% and around the Canadian average of 3.4%, [28] while the age-standardized prevalence of stroke in Nova Scotia was 2.4% and around the Canadian average of 2.5%. [29] In sum, CVD poses significant health threats and Nova Scotia is a province facing a relatively high CVD burden.

Table 2.1 Age-standardized death rates due to CVD, by Atlantic Canada and Canada overall, 2010-12 (per 100,000 population)

	Mortality rate	PYLL rate[a]
NS	216.6	828.6
PE	230.8	887.0
NB	211.6	809.8
NF	256.8	1,014.2
CA	193.1	677.5

Sources: Statistics Canada, Vital Statistics - Death Database. [7]
Note: PYLL, potential years of life lost; NS, Nova Scotia; PE, Prince Edward Island; NB, New Brunswick; NF, Newfoundland; CA, Canada.
a. PYLL measures the number of years that a person did not potentially live due to premature death, which is defined relative to a reference age of 75 for this indicator.

2.1.2 Economic Impact of CVD

CVD has a substantial economic impact. In 2008, CVD was estimated to cost the Canadian economy $12.3 billion in 2010 constant dollars—$11.9 billion in direct, health-related costs, and roughly $395.4 million in indirect costs from lost productivity due to disability or as a consequence of premature death (Table 2.2). [2,3] This placed CVD second in total costs among all diagnostic categories, only surpassed by neuropsychiatric conditions at $12.9 billion. Among the direct costs, hospitalization was the largest contributor at $5.2 billion, while among indirect costs, the largest contributor was the cost associated with morbidity, with $300.8 million lost due to illness and/or injury. [2,3] The estimated cost of CVD in Nova Scotia was $387.8 million. This accounted for 15.4% of all illness costs, representing the single highest diagnostic category in regards to economic impact. [2] The vast majority was spent on healthcare, where $385.2 million was directly spent on hospitalization, prescription drugs, and physician care (Table 2.2).

Table 2.2 Cost estimates due to CVD, by cost component, Atlantic Canada and Canada overall, 2008 ($'000,000 2010 Constant Dollars)

	NS	PE	NB	NF	CA
Direct costs					
Hospital care	181.6	24.7	139.9	115.9	5,174.6
Drugs	140.8	19.9	109.7	80.9	4,362.5
Physician care	62.8	8.8	49.7	32.4	2,401.5
Total direct costs	**385.2**	**53.4**	**299.3**	**229.2**	**11,938.7**
Indirect costs					
Morbidity[a]	NA	NA	NA	NA	300.8
Mortality	2.6	0.3	1.8	1.7	94.6
Total indirect costs	**2.6**	**0.3**	**1.8**	**1.7**	**395.4**
Total cost of CVD	**387.8**	**53.7**	**301.1**	**230.8**	**12,334.1**

Source: Public Health Agency of Canada, Economic Burden of Illness in Canada (EBIC), 2005-2008. [2,3]
Note: NS, Nova Scotia; PE, Prince Edward Island; NB, New Brunswick; NF, Newfoundland; CA, Canada; NA, Not Available.
a. Provincial morbidity costs attributed to CVDs are not released due to guidelines restricting the release of data based on small cell counts. Therefore, provincial indirect and total combined costs omit this cost component.

By factoring in population sizes into these cost estimates and estimating health expenditure per capita, it is seen that Atlantic Canada bears a disproportionate financial burden of CVD, which is suggested by the epidemiological data. For example, the 2008 health expenditure associated with CVD, expressed in 2010 constant dollars, was $414.4 per capita in Nova Scotia (author's calculations). [2,30] Similar trends are seen for all of Atlantic Canada, ranging from $387.0 per capita in Prince Edward Island to $451.2 per capita in Newfoundland. Conversely, the estimate was $360.9 per capita for Canada overall. Similarly, in the most populous province of Ontario, the estimate was $352.8 per capita, while in British Columbia—traditionally the province experiencing the least CVD burden—the estimate was $359.1 per capita. [2,30] These cost figures thus demonstrate the taxing effect of CVD on the economy and healthcare system, particularly in Nova Scotia and Atlantic Canada.

The high rates of CVD in Nova Scotia can be studied downstream at the individual and healthcare level, identifying individuals already living with CVD and targeting them with education and motivational interventions. This has been the traditional approach to addressing CVD. [9,10,31] Although this individually targeted and

resource-intensive approach has been demonstrated to result in behavioural modifications, the ability to achieve sustained behavioural change has been less evident, even with interventions focused on high-risk and highly motivated individuals. [32-35] Developing on this, the spatial inequalities suggest that broader avenues must be targeted to address disparities and total CVD burden. Indeed, there is a significant opportunity in going upstream by examining CVD occurrence in relation to place, trying to identify where CVD is more prevalent and the salient characteristics of these areas. This approach has broad potential for CVD prevention and population health promotion, and can be facilitated through an understanding of spatial epidemiology.

2.2 SPATIAL EPIDEMIOLOGY

Where one lives matters to their health. Yet, historically, place has received less attention than person and time in epidemiology. [36,37] However, the increasing availability of public health databases with geographically indexed data, coupled to advances in geographic information systems (GIS)—software tools used to manipulate and display spatial data—has stimulated this research paradigm. [14] Collectively, these developments have led to the growth of spatial epidemiology, which is a conceptual framework and a sub-discipline of health geography that focuses on the analysis of variation in health outcomes within small areas. [13] This entails the analysis of geographically indexed health data with respect to numerous individual and contextual risk factors. [12] Its overall objective is to capture the spatial patterns of health and disease, which is promoted through disease mapping. [14]

2.2.1 Disease Mapping

Disease mapping at a small area scale was the focus of this thesis. Crude counts of disease serve as the basis for estimating disease burden in an area. However, counts will be affected by variation in the underlying population "at risk" in each area. [13] The composition (i.e., age, sex, SES, disease susceptibility, etc.) of the background population will vary from one area to another, and these differences will influence the risk of disease. Hence, this underlying variation that may confound observed differences in disease risk must be accounted for through adjustment. Adjustment allows for fair

comparisons between areas and teases apart how each characteristic adjusted for contributes to variation in rates. [13]

Although the disease map is a good communication device, there is also potential for it to mislead. Different scales of resolution—the ratio of the measured distance on the map to that of the real environment on the ground— [38] using the same data can produce very different spatial patterns of disease. This is known as the modifiable areal unit problem (MAUP), which arises due to the uncertainty induced by the aggregation procedure. [39] Disease mapping is also challenged by the small numbers problem. When mapping disease to small geographical areas some of these areas may have sparse data within them, and extreme values in rates may appear by chance, especially if the underlying population density is low. [40] Thus, rate estimates become unstable due to sampling variability. [14] To reduce some of this random noise, methods based on Bayesian statistics have been developed, with unstable rate estimates smoothed towards a global (or local) average by "borrowing strength" from more stable estimates—thus unmasking the more meaningful patterns in underlying risk. [14]

2.2.2 Residential Environments (Areas) and CVD Risk

CVD has many risk factors. Non-modifiable risk conditions include biological and genetic endowment (age, sex, and family history). Age is the dominant risk condition, with rates of all CVDs increasing with advancing age. [20] This is troubling as 28% of Nova Scotia's population is projected to be over the age of 65 by 2031. [4] Men display a ten-year lead on women in the development of IHD [20] and have a nearly three-fold higher mortality rate due to CVD under the age of 65, although sex differences lessen with increasing age, with few differences among the oldest age strata (85+). [1] Lastly, family history is also important, with familial factors and molecular defects in vascular physiology increasing susceptibility to atherosclerosis and CVD progression. [16,20] Conversely, modifiable risk factors include: smoking; alcohol consumption; physical inactivity; stress; hypertension (blood pressure >140/90 mm Hg); and poor nutrition leading to elevated blood lipids, glucose, and resulting obesity and diabetes. [20,41,42]

With a myriad of risk factors, CVD has served as a paradigm of risk factor epidemiology. However, an individualistic outlook fails to capture the broader disease

determinants or contexts that help shape geographic variations in disease. [43,44] A concomitant macro level paradigm has emerged that has framed CVD within environmental contexts, emphasizing the role of physical, social, cultural, political, or economic factors that help shape geographic differences in CVD. [10] These developments have necessitated the development of theories on the health-promoting and health-damaging features of environments, and how environments contribute to the CVD risk of individuals. The majority of studies to date considering CVD in the context of place have examined the association between neighbourhood SES and CVD risk. On the whole, these studies have demonstrated that living in more socioeconomically deprived or disadvantaged neighbourhoods is associated with greater CVD mortality and the prevalence and incidence of outcomes and established behavioural risk factors. [1,45] These relationships have been observed even after controlling for individual-level measures of SES. [9,10,45]

It has been argued that the SES of areas may serve as a proxy for more specific physical and social features of environments that may impact CVD risk, [9,37,46,47] with putative pathways linking residential environments as determinants of CVD depicted in the framework in Figure 2.1. Aspects of the physical (or built) environment may include things related to physical activity (e.g., accessibility of recreational facilities), diet (e.g., availability of healthy foods or unhealthy food advertising), and environmental exposures (e.g., pollution and noise). [9,10,37] This is supported by evidence of higher levels of smoking, physical inactivity, and overweight or obesity reported by individuals living in rural areas. [1] Moreover, the prevalence of many risk factors and CVD outcomes is higher among Aboriginal Peoples (First Nations, Inuit, and Métis) compared to the general population, who are often located on rural and remote reserves. [1,20] The social environment may influence CVD risk through things such as social norms (e.g., contagion effects of residential social norms influencing individual CVD-related behaviour), psychosocial stressors (e.g., safety and violence), and social connections/organization (e.g., social ties, social support, and social cohesion). [9,10,37,48,49] In the chain of causation, distal factors influence intermediate risk factors (mainly behaviours and stress), which propagate into proximal risk factors (e.g., hypertension) that directly precipitate into clinical CVD. [9,16,43,44]

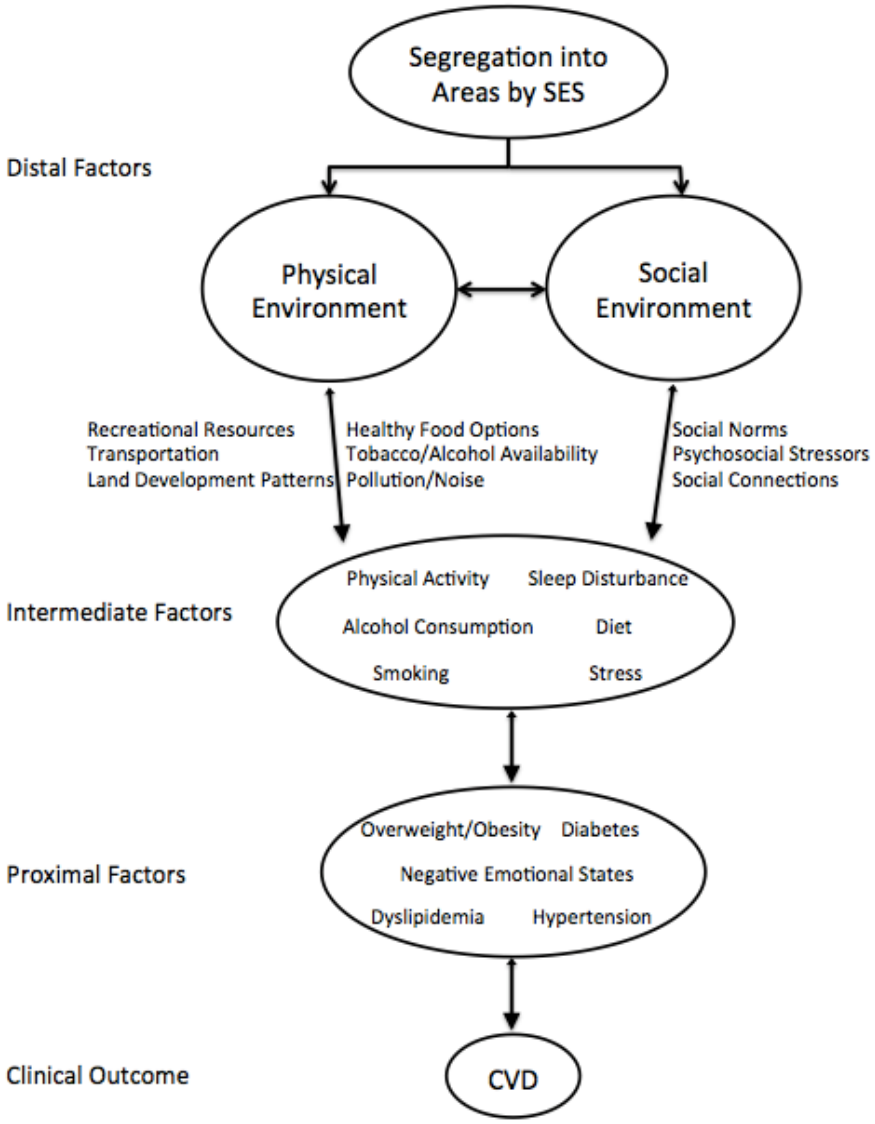

Figure 2.1 Flowchart depicting possible pathways linking residential environments to CVD. Double-headed arrows indicate reciprocal, although unequal, relationships. Adapted from sources: [9,10,16,37,43,44,49]

It is also important to recognize interrelationships between the different levels of risk factors, which are represented by the double-headed arrows in the figure. Intermediate risk factors such as a low quality-diet may be due to limited choices for healthy food options in more disadvantaged neighbourhoods. However, it may be that there are less healthy food options in these neighbourhoods because of limited local demand for healthy foods, such that an individual's choices are constrained by the

decisions of others. These interrelationships capture a major theme in place effects research—contextual versus compositional effects—that is represented by a desire to ascribe variation in outcomes to a correct source.

2.2.3 Contextual Versus Compositional Sources of Variation

Variations in health outcomes exist across time and space. The question is not whether variations between areas exist, as they always will, but instead what is their underlying source? SARV in poor health outcomes can arise from exogenous factors that are intrinsic to areas themselves and which also include collective understandings, beliefs, and norms (a contextual explanation), or they may be due to endogenous differences between certain types of people living in these areas (a compositional explanation). [50,51] This distinction has resulted in the desire for disentangling contextual from compositional effects on health. [45] The task is not only on identifying some notion of "need", but also to invest in public health interventions that can best address the sources of this need. This is rooted in pragmatism, as policy agendas and fiscal restraints means that decisions makers must appropriate limited resources towards interventions that deliver the best bang for the buck.

This contextual-compositional distinction is oversimplified. There has been a tendency in health research to ascribe most SARV in health outcomes to compositional differences between areas, with contextual explanations being a relative afterthought. [52] This has been the implicit conclusion in much of epidemiology, insofar as researchers have held a guarded view of ecological data due to the potential for committing the ecological fallacy—incorrectly drawing inferences on individual-level associations and behaviours based on area-level data. [53] This view has partly developed from the unavailability of individual-based data (necessitating the use of aggregated data as a proxy for individual-level variables), but has also been promoted in part by limitations of traditional statistical modeling techniques. [54] Yet, a distinction must be made between the ecological fallacy (the improper use of aggregate data) and the ecological perspective (the analysis of the effects of physical and social environments on health outcomes of individuals and populations). [52]

In reality, compositional and contextual effects are interdependent and mutually reinforcing. Areas provide the context that shapes the distribution of more direct CVD risk factors that are difficult to change without modifying the environment that promotes them. [55] This reciprocal relationship is layered with complexity: different contexts may be experienced differentially by different groups (i.e., there is contextual heterogeneity); different groups may be differentially variable within the same context (i.e., individual heterogeneity); individual differences may interact with contexts differentially based on contextual characteristics (i.e., individual-contextual interactions); individual health differences may depend on multiple scales of aggregation from proximate environments to macroecologic settings (i.e., multiple hierarchical contexts); individuals experience a number of overlapping but nonhierarchical contexts such as where they live and where they work (i.e., contexts have cross-classified structures); and contextual explanations of health depend on spatiotemporal trends and historical change in areas. [48,50] SARV in health is a byproduct of this complexity, and policy makers evidently need to invest in both people and places. [48] Interventions targeting CVD will not only have to target individuals, but will also have to tackle wider societal conditions of poverty, powerlessness, and lack of social capital.

2.3 RATIONALE

CVD in Nova Scotia is a tangible problem. To address this problem, a strategy for Chronic Disease Prevention and Management was developed by the Department of Health and Wellness (DHW). A key component of the overall strategy is the emphasis on a population health approach to chronic diseases that goes beyond any one sector of society, requiring intersectoral collaboration and action to truly effect change. [56] In support of this strategy, the DHW formed Cardiovascular Health Nova Scotia (CVHNS), which is the key provincial program aimed at improving the cardiovascular health and care of Nova Scotians. [57] CVHNS has created a strategic plan to address CVD in Nova Scotia, with many recommendations focused on the broader determinants of health and on upstream prevention (e.g., the need to create supportive environments, using evidence to inform healthy public policy, and the importance of knowledge exchange for decision support). [58] This is where estimates of CVD prevalence for small areas can find their

utility, by generating an evidence base on the spatial distribution of CVD, which can then be used to inform successful healthy public policy and prevention strategies.

SARV research is context dependent, requiring a review of the current state of knowledge on small area CVD prevalence estimates in Nova Scotia. Using data from the Improving Cardiovascular Outcomes in Nova Scotia (ICONS) database, CVHNS produced two maps of rates for acute coronary syndrome—a subcategory of IHD—and CHF. [59] Although rate variation was documented, with higher rates in northern and northeastern Nova Scotia and lower rates in central Nova Scotia compared to the provincial average, the data was from 2002-05 and was mapped according to district health authority (DHA). These administrative boundaries no longer exist, with the DHAs amalgamated into the single Nova Scotia Health Authority (NSHA). Moreover, rates were estimated for these nine large DHAs, which do not capture the finer resolution more typically associated with SARV research.

In late 2016, the Nova Scotia Health Atlas was launched. [15,60,61] This interactive, web-based mapping tool visualizes differences in the distribution of various health indicators across small areas in Nova Scotia. It was created through a partnership between NSHA, DHW, the Maritime SPOR SUPPORT Unit (MSSU), and researchers at Dalhousie University. [62] Also, it aggregated data that was used for a parent study to this thesis entitled "Small Area Variation in Rates of High-Cost Healthcare Use Across Nova Scotia," [15] which was produced in collaboration between the MSSU and the Nova Scotia Primary and Integrated Health Care Innovations Network (NS-PHICI). [63] The Atlas currently maps rates of IHD and CHF according to small geographic areas in Nova Scotia—cross-tabulated by age and sex. [15] Thus, the Atlas provides timely information on CVD that can be used by healthcare stakeholders such as CVHNS to meet the cardiovascular health needs of the province.

The genesis of this thesis laid within these recent developments on the health and CVD status of Nova Scotia. The main objective of this study was to not only document SARV in CVD prevalence in Nova Scotia, but also to quantify and describe the performance of areas relative to each other and to the province as a whole. As age and sex are key predictors of CVD, this study described how estimates vary according to the demographics of areas to facilitate comparisons. Although advancing age is the dominant

risk condition for CVD, [20] the premature onset of CVD in younger age groups is of concern due to the ongoing health and economic impacts that it creates. Thus, this study also compared how areas vary in CVD prevalence for younger age groups with a goal of identifying areas with higher prevalence. This could generate hypotheses and implicate factors associated with the premature onset of CVD, including life course and environmental factors, accelerated aging, and premature mortality. This may have also indicated that these areas have difficulties with the prevention of the early onset of CVD, which could provide some directions for policy and planning that could have significant meaning towards reducing the burden of CVD.

Finally, as the framework above depicted, aspects of both the physical and social environment have been linked to CVD. Thus, variables of area SES were included in the analysis to describe how estimates vary according to characteristics of areas and to see how much of the residual variance was explained by these variables. This directly identified the importance of these variables to SARV in CVD prevalence, which has broad potential for prevention strategies aimed at addressing inequalities in CVD. Collectively, this thesis provided detailed information on CVD, which not only complements the work that has already been accomplished, but further contributed to the knowledge base on a disease that significantly impacts the health and economic well-being of Nova Scotia and its citizens.

2.4 OBJECTIVES

The primary objectives of this study were to:

1. Estimate area-specific crude and age-sex adjusted prevalence for the following CVD-component conditions:
    a. IHD
    b. CHF
    c. Hypertension (as a risk factor for CVD)
2. Assess how small area rate variation in CVD prevalence differs between younger and older age groups.
3. Estimate the extent to which area SES contributes to area variation in CVD prevalence.

CHAPTER 3. METHODS

3.1 STUDY DESIGN

This was a descriptive, population-based, cross-sectional study informed by the conceptual framework of spatial epidemiology. The unit of analysis was individuals, nested within 78 geographic areas of the province. [64] The study population included all residents of Nova Scotia who were eligible for provincial healthcare coverage for at least one year anytime between the fiscal years 2010-11 to 2012-13. As CVD outcomes are relatively uncommon in younger age groups (e.g., the self-reported prevalence of IHD among Canadians between ages 12-39 was 0.81% in 2013-14), [1,65,66] an age cut-off of 40 was used. Thus, to satisfy inclusion criteria, residents were 40 years of age and above after April 1, 2010, which is the start date of data years in the data source that was used (fiscal years run from April 1 to March 31), [67] and is also the date from which collection of CVD outcomes in this study commenced.

Cases of CVD (IHD, CHF, and hypertension) were extracted from administrative databases to estimate crude and adjusted CVD prevalence. Covariates in the study included patient demographics (e.g., age and sex) and characteristics of areas in the study (e.g. measures of SES). Multilevel models, which have become the statistical tools of choice in SARV research, were used to estimate and adjust CVD prevalence. Individuals were allocated to distinct areas based on postal code of residence, with GIS used to map prevalence and depict variations in CVD.

3.2 DATA AND DATA SOURCES

Individual-level data on CVD, patient demographics, and area of residence were obtained from available data assembled for a parent study entitled "Small Area Variation in Rates of High-Cost Healthcare Use Across Nova Scotia." This data was accessed through Health Data Nova Scotia (HDNS) at Dalhousie University. Administrative databases used included the Canadian Institute for Health Information (CIHI) Hospital Discharge Abstract Database (DAD) and the Medical Services Insurance (MSI) Physician Billings database (MED), with cases of CVD identified using codes from the International Statistical Classification of Diseases and Related Health Problems (ICD). [67] MSI patient registration and eligibility files were used to identify the study population.

These files contain information on a person's age and sex, eligibility date, date of death, and postal code of residence. Lastly, the 2011 Census of Population and National Household Survey (NHS) were accessed to obtain information on additional population demographics as well as socioeconomic characteristics of areas.

3.3 KEY VARIABLES AND MEASURES

3.3.1 CVD Outcomes and Individual-Level Variables

Individual-level variables in this study included CVD outcomes and patient demographics. Age was categorized into 10-year age groups (with a single category over the age of 80) for age-sex adjustment, and was also dichotomized into groups over and under 60 years of age to assess differences in SARV in CVD prevalence between relatively younger and relatively older age groups. Outcomes of interest in this study included dichotomous variables (i.e., prevalent case: yes/no) of IHD, CHF, and hypertension, which are component conditions of CVD that were conceptualized as a partially representative collective of this overall disease. In order for conclusions from this study to be valid and reliable, accurate ascertainment of CVD was essential, factoring in identified errors either through correction or discussion of biased results.

Cases of each CVD-component condition were extracted from the administrative databases for the parent study using a validated case algorithm. Case algorithms are specific combinations of administrative database elements that produce a set of definition criteria used to identify diseases in administrative databases. The varying elements include: data source, number of years of administrative data, diagnostic or procedural code(s) used, and number of data records (i.e., contacts) with a diagnostic code(s). [68] Case algorithms that were used are those that have been validated by the Canadian Chronic Disease Surveillance System (CCDSS). The CCDSS, supported by the Public Health Agency of Canada (PHAC), is a collaborative network of surveillance systems that uses administrative databases to track chronic diseases. [69] Appendix A presents the case algorithms that were used and includes: descriptions of the diagnostic and procedure codes, parameters of the algorithms, their content validity, the diagnostic fields used from which records were extracted, and the reported prevalence of the conditions (in the specific populations tested) in the administrative databases from the algorithms used.

In order to accurately estimate CVD prevalence at a small-area scale, it was imperative to assess the ability of these algorithms to produce results that reflected the constructs being measured. As Appendix A reveals, the algorithms do not have perfect accuracy, with none of sensitivity, specificity, nor the predictive value of a positive or negative test equalling 100%. Thus, misclassification error could bias area prevalence estimates. To address this bias, prevalence estimates were corrected for misclassification using the Rogan and Gladen formula: [70]

$$\pi_j = \frac{p_j + C - 1}{S + C - 1} \quad (1)$$

Where the estimator corrected for misclassification error (π_j) gives an estimate of the "true" prevalence of CVD in area j, and is a function of the "apparent" (observed) estimate of CVD prevalence (p_j) and the sensitivity (S) and specificity (C) of each algorithm from the validation studies. In the case where $S = C = 1$ (i.e., perfect diagnostic accuracy), then $p_j = \pi_j$, and the observed estimate of CVD prevalence would be an unbiased estimator of the "true" prevalence. Otherwise, p_j will be biased, and the magnitude of the bias depends on π_j, S, and C. Figure 1 in the Appendix of a publication by Rogan and Gladen [70] displays the relationship between p_j and π_j at different values of S and C. Even at relatively high levels of S and C, p_j will generally be biased, particularly when "true" prevalence of a disease is low, in which case p_j overestimates π_j ($p_j > \pi_j$). Estimates from π_j and p_j were compared to provide a quantitative assessment of the robustness of estimates from the naïve estimator (p_j). [71] Although a crude adjustment, it was expected that the estimates following correction for misclassification (π_j) would give a more accurate picture of CVD prevalence in comparison to the naïve estimator that inaccurately assumed perfect database information. [72]

3.3.2 Small Area Units and Area-Level Variables

Spatial boundaries of small areas that were used for mapping matched those in the Atlas and parent study, and were delimited using the first three characters of the postal

code—identifying the forward sortation area (FSA). [73] Cartographic boundary files of FSAs are readily available from Statistics Canada for GIS software. [74]

Area-level variables that were assessed in this study included distal factors hypothesized to contribute to SARV in the prevalence of the CVD outcomes. [1,9,10,20] These distal risk factors represented aggregated characteristics of areas, and the following eight variables were obtained from the 2011 Census and NHS for each FSA: proportion of population aged 65+ (control variable for the concentration of elderly residents in an area); proportion of household's with a total income less than $20,000; unemployment rate of population aged 15+ (proportion of total population in the labour force that is unemployed, which does not include discouraged workers who are no longer looking for work and are thus not in the labour force); education of population aged 15+ (proportion of population with no high school diploma, and proportion of population with a postsecondary certificate, diploma, or degree); proportion of census families headed by a lone mother; proportion of persons in private households or dwellings living alone; and proportion of population in private households identifying as Aboriginal peoples. These area-level variables were selected and categorized in this manner because previous studies have found associations between similarly categorized variables and CVD. [31,75-81] Also, they were included as proportions rather than medians to weight the data by unequal FSA populations. [75] Finally, they were linked to individuals in the study population at the FSA level using matching FSA codes.

3.4 ANALYSIS

3.4.1 Descriptive Statistics

Frequencies and proportions (%) were tabulated and described for the demographic characteristics of the baseline study population, the SES characteristics of areas, and the observed prevalence of the CVD-component conditions across individual- and area-level characteristics.

3.4.2 Regression Modeling Approach

Study objectives were met by fitting a random intercept (multilevel), logistic regression model of the following form:

$$\text{logit}(p_{ij}) = \mathbf{X}_{ij}^{T}\beta + u_j \qquad u_j \sim N(0, \sigma_u^2) \tag{2}$$

Where the dependent variable models the log odds ratio (OR) of CVD for individual *i* in area *j* with a specific covariate pattern, and can be transformed to give the predicted probability of CVD in each individual (p_{ij}). The p_{ij} was linked to each individual's specific covariate pattern through the linear predictor ($\mathbf{X}_{ij}^{T}$), which is a vector of known predictors for each individual that includes individual-level and area-level variables. These variables were estimated with fixed effect coefficient vectors β, and can be interpreted as the partial effects of a set of predictor variables on the p_{ij}. The random effect (u_j) captures variation between-areas in the intercept (of the dependent variable), and is assumed to follow a normal distribution with zero mean and variance σ_u^2. [50,82-84]

SARV in CVD prevalence—and performance between areas—was estimated by assigning values to each area based on the random intercept u_j, which measures the deviation of an area's mean from the overall mean of the response variable. As the estimation of rates for small areas can encounter statistical challenges due to few observations (i.e., small numbers problem), empirical Bayes methods were used to produce empirical best linear unbiased predictor (EBLUP) estimates. These are precision-weighted estimators that are used to "smooth" risk estimates. [12,85] In essence, for FSAs with fewer observations, observed estimates would have higher sampling variability (i.e., less precision) and were given lower credibility, resulting in the (model-based) fitted estimates being shrunk towards the overall mean of the study population as predicted by the model. Conversely, estimates for FSAs with many observations were considered more stable, and thus the fitted estimates were subject to less to shrinkage. [14,50,85]

To estimate and standardize CVD prevalence rates (for objectives 1 and 2), the following multistep regression modeling approach was conducted in SAS, version 9.4 (Cary, North Carolina, USA):

I) First, the crude CVD prevalence for the whole study population (p) was estimated, which equaled the proportion of the total population that has each CVD.

II) Next, regression models were sequentially fitted using the GLIMMIX procedure in SAS:

a) Model 1 was a null, two-level model that only included a random intercept for area. This variance components model estimated unadjusted random effects u_j for each area, and was used to estimate the proportion of the total variation that was between FSAs.

b) Model 2 added an age-sex interaction term to Model 1, with the predicted probabilities for each FSA (p_j) from this random intercept model used to meet the first half of the first study objective—*estimate area-specific crude prevalence for IHD, CHF, and hypertension.*

c) Next, the product from step I) and the predicted random effects from step II.b) was computed. This produced directly standardized prevalence estimates for each FSA, using the p as a standard and the u_j as the FSA deviation from the p. This was used to meet the second half of the first study objective—*estimate area-specific age-sex adjusted prevalence for IHD, CHF, and hypertension*.

d) The population was stratified on sex and on age < 60 and ≥ 60. Categorized age was included in the random intercept models for these stratified populations, with a similar process to the one in step II.c) used to compute age-adjusted prevalence estimates for each FSA. These random intercept models were fitted to meet the second objective—*assess how small area rate variation in CVD prevalence differs between younger and older age groups.*

III) Finally, using the sensitivity and specificity of each case algorithm from the validation studies, the "naïve" FSA prevalence estimates from steps II.b-d) were corrected for misclassification error using the Rogan and Gladen estimator in equation (1). This produced corrected CVD prevalence estimates for FSAs (π_j), which improved their validity.

To measure the association between CVD and individual and area-level variables, three separate models were added to Model 2 from step II.b):

I) Model 3 added measures of area social isolation (% of single mother census families, % of persons living alone in private households, and % of population with Aboriginal identity) and a control variable of the % of the population greater than 65 years of age.
II) Model 4 added measures of area material deprivation (% of households with a total income of less than $20,000; % of population that is unemployed; % of population with a post-secondary certificate, diploma, or degree; and % of population with no high school diploma) and a control variable of the % of the population greater than 65 years of age.
III) Model 5 was a full model that added the eight area-level variables concurrently.

Models 3-5 were fitted to see how much of the remaining residual variance was explained by these variables and addressed the final study objective—*estimate the extent to which area SES contributes to area variation in CVD prevalence.* To accomplish this last objective in the study, three measures of area variation were computed, including the variance partitioning coefficient (VPC), median odds ratio (MOR), and interval odds ratio (IOR). The VPC estimates the proportion of the total variance (between and within areas) that is due to differences between areas and provides insight on the level of hierarchy at which the action lies. VPC thus depends on the partitioning of total residual variation into components that can be attributed to different levels. [86] Estimating the VPC for multilevel logistic regression models is challenged due to an inherent lack of scale for binary responses, as the between-individual variance (σ_ε^2) depends on the predictor variables (i.e., it is heteroskedastic). Thus, there is no unique way of defining the VPC. [84,87-89] There are alternative approaches available, [84,86,90] and this study employed the latent variable approach to give an approximate estimate of the VPC:

$$VPC = \frac{\sigma_u^2}{\sigma_u^2 + \sigma_\varepsilon^2}$$

$$= \frac{\sigma_u^2}{\sigma_u^2 + 3.29} \quad (3)$$

Where the between-area variance (σ_u^2) equals the intercept variance (σ_{u0}^2) while the σ_ε^2 follows a logistic distribution and is fixed at 3.29. VPC values close to 1 (100%) indicate maximum heterogeneity between areas in regards to the probability of CVD in individuals, while those close to 0 indicate a homogenous probability among areas. [84,86,90] Utilizing the latent variable approach was reasonable in this study as the binary response variable of whether an individual has CVD or not was seen as being derived from a truncation of an underlying continuous scale of the propensity to have CVD. [90] However, the distinction between σ_ε^2 and σ_u^2 is not clear in multilevel logistic regression models, and the interpretation of the VPC is difficult for binary outcomes. [91,92] Thus, the MOR and IOR were also calculated to aid interpretation of the between-area variation and to understand the importance of areas on CVD prevalence.

The MOR is defined as the median value of all possible ORs when comparing an individual from an area with a higher propensity for CVD with an individual from an area with a lower propensity for CVD. It quantifies the between-area variation when comparing two individuals from two randomly chosen areas and can be conceptualized as the median amount by which the probability of CVD changes when an individual moves from one area to another. This gives the extent to which the individual probability of having CVD is determined by residential areas. [91,92] The basic aim of the MOR is to translate the σ_u^2 onto the OR scale, giving it a more intuitive interpretation that allows the magnitude of the random effects of areas to be directly compared with the fixed effects of individual-level variables (that vary within areas). [91,92] The MOR was computed as:

$$MOR = exp^{0.6475*\sqrt{2\sigma_u^2}} \quad (4)$$

The MOR is ≥ 1. If it is equal to 1, then there is no between-area variation and no differences would be seen between areas in the probability of individuals having CVD. If the MOR is large, then considerable between-area variation exists, meaning areas would

be relevant to understanding area variations of the individual probability for having CVD. [91,92]

For area-level variables that vary across areas (but not between individuals within-areas), usual OR interpretations are incorrect. This is because it is necessary to compare individuals with different area random effects. Thus, the IOR was computed to quantify the fixed effects of area-level variables on the outcome. It is an interval that covers 80% of the ORs when comparing two randomly chosen individuals from different areas with different area-level variables (that are constant within areas). [91,92] The lower and upper bounds of the IOR were computed as:

$$IOR_{\text{lower}} = exp^{\beta + \sqrt{2\sigma_u^2} * (-1.2816)} \quad (5)$$

$$IOR_{\text{upper}} = exp^{\beta + \sqrt{2\sigma_u^2} * (1.2816)} \quad (6)$$

The IOR is not a common confidence interval (CI). If the interval of the IOR is narrow, then the between-area variation is small, and if it is wide, then the between-area variation is large (which repeats information given by the MOR). If the IOR contains 1, then the area random effect is large relative to the fixed effect of the area-level variables. If it does not contain 1, then the fixed effect of area-level variables is large in comparison with the area random effect. [91,92] Thus, the IOR was used to estimate the extent to which area SES contributes to area variation in CVD prevalence.

3.4.3 Cartographic Displays

To visualize CVD prevalence (for each of the component conditions), choropleth thematic maps were created using ArcGIS 10.2.2 (Esri Incorporated). Choropleth maps are used to depict data in discrete, areal form, and are usually used to depict standardized data. In this study, data was classified into equal intervals, with prevalence being depicted on the map using percentages. [93] Only the smoothed—and where it was possible—corrected prevalence was mapped and included for comparison to assess SARV in estimates.

CHAPTER 4. RESULTS

4.1 DESCRIPTIVE STATISTICS

The descriptive characteristics of the study population, areas, and the population with CVD-component conditions were first tabulated and described (Tables 4.1–4.3). Table 4.1 shows the distribution of individuals in the study population across individual-level, compositional characteristics. The overall study population in the administrative databases consisted of 526,103 individuals over 40 years of age. 1,885 individuals were missing a three digit FSA code and were thus excluded from further analyses. Although the age distribution was right-skewed, a large proportion of individuals were above 60 years of age (40.2%) in the study population. This is similar to the demographic distribution of the overall population of Canada for the same time period (i.e., 41.6% of all Canadians above 40 years of age were above 60 years of age between 2011-11 to 2012-13). [30]

Table 4.1 Compositional characteristics of the study population, Nova Scotia, 2010-11 to 2012-13 (*n* = 526,103[a])

	n	%
Age group (years)		
40-49	159,498	30.3
50-59	154,926	29.4
60-69	113,266	21.5
70-79	61,576	11.7
80+	36,837	7.0
Sex		
Female	274,462	52.2
Male	251,641	47.8

a. Total size of study population found in administrative databases.

Table 4.2 summarizes aggregate, contextual characteristics of FSAs found in the 2011 Census of Population and NHS that were used as area-level measures of SES in regression modeling to meet the final study objective. Most of these area-level variables have distributions that are right-skewed, with large maximum values pulling the mean away from the median. However, the interquartile range (Q3-Q1) indicates that the middle half of most of these variables extends across a more narrow range of 7-10%.

Some noticeable patterns emerge for these contextual characteristics. In general, more urbanized FSAs around Halifax and Dartmouth had the highest overall SES. This was most evident on measures of material deprivation (e.g., these FSAs had some of the lowest proportions for total household income less than $20,000, unemployment rate, and no high school diploma, while also having some of the highest proportions of post-secondary education). These areas were followed by FSAs classified as medium SES, which were primarily localized to the centre of mainland Nova Scotia in counties such as Hants, Colchester, Kings, Cumberland, Pictou, and Antigonish. Conversely, FSAs that generally had the lowest SES were more rural and remote, both on Western parts of mainland Nova Scotia and Cape Breton Island, particularly FSAs in and around Sydney and Richmond County.

Table 4.2 Characteristics of FSAs, Nova Scotia, 2011 Census Program (*n* = 78)

	Mean	Min	Q1	Median	Q3	Max
Census families						
% Single mother families	14.8	6.5	10.4	13.3	17.5	42.9
Persons in households						
% Living alone	11.7	3.5	7.7	11.5	14.0	31.4
Population by identity						
% Aboriginal identity	4.4	0.0	1.5	2.5	3.3	99.2
Population age group						
% > 65 years of age	16.6	2.6	13.0	17.3	20.5	26.1
Household total income						
% < $20,000	14.3	0.0	8.1	14.8	18.7	44.9
Labour force (ages 15+)						
% Unemployed	12.2	2.2	7.8	10.5	15	40.0
Education (ages 15+)						
% Post-secondary	53.3	33.2	48.2	53.2	59.4	74.3
% No high school diploma	23.1	4.3	17.1	21.6	27.9	61.9

Source: Statistics Canada, 2011 Census of Population and National Household Survey (NHS). [94,95] All computations, use and interpretation of these data are entirely those of the author(s).
Note: FSA, Forward Sortation Area.

Table 4.3 shows the bivariate tabulation of the "apparent" (not corrected for misclassification error) prevalence of the CVD-component conditions across different subgroups of both individual-level and area-level variables. The overall, uncorrected prevalence of the conditions was 5.2% for IHD, 1.7% for CHF, and 18.4% for

hypertension. Using the Rogan and Gladen formula, the corrected overall prevalence of the conditions was computed to be 4.3% for IHD and 20% for hypertension. The overall prevalence of CHF was not corrected for misclassification as the low prevalence of this condition resulted in a negative numerator in the Rogan and Gladen estimator formula and implausible estimates. [96] Thus, the presented results estimating SARV in CHF prevalence are based on the estimates that were not corrected.

There was large variation in the prevalence of the conditions across the subgroups of the variables, particularly the individual-level variables of age and sex. However, the prevalence of all three conditions consistently increased across increasing age groups, and males had a higher prevalence of all three conditions than females. To measure the prevalence of the CVD-component conditions across area-level variables, tertiles were first used to rank the FSAs into three categories (low, medium, or high) on each area-level variable of interest (i.e., low-ranked FSAs had the lowest proportions for that particular area-level variable, which equated to a higher SES for most of the variables except for post-secondary education, in which case low-ranked FSAs had lower proportions for post-secondary education and therefore lower SES). Subsequently, the prevalence of the CVD-component conditions was measured across the three categories of ranked FSAs for each area-level variable. Overall, Table 4.3 shows that across most of the area-level variables, the category of FSAs ranked low had lower observed prevalence than higher ranked FSAs. In addition, the observed prevalence sequentially increased when moving across the categories of ranked FSAs from low to high. The exceptions were for % of persons living alone in private households (highest prevalence in the medium ranked FSAs), % of population by Aboriginal identity (lowest prevalence in the medium ranked FSAs), and for % of population with a post-secondary certificate, diploma, or degree (where the pattern was reversed).

Table 4.3 Observed prevalence of CVD-component conditions across individual- and area-level variables, Nova Scotia, 2010-11 to 2012-13 (*n* = 526,103[a])

	IHD		CHF		Hypertension	
Variable	***n***	**Pr (%)**	***n***	**Pr (%)**	***n***	**Pr (%)**
Overall	27,246	(5.2)	9,024	(1.7)	96,987	(18.4)
Individual-level						
Age group (years)						
40-49	1,972	(1.2)	246	(0.2)	12,796	(8.0)
50-59	5,291	(3.4)	793	(0.5)	24,195	(15.6)
60-69	7,757	(6.9)	1,815	(1.6)	27,707	(24.5)
70-79	6,994	(11.4)	2,556	(4.2)	19,753	(32.1)
80+	5,232	(14.2)	3,614	(9.8)	12,536	(34.0)
Sex						
Female	9,699	(3.5)	4,463	(1.6)	50,231	(18.3)
Male	17,547	(7.0)	4,561	(1.8)	46,756	(18.6)
Area-level[b]						
Census families (% single mother families)						
Low	8,174	(4.8)	2,525	(1.5)	28,164	(16.5)
Medium	9,206	(5.3)	2,936	(1.7)	33,135	(19.1)
High	9,861	(5.5)	3,560	(2.0)	35,663	(19.8)
Persons in households (% living alone)						
Low	7,796	(4.4)	2,270	(1.3)	30,269	(17.0)
Medium	11,305	(6.4)	3,763	(2.1)	38,128	(21.6)
High	8,140	(4.8)	2,988	(1.8)	28,565	(16.9)
Population by identity (% Aboriginal identity)						
Low	8,387	(5.1)	2,747	(1.7)	31,327	(19.1)
Medium	8,850	(4.7)	2,937	(1.6)	32,627	(17.2)
High	10,004	(5.9)	3,337	(2.0)	33,008	(19.4)
Population age group (% > 65 years of age)						
Low	6,800	(3.9)	2,179	(1.2)	27,200	(15.5)
Medium	9,767	(5.7)	3,052	(1.8)	33,846	(19.6)
High	10,674	(6.1)	3,790	(2.2)	35,916	(20.4)
Household total income (% < $20,000)						
Low	8,131	(4.4)	2,489	(1.3)	31,877	(17.0)
Medium	8,814	(5.7)	2,855	(1.9)	29,560	(19.2)
High	10,287	(5.6)	3,674	(2.0)	35,502	(19.4)
Labour force (ages 15+) (% unemployed)						
Low	6,616	(3.9)	2,138	(1.3)	27,501	(16.0)
Medium	9,444	(5.2)	3,111	(1.7)	32,296	(17.8)
High	11,181	(6.5)	3,772	(2.2)	37,165	(21.7)
Education (ages 15+) (% post-secondary)						
Low	11,024	(6.4)	3,750	(2.2)	36,723	(21.2)
Medium	9,314	(5.1)	2,971	(1.6)	33,423	(18.3)
High	6,903	(4.1)	2,300	(1.4)	26,816	(16.0)

Variable	IHD		CHF		Hypertension	
	n	Pr (%)	*n*	Pr (%)	*n*	Pr (%)
Education (ages 15+) (% no high school diploma)						
Low	7,117	(4.1)	2,352	(1.4)	28,123	(16.1)
Medium	8,625	(5.2)	2,714	(1.6)	30,602	(18.5)
High	11,499	(6.3)	3,955	(2.2)	38,237	(20.8)

Source: Area-level variables: Statistics Canada, 2011 Census of Population and National Household Survey (NHS). [94,95] All computations, use and interpretation of these data are entirely those of the author(s).
Note: Pr, Prevalence.
a. Total size of study population found in administrative databases.
b. Area-level variables at FSA level and categorized using tertiles into low, medium, and high.

4.2 CATERPILLAR PLOTS AND MAPS DEPICTING SARV IN CVD PREVALENCE

Estimated SARV in the prevalence of the CVD-component conditions was depicted using caterpillar plots and maps in Figures 4.1–4.8 at the end of *Section 4.2*. Caterpillar plots sort FSAs vertically from the highest to the lowest point prevalence estimate and include their 95% CIs. The vertical line in the middle represents the prevalence for the overall population, which is centred at 0 as the prevalence for FSAs is estimated as a relative percentage change to this average. This is depicted by the x-axis, where estimates lying to the right of the vertical line represent FSAs with a relative percentage increase (i.e., areas with higher prevalence estimates), while estimates to the left of 0 represent percentage decreases (i.e., areas with lower prevalence estimates). If the CI of an FSA does not cross the vertical line, then that FSA is significantly different than the provincial average. Maps accompany each caterpillar plot, depicting the spatial distribution of SARV in CVD prevalence by plotting the relative prevalence: FSAs that are above average in prevalence are depicted in red, FSAs with below average prevalence are depicted in blue, while white areas were near the average. As the maps are based solely on the point estimates, it is important to interpret them in conjunction with their accompanying caterpillar plots. This is because the caterpillar plots quantify how much variation there is between areas and also show statistical significance. This is hidden in maps, so both should be interpreted together to get the full picture of the SARV.

4.2.1 IHD

Pronounced SARV in IHD prevalence was observed across Nova Scotia by FSA, which is captured through caterpillar plots and maps in Figures 4.1–4.4. First, Figure 4.1a is a caterpillar plot of SARV in crude, unadjusted prevalence of IHD. Striking SARV is observed, with approximately equal numbers of FSAs above and below the provincial average and ranging in prevalence from a high of +266% to a low of -73%. The CIs show that most FSAs are significantly different from the provincial average, and their ranges also indicate that the majority of estimates below average are precise and are thus more reliable, while many estimates above the provincial average are less precise and are thus less likely to be reliable. Many estimates above the provincial average are from FSAs with smaller populations, and have thus likely also been more precision-weighted towards the provincial average using the EBLUP estimates. This may indicate that these areas have even higher true prevalence than that which is estimated, which would result in greater SARV. The accompanying map (Figure 4.2a) depicts significant spatial variation in IHD prevalence. Higher relative prevalence estimates are seen in Western and Northern FSAs of mainland Nova Scotia and across Cape Breton Island, where a large portion of FSAs have relative prevalence estimates greater than 50% above the provincial average. Conversely, FSAs around Halifax have much lower prevalence, with the majority of these FSAs in the lowest two relative prevalence categories.

To assess the contribution of demographics to SARV in IHD prevalence, estimates were adjusted for age and sex to remove their influence on the observed variation in prevalence. Figure 4.1b presents the age-sex adjusted caterpillar plot for SARV in IHD prevalence. Again, variation is observed, with many FSAs having significantly different prevalence estimates from the provincial average. But adjustment resulted in noticeably different IHD prevalence estimates for many FSAs. 50 of the 78 FSAs (64.1%) had lower IHD prevalence following adjustment, with the greatest decrease in prevalence being -80% for an FSA. These areas likely have older population age structures relative to the provincial average. Conversely, the remaining portion of FSAs had prevalence increases following age-sex adjustment, highlighted by a markedly increased prevalence of +217% for one FSA (whose estimate was not included in the age-sex adjusted caterpillar plot to maintain legibility of the remaining estimates). These

areas likely have younger population age structures. In congruence with these results, adjustment also drastically altered the spatial pattern of SARV in IHD prevalence (Figure 4.2b). The majority of FSAs experienced reductions in relative prevalence following adjustment, which can be seen by the now lighter red coloured FSAs in Northern and Western parts of Nova Scotia and on Cape Breton Island, as well as many of the previously white FSAs now becoming a shade of blue. Conversely, many of the FSAs in and around Halifax had increased prevalence estimates following adjustment, as can be seen by the less dark shades of blue for these FSAs. However, there are exceptions to these patterns, such as an area in rural South East of Halifax County (FSA: BOJ) having a decreased estimate following adjustment, while Sydney Southwest (FSA: B1L) on Cape Breton Island had an increased estimate. But collectively, demographics played a significant role in explaining the SARV that was observed for crude prevalence.

Figures 4.3 and 4.4 present caterpillar plots and maps that were used to compare SARV in age-adjusted IHD prevalence between younger and older age groups. However, the interest lies in the premature onset of CVD in the younger age groups. Thus, the results for the older age groups are not included here, but are included in Appendix B for reference when making comparisons. Estimates for the female group less than 60 years of age were not corrected for misclassification as IHD prevalence in this group was only 1.3%, which again resulted in implausible estimates in the Rogan and Gladen estimator formula. Also, the caterpillar plots for both younger age and older female groups omit the estimate of the FSA with the highest prevalence estimate. This is because it was much higher than the rest of the estimates, so it was omitted to maintain legibility of the other estimates.

Overall, SARV for all four age-sex subgroups generally followed the variation found in the full study population, with lower relative prevalence estimates in FSAs around Halifax and higher estimates around Sydney. There were exceptions to this pattern, particularly among the younger age groups of interest. Lower IHD prevalence was estimated on Cape Breton Island (relative to surrounding areas) for younger females in FSAs B1C, B1J, B1K, B1M, B1R, B1X, and B1Y, and younger males in FSAs B1J, B1M, B1P, B1R, B1S, and B1X. Conversely, higher IHD prevalence was estimated

around Halifax for younger females in FSA B3R, and younger males in FSAs B3R and B4C.

Patterns were also similar between the four subgroups, in that FSAs with low or high IHD prevalence in one subgroup often had prevalence estimates in the same direction for all subgroups. There were exceptions to this pattern as well, with greater variation existing between the subgroups for some FSAs. For example, Halifax South (FSA: B3R) located just Southwest of Halifax Peninsula had far above average relative prevalence for younger females and above average prevalence for younger males, but just average and far below average relative prevalence for older females and older males, respectively. This is an area that may be experiencing issues with prevention of the early onset of CVD. Conversely, an area such as Amherst (FSA: B4H) located on the border with New Brunswick had average and below average relative prevalence for younger females and males, but had above average relative prevalence for both older females and males. This may indicate that this is an area that is dealing less with issues of early onset and more with population ageing and CVD management. There are also areas such as the Guysborough area (FSA: BOH) in rural and North East mainland Nova Scotia that have less uniform patterns. This area has average relative prevalence for younger females and far above average prevalence for older females, yet has very far above average relative prevalence for younger males and only above average relative prevalence for older males. Thus, this area is experiencing sex differences in IHD prevalence, with potential issues of ageing that is more specific to females, and potential issues of premature onset of CVD that is more specific to males.

It is also apparent that there is a slightly wider range of relative prevalence estimates for the two younger age groups relative to the two older age groups, especially among males. This is evidenced by a wider range of prevalence estimates in the caterpillar plots and generally a greater numbers of darker red and blue FSAs in the maps, respectively. However, there are exceptions, particularly among females, with many areas around Halifax having lower relative prevalence for older females in comparison to younger females. One possible explanation for these patterns in SARV is that there were much lower IHD prevalence estimates in the two younger age groups relative to the two older age groups, resulting in less precise and more variable estimates relative to the

provincial average. However, this would also mean that with greater variability, estimates for these younger age groups would have been more precision-weighted towards the population average than for the older age groups, which contradicts the results. Perhaps what is occurring is that even following shrinkage, SARV in IHD prevalence remains more pronounced for the younger age groups in comparison to the older age groups, and that there is much more true variation in IHD prevalence for these younger age groups.

4.2.2 CHF

For multiple reasons, the results for SARV in CHF prevalence were deemed unreliable, and thus not presented in this section. First, the case algorithm for CHF had low diagnostic accuracy (e.g., the positive predicative value (PPV) was only 55.6%), which would have resulted in high rates of misclassification. Thus, there was low confidence in the ability of the case algorithm to identify the true cohort of individuals with CHF. As aforementioned, the low overall prevalence of CHF in the study population meant that neither the overall prevalence estimate nor the FSA estimates could be corrected for misclassification, as the Rogan and Gladen estimator resulted in implausible negative estimates. Thus, this combined uncertainty meant that these results were treated as suspect. However, they were inserted into Appendix B for reference.

From the conclusions that can be made, it was seen that similar to IHD, significant SARV in crude CHF prevalence was observed across FSAs. This variation followed a general pattern that was similar to the pattern observed for IHD, with higher relative prevalence estimates in certain central FSAs of mainland Nova Scotia, much higher relative prevalence estimates on Cape Breton Island, and much lower relative prevalence estimates around Halifax. However, in comparison to variation observed for IHD, significantly less variation was observed for CHF, with a narrower range of relative prevalence estimates (from +104% to -64%) in the caterpillar plot and visibly much less variation in the map (evidenced by many fewer FSAs in the darkest coloured categories).

Probable factors driving the difference between SARV in CHF and IHD prevalence are that CHF is more strongly age-related, only becoming increasing prevalent above 65 years of age, [65] and because CHF lie towards the distal end of the CVD continuum, often preceded by many other and often fatal CVD episodes such as AMIs. [16]

Thus, less variation is expected for CHF, being more consistently prevalent across FSAs once CHF develops in older age groups. This explanation is supported by the age-sex adjusted results, which show markedly reduced SARV. The FSA estimates are much closer to the vertical line in the caterpillar plots following adjustment, and the range of relative prevalence estimates decreases to +86% to -34%. 47 of the 78 FSAs (60.3%) had lower CHF prevalence following adjustment, with the greatest decrease in prevalence being -53% for an FSA. Again, these areas likely have older population age structures relative to the provincial average. The remaining portion of FSAs had prevalence increases following adjustment (highest being +94%), and these areas likely have younger population age structures. The overall reduction in SARV in CHF prevalence following adjustment is even starker in the included maps, as a large portion is now covered by FSAs in white colour. Similar to the age-sex adjusted map for IHD, adjusting CHF prevalence estimates resulted in lower prevalence estimates in many FSAs in and around Sydney and higher prevalence estimates in many FSAs in and around Halifax.

Lastly, comparisons between younger and older age groups for CHF could not be made because estimates could not be obtained for both younger groups. This is because there were only a total of 395 females and 644 males below 60 years of age with CHF, with many FSAs with zero cases within them. Thus, the regression models were unable to converge and these results are unavailable. Although results for the two over age 60 groups were obtained, they were omitted, as SARV for these subgroups was very similar to the age-sex adjusted results for the overall population. Therefore, these were not necessarily unique or of interest to the main objective of assessing SARV in CHF prevalence for younger age groups to assess variation in premature onset of this disease.

4.2.3 Hypertension

Figures 4.5–4.8 describe SARV in hypertension prevalence. All presented results were corrected using the Rogan and Gladen estimator formula. Similar to IHD and CHF, striking SARV in crude, unadjusted prevalence of hypertension was observed. Relative prevalence of hypertension ranged from a high of +117% above the provincial average to a low of -79% below the provincial average in Figure 4.5a, which is more similar to the range observed for CHF than for IHD. A large majority of FSAs had precise and stable

prevalence estimates, which was evidenced by very tight confidence bands around point estimates. This is due to the high overall prevalence of hypertension in the population, producing high credibility estimates that would have experienced less shrinkage towards the population average relative to the estimates for IHD and CHF. The map (Figure 4.6a) reveals that the spatial pattern of SARV in hypertension prevalence across FSAs generally followed those for IHD and CHF, with higher prevalence around Sydney and Northern Nova Scotia and lower prevalence around Halifax and central Nova Scotia.

Age-sex adjustment also resulted in substantial changes in SARV, with 60 of the 78 FSAs (76.9%) having lower prevalence estimates for hypertension compared to crude estimates, and the greatest decrease equaling -55% (Figure 4.5b). This can be seen in a comparison of the caterpillar plots, where there appears to be a slight shift in the overall distribution of the plot towards lower relative prevalence estimates. This is also observable in a comparison of the maps, where many FSAs experienced colour changes towards lower relative prevalence following adjustment, particularly in Western and Northern Nova Scotia and Cape Breton Island (Figure 4.6b). However, similar to IHD and CHF, many FSAs around Halifax had increased prevalence following adjustment, again suggesting that these areas have younger population age structures relative to the provincial average. It also appears that age-sex adjustment resulted in less overall change in SARV in prevalence for hypertension as compared to IHD or especially CHF. Many FSAs, particularly those on Cape Breton Island, experienced minimal change in relative prevalence estimates, characterized by few colour changes in these areas. A probable explanation for this phenomenon is that unlike CHF or IHD, where age is an important contributor that drives prevalence, hypertension is a lifelong condition, appearing as an early risk factor for downstream CVD events on the CVD continuum. [16] Thus, SARV in hypertension prevalence is expected to change less consistently when adjusting for age and sex as compared to when doing so for CHF and IHD. This means that age is likely a much less significant factor contributing to SARV in hypertension prevalence as compared to its relative importance for SARV in IHD and especially CHF prevalence.

Finally, Figures 4.11 and 4.12 present caterpillar plots and maps used to assess SARV in age-adjusted hypertension prevalence for younger age groups (with the older age group results in Appendix B). The overall patterns of SARV in hypertension

prevalence for all four subgroups generally followed the variation found in the full study population, matching IHD. Again, there were exceptions, with lower hypertension prevalence estimated on Cape Breton Island for younger females in FSAs B1R and B1S, and younger males in FSAs B0E, B1J, B1L, B1P, and B1N, and B1S. Conversely, higher hypertension prevalence was estimated around Halifax for younger females in FSAs B2Z, B3G, and B3T, and younger males in FSAs B2Z, B2V, and B3T. Second, patterns were also similar between the subgroups, in that FSAs with low or high CVD prevalence in one subgroup often had prevalence estimates in the same direction for all subgroups. However, similar to IHD, there were exceptions. As one example, Sydney North (FSA: B1N) had far above average relative prevalence for younger females and above average prevalence for older females, but had average prevalence for younger males and above average prevalence for older males. Thus, similar to Guysborough for IHD, this is an area experiencing sex differences, but with reverse patterns (i.e., greater potential issues of ageing and CVD management specific to males and greater potential issues of premature onset of CVD in females). Lastly, similar to IHD, there was a wider range of relative prevalence estimates for the two younger age groups relative to the two older age groups. A possible explanation for this is that because hypertension is less age-related, it may manifest more inconsistently in the younger age groups relative to the older age groups—where it is more consistently prevalent.

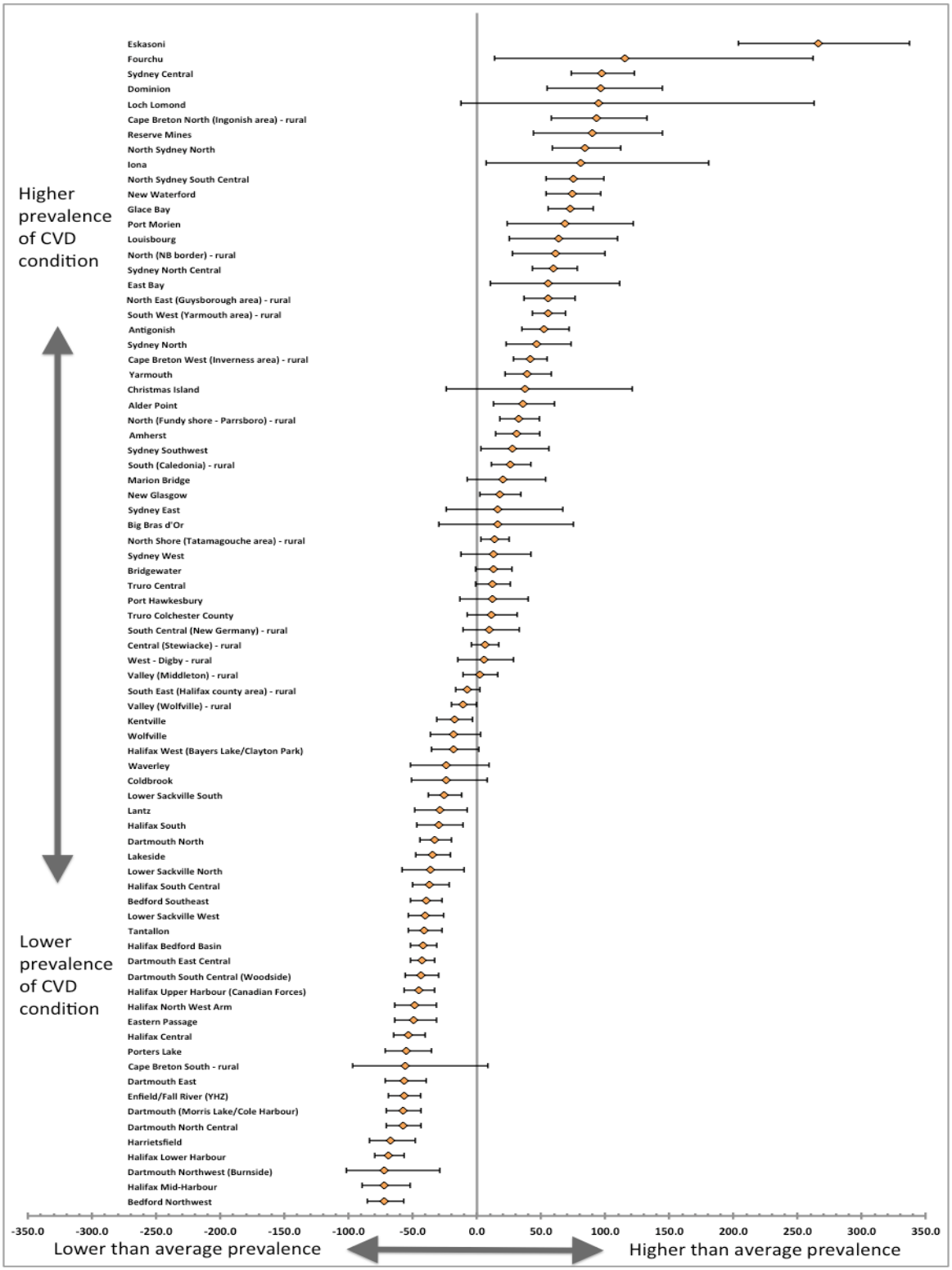

Figure 4.1a Caterpillar plot depicting SARV in crude (unadjusted) IHD prevalence by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error

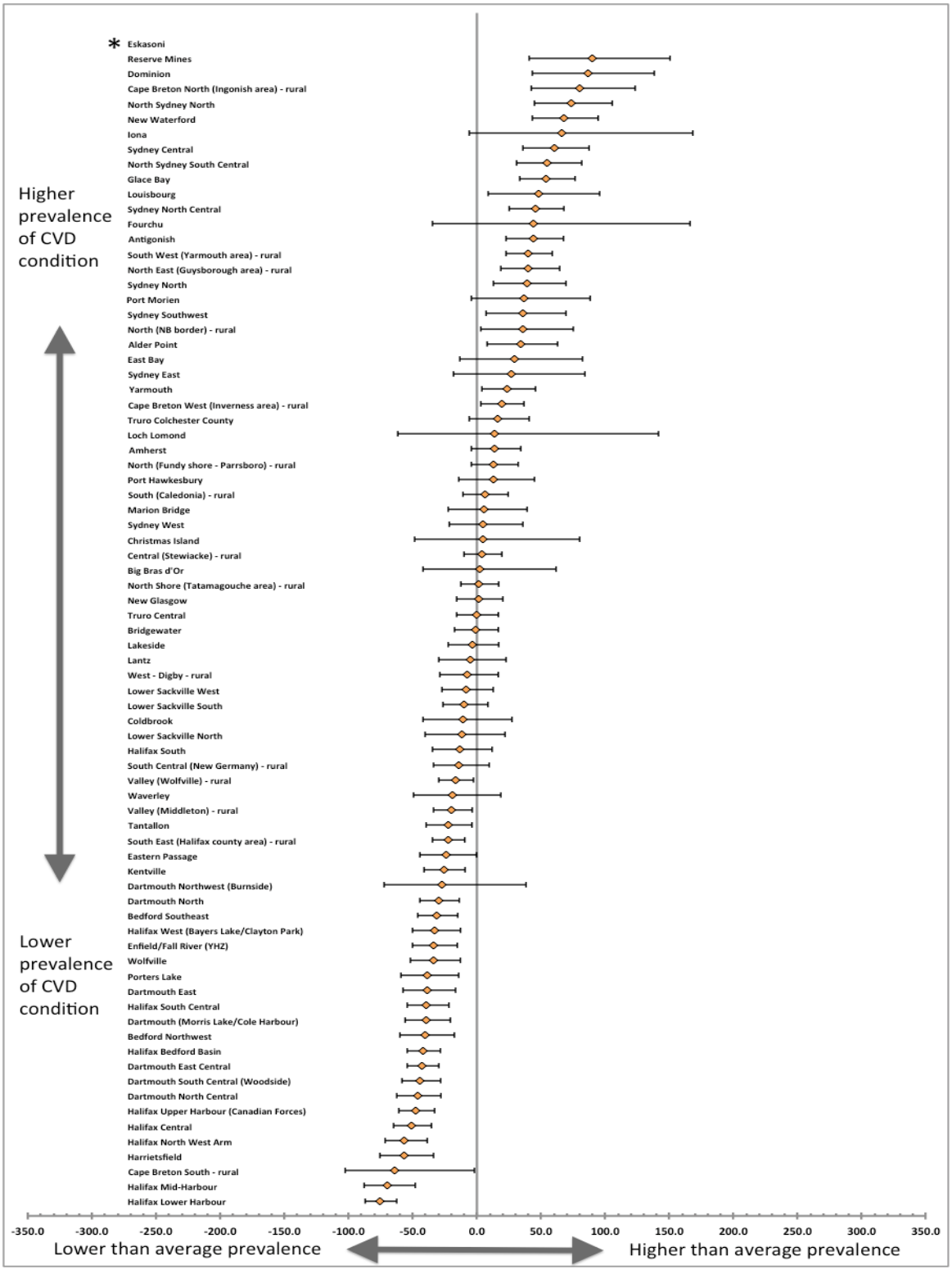

Figure 4.1b Caterpillar plot depicting SARV in age-sex adjusted IHD prevalence by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error. * indicates data not shown for that particular FSA

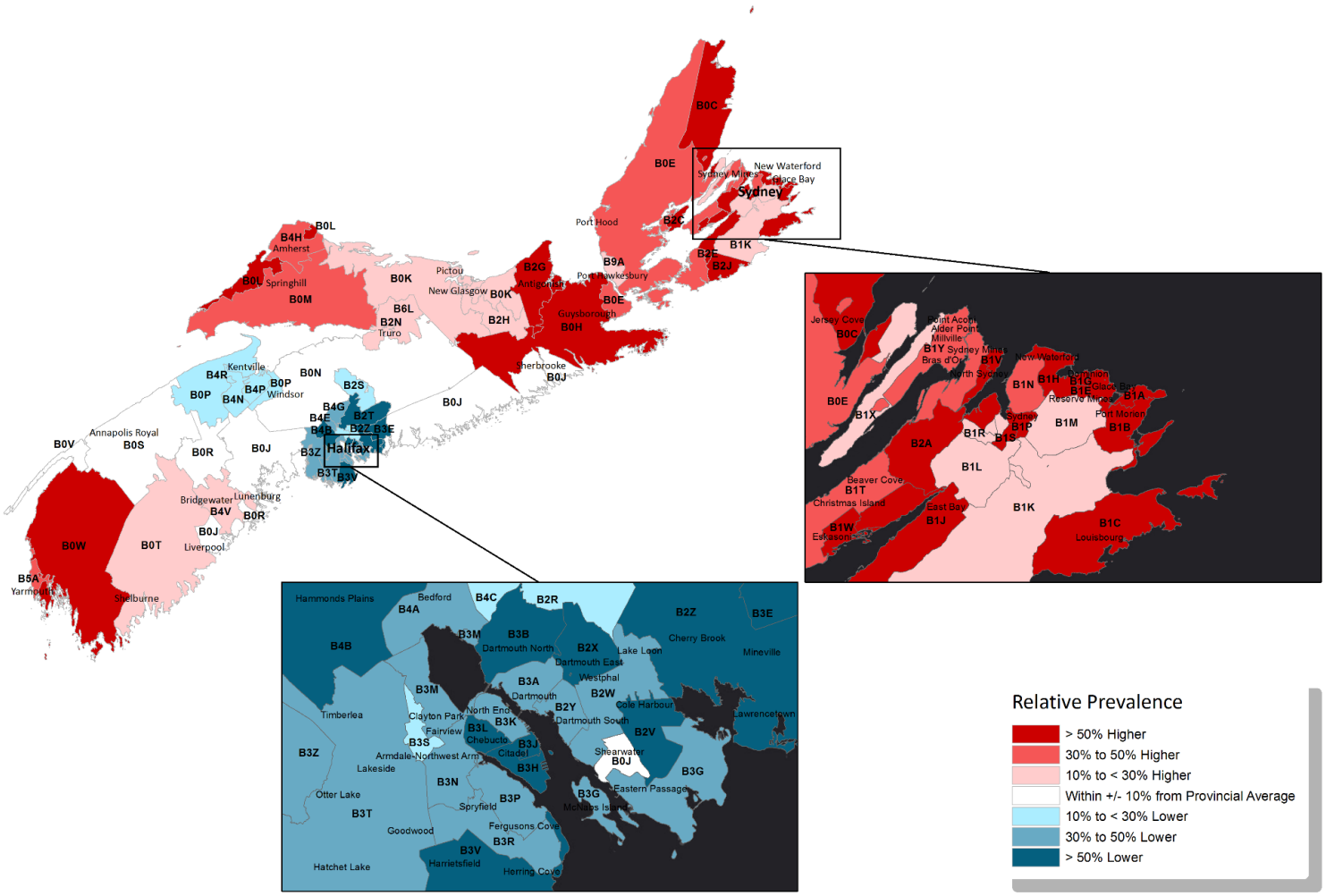

Figure 4.2a Map showing SARV in crude (unadjusted) IHD prevalence relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error

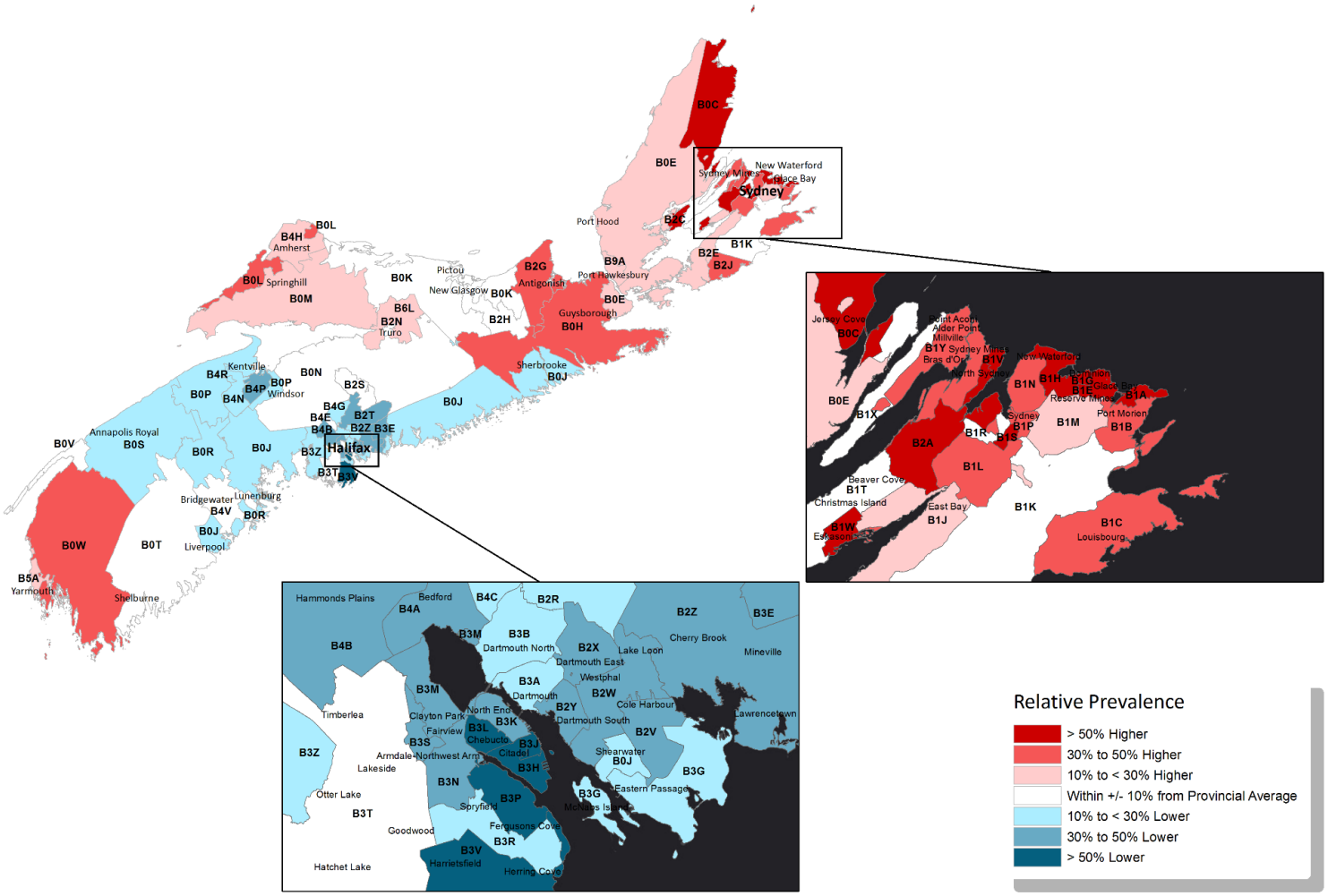

Figure 4.2b Map showing SARV in age-sex adjusted IHD prevalence relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error

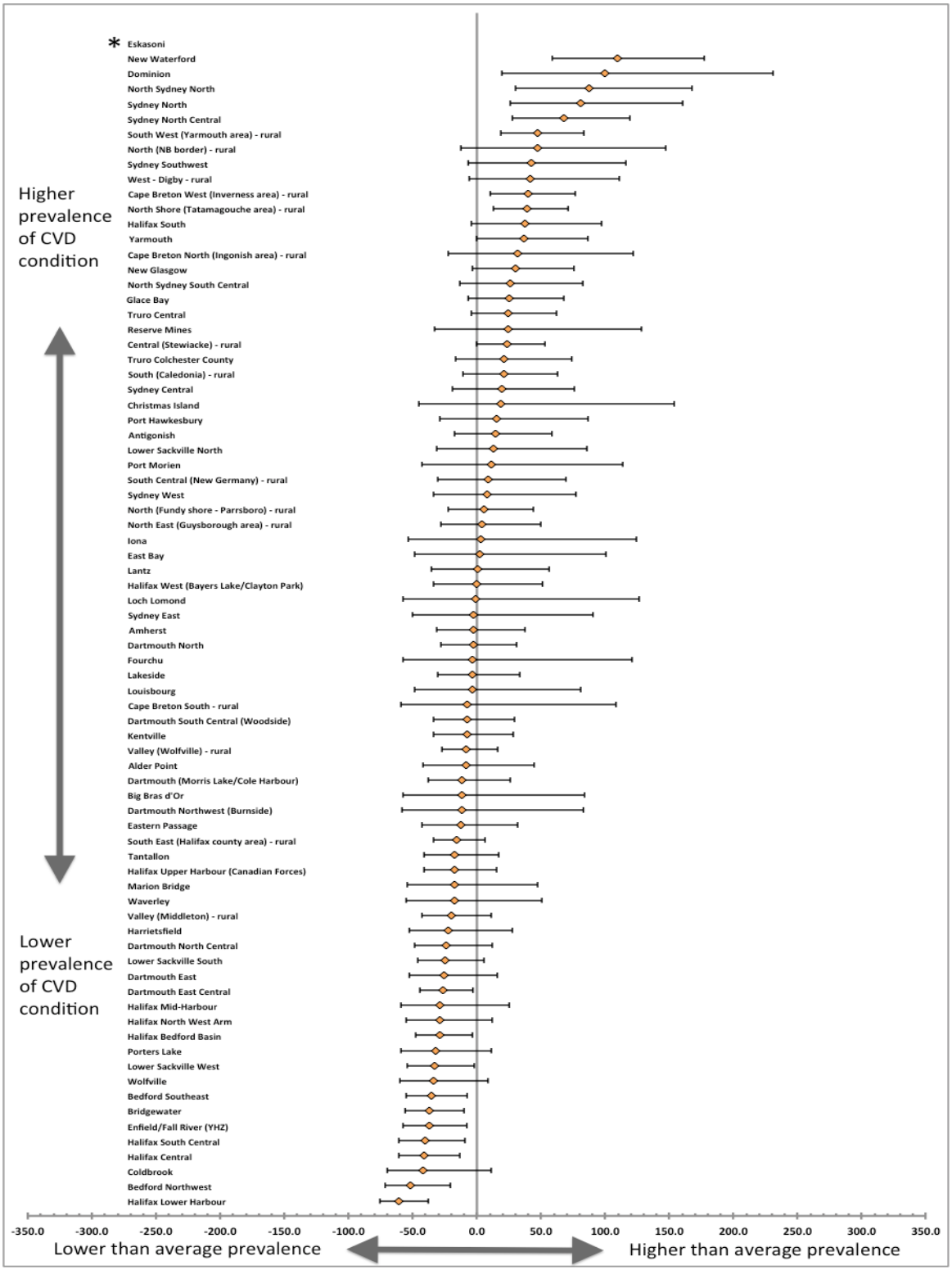

Figure 4.3a Caterpillar plot depicting SARV in age-adjusted IHD prevalence for females less than 60 years of age by FSA, Nova Scotia, 2010-11 to 2012-13. Not corrected for misclassification error. * indicates data not shown for that particular FSA

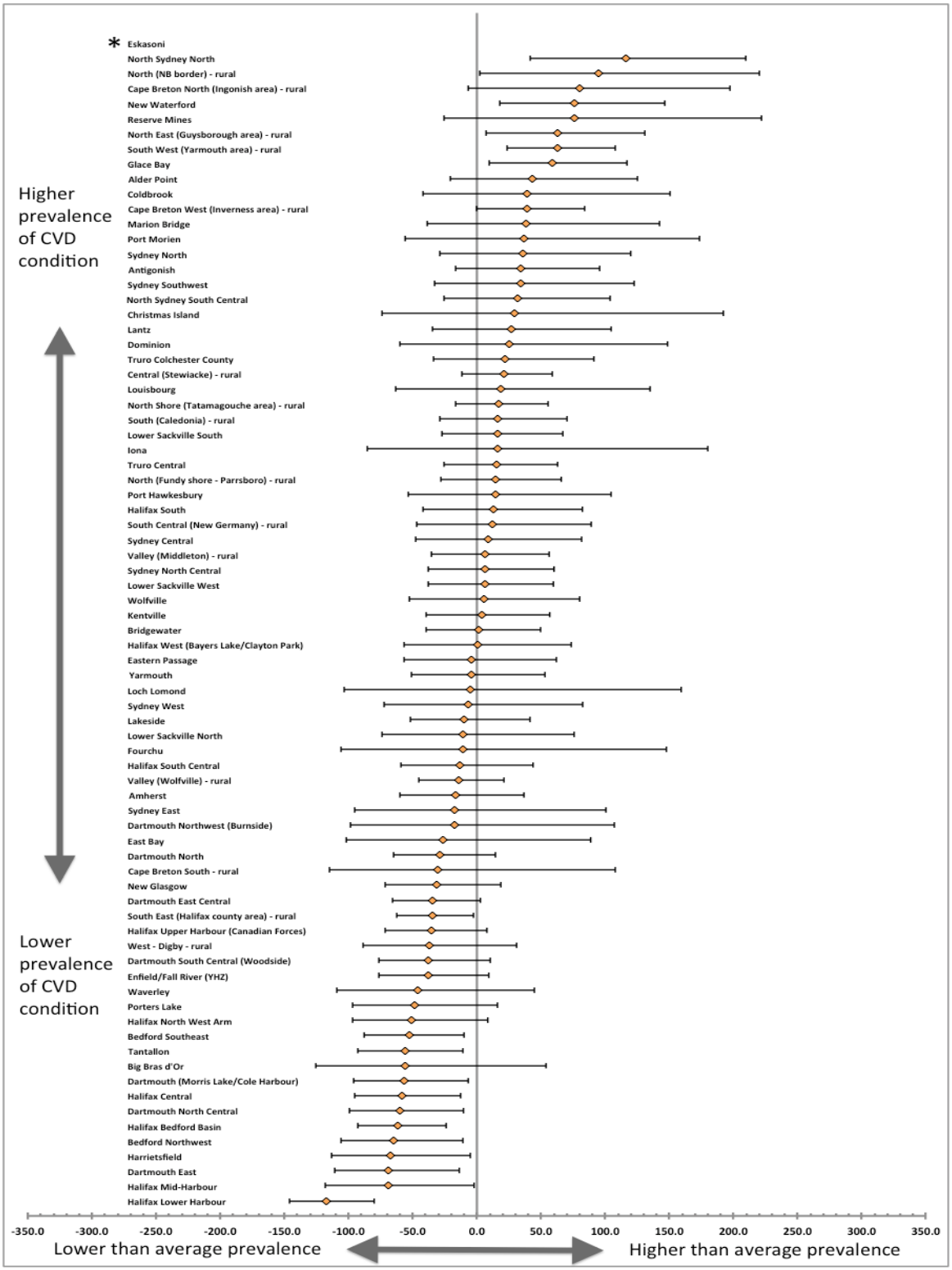

Figure 4.3b Caterpillar plot depicting SARV in age-adjusted IHD prevalence for males less than 60 years of age by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error. * indicates data not shown for that particular FSA

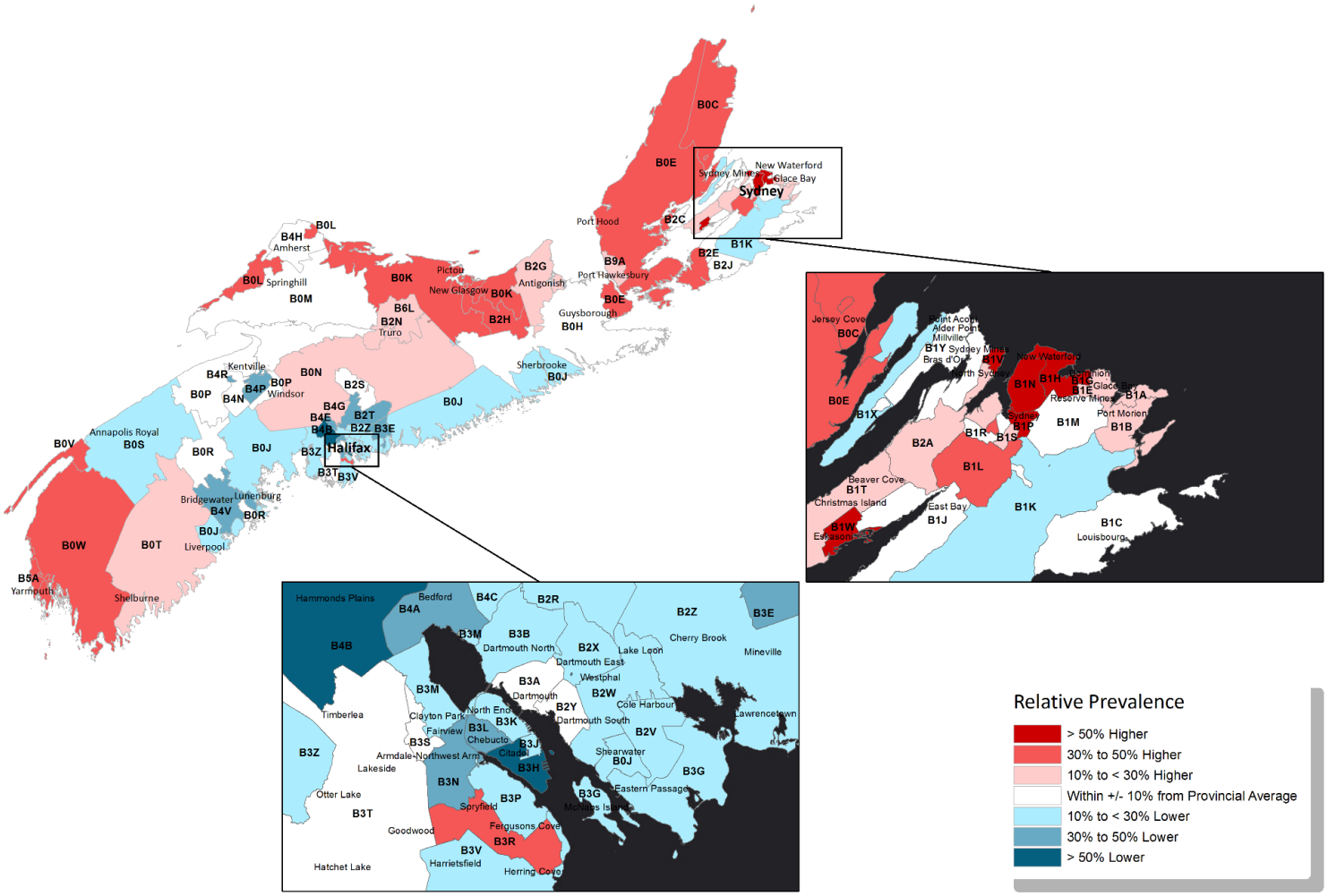

Figure 4.4a Map showing SARV in age-adjusted IHD prevalence for females less than 60 years of age relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Not corrected for misclassification error

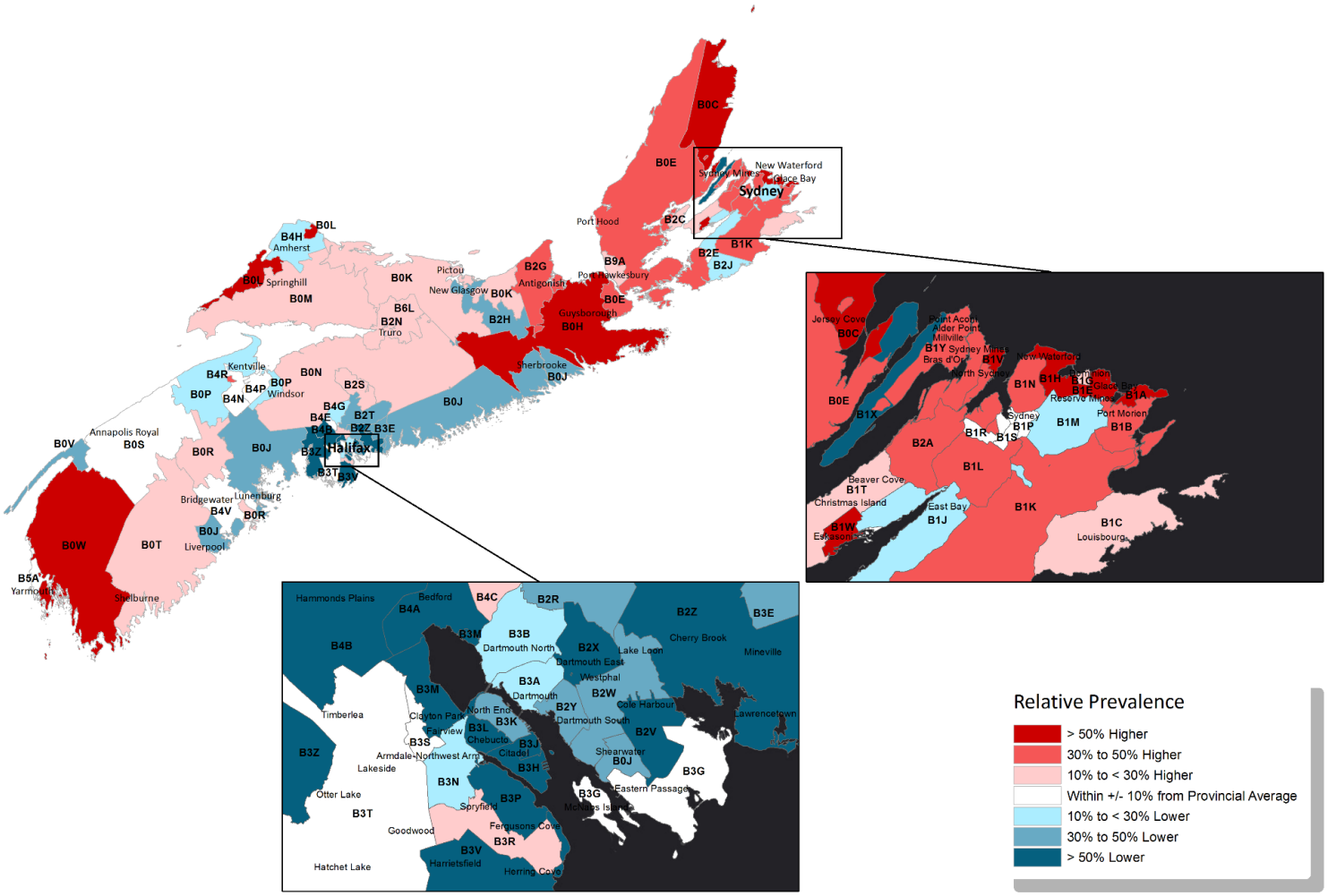

Figure 4.4b Map showing SARV in age-adjusted IHD prevalence for males less than 60 years of age relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error

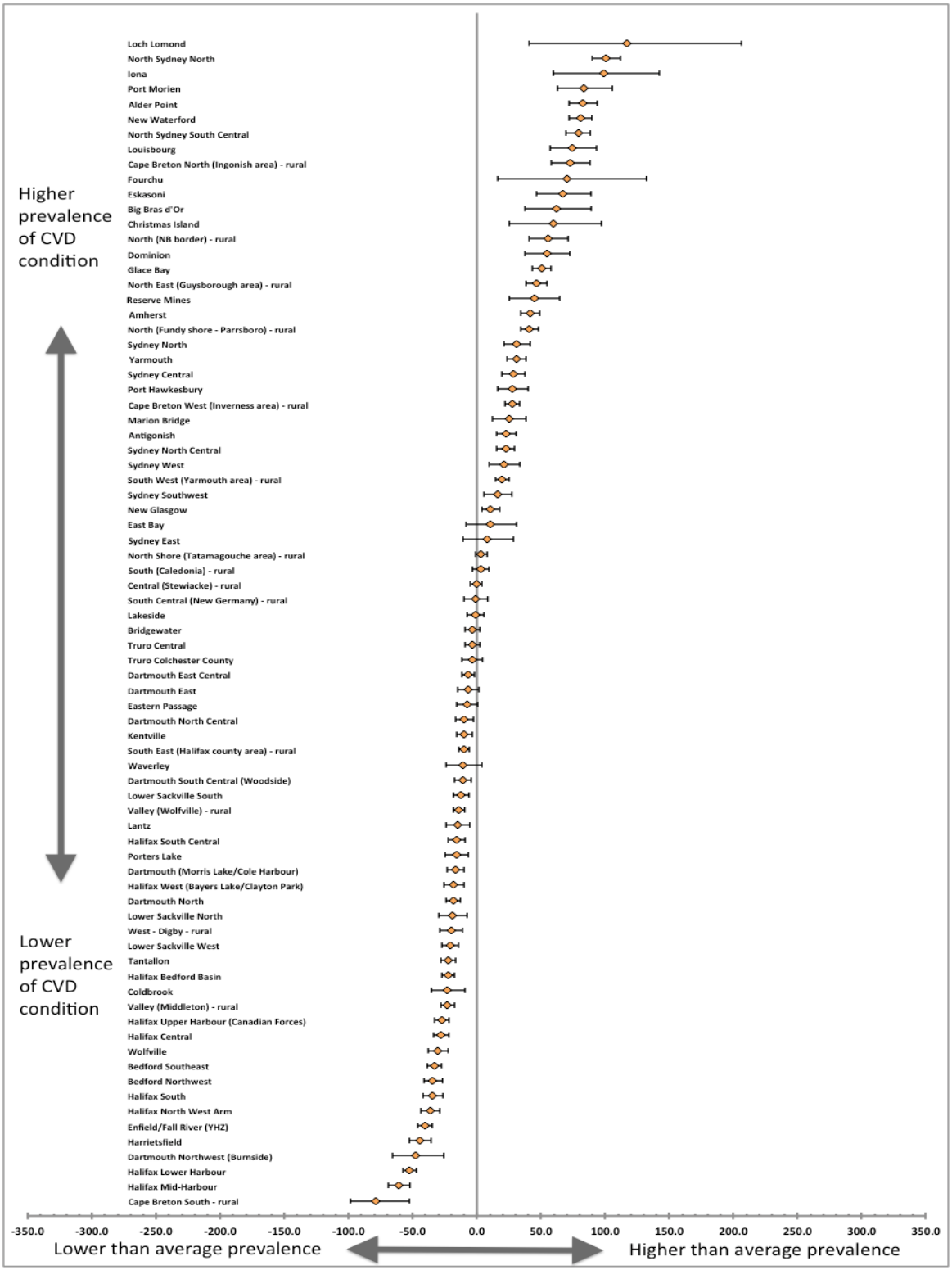

Figure 4.5a Caterpillar plot depicting SARV in crude (unadjusted) hypertension prevalence by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error

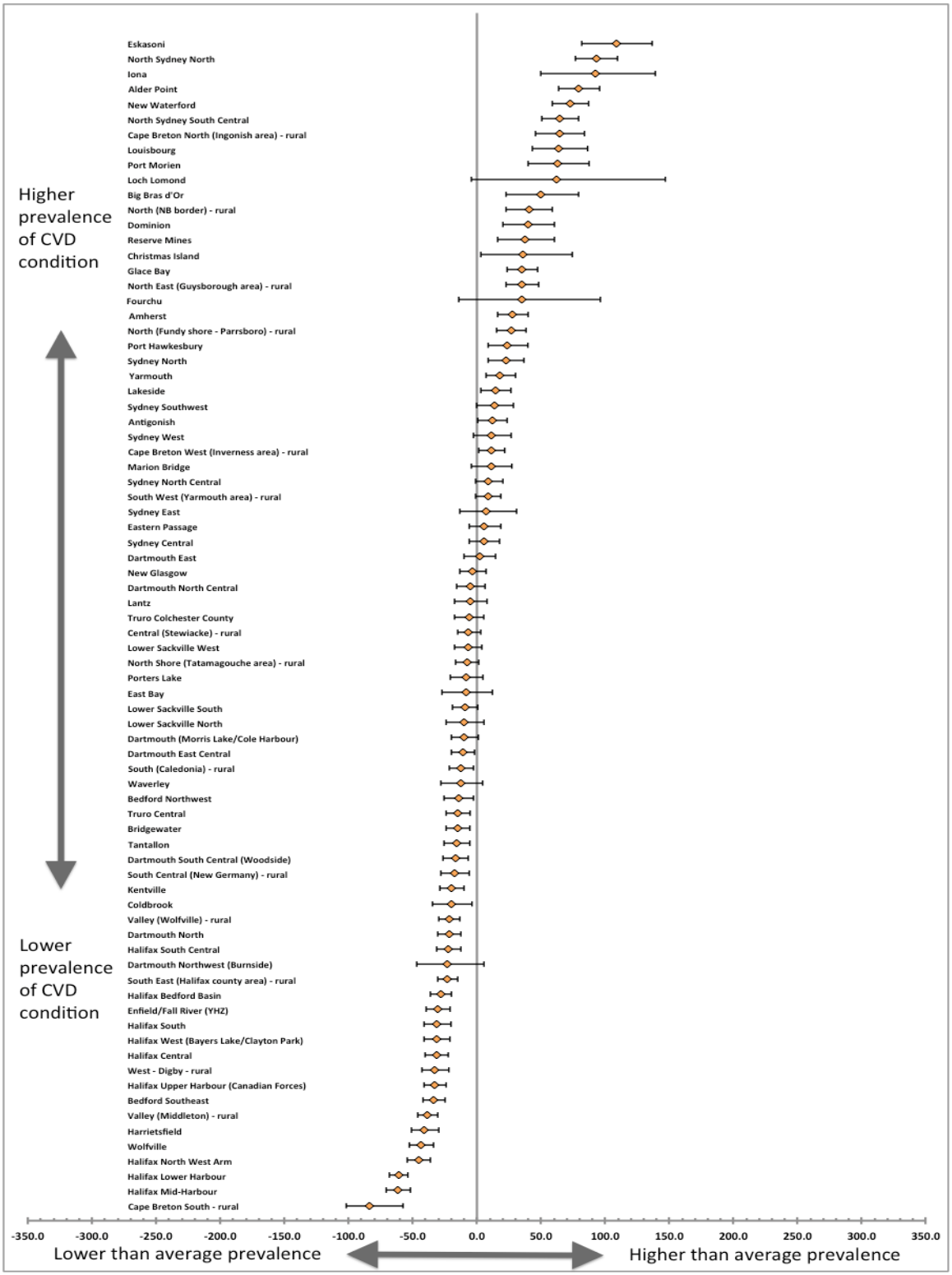

Figure 4.5b Caterpillar plot depicting SARV in age-sex adjusted hypertension prevalence by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error

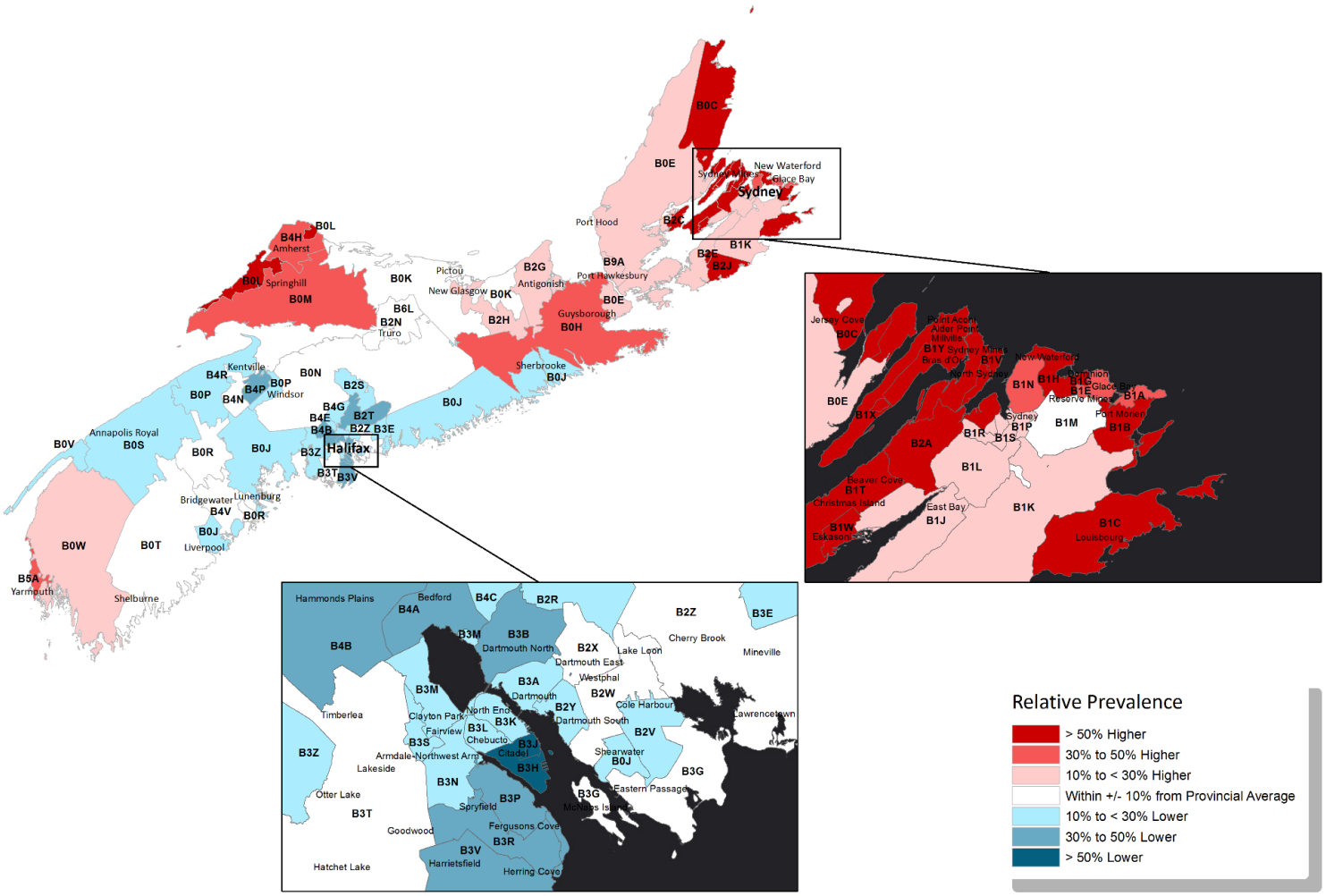

Figure 4.6a **Map showing SARV in crude (unadjusted) hypertension prevalence relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error**

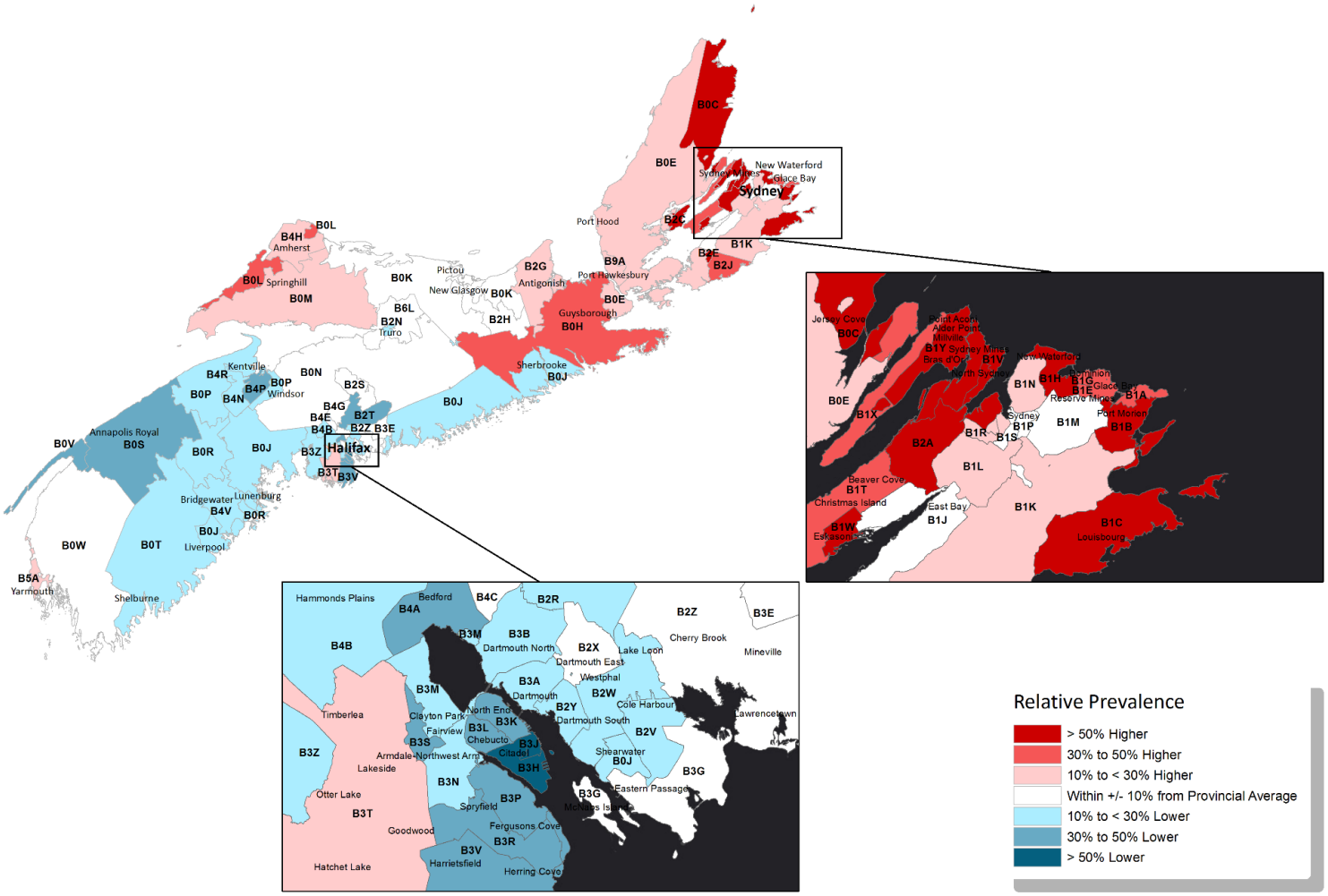

Figure 4.6b Map showing SARV in age-sex adjusted hypertension prevalence relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error

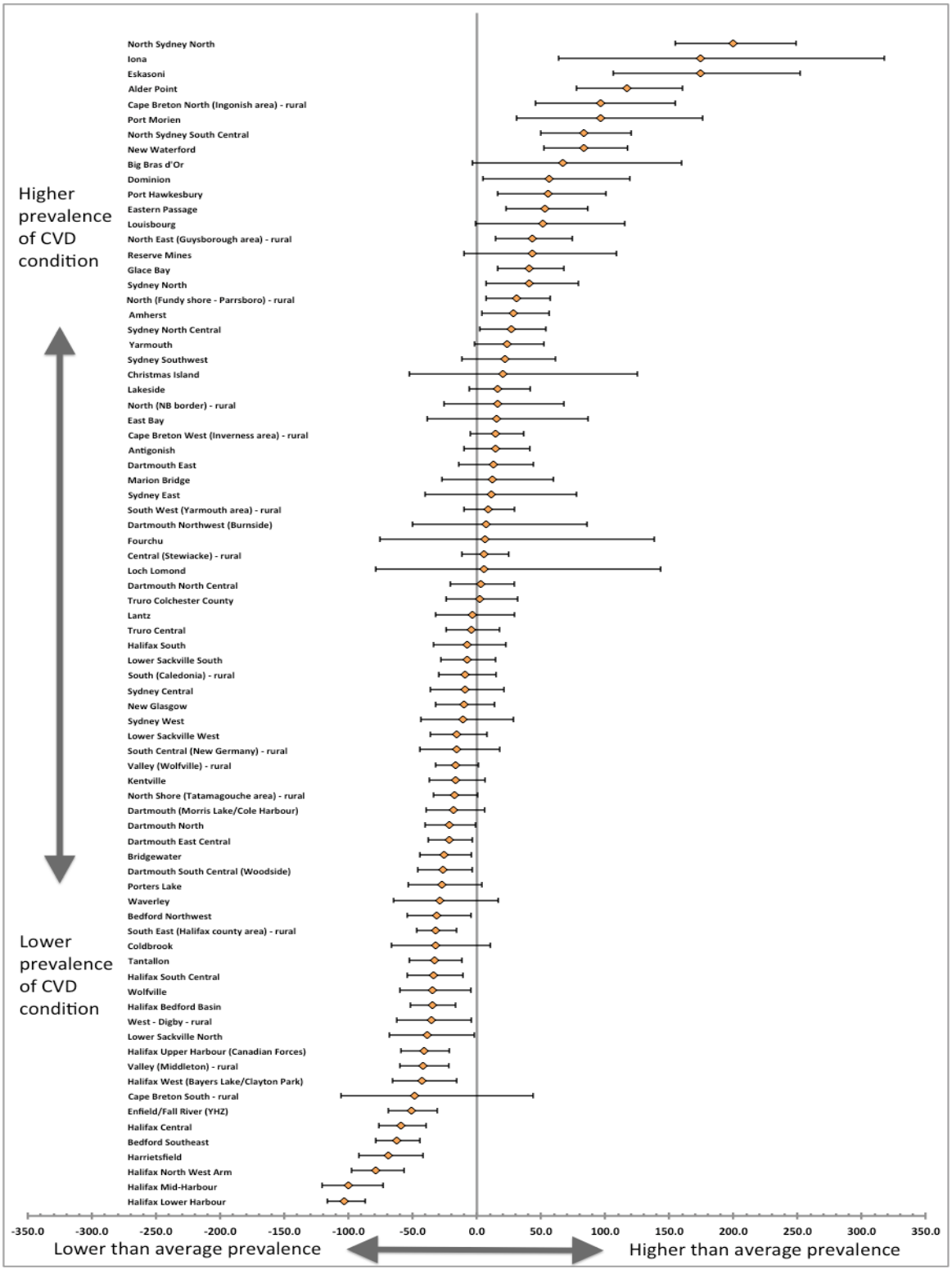

Figure 4.7a Caterpillar plot depicting SARV in age-adjusted hypertension prevalence for females less than 60 years of age by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error

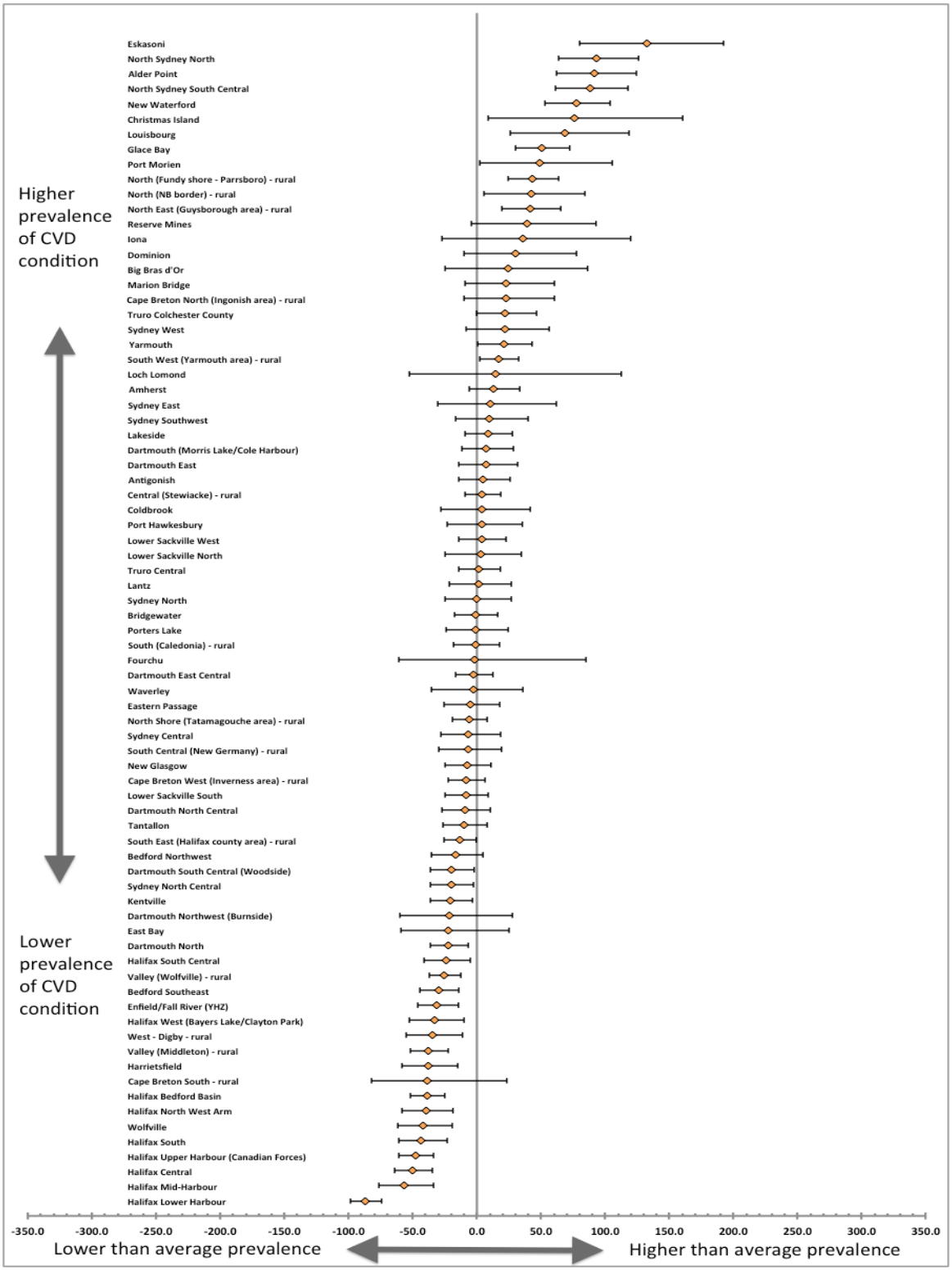

Figure 4.7b Caterpillar plot depicting SARV in age-adjusted hypertension prevalence for males less than 60 years of age by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error

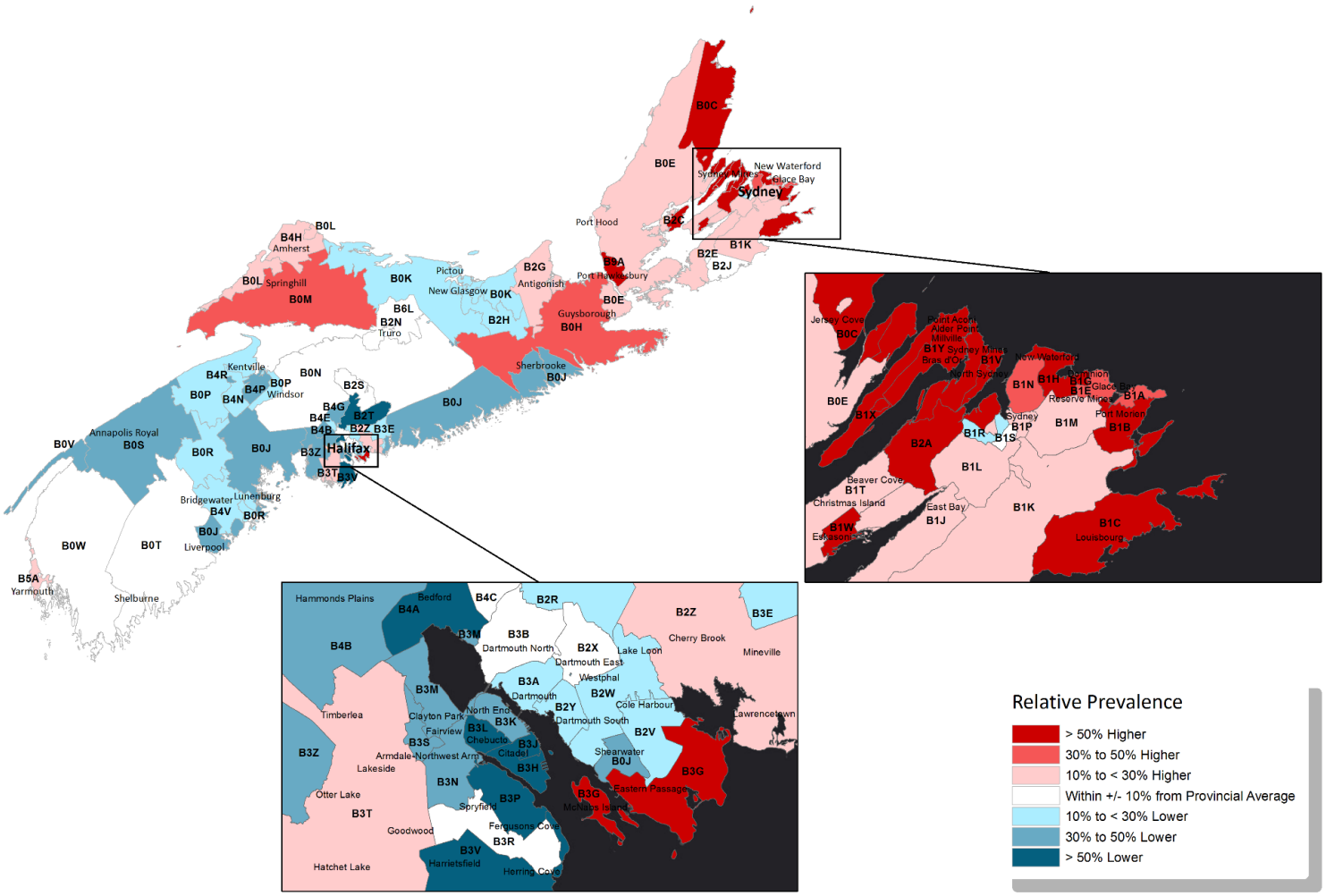

Figure 4.8a Map showing SARV in age-adjusted hypertension prevalence for females less than 60 years of age relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error

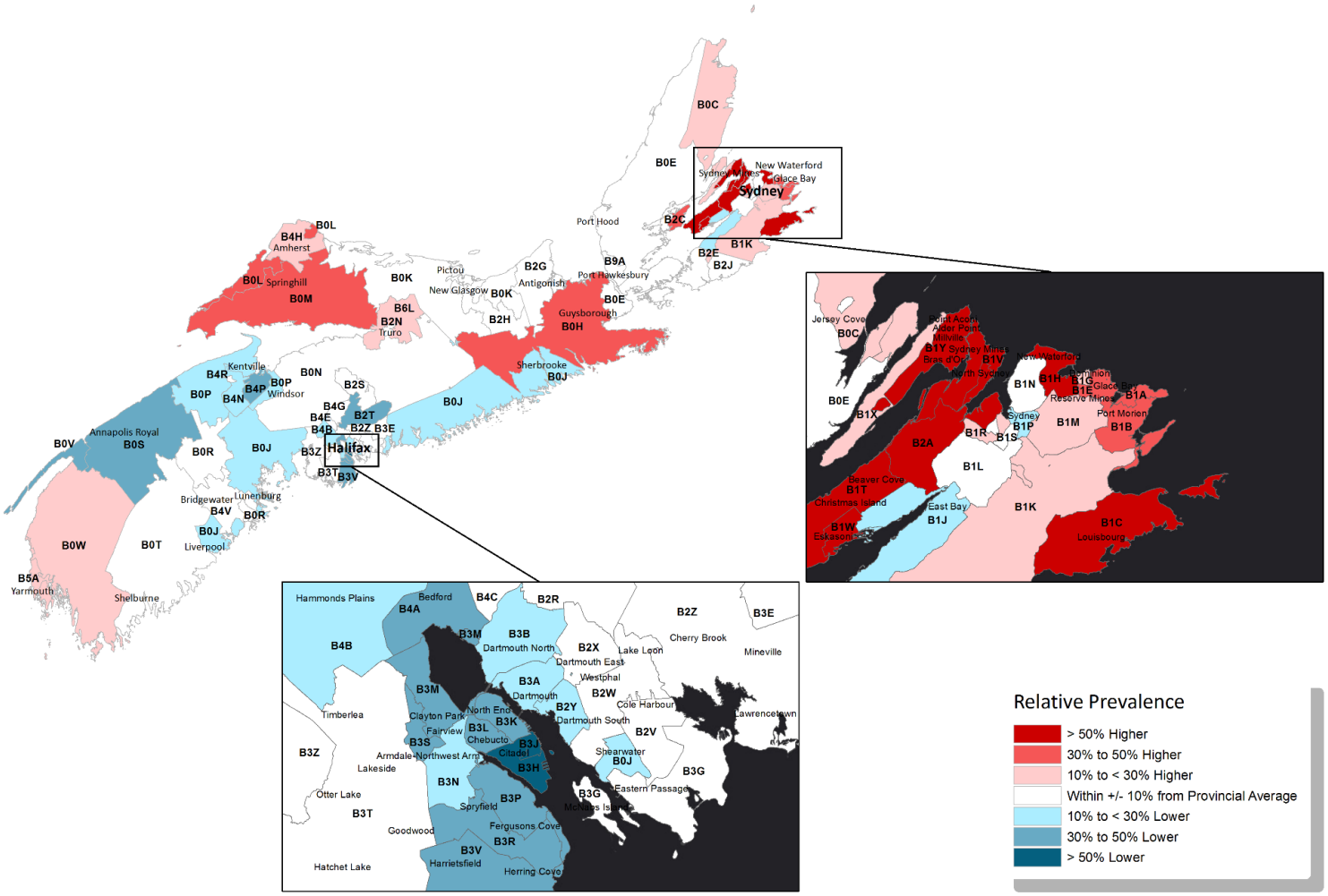

Figure 4.8b Map showing SARV in age-adjusted hypertension prevalence for males less than 60 years of age relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error

4.3 ASSOCIATIONS BETWEEN CVD AND INDIVIDUAL AND AREA-LEVEL VARIABLES

Measures of area variation and associations between IHD, CHF, and hypertension and individual- and area-level variables are presented in Tables 4.4–4.6 at the end of *Section 4.3*. For all three CVD-component conditions, models indicate substantial between-area variation in prevalence, and that FSAs have large and significant effects on the individual probability of having CVD. Looking at the tables, the VPC in the null model (Model 1)—prior to the addition of covariates—was 3.80 for IHD, 3.58 for CHF, and 3.83 for hypertension. This means that about 3.5% to 3.8% of the total variation in the propensity to have each CVD can be attributed to differences between areas (i.e., lies at the area level), and that most of the variation occurs at the individual level. Although these VPC measures may seem small, the MORs (which translate between-area variance onto the OR scale for more intuitive comparisons of random effects with fixed effects) were 1.41 for IHD, 1.39 for CHF, and 1.41 for hypertension. This means that when randomly picking out two persons in different areas, the area effects of FSAs increased by about 1.39-1.41 times the individual odds of having CVD when moving to the area with increased probability of CVD. [91] This is a relatively large effect. Moreover, although the addition of covariates to increasingly complex models sequentially reduced the MORs (and VPCs), they all remained significantly above 1 in all of the models. This indicates both the presence of residual, unexplained variation, and that areas retained significance in explaining the individual probability of having CVD.

Area-level variables had large effects relative to individual-level, compositional variables in explaining between-area variation in CVD prevalence. This can be shown in a couple of ways. One way is by assessing how measures of variation changed between increasingly complex models as more variables were added to the models. It can be seen that age-sex adjustment in Model 2 resulted in only modest decreases for all three measures of between-area variation for all three conditions (e.g., the VPC and MOR for IHD only decreased from 3.80 to 3.21 and from 1.41 to 1.37 from Model 1 to 2). Following age-sex adjustment, area effects retained their importance for explaining variation in CVD prevalence. However, further adjustments with area-level variables resulted in much larger reductions in these measures of area variation in comparison to

the individual-level variables. For example, adding area measures of social isolation (Model 3) significantly reduced the VPC from 3.21 to 0.72 and the MOR from 1.37 to 1.16 for IHD. Similarly, adding area measures of material deprivation (Model 4) also reduced the VPC from 3.21 to 1.08 and the MOR from 1.37 to 1.20. Finally, the full model with all area-level variables included drastically attenuated the VPC from 3.21 to 0.54 and the MOR from 1.37 to 1.14. Thus, area-level variables have large effects on variation in prevalence relative to individual-level variables. Also, area measures of social isolation account for more between-area variation in IHD and CHF prevalence than measures of material deprivation, while the opposite is true for hypertension.

An alternative method of determining the relative contribution of area-level SES to variation in CVD prevalence is by assessing fixed effect ORs. It can be seen that many of the area SES variables achieved statistical significance at the $\alpha = 0.05$ level, and many remained significant with sequential modeling using increasingly complex models. However, caution is warranted when interpreting the meaning of associations for area-level variables, as usual OR interpretations are incorrect for these variables. Thus, the IOR for each area-level variable was used to help guide interpretations, by integrating the between-area variance with the fixed effects of the area-level variables. [84,85] First, it can be seen in the tables that the IOR is wide for many area-level variables (e.g., IOR of % of single mother families in Model 3 for IHD is 7.48–13.10). This means that the unexplained (residual) between-area variation in the individual propensity to have CVD is large, indicating that the likelihood is high of finding a person in a FSA with a low proportion of single mothers who presents a higher odds of IHD than a person in a FSA with a high proportion of single mothers. However, it can also be seen that the IOR for many variables does not include 1, which indicates that the fixed effect of the area-level variable under consideration is large in comparison to the residual between-area variation. For the example, this means that a fairly large proportion of the (large) between-area variation in the individual propensity for CVD can be explained by % of single mother families at the FSA level. This indirectly supports the contribution of certain measures of area SES to area variation in CVD prevalence.

Table 4.4 ORs (95% CIs) of IHD associated with individual- and area-level variables, Nova Scotia, 2010-11 to 2012-13

Covariate	Model 1[a]	Model 2[a]	Model 3[a]	Model 4[a]	Model 5[a]
			Fixed effects OR (95% CI)		
			Individual-level		
Age-sex group (vs. females 60-69)					
Females 40-49		0.19 (0.17–0.21)*	0.19 (0.17–0.21)*	0.19 (0.17–0.21)*	0.19 (0.17–0.21)*
Females 50-59		0.46 (0.42–0.49)*	0.46 (0.42–0.49)*	0.46 (0.42–0.49)*	0.46 (0.42–0.49)*
Females 70-79		2.06 (1.94–2.19)*	2.06 (1.94–2.19)*	2.06 (1.94–2.19)*	2.06 (1.94–2.19)*
Females 80+		3.37 (3.11–3.64)*	3.36 (3.11–3.64)*	3.36 (3.11–3.64)*	3.36 (3.11–3.64)*
Males 40-49		0.45 (0.42–0.49)*	0.45 (0.42–0.49)*	0.45 (0.42–0.49)*	0.45 (0.42–0.48)*
Males 50-59		1.34 (1.26–1.41)*	1.34 (1.26–1.41)*	1.34 (1.26–1.41)*	1.34 (1.26–1.41)*
Males 60-69		2.71 (2.58–2.85)*	2.71 (2.58–2.85)*	2.71 (2.58–2.84)*	2.71 (2.58–2.84)*
Males 70-79		4.55 (4.31–4.80)*	4.55 (4.31–4.80)*	4.55 (4.31–4.79)*	4.55 (4.31–4.80)*
Males 80+		5.59 (5.19–6.01)*	5.59 (5.19–6.01)*	5.59 (5.19–6.01)*	5.59 (5.19–6.01)*
			Area-level		
Single mother families			9.90 (4.23–23.17)*		5.90 (2.52–13.84)*
IOR (80% interval)			[7.48–13.10]		[4.63–7.53]
Living alone			0.06 (0.02–0.19)*		0.28 (0.08–0.99)*
IOR (80% interval)			[0.04–0.08]		[0.22–0.35]
Aboriginal identity			3.92 (2.92–5.26)*		3.29 (2.20–4.92)*
IOR (80% interval)			[2.96–5.19]		[2.58–4.19]
Population > 65 years of age			27.80 (11.20–69.02)*	0.58 (0.07–5.00)	6.00 (1.91–18.84)*
IOR (80% interval)			[21.00-36.80]	[0.41–0.82]	[4.71–7.65]
Household total income < $20,000				0.57 (0.21–1.55)	0.31 (0.13–0.78)*
IOR (80% interval)				[0.40–0.80]	[0.25–0.40]
Unemployment				1.03 (1.02–1.04)*	1.02 (1.01–1.02)*
IOR (80% interval)				[0.73–1.45]	[0.80–1.30]
Post-secondary education				0.94 (0.20–4.42)	1.24 (0.49–3.18)
IOR (80% interval)				[0.67–1.32]	[0.98–1.59]
No high school diploma				6.97 (0.93–52.25)	3.16 (0.89–11.24)
IOR (80% interval)				[4.95–9.83]	[2.48–4.03]

Covariate	Model 1[a]	Model 2[a]	Model 3[a]	Model 4[a]	Model 5[a]
Measures of area variation and random effects					
Variance[b] (SE)	0.130 (0.023)	0.109 (0.020)	0.024 (0.005)	0.036 (0.008)	0.018 (0.004)
VPC (%)	3.80 (2.52–5.05)	3.21 (2.08–4.31)	0.72 (0.43–1.02)	1.08 (0.61–1.55)	0.54 (0.31–0.78)
MOR (95% CrI)	1.41 (1.32–1.49)	1.37 (1.29–1.44)	1.16 (1.12–1.19)	1.20 (1.15–1.24)	1.14 (1.10–1.16)

Note: OR, odds ratio; CI, confidence interval; IOR, interval odds ratio; SE, standard error; VPC, variance partitioning coefficient; MOR, median odds ratio; CrI, credible interval.

a. Model 1 is an intercept only model. Model 2 is adjusted for individual-level variables (age-sex interaction term). Model 3 adds measures of area social isolation to Model 2 (% of single mother census families, % of persons living alone in private households, and % of population with Aboriginal identity), and a control variable of the % of the population greater than 65 years of age. Model 4 adds measures of area material deprivation to Model 2 (% of households with a total income of less than $20,000, % of population that is unemployed, % of population with a post-secondary certificate, diploma, or degree, and % of population with no high school diploma), and a control variable of the % of the population greater than 65 years of age. Model 5 is a full model that adjusts for all individual- and area-level variables.

b. Measuring area-level variance (σ_u^2).

*$p < 0.05$.

Table 4.5 ORs (95% CIs) of CHF associated with individual- and area-level variables, Nova Scotia, 2010-11 to 2012-13

Covariate	Model 1[a]	Model 2[a]	Model 3[a]	Model 4[a]	Model 5[a]
Fixed effects OR (95% CI)					
Individual-level					
Age-sex group (vs. females 60-69)					
Females 40-49		0.10 (0.08–0.12)*	0.10 (0.08–0.12)*	0.10 (0.08–0.12)*	0.10 (0.08–0.12)*
Females 50-59		0.29 (0.25–0.34)*	0.29 (0.25–0.34)*	0.29 (0.25–0.34)*	0.29 (0.25–0.34)*
Females 70-79		2.79 (2.50–3.13)*	2.78 (2.49–3.12)*	2.79 (2.49–3.12)*	2.79 (2.49–3.12)*
Females 80+		7.67 (6.83–8.61)*	7.65 (6.82–8.59)*	7.67 (6.83–8.61)*	7.66 (6.83–8.60)*
Males 40-49		0.15 (0.13–0.17)*	0.15 (0.13–0.17)*	0.15 (0.13–0.17)*	0.15 (0.13–0.17)*
Males 50-59		0.51 (0.45–0.59)*	0.52 (0.45–0.59)*	0.52 (0.45–0.59)*	0.52 (0.45–0.59)*
Males 60-69		1.54 (1.36–1.74)*	1.53 (1.36–1.73)*	1.53 (1.36–1.73)*	1.53 (1.36–1.73)*
Males 70-79		4.00 (3.56–4.45)*	3.97 (3.55–4.45)*	3.97 (3.55–4.44)*	3.97 (3.55–4.44)*
Males 80+		9.86 (8.58–11.34)*	9.85 (8.57–11.33)*	9.85 (8.57–11.33)*	9.85 (8.57–11.33)*

Covariate	Model 1[a]	Model 2[a]	Model 3[a]	Model 4[a]	Model 5[a]
Area-level					
Single mother families			21.06 (7.42–59.76)*		10.16 (3.71–27.77)*
IOR (80% interval)			[16.63–26.67]		[8.33–12.38]
Living alone			0.17 (0.05–0.59)*		0.55 (0.11–2.63)
IOR (80% interval)			[0.14–0.22]		[0.45–0.67]
Aboriginal identity			1.78 (1.21–2.62)*		1.25 (0.82–1.90)
IOR (80% interval)			[1.41–2.25]		[1.02–1.52]
Population > 65 years of age			9.27 (3.26–26.33)*	0.64 (0.21–1.99)	1.69 (0.54–5.27)
IOR (80% interval)			[7.32–11.73]	[0.50–0.83]	[1.39–2.06]
Household total income < $20,000				1.27 (0.60–2.70)	0.68 (0.21–2.20)
IOR (80% interval)				[0.98–1.64]	[0.56–0.83]
Unemployment				1.02 (1.01–1.03)*	1.01 (1.00–1.02)*
IOR (80% interval)				[0.79–1.32]	[0.83–1.23]
Post-secondary education				0.23 (0.05–1.06)	0.87 (0.24–3.15)
IOR (80% interval)				[0.18–0.30]	[0.71–1.05]
No high school diploma				1.17 (0.21–6.45)	2.76 (0.70–10.94)
IOR (80% interval)				[0.90–1.50]	[2.26–3.36]
Measures of area variation and random effects					
Variance[b] (SE)	0.122 (0.024)	0.060 (0.013)	0.017 (0.005)	0.020 (0.006)	0.012 (0.004)
VPC (%)	3.58 (2.23–4.89)	1.79 (1.04–2.53)	0.51 (0.22–0.81)	0.60 (0.25–0.96)	0.36 (0.13–0.60)
MOR (95% CrI)	1.39 (1.30–1.48)	1.26 (1.19–1.32)	1.13 (1.08–1.17)	1.14 (1.09–1.18)	1.11 (1.06–1.14)

Note: OR, odds ratio; CI, confidence interval; IOR, interval odds ratio; SE, standard error; VPC, variance partitioning coefficient; MOR, median odds ratio; CrI, credible interval.

a. Model 1 is an intercept only model. Model 2 is adjusted for individual-level variables (age-sex interaction term). Model 3 adds measures of area social isolation to Model 2 (% of single mother census families, % of persons living alone in private households, and % of population with Aboriginal identity), and a control variable of the % of the population greater than 65 years of age. Model 4 adds measures of area material deprivation to Model 2 (% of households with a total income of less than $20,000, % of population that is unemployed, % of population with a post-secondary certificate, diploma, or degree, and % of population with no high school diploma), and a control variable of the % of the population greater than 65 years of age. Model 5 is a full model that adjusts for all individual- and area-level variables.

b. Measuring area-level variance (σ_u^2).

*$p < 0.05$.

Table 4.6 ORs (95% CIs) of hypertension associated with individual- and area-level variables, Nova Scotia, 2010-11 to 2012-13

Covariate	Model 1[a]	Model 2[a]	Model 3[a]	Model 4[a]	Model 5[a]
			Fixed effects OR (95% CI)		
			Individual-level		
Age-sex group (vs. females 60-69)					
Females 40-49		0.25 (0.24–0.26)*	0.25 (0.24–0.26)*	0.25 (0.24–0.26)*	0.25 (0.24–0.26)*
Females 50-59		0.53 (0.52–0.55)*	0.53 (0.52–0.55)*	0.53 (0.52–0.55)*	0.53 (0.52–0.55)*
Females 70-79		1.57 (1.52–1.63)*	1.57 (1.52–1.63)*	1.57 (1.52–1.63)*	1.57 (1.52–1.63)*
Females 80+		1.76 (1.66–1.86)*	1.76 (1.66–1.86)*	1.76 (1.66–1.87)*	1.76 (1.66–1.87)*
Males 40-49		0.31 (0.30–0.33)*	0.31 (0.30–0.33)*	0.31 (0.30–0.33)*	0.31 (0.30–0.33)*
Males 50-59		0.65 (0.63–0.68)*	0.65 (0.63–0.68)*	0.65 (0.63–0.68)*	0.65 (0.63–0.68)*
Males 60-69		1.07 (1.03–1.11)*	1.07 (1.03–1.11)*	1.07 (1.03–1.11)*	1.07 (1.03–1.11)*
Males 70-79		1.44 (1.37–1.50)*	1.44 (1.37–1.50)*	1.44 (1.37–1.50)*	1.44 (1.37–1.50)*
Males 80+		1.51 (1.42–1.61)*	1.52 (1.42–1.61)*	1.51 (1.42–1.61)*	1.51 (1.42–1.61)*
			Area-level		
Single mother families			10.78 (3.41–34.08)*		11.68 (3.39–40.22)*
IOR (80% interval)			[7.19–16.16]		[8.33–16.39]
Living alone			0.05 (0.02–0.16)*		0.14 (0.03–0.69)*
IOR (80% interval)			[0.03–0.08]		[0.10–0.19]
Aboriginal identity			1.38 (0.89–2.12)		0.81 (0.47–1.37)
IOR (80% interval)			[0.92–2.06]		[0.57–1.13]
Population > 65 years of age			19.70 (5.94–65.33)*	0.99 (0.30–3.31)	3.17 (0.71–14.25)
IOR (80% interval)			[13.14–29.52]	[0.67–1.47]	[2.26–4.45]
Household total income < $20,000				0.23 (0.11–0.52)*	0.35 (0.08–1.47)
IOR (80% interval)				[0.16–0.35]	[0.25–0.49]
Unemployment				1.03 (1.02–1.04)*	1.02 (1.01–1.03)*
IOR (80% interval)				[0.70–1.53]	[0.73–1.44]
Post-secondary education				1.90 (0.43–8.44)	7.70 (1.72–34.51)*
IOR (80% interval)				[1.28–2.81]	[5.49–10.80]
No high school diploma				9.12 (1.29–64.80)*	12.80 (1.61–101.3)*

Covariate	Model 1[a]	Model 2[a]	Model 3[a]	Model 4[a]	Model 5[a]
IOR (80% interval)				[6.16–13.51]	[9.12–17.94]
Measures of area variation and random effects					
Variance[b] (SE)	0.131 (0.024)	0.117 (0.021)	0.050 (0.009)	0.047 (0.009)	0.035 (0.007)
VPC (%)	3.83 (2.49–5.13)	3.43 (2.25–4.59)	1.50 (0.97–2.01)	1.41 (0.88–1.93)	1.05 (0.64–1.46)
MOR (95% CrI)	1.41 (1.32–1.49)	1.38 (1.30–1.46)	1.24 (1.19–1.28)	1.23 (1.18–1.27)	1.19 (1.15–1.23)

Note: OR, odds ratio; CI, confidence interval; IOR, interval odds ratio; SE, standard error; VPC, variance partitioning coefficient; MOR, median odds ratio; CrI, credible interval.

a. Model 1 is an intercept only model. Model 2 is adjusted for individual-level variables (age-sex interaction term). Model 3 adds measures of area social isolation to Model 2 (% of single mother census families, % of persons living alone in private households, and % of population with Aboriginal identity), and a control variable of the % of the population greater than 65 years of age. Model 4 adds measures of area material deprivation to Model 2 (% of households with a total income of less than $20,000, % of population that is unemployed, % of population with a post-secondary certificate, diploma, or degree, and % of population with no high school diploma), and a control variable of the % of the population greater than 65 years of age. Model 5 is a full model that adjusts for all individual- and area-level variables.

b. Measuring area-level variance (σ_u^2).

*$p < 0.05$.

CHAPTER 5. DISCUSSION

5.1 INTERPRETATION OF MAJOR FINDINGS

The overarching aim of this study was to assess SARV in CVD prevalence in Nova Scotia, including describing how estimates varied according to population demographics, and to estimate the contribution of area SES to the variation observed. To the best of our knowledge, this was the first study in Nova Scotia to assess SARV in CVD prevalence using an administrative, health care utilization data source. Access to this population-based, chronic disease data facilitated a comprehensive assessment of the burden of CVD in Nova Scotia. This has not been conducted previously due to the unavailability of population-based datasets with large sample sizes that promote research using small area estimation methods.

Pronounced spatial variation in the prevalence of all three CVD-component conditions under study (IHD, CHF, and hypertension) was observed across FSAs, with up to a ~4.5-fold difference in "apparent" crude IHD prevalence between FSAs with the highest prevalence estimates (13.8%) and FSAs with the lowest prevalence estimates (2.9%). This increased to a striking ~13-fold difference in crude IHD prevalence following correction (15.8% vs. 1.2%), which introduced extra variability due to misclassification error.[72] These results are consistent with literature examining SARV in CVD prevalence for other settings, which also found substantial variation between areas.[97,98] In one United States study, an approximately 3-fold difference in the "apparent" prevalence of poor cardiovascular health was observed between the highest prevalence in the State of Louisiana (~17%) and the lowest prevalence in the State of Colorado (~6%).[98] However, typical of the literature,[72] the US study only provided naïve estimates, only accounting for uncertainty due to random variation. Yet as our results indicate, considerable differences may exist in estimates following correction. Although it is not guaranteed that the corrected estimates are necessarily more accurate than their naïve counterparts, correction acknowledges the uncertainty associated with administrative data and enables prevalence estimates to be assessed for their robustness.

This study identified prominent pockets of FSAs with lower than average prevalence estimates for all three conditions, particularly around Halifax and more urbanized areas in Hants and Kings Counties. Conversely, more rural FSAs on Cape

Breton Island, followed by Northern and Western FSAs on mainland Nova Scotia, had some of the highest prevalence estimates for all three conditions. Again, spatial variation in CVD prevalence, incidence, and/or mortality has been reported in the literature for other settings, with noticeable SARV documented. This was particularly true in the US and the UK, where lower age-adjusted rates of CVD were documented in more urban centres in the northeast and the west of the US compared to more rural areas in the south, [76,98-100] while in the UK lower age-adjusted rates were observed in the more populated south compared to the more rural north and Scotland. [101-103]

The observed spatial patterns in SARV in CVD prevalence in Nova Scotia may be better understood through a historical and socio-ecological lens. The highest estimates in CVD prevalence were observed in FSAs that are in counties whose economies have traditionally been driven by the resource industry (coal and gold mining). For coal mining, this primarily includes four smaller coalfields in Inverness County, Pictou coalfields in Pictou County, coalfields around Springhill in Cumberland County, and especially numerous coalfields and steel mills that have made up what is known as "Industrial Cape Breton" over the last 250 years. [104] Gold districts have existed more within central Nova Scotia, stretching from Guysborough County down through Eastern Shore, up towards Hants, and then in Lunenburg, Queens and Yarmouth Counties. [105] Exposure to coal dust is associated with an increased risk of IHD mortality after adjusting for age, smoking, and body mass index (BMI), [106,107] while gold mining has been found to increase the relative risk for IHD after adjusting for blood pressure, smoking, and BMI. [108] Thus, it is speculated that these exposures may have contributed to a historical trend in increased CVD prevalence within these Nova Scotian Counties, which can be observed in the maps of crude IHD prevalence and to a lesser degree among older male groups.

The mining industry has declined in Nova Scotia in the last half century, [104] with currently no large coal mines in operation, [109] while active gold districts are a fraction of historical levels. [110] This decline has contributed to more recent social and economic trends that more likely underpin the current landscape of SARV in CVD prevalence. Nova Scotia is a province that is experiencing both a population decline as well as population ageing. However, there are inequalities, and there is a net outmigration of individuals (especially those below 30 years of age) from remote and rural areas to

Halifax Regional Municipality and to other provinces. Thus, while Halifax and nearby counties such as Hants, Kings, and Colchester have experienced population increases since mid-1990s; the majority of counties in Nova Scotia have experienced population decreases during the same time as well as population ageing. [111,112] This is especially true for the counties that had FSAs with the highest estimated CVD prevalence, particularly Guysborough, Inverness, Richmond, Shelburne, Cape Breton, and Victoria Counties. These are also counties that have relatively lower SES compared to central Nova Scotia. [111,112] As a result, the populations remaining in these counties are proportionally older, have lower SES, and are more likely to be less healthy, which corresponds with the higher CVD prevalence estimates seen for these areas. Thus, the social and economic milieu of Nova Scotia likely plays a large role in the geographic patterns of CVD prevalence that were observed.

Removal of the influence of population demographics (e.g., age and sex) through adjustments resulted in significantly different estimates in many areas, which corroborates some of these possible social and economic explanations. For example, prevalence estimates fell for the majority of FSAs following adjustments for all three conditions, especially for areas with higher relative prevalence estimates across Cape Breton Island and Western and Northern parts of mainland Nova Scotia. Yet for all three conditions, many areas experienced an increase in prevalence following adjustment, particularly FSAs in and around Halifax that had crude estimates below the provincial average. There are however exceptions to these patterns, such as the FSAs B1L (Sydney Southwest), B1M (Sydney East), and B1W (Eskasoni) on Cape Breton Island having increased prevalence estimates for IHD following adjustment. Conversely, FSAs in and around Halifax such as B2W (Dartmouth East Central), B3K (Canadian Forces Base in Halifax Upper Harbour), B3L (Halifax Central), and B3S (Bayers Lake/Clayton Park in the West of Halifax) had decreased prevalence estimates for hypertension following adjustment. Adjustments for demographics are routinely conducted in spatial epidemiology (e.g., all of the studies cited above from the US and the UK presented age-adjusted rates) [76,98-103] to permit fairer comparisons between areas. This is because it removes the influence of underlying differences in population demographic structures that would otherwise confound observed differences in disease risk. [13] Although changes

in prevalence following adjustment are not surprising and are to be expected, the particular pattern of changes that were observed was of interest. This is because it provided clues on the possible demographic factors that contribute to and explain variation in CVD prevalence, resulting in some important policy implications.

This study also assessed SARV in CVD prevalence in younger age groups to identify areas that may be experiencing the premature onset of CVD. After stratifying the overall population into younger and older age groups, estimates were then compared between these two groups, which identified persistent patterns for the younger age groups of interest. First, overall patterns for the younger age groups generally followed the variation found in the full study population for both IHD and hypertension. Second, greater variation was observed for the younger age groups versus the older age groups. Only one other study was identified in which age-specific estimates were made for SARV in CVD.[99] The study reported increased clustering in age-adjusted CHD among a younger age group (aged 40) in comparison to an older age group (aged 70) in the "Coronary Valley" of the United States (i.e., Kentucky and its neighbouring states). However, this study examined mortality rather than prevalence, and this was a minor finding in that study as the data was not shown for these results. Although these differences to the present study restrict the cross-setting comparisons that can be made, it can still be seen in some of the maps that many adjacent FSAs had greater clustering of similar prevalence estimates for the younger age groups than the older age group (e.g., greater clustering of FSAs around Sydney with more similar hypertension prevalence estimates for females less than 60 years of age versus females greater than 60 years of age). Ultimately, further research will need to identify more specific reasons for why some areas are further above average and why others are further below average for the early onset of CVD in comparison to variation in CVD prevalence in older age. But the collective patterns for younger age groups, and especially their aberrations, result in important policy implications that are outlined in *Section 5.2*.

Finally, sequential modeling revealed that FSAs and their characteristics have large effects on the individual probability of having CVD and on between-area variation in CVD prevalence. Between-area variation was computed to account for 3.5% to 3.8% of the total variation in the propensity to have each CVD-component condition in the null

models. The magnitude of these measures is consistent with previous investigations of CVD that employed multilevel logistic regression models. [78,81] The MORs were also above 1 in the null model and remained significantly above 1 after sequential adjustment, indicating the presence of unexplained variation and that area effects retained their significance in explaining the individual probability of having CVD. The results also indicate that area-level variables are a larger component of SARV in CVD prevalence than are individual-level variables. Moreover, area measures of social isolation appeared to account for slightly more of this variation in comparison to area measures of material deprivation for IHD and CHF, while the opposite was true for hypertension. This was evidenced by greater decreases in the σ_u^2, VPC, and MOR when adjusting from Models 2 to Models 3 for the conditions than when adjusting from Models 2 to Models 4 (opposite for hypertension). The nuances of these differences can ultimately be used to direct health policy towards specific avenues of population-based, CVD interventions.

These final results are congruent with a body of literature that has developed over the last two decades that has documented SARV in different CVD-component conditions as well as their associated risk factors. A scoping study by Riva et al. [45] synthesized the literature on multilevel investigations of small area effects on self-rated health, cardiovascular disease and its risk factors, and mortality among adults. Of the 86 studies identified, 32 studies specifically investigated cardiovascular morbidity and risk factors, with 23 examining the direct main effect of area/neighbourhood deprivation on CVD and risk factors. In all of these studies, at least one indicator of area deprivation was associated with increased risk for CVD as well as at least one of the risk factors investigated—consistently shown to be independent of individual characteristics after their adjustment. [45] With these findings summarized, some of the potential policy implications can be explored.

5.2 POTENTIAL POLICY IMPLICATIONS OF FINDINGS

CVD has a large disease burden in Nova Scotia; being a disease category that manifests with the second highest impact on the local economy. Thus, significant improvements can be made to the current state of affairs with even modest changes to the CVD landscape, and addressing heart disease (and stroke) have been considered

Canada's number one health opportunity. [113] In Nova Scotia, this opportunity can be promoted by translating the present findings as partial evidence towards an upstream approach to CVD that is focused on population-based health promotion and disease prevention. This is especially important for areas with evidence of early onset of CVD. As aforementioned, this study follows on the heels of a parent study entitled "Small Area Variation in Rates of High-Cost Healthcare Use Across Nova Scotia," in which case significant SARV in high-cost use was observed. [63] As the current study employed similar statistical methods and adjustments using the same data source but for a different outcome, some of the suggested policy implications for the observed SARV in CVD prevalence were built off of those made in the parent study.

One of the findings from this parent study was that six distinct types of areas were identified based on sequential adjustments, and it was suggested that each be targeted with a different policy priority. In addition to areas that were labeled as having high or low rates of high-cost use due to demographics or due to other reasons (which were further assessed for factors such as levels of continuity of care), some areas were labeled as having high or low rates of high-cost use due to chronic disease patterns. In support of these findings, many areas that were identified as having a high prevalence of the CVD-component conditions in the present study coincided with areas that were identified as having high rates of high-cost use from chronic disease. This includes many FSAs around Sydney and up towards Glace Bay on Cape Breton Island, as well as some large FSAs on mainland Nova Scotia such as rural South Caledonia (FSA: B0T) in Western Nova Scotia and Guysborough area (FSA: BOH) in rural and North East mainland Nova Scotia. Conversely, many FSAs in and around Halifax Peninsula and in Kings County were identified as having low rates of high-cost use from chronic disease, which also coincided with FSAs that were identified as having a lower prevalence of the CVD-component conditions. The congruence between these studies is noteworthy because it confirms that CVD is a disease category that contributes significantly to SARV in high-cost use, and that policy designed to ameliorate inequalities in CVD prevalence will have the beneficial effect of also partially addressing SARV in rates of high-cost use.

The findings from this study have important policy implications for targeting areas that could be integrated into the overall strategic plan of CVHNS. This strategic

plan includes: a mission of improving the cardiovascular health and care of Nova Scotians, a vision where cardiovascular health and care are fully integrated across the life-course and are focused on the determinants of health, and values that are community-based and lead to decisions that promote community health. [58] This study indicated that healthy public policy targeted at CVD should give some consideration to the influence of population demographics on CVD prevalence and on the interventions to be created. First, it was observed in the study that following adjustment for demographics, SARV in CVD prevalence changed for all three conditions, with the majority of FSAs having reduced prevalence estimates. These areas likely have older population age structures relative to the provincial average that are partially driving the crude prevalence estimates, which are reduced once the influence of the older age structures for these FSAs are removed through adjustment. Thus, areas that have higher than average CVD prevalence and older population age structures will need to be prioritized for some resource and service allocation. For example, funds could be appropriately allocated towards healthcare services that increase availability and access to acute and chronic cardiovascular care, as well as towards CVD management, rehabilitation, and end-of-life planning.

Logical strategies that stem from this conclusion, which are goals outlined by CVHNS, are to promote the development of standards for access, delivery models that address gaps in service guidelines, and the ongoing development of cardiovascular health service providers. This would increase and improve the availability and accessibility of comprehensive, and high quality, cardiovascular care. [58] This is particularly relevant to these areas that were identified as having above average CVD prevalence and older population age structures, as many of these areas are rural and/or remote areas (e.g., the large FSAs B0E on Cape Breton Island and B0W in Western mainland Nova Scotia). These types of areas will have aging populations, declining younger populations, and issues regarding access to health care that must be a priority focus in these areas. However, the fact that these areas may have older populations and above average CVD prevalence is not all too surprising, as age is the dominant risk condition for CVD. Thus, although policy should continue to improve access to cardiovascular care in these areas, these areas do not necessarily need to be prioritized as having excess risk solely based on the expected results from the underlying population age structure.

On the other hand, adjustment also resulted in a smaller proportion of FSAs that experienced increased prevalence estimates. These areas likely have younger population age structures that partially keep crude prevalence estimates relatively low, which increase following adjustment and the removal of these influences (e.g., the FSA B3T just West of Halifax Peninsula, which experiences increased prevalence following age-sex adjustment for both IHD and hypertension). The important implication for these areas is that health policy will need to place more emphasis on long-term prevention of potentially premature onset of CVD. This is an important concern from a population health perspective, as earlier onset of CVD has longer impacts on an individual's overall quality of life, and results in greater morbidity costs for these individuals and society as a whole. Evidence has shown that early detection and treatment of modifiable risk factors for CVD (e.g., smoking, physical inactivity, poor diet, etc.) could stop or delay its progression. A further benefit to this approach is that reducing modifiable risk factors for CVD, which collectively make up metabolic syndrome, could also reduce diabetes. [16,114] Thus, proactive policy agendas focused on CVD prevention, through modifications in individual-level risk factors across the life course, could result in multiple and concomitant public health benefits. However, despite the importance of demographics to CVD prevalence, as the reductions in the VPC between models showed, population demographics per se play a small role in SARV in CVD prevalence in Nova Scotia. Thus, policy will need to focus on broader, more important components that contribute to the premature onset of CVD in some areas.

Healthy public policy targeted at CVD needs to consider where people live, work, and play. A key finding from this study was that areas had large effects on the individual probability of having CVD, and that both social and material area-level variables contributed significantly to variation observed. Thus, an important conclusion from this study is that policy should be focused on the broader social and material determinants of health needed to create heart healthy environments, and to address those socioeconomic determinants that result in accelerated aging and early onset of CVD. According to the Canadian Heart Health Strategy and Action Plan (CHHS-AP), creating heart healthy environments means creating supportive environments that make healthy choices easy for all—both early on in life and across the life course. [113] Resources could be allocated

towards activities focused on: reducing socioeconomic disparities and overall poverty, improving employment and working conditions, improving the built environment to encourage access to physical activity, increasing access to affordable housing, reducing food insecurity and improving access to healthy food choices, improving access to early childhood education and care, improving access to literacy training, reducing tobacco exposure, and improving social status and social support networks. [113] Investments in these interventions will have an indirect overall effect of chronic disease prevention, translating into long-term health and economic benefits in Nova Scotia.

An important recommendation by the CHHS-AP is to end the CVD crisis that exists among Aboriginal Peoples. [113] According to the 2011 Census, FSAs with the highest proportion of Aboriginal Peoples typically had lower SES and younger population age structures, while also having some of the highest CVD prevalence estimates in our study. For these areas, a culturally competent approach to healthy public policy is required, ensuring that Aboriginal Peoples are at the centre of decision-making in the development of solutions and plans. Recommendations put forth by the CHHS-AP that could be integrated into the strategic plan of CVHNS include the development of a network of Aboriginal/indigenous centres for CVD (and chronic disease) prevention and management. These centres could create heart healthy Aboriginal/indigenous communities through numerous ways. First, they could help Aboriginal/indigenous Peoples lead healthier lives (e.g., by promoting practices that limit the sale of tobacco to minors). Next, they could promote reforming health services to increase access to patient-centred cardiovascular care. Furthermore, they could increase the knowledge infrastructure to enhance CVD prevention and care among Aboriginal Peoples (e.g., by improving screening and surveillance within Aboriginal/indigenous health service agencies). Finally, they could help promote the development of a culturally competent, health service workforce by offering the right education and skills. [113] The collective sum of these efforts could result in significant cardiovascular health improvements among Aboriginal Peoples, while inaction would create a disservice to those who could benefit most from these health policies.

Finally, as the caterpillar plots and maps may indicate, healthy public policy should focus on creating interventions that are guided by relative levels of need. Not all

areas above average in CVD prevalence are equally above average, and not all areas below average in CVD prevalence are equally below average. Gradients in the figures should help policy decision-makers observe that some areas are not only dealing with greater CVD prevalence, but that they are dealing with much greater CVD prevalence. This means that resources will need to be appropriated accordingly, with these areas likely needing greater investments for addressing CVD. Collectively, these policy implications could be incorporated into the current strategies of CVHNS that are focused on the broader determinants of health and on upstream prevention (e.g., the need to create supportive environments, using evidence to inform healthy public policy, and the importance of knowledge exchange for decision support). [58]

5.3 STRENGTHS

This study contained several important strengths. A strength of this study was that estimating CVD prevalence for small areas has broad applications to an upstream approach that is focused on CVD surveillance and prevention. This approach is consistent with a shift in focus on intervening in the CVD continuum and stopping CVD prior to its development in individuals. [114] To accomplish its objectives, this study took a population health approach that identified areas most affected by CVD. Moreover, by describing how SARV changes with population demographics and area SES, another strength of this study was that it examined the contribution of these variables to variation. This generated hypotheses that can be used by CVHNS to help meet its objectives and to address inequalities in CVD prevalence. To facilitate this approach, another strength of this study was the use of administrative databases. As the administrative databases contain all individuals eligible for health care coverage, nearly 100% of the Nova Scotian population was captured. The data is not specific to one geographical area (i.e., can be generalized to whole provincial population), and it includes important subpopulations such as residents of institutions that may be missed in surveys. [68,115] Thus, as population-based data, administrative databases are an essential tool for capturing disease burden and to plan appropriate healthy public policy decisions. [116]

Another important strength of this study was that it corrected most prevalence estimates for misclassification error using the Rogan and Gladen estimator formula. This

is infrequently conducted in research focused on small area health variations, yet its absence results in applying case definitions with the inappropriate assumption of perfect accuracy. Using multilevel (random intercept) models in this study was another strength, as it recognized the hierarchy of individuals being nested within areas. The ability of multilevel models to take into account the non-independence of observations in correlated data, through the specification of random effects, produces correct estimates of standard errors and CIs and results in valid inferences from a study. [117] By comparison, standard regression models such as ordinary least squares (OLS) regression assume that individuals are sampled independently, with residual error terms that are independently distributed. These models are unable to account for the spatial correlation of data and the violation of the independence assumption, which would underestimate standard errors, produce too narrow CIs, and artificially significant results. [118] Finally, generating maps was also a strength, as this provides a simple visual aid that can be used to assess inequalities and to direct further research and/or action on healthy public policy.

5.4 LIMITATIONS

This study contained several important limitations. The first limitation is that despite administrative databases providing a population-based source of data, they were designed for health system management and billing purposes, rather than research. [68] Thus, estimating CVD prevalence depended on the application of case algorithms with imperfect diagnostic accuracy, which would have resulted in misclassifications. [72,119,120] Ideally the administrative database case algorithms would have 100% sensitivity and specificity, thus removing false negative and false positive outcomes. As displayed in Appendix A, they do not have perfect accuracy, and these test characteristics in turn depend on the different possible combinations of various administrative database elements used to define a case algorithm. Different selections create different arbitrary cut-off points that play on chance, resulting in a trade-off between the sensitivity and specificity of an algorithm (e.g., requiring more contacts with a code will improve specificity at the expense of sensitivity). [68] The objective of validation studies is to assess the diagnostic performance of algorithms in order to identify algorithms that best balance

this trade-off. With imperfect accuracy, misclassification errors would have inevitably occurred.

A key limitation in this study is that although this study corrected prevalence estimates for misclassification error, not all prevalence estimates could be corrected for using the Rogan and Gladen estimator formula. This is because the combination of the low observed, "apparent" prevalence of a condition such as CHF, and the specificity of the case algorithm used for this condition, was less than 1. This resulted in the formula producing negative estimates, which are implausible. [82,96] Furthermore, perhaps the biggest limitation in this study is that even among those estimates that could be corrected, there were no sensitivity and specificity measures for each area, so both of these indices and misclassifications were thus assumed to be fixed. These validation indices are expected to differ to some degree between each area and its different subpopulations, and there should be variation in misclassifications between areas.

It is expected that diagnostic accuracy will vary between physicians due to subjective clinical judgement (e.g., a cardiologist may diagnose "other acute ischemic heart diseases' more accurately than a family physician), which can result in differential rates of misdiagnosed or undiagnosed cases. [121] Diagnostic accuracy may also vary between different areas, with difficult diagnoses requiring complex instrumentation and advanced expertise that may be more readily available at tertiary hospitals in comparison to community settings. Any error along the process from disease diagnosis to code assignment will result in misclassification errors, increasing in probability with increasing complexity of the process. [121] This will affect the diagnostic accuracy of the case algorithms, precipitating into observed CVD prevalence that would be a biased estimate of "true" CVD prevalence as a function of variation in coding practices. This would result in differentially biased prevalence estimates across areas in an exact manner that was impossible to correct for, and the variation in CVD prevalence that was observed may partly be due to a variation in misclassifications.

One way to overcome the limitation of negative prevalence estimates from the Rogan and Gladen formula is to adopt a Bayesian approach. In this case, the sensitivity and specificity are treated as random variables and are assumed to have intrinsic probability distributions that reflect their uncertainty. This information can be combined

with the data (observed prevalence) to obtain a posteriori probability distribution of the true prevalence. The prior information on the distributions for the sensitivity and specificity can be obtained from validation studies (e.g., sensitivity and specificity are measured with error, and studies will include a range of values) or from clinical expertise. [96] In this study, the particular algorithms employed have received some validation work, but it has not been extensive. Moreover, besides hypertension, the algorithms for IHD and CHF have not been officially adopted as the standard case algorithms used for diagnosis by the PHAC through the CCDSS, perhaps reflecting their diagnostic inaccuracies. Among the validation studies conducted, a 95% CI was reported for each index, providing a range of estimates. A sensitivity analysis was attempted using different values for these indices in an attempt to obtain plausible, corrected estimates. However, for all estimates that could not be initially corrected, no combination of different parameter estimates from these 95% CIs could produce plausible estimates. Thus, we were left with reporting estimates that were not corrected for misclassification error (particularly for CHF), limiting our confidence in these results and why we treated them as suspect. Furthermore, the fact that some estimates in this study were corrected for while others could not be may raise some questions on the validity of this correction method and/or the overall robustness of this study's results. These concerns are valid, and ultimately what is important is to be cautious in the interpretation of these results.

The use of FSAs for area boundaries was another limitation in this study. The parent study found that using FSAs is a less than ideal method of geocoding individuals from the databases onto the map. This is because FSAs can be large, particularly in rural areas, which can produce area boundaries that are not socially meaningful. [122] Another limitation of FSAs is that they are administrative definitions that are not tied into health planning. A more appropriate definition of areas would have been to use the recently developed "community cluster" health planning geographies that are built around community health-service usage. [15,60-62] Keeping the limitations in mind, this study still employed the FSA definition of areas to maintain consistency and comparability of results with the first iteration of the parent study as well as most of the Health Atlas. However, this also points to the crucial dependency of scale, where there is a trade-off between making regions large enough to produce stable prevalence estimates, while at

the same time keeping them as small and homogenous as possible in terms of SES and other important characteristics. [14] Moreover, it is important to stress the MAUP and that choosing any territorial unit to delimit areas in this study could produce results that would differ from when choosing another territorial unit (i.e., there is no perfect definition of small areas or their boundaries). [38-40]

Another limitation was that of small numbers. As aforementioned, areas may have sparse data within them, which can lead to unstable rate estimates. [14] Although empirical Bayes estimates were used to reduce some of this random noise from the disease map, a challenge is that there is a bias versus precision trade-off when shrinking estimates. That is, although the EBLUP estimates were more accurate for areas with fewer observations, with smaller error variance, these estimates were conditionally biased towards the mean of the study population as predicted by the model and away from the true area estimate. [85] A further limitation was that the study included a limited set of covariates, and those that were included were broken down into fairly large categories. Thus, there was likely some statistical under adjustment in the models and the associations that were observed. A richer set of covariates would have explained a greater proportion of the SARV observed as reflected in greater measures of area variation. An additional limitation is that this study employed a cross-sectional design. A more longitudinal design with more years of data would have enabled more thorough investigations with an explanatory focus. [45]

Finally, prevalence measures the number of cases of a disease that exist in a population at a specified time, and is a function of both incidence and duration (i.e., mortality rate of cases). [123] This presents a challenge for interpreting prevalence estimates for small areas as areas may be identified as having higher prevalence of CVD simply due to earlier diagnosis along the CVD pathophysiological continuum in an area and/or due to better management of conditions and increased survival of diagnosed cases. As such, comparisons in performance between areas (i.e., SARV in CVD prevalence) were limited to observed or "apparent" prevalence (which was corrected) rather than the underlying "true" prevalence. This is especially problematic when individuals who may already have early signs of IHD are either unaware of this, or are aware but delay visiting a doctor. These individuals will be missed by the administrative databases, thus underestimating CVD prevalence. Thus, although prevalence is a good measure of

overall disease burden, it may not on its own tell policy makers what to do, and a high prevalence of disease in an area paradoxically may not necessarily be a bad thing (e.g., there could be better screening rates in an area). It is important to be aware of these caveats when interpreting prevalence estimates, and an effort must be made to tease apart the intricacies that underlie estimates to determine what to do from a policy and planning perspective.

5.5 FUTURE DIRECTIONS AND CONCLUSION

This study adopted an approach used in other studies that base need for care on observed prevalence estimates of a disease. It makes noteworthy contributions to the broader and ongoing goal of developing a comprehensive Health Atlas for Nova Scotia. Similar to the conclusions made in the parent study, the findings from this study should only serve as an initial and ongoing step towards reducing the health and economic impact of CVD in Nova Scotia. The momentum generated from previous studies and carried through this study should now be carried forward into future work.

A key finding from this study is that many areas were identified with evidence of early onset of CVD. Future work should explore the possible factors that may be contributing to these patterns in greater detail, and use this information to inform population-wide health interventions. This work should assess the impact of modifiable risk factors (e.g., smoking, drinking, physical inactivity, poor diet, stress, etc.) on SARV in CVD prevalence. This could be accomplished by linking individual-level data from a population-based data source such as the Canadian Community Health Survey (CCHS).[81] However, as sample sizes in the CCHS only permit analyses for larger areas (e.g., health regions), a more ideal approach would be to conduct exploratory research by collecting primary data on lifestyle, social conditions, and overall well-being for smaller areas that would enable these small area analyses, or to use other data such as electronic medical records. A starting point would be to assess the contribution of diabetes to SARV in CVD prevalence. This is because diabetes is a strong risk factor for CVD, conferring risk of future CVD events equivalent to the risk of a prior AMI, and because maintaining glycemic control has been shown to delay the onset of CVD.[16] This recommendation is also feasible as a variable for diabetes status is already included in the dataset of the

parent study. This variable was coded using a case algorithm with high validity [124] that previously resulted in an official report on diabetes in Canada by the CCDSS. [125]

As the "community cluster" shapefiles have now been developed and released on the Health Atlas website, another recommendation for future work would be to repeat the analyses using these alternative geographic areas that are tied into health planning. This would enable the DHW, through CVHNS, and those concerned with health policy to directly address SARV in CVD prevalence with limited healthcare resources that are appropriately guided towards areas of population need. Furthermore, the study could be expanded to include more years of administrative healthcare utilization data, which could provide a temporal dimension to assessments that could generate more stable and certain prevalence data over time. Finally, the substantial potential for misclassification error in this study means that future work should incorporate a more robust method for corrections. A starting point could adopt the Bayesian approach and seek other possible algorithms with improved validation indices that produce plausible estimates or by seeking clinical expertise for this information.

To conclude, CVD is a disease that has a significant impact on Nova Scotia and its citizens. As Nova Scotia has an aging healthcare infrastructure system and a population age structure that will progressively become older and older, the already substantial CVD problem will only become magnified. Work employing small area analysis can play an important role in identifying areas of greatest need and can also provide explanations for factors that drive observed variation in prevalence estimates. Collectively, multipronged approaches that include analyses of SARVs, in addition to the synergy of intersectoral collaborations, will ultimately be needed to continue to effectively address the CVD problem in Nova Scotia.

5.6 HEALTH DATA NOVA SCOTIA DISCLAIMER

"The data (or portions of the data) used in this report were made available by Health Data Nova Scotia of Dalhousie University. Although this research is based on data obtained from the Nova Scotia Department of Health and Wellness, the observations and opinions expressed are those of the authors and do not represent those of either Health Data Nova Scotia or the Department of Health and Wellness"

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APPENDIX A. Descriptions and Diagnostic Accuracy of Case Algorithms that were used to Identify CVD Conditions of Interest

Table A.1 Diagnostic and procedure codes and their descriptions that were used to identify CVD conditions of interest

	Diagnostic Codes		Procedure Codes	
Condition	**ICD-9**	**ICD-10**	**CCI**	**Description**
IHD				
	410	I21		Acute myocardial infarction
	411			Other acute and subacute forms of ischemic heart disease
	412			Old myocardial infarction
	413	I20		Angina pectoris
	414			Other forms of chronic ischemic heart disease
		I22		Subsequent myocardial infarction
		I23		Certain current complications following acute myocardial infarction
		I24		Other acute ischemic heart diseases
		I25		Chronic ischemic heart disease
			1.IJ.50	PCI; dilation, coronary arteries
			1.IJ.54	PCI; removal of device, coronary arteries
			1.IJ.57.GQ	PCI; extraction, coronary arteries
			1.IJ.76	CABG; bypass, coronary arteries
CHF				
	428	I50		Heart failure
Hypertension[a]				
	401	I10		Essential (primary) hypertension
	402	I11		Hypertensive heart disease
	403	I12		Hypertensive renal disease
	404	I13		Hypertensive heart and renal disease
	405	I15		Secondary hypertension

Sources for Descriptions: ICD-9 codes: [126,127]; ICD-10 codes: [128,129]; CCI codes: [129,130]

Note: ICD-9 & ICD-10: International Statistical Classification of Diseases and Related Health Problems (Ninth and Tenth Revisions); CCI: Canadian Classification of Health Interventions; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass graft.

a. Pregnancy-induced hypertension and primary pulmonary hypertension excluded. [115]

Table A.2 Diagnostic accuracy of case algorithms that were used to identify CVD conditions of interest

Condition	Validation Source	Case Algorithm	Diagnostic Fields Used	κ	Sensitivity %	Specificity %	PPV %	NPV %	Reported Prevalence %[a]
IHD									
	Tu et al. [131]	One or more hospitalizations or procedure code or two or more physician claims within one year	All	0.73	77.0	97.5	75.3	97.7	9.0
CHF									
	Schultz et al. [65]	One or more hospitalizations or two or more physician claims within one year	All	0.65	84.8	97.0	55.6	99.3	4.3
Hypertension									
	Tu et al. [132]	One or more hospitalizations or two or more physician claims within two years	All	0.70	72.3	95.0	87.2	87.9	32.5
	Quan et al. [133]			0.71	75.0	94.0	81.0	92.0	21.5
	Lix et al. [68] [b]			0.67	69.4	95.2	76.8	93.2	14.3

Note: PPV: Positive Predictive Value; NPV: Negative Predictive Value; κ: Kappa statistic.

a. Validation studies conducted using administrative databases in Ontario, Manitoba, Alberta, and British Columbia, and thus prevalence estimates are specific to the specific populations used in these studies. Prevalence of hypertension from Tu et al. [132] obtained through correspondence with study author.

b. Published study only included ICD-9 codes. However, correspondence with study author revealed that subsequent unpublished work with ICD-10 codes included did not change validation indices, and this study is now included as one of the validation studies for hypertension by the PHAC. [115]

APPENDIX B. Additional Caterpillar Plots and Maps Depicting SARV in CVD Prevalence

B.1 IHD

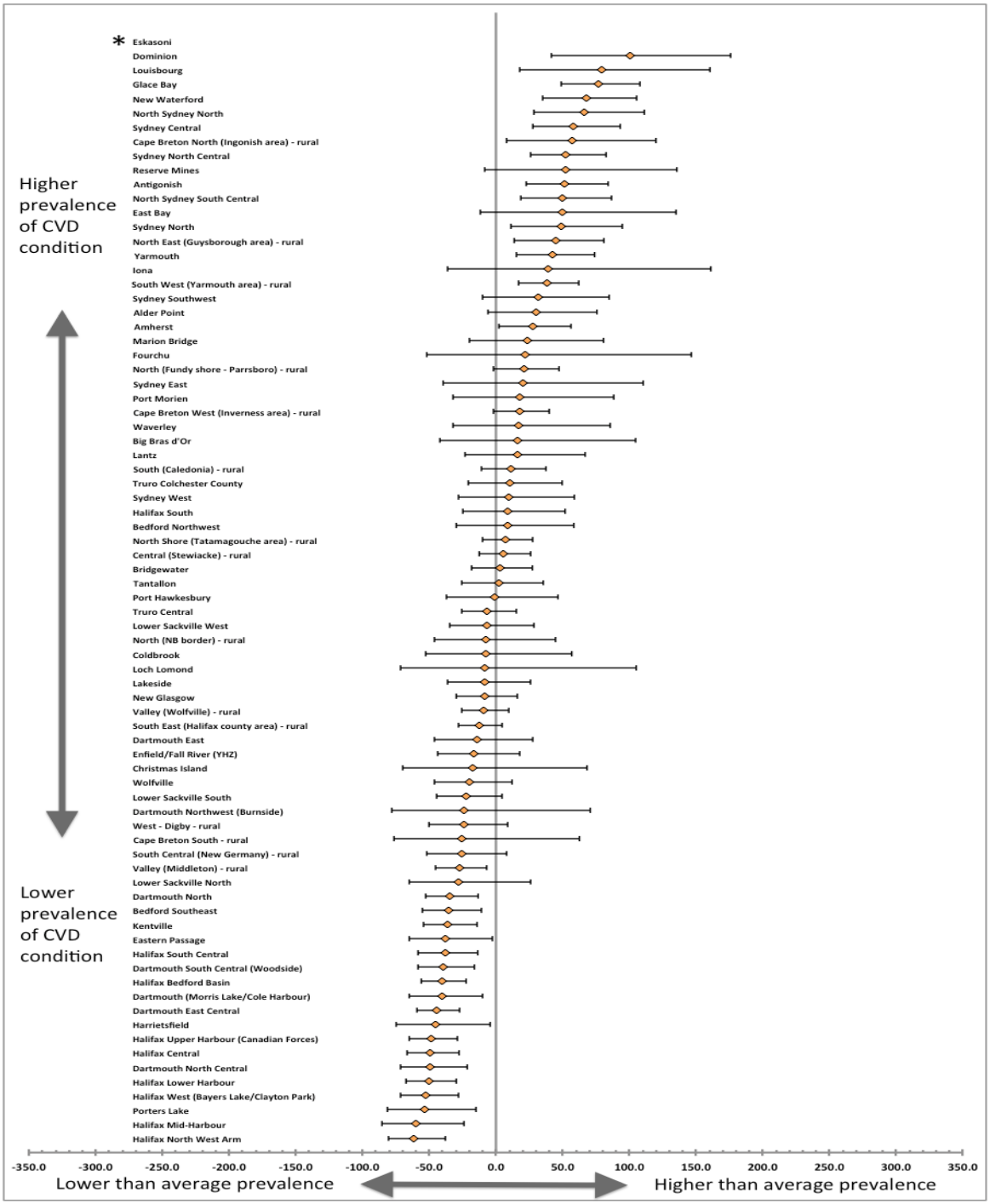

Figure B.1a Caterpillar plot depicting SARV in age-adjusted IHD prevalence for females greater than or equal to 60 years of age by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error. * indicates data not shown for that particular FSA

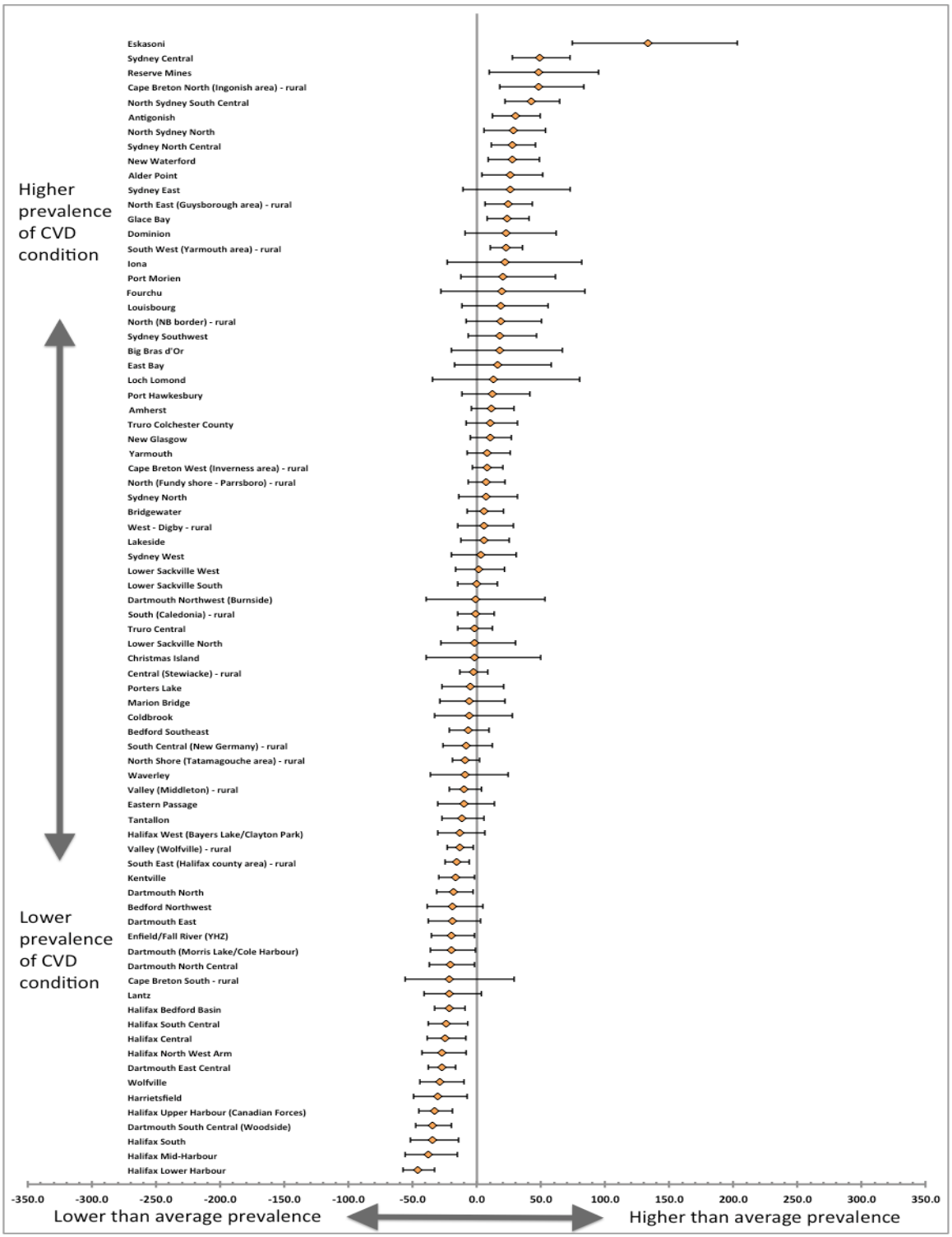

Figure B.1b Caterpillar plot depicting SARV in age-adjusted IHD prevalence for males greater than or equal to 60 years of age by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error

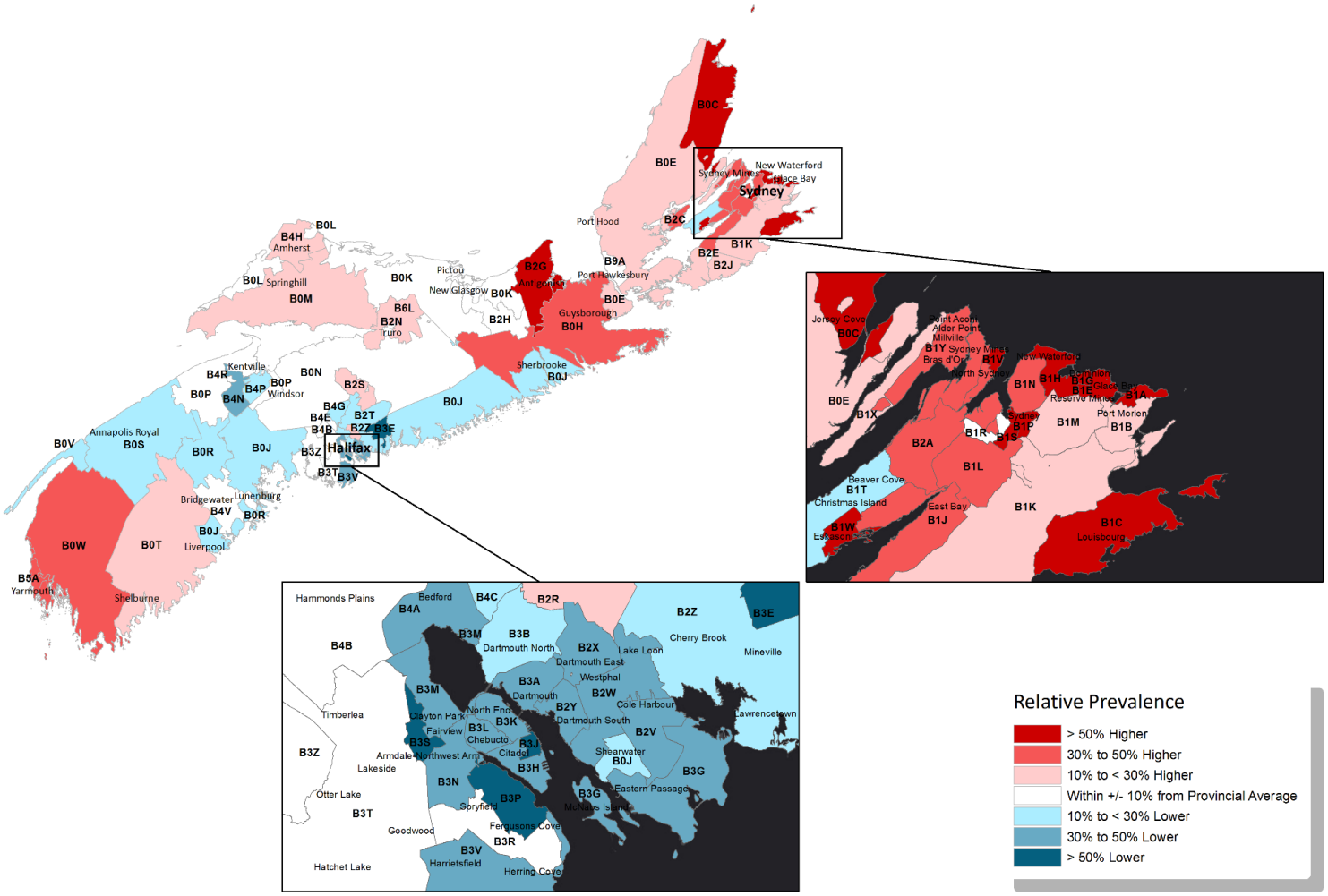

Figure B.1c Map showing SARV in age-adjusted IHD prevalence for females greater than or equal to 60 years of age relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error

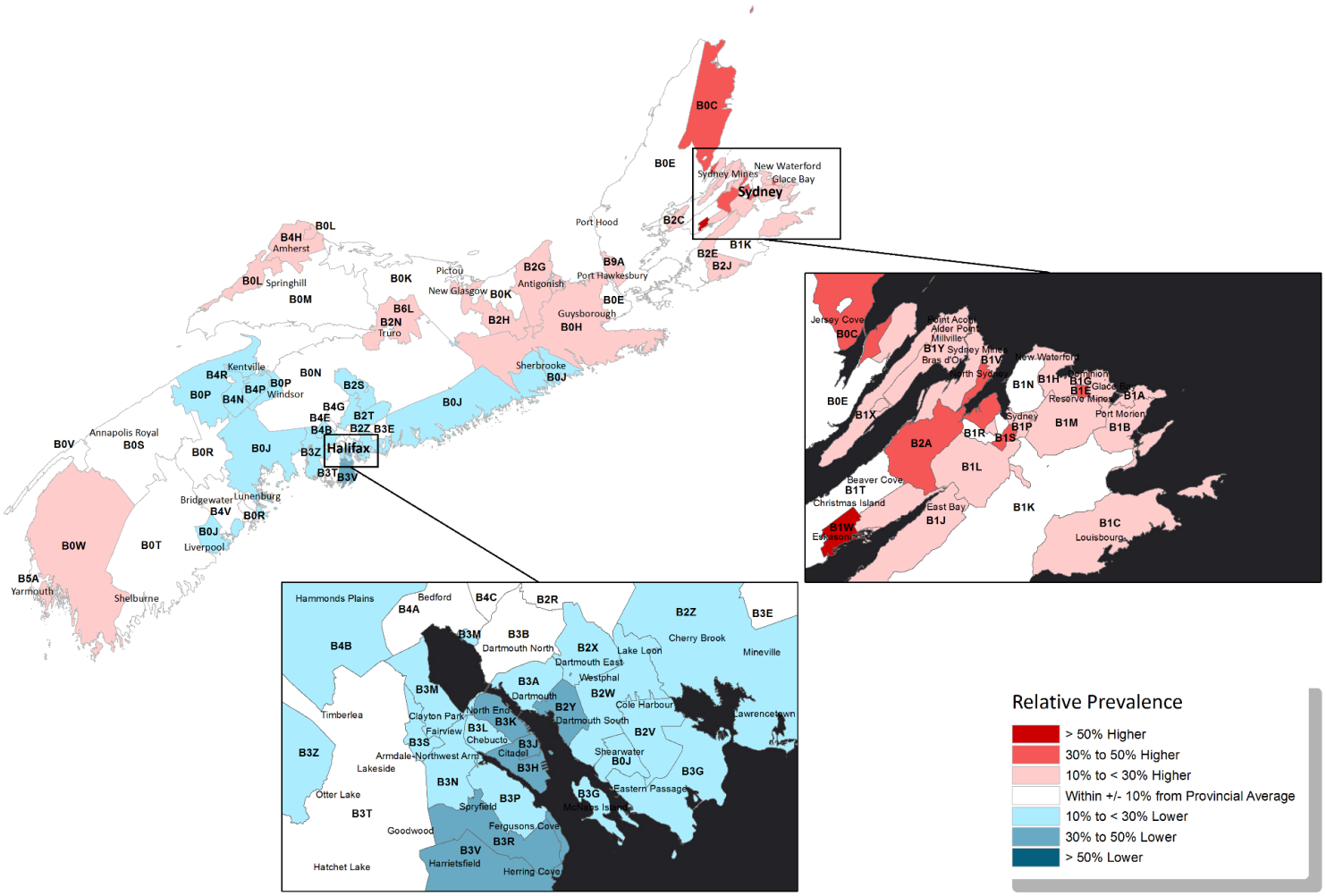

Figure B.1d Map showing SARV in age-adjusted IHD prevalence for males greater than or equal to 60 years of age relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error

B.2 CHF

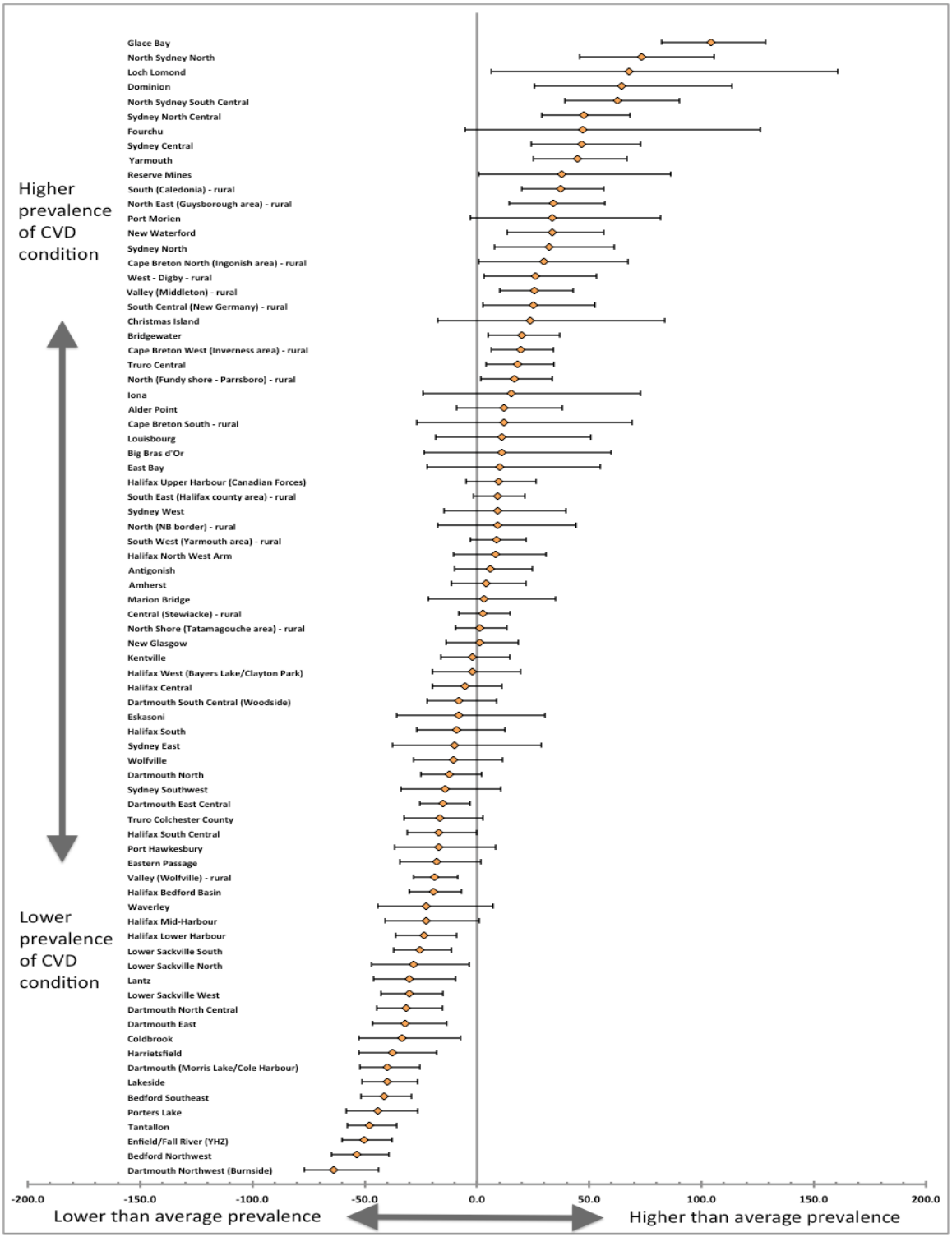

Figure B.2a Caterpillar plot depicting SARV in crude (unadjusted) CHF prevalence by FSA, Nova Scotia, 2010-11 to 2012-13. Not corrected for misclassification error

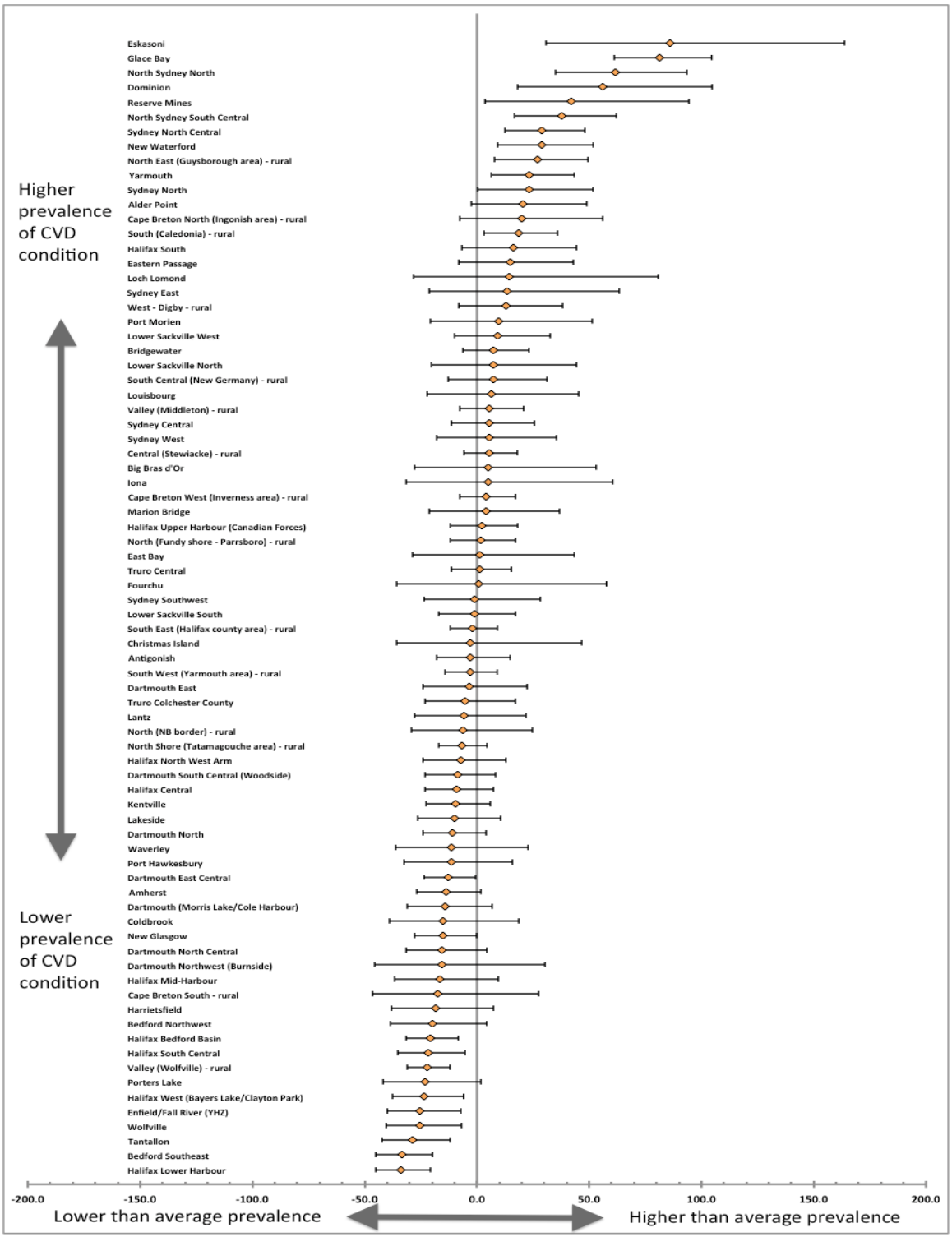

Figure B.2b Caterpillar plot depicting SARV in age-sex adjusted CHF prevalence by FSA, Nova Scotia, 2010-11 to 2012-13. Not corrected for misclassification error

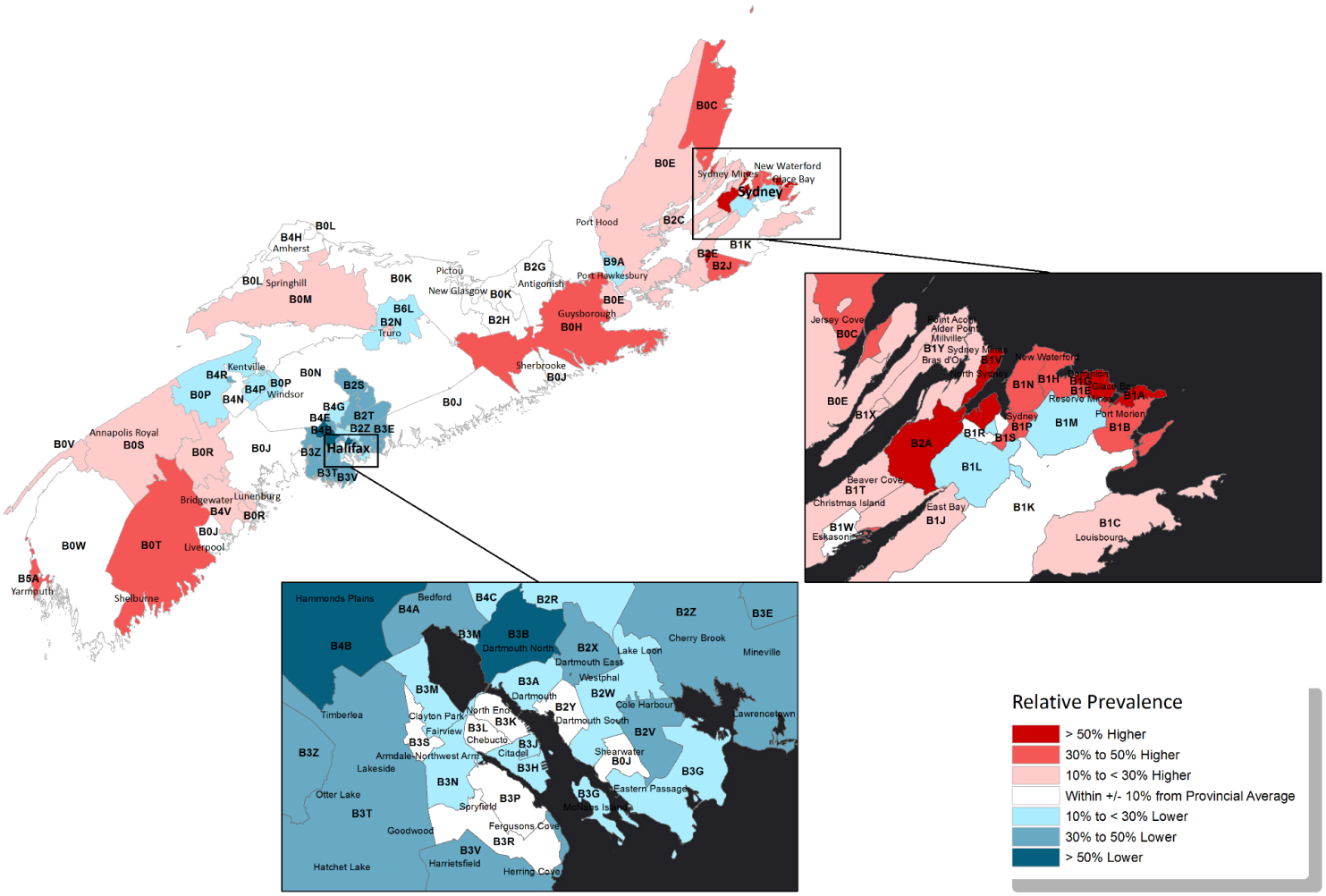

Figure B.2c Map showing SARV in crude (unadjusted) CHF prevalence relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Not corrected for misclassification error

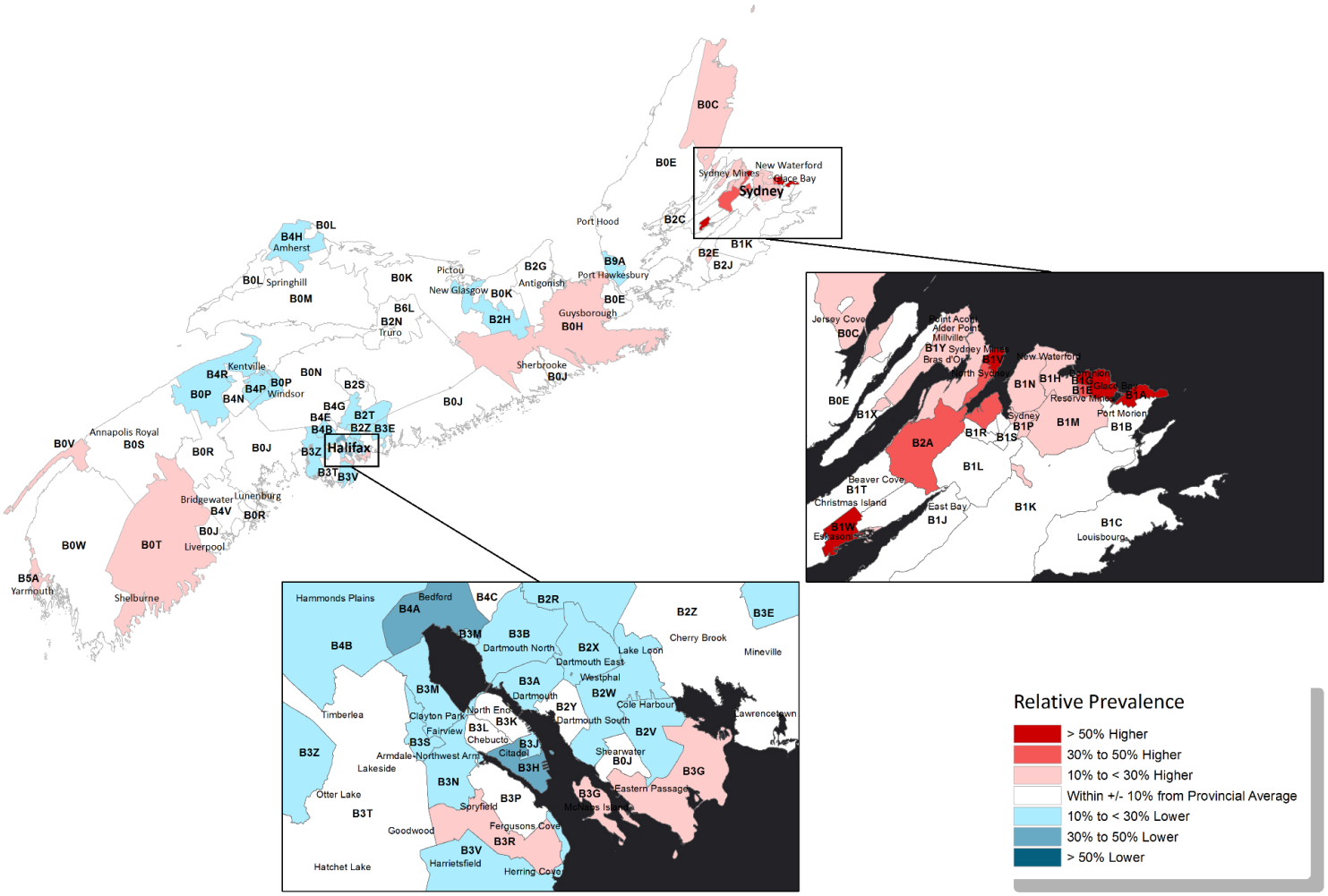

Figure B.2d Map showing SARV in age-sex adjusted CHF prevalence relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Not corrected for misclassification error

B.3 Hypertension

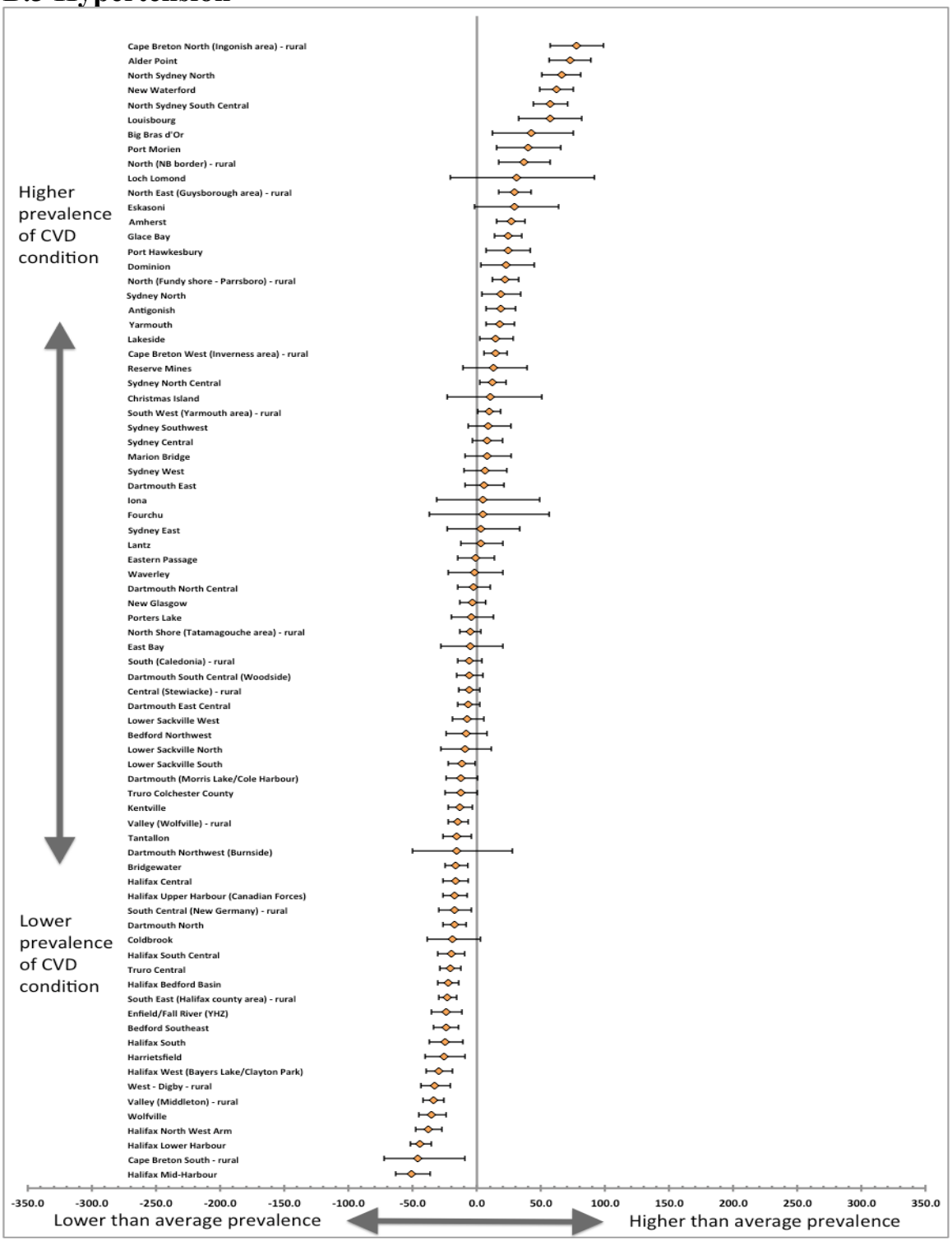

Figure B.3a Caterpillar plot depicting SARV in age-adjusted hypertension prevalence for females greater than or equal to 60 years of age by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error

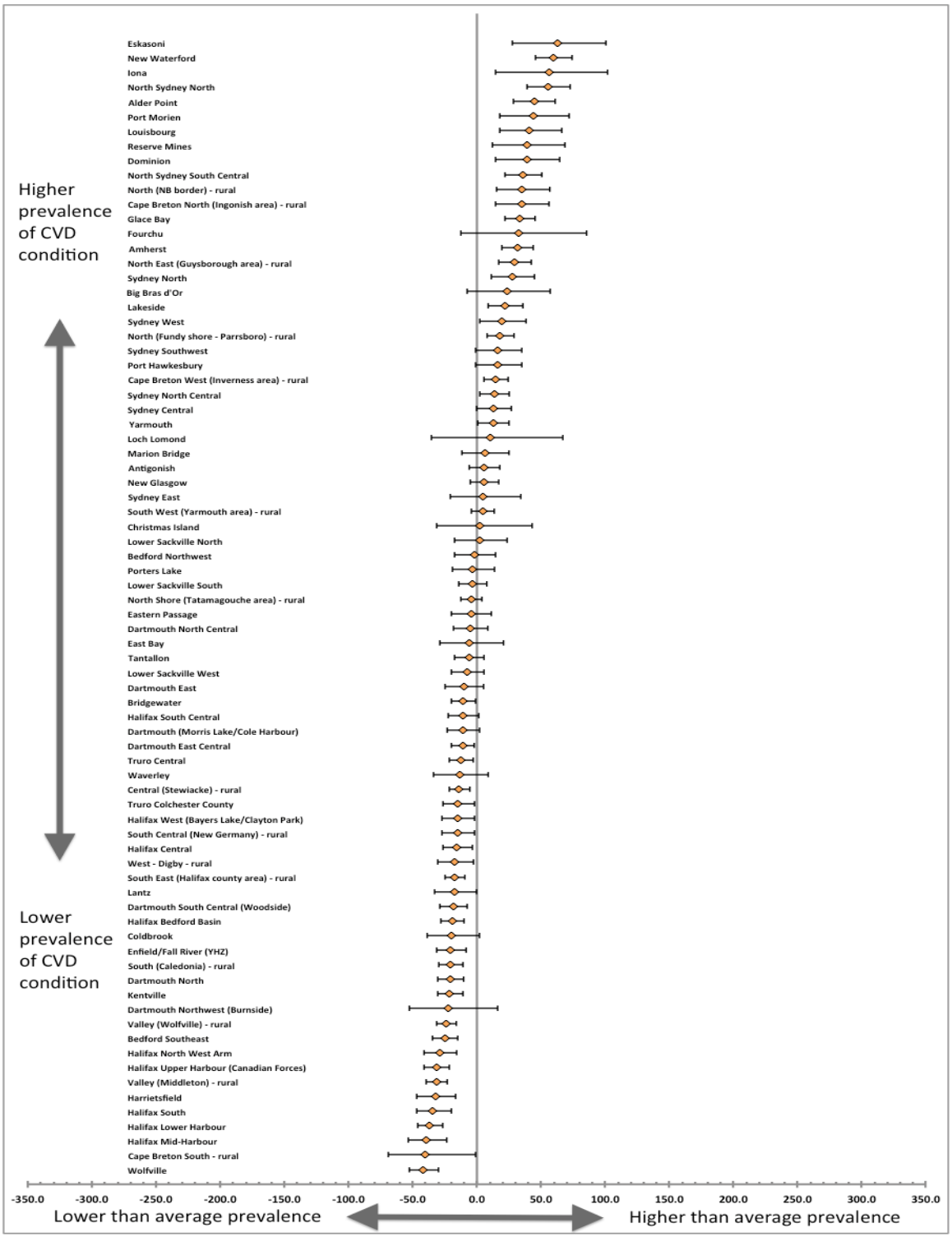

Figure B.3b Caterpillar plot depicting SARV in age-adjusted hypertension prevalence for males greater than or equal to 60 years of age by FSA, Nova Scotia, 2010-11 to 2012-13. Corrected for misclassification error

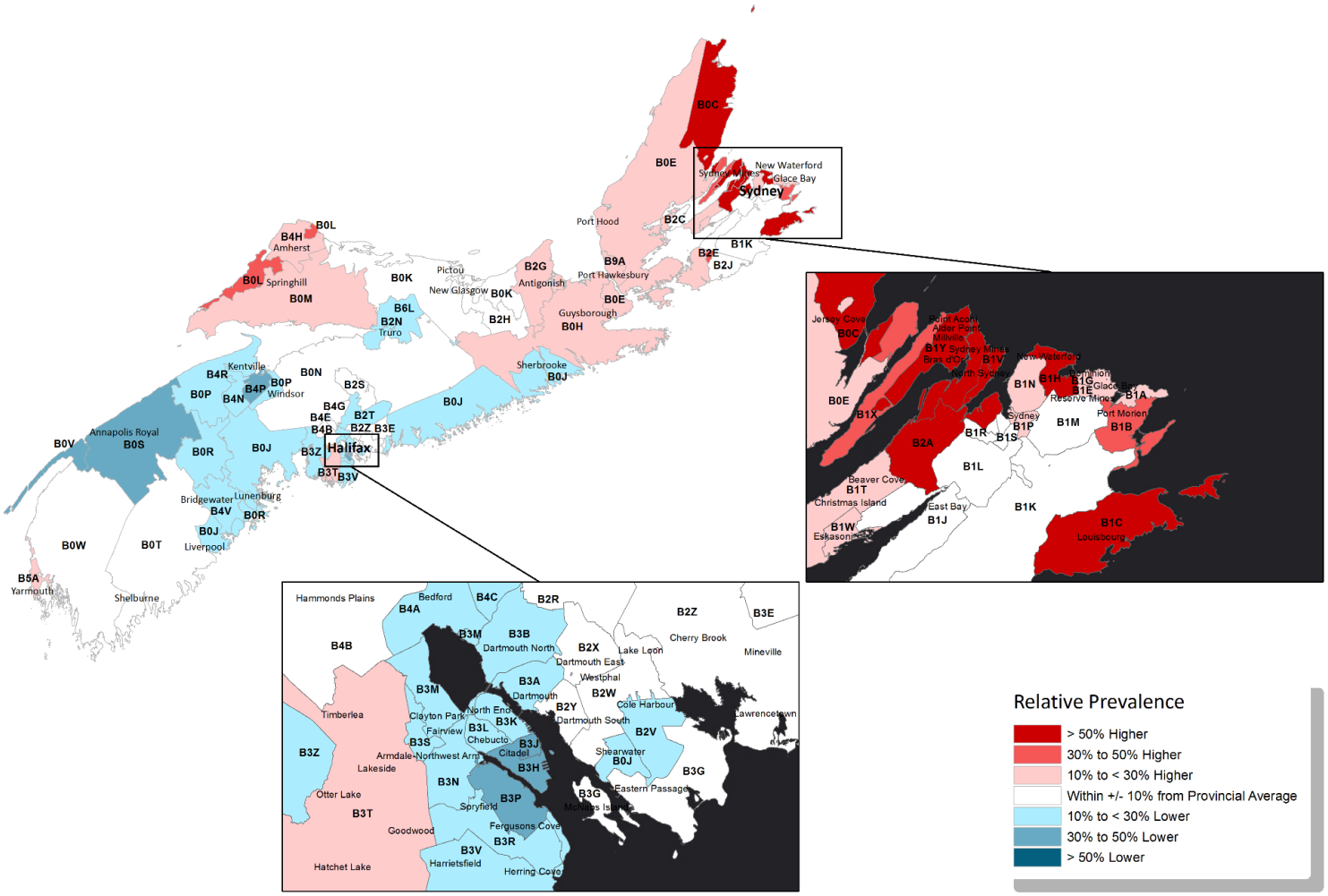

Figure B.3c Map showing SARV in age-adjusted hypertension prevalence for females greater than or equal to 60 years of age relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error

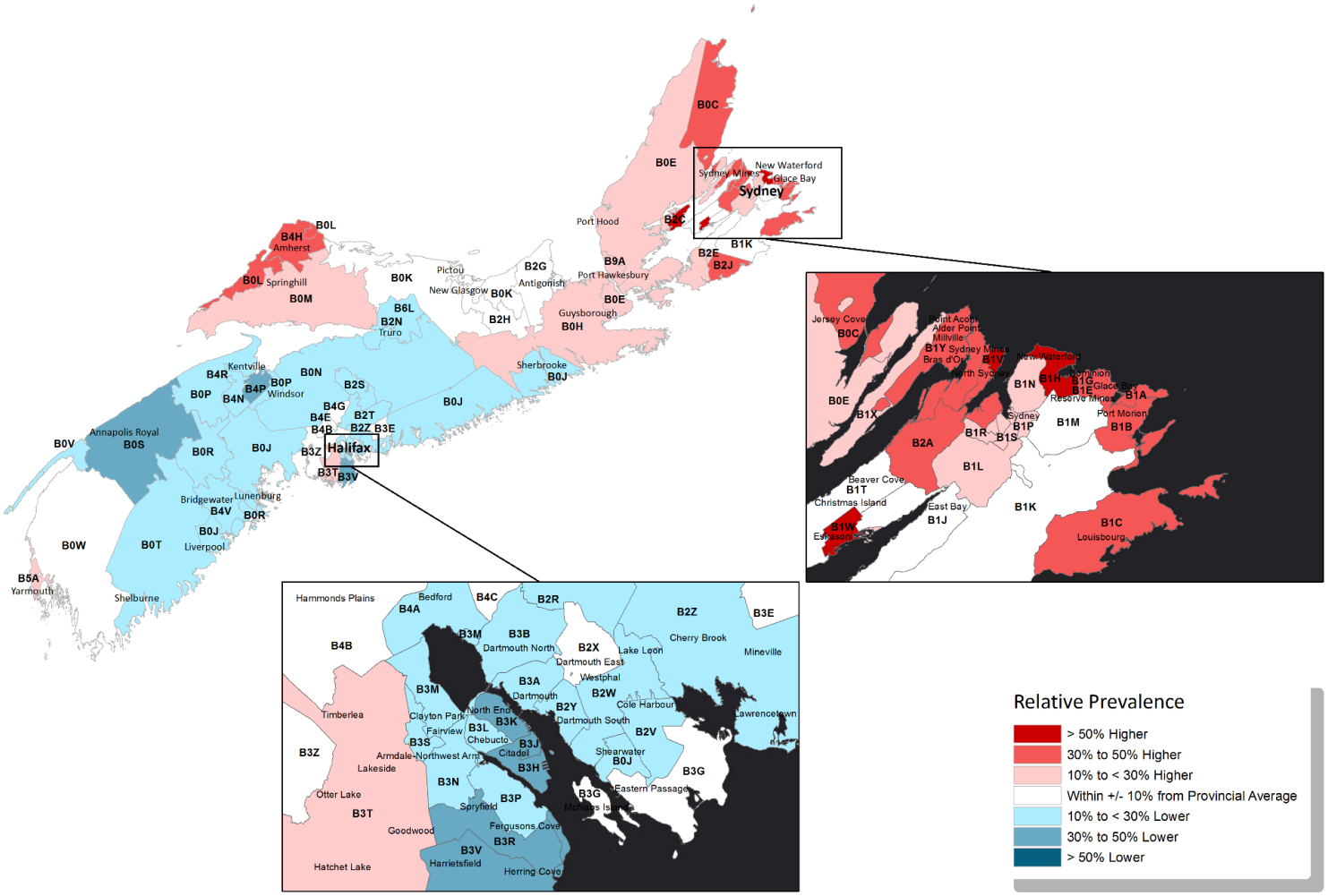

Figure B.3d Map showing SARV in age-adjusted hypertension prevalence for males greater than or equal to 60 years of age relative to the provincial average by FSA, Nova Scotia, with inset maps of FSAs surrounding Halifax and Sydney, 2010-11 to 2012-13. Corrected for misclassification error