TOWARDS AN UNDERSTANDING OF GEOGRAPHIC VARIATION IN CARDIOVASCULAR DISEASE MORTALITY AND MORBIDITY IN ONTARIO, 1986 – 1994

BY

GODWIN A. DJIETROR, B.A. (HONS), M.A.

A Thesis

Submitted to the School of Graduate Studies

in Partial Fulfillment of the Requirements

for the Degree

Doctor of Philosophy

McMaster University

TOWARDS AN UNDERSTANDING OF GEOGRAPHIC VARIATION IN CARDIOVASCULAR DISEASE MORTALITY AND MORBIDITY IN ONTARIO, 1986 – 1994

DOCTOR OF PHILOSOPHY (2003) MCMASTER UNIVERSITY
(Geography) Hamilton, Ontario

TITLE: Towards an Understanding of Geographic Variation in Cardiovascular Disease Mortality and Morbidity in Ontario, 1986-1994.

AUTHOR: Godwin Aklama Djietror, B.A. (Hons) (University of Ghana)
M.A. (The University of Toledo)

SUPERVISOR: Dr. Susan J. Elliott

NUMBER OF PAGES: xii; 301

ABSTRACT

This thesis aims to understand geographic variations in cardiovascular disease (CVD) mortality and morbidity in Ontario between 1986 and 1994. While cardiovascular disease is the leading cause of preventable mortality and morbidity in Canada and Ontario, a large proportion of the regional variations in the outcomes remain unexplained. Using the public health units in the Province as the units of analysis, the research addressed three specific objectives: 1) to describe the spatial and temporal variations in CVD mortality and morbidity in Ontario, 1986-1989 and 1990-1994; 2) to examine the prevalence and distribution of a broad range of potential CVD risk factors in Ontario; and, 3) to model geographic variations in CVD mortality and morbidity in Ontario. The data used come from a variety of sources, including the Canadian Institute of Health Information, the 1990 Ontario Health Survey, the 1991 census of Canada, the Child Care Services Division of the Ontario Ministry of Community and Social Service, Customs and Revenue Canada, and the Municipal Financial Returns of the Ontario Ministry of Municipal Affairs. Informed by the population health perspective, a socio-ecological conceptual model was developed to guide the research. It is composed of seven CVD risk constructs: economic characteristics, social capital, demographic characteristics, risk factor behaviours, psychosocial health and well-being, social support, and physiological characteristics. The research used GIS analytical techniques to examine the spatial

patterns of CVD mortality and morbidity in the province during the study period. The results show little or no temporal change in the spatial pattern of the rates of CVD outcomes. The results show, further, that there were clusters of elevated rates (or 'hot spots') of CVD mortality and morbidity, which were largely concentrated in the northern part of the Province. Results also show that the prevalence of CVD risk factors varied markedly across the public health units with respect to age, sex, and level of education, type of public health unit, and the relative location of public health units in the province. The modelling results show that, overall, variables of the socio-economic and psychosocial environments played a far more significant role in explaining geographic variations in CVD outcomes than physiological variables and CVD risk factor behaviours. The implications of these findings are that strategies aimed at reducing the rate of cardiovascular disease mortality and morbidity in the Province need to place more emphasis on northern Ontario as well as focus more on such risk factors as the socioeconomic and psychosocial environments. This thesis has made a theoretical contribution by developing a conceptual model within the context of the population health perspective to guide the investigation of the underlying causes of geographic variations in cardiovascular disease outcomes. It has also made a methodological contribution by illustrating how GIS and spatial analytical techniques can be applied in identifying local clusters of elevated rates (or 'hot spots') of chronic disease outcomes. Substantively, this research has expanded the range of potential determinants of CVD outcomes in Ontario. It has also provided a basis for rethinking the emphasis on individual level, physiological and behavioural characteristics in CVD risk factor research and heart health programming.

ACKNOWLEDGEMENTS

I would like to thank all who have contributed in diverse ways to the successful completion of this thesis. First, I would like to thank the Heart and Stroke Foundation of Ontario who funded and sponsored the research. I would also like to thank the School of Geography and Geology and the School of Graduate Studies, McMaster University for giving me the opportunity and support in studying for this degree.

I would like to thank my supervisor, Dr. Susan Elliott, very sincerely for her guidance and support throughout my program. The successful completion of this thesis was made possible by her flexibility, understanding, encouragement, and counsel. Thanks also to my committee members – John Eyles, Stephen Walter, and Mike Jerrett – who also made time for me in their busy schedules and provided guidance and useful suggestions.

I would also like to thank all my McMaster friends, and particularly, the teaching and non-teaching staff of the School of Geography and Geology, who have all contributed in various ways to create a conducive environment in which to pursue my doctoral programme. A very sincere thank-you to all of you.

Completion of this thesis would not have been possible without the invaluable support, understanding, and patience of my wife, Felicia, and my lovely daughters, Kabuki, Kabukuor, Aluayo, and Manyeyo. Your encouragements, inspiration, and, above all, your prayers, are deeply appreciated.

This thesis is dedicated to my family, my siblings and mother, all my teachers past and present, and to the memory of my father.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND AND CONTEXT OF RESEARCH

Cardiovascular disease (CVD) is the leading cause of preventable mortality and morbidity in Canada and many industrialized societies (Heart and Stroke Foundation of Canada [HSFC] 1997b). In 1999, CVD accounted for 36 per cent of all deaths in Canada (Health Canada 1999). In Ontario, it accounted for 36.7 per cent of total deaths. In comparison, cancer accounted for 28.6 per cent of all deaths in the country, and 28.5 per cent of all deaths in Ontario (Health Canada 1999). It has been reported that during fiscal year 1996/97, cardiovascular disease diagnoses accounted for 13 per cent of all hospital admissions and 18 per cent of all in-patient resource utilization in Ontario.

In Canada the number of hospital separations for CVD events in 1999 was 450,455 (i.e., 15% of all separations). This highlights the CVD morbidity burden. Comparatively, the total number of hospital separations in that year was 220,969 (7.4%) for cancers; 246,906 (8.2%) for accidents, poisons, and violence; 282,417 (9.4%) for respiratory diseases; 324,295 (10.8%) for digestive diseases; and 173,556 (5.8%) for mental disorders. The total of direct and indirect costs of CVD to the nation in 1993 was $19.7 billion. Again the comparative figures for cancers, injuries, and respiratory diseases were $14.1 billion, $14.3 billion, and $12.2 billion respectively (HSFC 1997a).

It is estimated that CVD costs Ontario $5.5 billion per year and is responsible for 24 per cent of total person years of life lost in the province (Chang and Young 1999).

The resulting economic burden of CVD mortality and morbidity are substantial for both Ontario and Canada, particularly when combining both direct (e.g., hospital care and medical services) and indirect (e.g., lost productivity and lost future earnings due to premature death) costs. In addition, there are profound personal and psychosocial impacts associated with the behavioural and emotional implications of CVD-related life-style changes. Although CVD death rates have been decreasing in Ontario and Canada (HSFC 1997a; Chan and Young 1999), the burden of heart disease in the nation is expected to increase as the population continues to age (Gallop and Naylor 1999; HSFC 1999).

The prevention and control of CVD has been a primary focus of international initiatives such as the Victoria Declaration on Heart Health (WHO 1992), which calls for regulatory and policy changes to enhance health promotion and disease prevention in populations; the Catalonia Declaration (WHO 1995), which urges the international community to invest in heart health; the Singapore Declaration (WHO 1998) that focuses on forging the will for heart health promotion; and the Osaka Declaration (WHO 2001), which calls for action to stem the global tide of cardiovascular disease. National policy documents, such as *Promoting Heart Health in Canada: A focus on heart health inequalities* (Health Canada 1993), have also had favourable impacts on heart health promotion. In Ontario, the document, *Opportunities for Health: Promoting Heart Health* (Ontario Ministry of Health 1993), which attempts to translate scientific insight into a

comprehensible form for all decision makers (Iannantuono 2002), represents an important policy initiative intended to shape the context for heart health. The primary motivation of these policy documents has been the fact that much cardiovascular disease is preventable. In Ontario, as much as 30 per cent of the observed variation in CVD mortality can be explained by the contributions of three modifiable risk factors; smoking, excess fat in diet, and physical inactivity (Jaglal et al. 1999). Indeed, 75% of Canadian adults have at least one modifiable risk factor for cardiovascular disease (HSFC 1997a). These findings illustrate significant scope for the initiation of preventive action to begin to modify behaviours. In Ontario, there has been increased emphasis on CVD prevention and control through the Public Health and Health Promotion Branches of the Ministry of Health and Long Term Care and non-governmental organizations, notably the Heart and Stroke Foundation.

An important feature of the mortality and morbidity burden of cardiovascular disease is that it varies significantly at various geographic scales: global, national, regional, sub-regional, and local. Variation in CVD outcomes is also observable across socio-economic classes (Marmot et al. 1991; Howard et al. 2000; Hemingway et al. 2000; Reddy et al. 2002). In Canada, geographic variations in CVD mortality and morbidity rates have been described in recent national and provincial reports (e.g., Foster and Edgell 1992; HSFC 1997a, 1997b, 1997c; Bondy et al. 1999).

While variations have been observed in CVD outcomes, it has been noted that a substantial proportion of these variations remains unexplained (Marmot and Mustard 1994). For example, as stated above, differing levels of modifiable risk factors such as

smoking, excess fat in diet, and physical inactivity can explain only about 30 per cent of the observed regional variation in cardiovascular death rates in Ontario (Jaglal et al. 1999).

The focus on these modifiable, individual level risk factors typifies the use of biomedical criteria to define disease and ill health, and the use of the individual as the basic unit of analysis. It also typifies the use of extensive explanatory designs that aim to identify characteristics that persons with a particular disease have in common. This focus neglects the influences of more fundamental determinants of health such as the social milieu (Wolfson et al. 1999), as well as economic, cultural, political, and even physical environments (Curtis and Taket 1996). Thus the large proportion of CVD deaths in Ontario that remains unexplained may be a reflection of the omission of these fundamental determinants of health from the list of explanatory variables. Therefore, in studying the regional variations in CVD outcomes in Ontario, it is important to broaden the range of potential determinants to include these variables.

1.2 OBJECTIVES

Given the above, the specific objectives of the research are:

1. to describe the spatial and temporal variations in CVD mortality and morbidity in Ontario, 1986-1989 and 1990-1994,
2. to examine the prevalence and distribution of a broad range of potential CVD risk factors in Ontario, and
3. to model geographic variations in CVD mortality and morbidity in Ontario.

Jaglal et al. (1999) have described the geographic and temporal variation in cardiovascular disease mortality and morbidity in Ontario at the county level. They report that during the period between fiscal year 1994/95 and 1996/97, the highest adjusted mortality rate of CVD for a county was 1.75 times as high as the lowest rate, and the highest adjusted mortality rate of IHD was 2.4 times that of the lowest rate. Their analyses also show that many of the counties with the highest CVD outcome rates are rural. In this research, the focus of the description of geographic and temporal variations in CVD outcome is on a wider time period, 1986-1994, and the units of analysis are public health units rather than counties. This analysis is also expanded to include ischemic heart disease and cerebrovascular disease, and a distinction is made between females and males.

Studies that have examined the prevalence of CVD risk factors have tended to focus on physiological variables, such as diabetes, obesity, hypertension, and plasma lipids, and on lifestyle variables, such as cigarette smoking, leisure time exercise, and eating habits. For example, in their study of the prevalence of self-reported CVD and risk factors among Ontario women aged 50 years and over, Hodgson and Jamieson (1997) focused on hypertension, diabetes, height and weight, physical activity, and smoking habits, to the exclusion of non-medical variables such as socio-economic status, psychosocial health, and social capital. In some studies (e.g., Diez-Roux et al. 1999), the latter group of variables is included as factors affecting the distribution of the classic CVD risk factors, i.e., smoking, hypertension, overweight, and physical inactivity (Gensini et al. 1998). This study expands the analysis of the prevalence of CVD risk

factors in Ontario to include these non-medical variables in the light of current evidence of their role in cardiovascular health outcomes (Kawachi et al. 1997; Lomas 1998; McCarthy 2000).

As indicated in Section 1.1, only a limited proportion of the regional variation in cardiovascular death rates in Ontario can be explained using the traditional risk factors. Meanwhile, evidence suggests that other factors, such as the socio-economic and psychosocial environment, have an impact on cardiovascular health. For example, in a systematic critical appraisal of the research literature on the role of psychosocial stress as a risk factor for women's coronary heart disease, Elliott (1995) concludes that there appears to be some evidence that psychosocial stress causes coronary disease in women, although there is a need for more etiologic research to reach a definitive conclusion. Using data from the Whitehall II study, Marmot and others (1997) assess the contribution of the psychosocial work environment, social support, and other variables to the inverse social gradient in mortality from coronary heart disease (CHD) among British civil servants (which was found in the first Whitehall study). They find that compared with men in the highest grade (administrators), men in the lowest grade (clerical and office-support staff) are 1.5 times as likely to develop CHD, and that the largest contribution to the socio-economic gradient in CHD frequency was from low control at work. They conclude that much of the inverse social gradient in CHD incidence can be attributed to differences in the psychosocial work environment. Although some studies have described the geographic variations in CVD mortality and morbidity rates in Ontario, the reasons for these variations are uncertain (Bondy et al. 1999). This appears to be because

attempts have not been made to link these variations to, for example, lifestyle, geographic, and clinical variables (HSFC 1997c), or other potential determinants such as social capital, psychosocial, and socio-economic variables. Therefore, this research investigates the extent to which the geographic variations in CVD outcome in Ontario during the time period studied were associated not only with classical risk factors such as smoking, poor diet, hypertension, obesity, diabetes, and physical inactivity, but also with non-traditional risk factors such as the psychosocial and socio-economic environments.

1.3 GEOGRAPHIC CONTEXT AND THEORETICAL PERSPECTIVE

There has been a shift in emphasis from traditional medical geography, which focuses on studying the spatial patterning of disease and death, and the spatial patterning of health service provision and utilization, towards the geography of health and health services (or contemporary medical geography), which emphasizes the role of human awareness, agency, and creativity, particular forms of social organization, and cultural values in health experience (Curtis and Taket 1998). This shift in perspective on health has been accompanied by a shift from positivist (quantitative) approaches in health research to more qualitative approaches that study health and illness in place (Elliott 1999). This research is placed partly within the realm of traditional medical geography. Statistical (i.e., quantitative) techniques are used to analyze regional variations in cardiovascular disease mortality and morbidity rates (objective 1), to explore the prevalence of CVD risk factors (objective 2), and to model the regional variations in CVD outcome (objective 3). The research is also placed partly within the realm of

contemporary medical geography. While many past studies of the determinants of cardiovascular disease have largely adopted the biomedical approach and focused mainly on physiological characteristics and lifestyle factors, this research extends the range of potential determinants of the variation in CVD outcome to include the influences of the socio-economic and psychosocial environments. Thus, from a theoretical point of view, this research takes into account the fact that health is now being approached in a holistic manner such that, in addition to cure and care through biomedicine, the influences of socio-economic, psychological, and cultural factors are also being considered. A useful model for investigating these contextual influences is the socio-ecological model of health, which requires that health be approached on the basis of how people perceive their health risks within the larger context of their lives (Elliott 1995). As Wilkinson (1996:13) argues, "... people's social and economically structured life processes remain the most powerful influences on health in the modern world ..."

Informed by the population health perspective, and drawing from Evans and Stoddart's (1990) population health framework (which emphasizes the relationship between the social environment, the physical environment, genetic endowment, individual response, and health), a conceptual model is developed to guide this research. A discussion of the population health perspective, including its empirical support and critiques, is provided in Chapter Two.

1.4 PRACTICAL IMPORTANCE OF STUDY

In recent years there has been increased public attention on degenerative diseases such as cancer, diabetes, and CVD. The importance attached to these diseases is underscored by governmental and non-governmental initiatives at both the national and local levels aimed, among others, at promoting public awareness and support for programs to prevent or lighten their burden on society. In Ontario, the Canadian Heart Health Initiative - Ontario Project, the Heart and Stroke Foundation of Ontario, and the Public Health and Health Promotion Branches of the Ministry of Health and Long Term Care, have emphasized CVD prevention and control and heart health promotion. The need for research on the determinants of CVD as input for these efforts cannot be over-emphasized. However, much of the current literature on these determinants focuses mainly on the more extensively researched risk factors for CVD, such as physical inactivity, tobacco use, and excess fat in diet, leaving the potential role of the lesser-known risk factors largely unstudied. The social environment influences health significantly (Marmot et al. 1987; Marmot and Mustard 1994; Hertzman et al. 1994); therefore, the concentration on these individual level risk factors can only result in partial explanation of variation in CVD mortality and morbidity - 30 per cent in Ontario, for example. This suggests that a large proportion of the determinants of CVD outcome in the Province are located in an area that is little researched. Meanwhile, survey data (i.e., the 1990 Ontario Health Survey and the 1991 Census) on a wide range of risk factors are available for the Province. Hence, the use of data on these risk factors in this study can contribute towards closing the information gap indicated above.

It has been noted that evidence points to the potential role played by the psychosocial environment in the variation in CVD outcome (Elliott and Dean 1998). Marmot and Mustard (1994) have also noted that the large differences in heart disease incidence both between and within societies, and the similarly large changes in these incidence rates over time, are indicative of the influence of the socio-economic and cultural features of those societies. In light of the limited explanation of the spatial variation in CVD outcome in Ontario, it is important to examine the potential role played by the psychosocial as well as socio-economic environment in the observed geographic variation in CVD outcome. The modelling component of the spatial data analysis performed in this research can help address this need by providing estimates of the relative contributions of various explanatory variables. Apart from augmenting the information base for heart health initiatives in the Province, the findings of this research can also be used in the allocation of resources for CVD prevention and control programs.

1.5 ORGANIZATION OF THE THESIS

This thesis is organized into six chapters. In the next chapter, current evidence on cardiovascular disease is reviewed and the theoretical perspective that informs the research is discussed in detail. The literature review includes the geographies of population health and individual and ecological level explanations of variations in cardiovascular disease outcomes. Chapter Three details the methodology used in this research. The chapter focuses on the preparation of the data for the study, definition of the variables used, and the analytical techniques adopted. The empirical evidence on

cardiovascular disease in the study area is presented in two chapters. The first of these, Chapter Four, describes the spatial and temporal patterns of CVD mortality and morbidity. It also describes "hot spots" of CVD outcome that are identified using GIS and spatial analytical techniques. The descriptions of the prevalence of CVD risk factors and the bivariate relationships between these and the various CVD outcomes studied are also included in Chapter Four. The prevalence of the risk factors is discussed under seven conceptual groupings, namely economic environment, social capital, demographic characteristics, psychosocial health and well-being, behavioural characteristics, social support, and physiological characteristics. Chapter Five contains the results of the statistical modeling of the geographic variations in CVD outcomes in Ontario during the period studied. The final chapter is devoted to the conclusions drawn from this research. It also summarizes and discusses the substantive, methodological and theoretical contributions of the study, and highlights future directions for research.

CHAPTER TWO

LITERATURE REVIEW

2.1 INTRODUCTION

This chapter contains a review of the theoretical and empirical literatures relevant to the research outlined in Chapter 1. The chapter begins with a discussion of developments in the geographical traditions related to health research, and the areas of study within these traditions, including a discussion of geographies of population health. The discussion of the evolution of the population health perspective continues with a critique of this conceptual framework for studying the determinants of cardiovascular health. The final section reviews the empirical literature on potential determinants of cardiovascular disease. This review includes individual level explanations of cardiovascular disease outcome, focusing on seven constructs of risk factors, namely economic characteristics, social capital, demographic characteristics, psychosocial health and well-being, risk factor behaviours, social support, and physiological characteristics. The chapter ends with a review of ecological level studies of regional variations in cardiovascular disease outcome.

2.2 GEOGRAPHIES OF HEALTH

Geographers have studied health from a variety of perspectives. The positivist perspective has played a significant role in geographic research on health. The

distinguishing feature of this perspective is an adoption of methods of natural science: observation, accurate measurement and recording, seeking order or spatial arrangement in a set of data, and seeking statistical regularities and associations (Gatrell 2002). Referred to as traditional medical geography, this perspective informs two main areas of study. The first is the study of the spatial patterning and diffusion of disease and death, using individual as well as area-level data. An example of this kind of health study is Cliff and Smallman-Raynor's (1992) description and explanation of geographical variation in the incidence of AIDS in Uganda. Using statistical regression techniques, the authors pursue three different hypotheses on the pattern of AIDS incidence in the study area: 1) that the disease is spread from urban to rural areas by return migrant workers, 2) that it is spread via major transport arteries, and 3) that the Ugandan military has played a major role in the spread of the AIDS in the country. As Gatrell (2002) points out, this study is a classic example of geographic inquiry from a positivist perspective, whereby a set of competing hypotheses are quantitatively tested in order to explain geographic variation in phenomena. From this perspective, many geographical studies of health are concerned with the spatial patterns of specific diseases and their relationships to particular environmental factors. Other related areas of health study are disease ecology and diffusion. Thomas (1992), for example, adopts a systems approach in giving an account of classical spatial diffusion models for infectious diseases.

The second area of study under traditional medical geography encompasses the spatial patterning of health service provision and utilization. Within this, three sub-areas of study can be identified. One of these is the study of the structure and spatial

distribution of health service facilities, such as hospitals and dentists' offices. Another sub-area is the study of patterns of inequality in the provision and utilization of health services. The third sub-area of study is the geographic inquiry into the factors that influence patients' service utilization behaviours, e.g., barriers to individuals' contact with formal health services. Such geographical investigations inform the location of health facilities, for instance, and the allocation of resources to these. Thus, they help address issues of equity and efficiency in the provision and utilization of health services (Curtis and Taket 1996). An example of this second main area of study within traditional medical geography is the study of childhood immunization uptake in a District Health Authority in the United Kingdom using multi-level modelling techniques (Jones et al. 1991). It has been argued that although the quantitative, multi-level modelling approach would identify the statistically significant covariates for childhood immunization uptake, appropriate policy responses would still need to be informed by insights into the reasons underlying the statistical associations (Curtis and Taket 1996). In Canada, Ross et al. (1994) use location-allocation modelling to evaluate the service provided by mammography facilities in the catchment area of the Ontario Breast Screening Program's Kingston Centre. The authors argue for the utility of a location-allocation approach to siting a women's preventive health care service, given the existence of criteria that make for a useful application of this statistical model – the need for the service, expected positive outcomes of access to the service, and a relatively immobile clientele. Employing this model, Ross and her colleagues consider the location of a second breast

screening facility in the catchment area and compared the actual distribution of services with a model-derived optimal solution.

There are three other approaches to geographic research on health, which contrast to traditional medical geography. These are collectively labelled as contemporary medical geography. Curtis and Taket (1996) point out that contemporary medical geography has its epistemological basis in various critiques of positivism, which is a hallmark of traditional medical geography. Gatrell (2002) refers to one of the approaches in contemporary medical geography as the social interactionist approach to health. Geographic inquiry informed by this perspective on health is characterised by emphasis on the influences on health and health-related behaviour of human awareness, agency, and creativity. As Gatrell suggests, " ... the emphasis is on the meaning of the illness or disease to the individual and the task for the researcher is to uncover or *interpret* these understandings and meanings that make it "rational" to act in a particular way ..." (p.32). An example of studies informed by the social interactionist perspective on health is the study of childhood accidents in Huddersfield in West Yorkshire, United Kingdom (Sparks et al. 1994). This study sets out to explain a steep social class gradient in the rates of accidents in two contrasting areas of the community – a high rate of childhood accidents in the area with low socio-economic status, and a low rate of accidents in the area with high socio-economic status. The focus of the research is the lay perceptions and day-to-day experiences of people living in the area, which are important in gaining insights into the social and psychological factors that influenced the accidents. Thus, the aim is to understand this health hazard from the point of view of the people directly

involved (Gatrell 2002). Another example of research conducted from a social interactionist perspective is Kearns' (1991) study of how health care in the Hokianga Special Medical Area on the North Island of New Zealand contributes to a sense of place. Kearns argues that while the provision of certain health services can enhance the community's sense of well-being to the extent that they own such facilities, the facilities can become important points – places – of social interaction in the community. Essentially, research conducted from the social interactionist perspective on health adopts qualitative rather than quantitative methods and techniques. Hence, the main criticism against this approach is that the research results are difficult to verify from a 'scientific' point of view. Another criticism is that this perspective on health focuses on human agency and creativity and neglects the influences on health of wider social structures.

The second approach to health studies under contemporary medical geography is the structuralist approach (or the political economy perspective). Here, the emphasis is on the wider socio-political, economic, and historical structures that influence health experience. From this perspective, the fundamental causes of unhealthy behaviours, such as smoking, excessive alcohol consumption, prostitution, and drug abuse, for example, are believed not to lie with the personal choices made by individuals; rather, they are rooted in the broader socio-economic and political structures that impinge on the lives of these individuals. Marxist theories of domination, class conflict, and oppression, inform this approach, and there is no room for human agency, creativity, and free will. From this perspective, therefore, the structure of society largely influences health and health service provision and utilization. For example, patriarchal domination in society, which plays a

large role in structuring women's health, is taken into consideration in examining women's experience of antenatal care. Another example is the role of colonialism in the diffusion of diseases, and later, the introduction of high-technology curative medicine (as opposed to preventive medicine), in the lands colonized. The colonial infrastructure, such as trade routes and communication networks, facilitated the spread of diseases while the health care delivery system was structured essentially to facilitate the colonization process. In 1897, for instance, Archdeacon Walker wrote: "I regard the medical work from its missionary aspect ... I consider how far it is likely to aid our work, not how much suffering will be relieved" (quoted in Gatrell 2002:39). The pursuit of such covert policy would, without doubt, be a significant factor in the health experience of colonial subjects.

The third approach to the geography of health within contemporary medical geography is known as the structurationist approach. This approach recognizes the dualism of structure and human agency. Thus, while social, political, and economic structures mould people's social practices and actions, individual creativity and actions (or inactions) can result in the creation and recreation of socio-political structures. For instance, the geographic locations and opening hours of immunization centres in a region can affect uptake rates significantly. On the other hand, poor attendance (both punctuality and regularity) can trigger changes in the patterning and operation of these health facilities (Gatrell 2002). Similarly, while structural changes in an urban economy, such as a cut-back in housing subsidies to low-income families can result in homelessness, teeming numbers of homeless people on the streets can lead to the

provision of shelters. From this perspective, therefore, both structure and agency have significant health and health care impacts, which are taken into consideration in health research.

In recent times, some health geographers have engaged with a post-structuralist approach to health studies. This perspective focuses on the exercise of control through the creation of expert knowledge and experience about individuals and societies within the context of power relations in society. This subtle exercise of control or power has the effect of channelling or constraining health thinking and action (Petersen and Lupton 1996). This perspective has informed health studies on representations of the body and of social groups (such as mental patients), health risk, and what being a healthy citizen means (Gatrell 2002). For example, Butchart (1998) examines the relations between socio-medical practices as power and the resultant knowledge of the African body in the mining compound. The post-structuralist approach to health studies seeks to question the rationality of the assumptions of scientific truth on which much public health research is based. Medical geographers who adopt the post-structuralist approach to health view the monitoring of asylum populations, the creation and operation of quarantines and isolation hospitals, and the mandatory wearing of car seatbelts or motor cycle helmets, for example, as constituting controls and the policing of health. Thus, in a subtle way, medical professionals and others in positions of authority are viewed as imposing their definition of health and health care, as well as how these should be experienced, on individuals. In this, there is little, if any, regard for human agency or the historical and political economic structures that impinge on health. Proposing a framework of

combined realist, post-structuralist and feminist approaches in the evaluation of vulnerability to diseases, Craddock (2000), for example, points out that it is not prostitution per se that poses a risk of HIV infection, but the power relations (e.g., who decides whether or not protective measures should be taken) that govern the terms of its practice. Craddock states:

> Risk, as the prologue to disease, must at all cost be seen as historically situated, structured by institutions, households, and nations, and shaped by an ever shifting and relentlessly demanding global economy. But it must also be recognized that these structures and economies mesh inextricably with the social ideologies and cultural codes of particular times and places. (p.164)

She notes that in this expanded scenario, diseases can be seen as more than just an aggregation of their symptoms; "they are also and always cultural products as well as being shaped by political economies and historical contingencies" (p.164).

The above discussion of the geographies of health indicates that there is diversity in the approach to the geography of health. The range and diversity in the approaches to medical geography (both traditional and contemporary), and the counteracting views on the question of categorizing these approaches (Curtis and Taket 1996; Gatrell 2002), suggest that the particular perspective or combinations of perspectives to adopt will be determined by the nature of the research question or questions to be addressed and the types of data available. In turn, these determine the appropriateness of the method(s) of inquiry that will be adopted (Elliott 1999). As Curtis and Taket (1996) point out, all these perspectives on health are present in medical geography today, and they interconnect. The authors suggest that to the extent that a distinction can be made between traditional and contemporary medical geography, the two perspectives within

the former should be characterized by a positivist approach to health research. They suggest that medical geographers who adopt perspectives within the latter should "adopt a stance which argues, in various ways, that notions of health, disease, and illness are problematic, and intimately linked to power relations in society" (p.22). This indicates that a distinction is to be made between quantitative and qualitative methods of investigation, which are respectively suited to traditional and contemporary medical geography. Other authors, however, caution against any rigid distinction between the perspectives on health. For example, Philo (1996:36) cautions:

> "Any attempt to categorize the theoretical approaches taken in medical geography is surely doomed to be flawed and partial, to illuminate some aspects of the intellectual landscape while obscuring others, and in doing so to be just one possible way of telling the story among many."

A combination of three distinct perspectives on health informs this research: logical positivism, social interactionism, and structuralism (see above). While the first perspective is reflected in the quantitative methods adopted, the last two are reflected in the conceptualisation of the research problem and hence the potential CVD risk factors explored in the study (Chapter Three). This research is geared towards understanding the geographic variations in cardiovascular disease mortality and morbidity in Ontario, Canada. These observed variations occur between the public health units in the Province. Therefore, the analyses are done at the ecological level. The conceptual framework that informs this level of analysis is the population health framework, which is reviewed below.

2.2.1 TRENDS IN GEOGRAPHIES OF POPULATION HEALTH

By the early 1970s, all developed nations had extensive and expensive formal health care systems, many of which were largely publicly funded. However, the associated health gains of these systems, which were based on the biomedical model, appeared to have fallen short of expectation (Evans and Stoddart 1990). Consequently, professional concern began to shift from expansion to evaluation and control of the existing systems, with a growing interest in alternative as well as more effective and less expensive approaches to health. Thus, the perspective on health that had emphasized the cure of disease through biomedicine began to broaden to include a consideration of 'non-medical' factors that influenced the health of society. This has led to the development of interest in enhancing the health of entire populations as opposed to expanding the formal health care system to better cater to individual health needs (Evans and Stoddart 1994). The broadening of the understanding of the determinants of health that occasioned the shift in the perspective on health was enhanced by the release of three health frameworks between 1974 and 1994.

The first of these frameworks is the Lalonde report, *A New Perspective on the Health of Canadians* (Lalonde 1974). Based on the 'health field' concept the report identifies four areas (or fields) – human biology, environment, lifestyle, and health care organization – as the underlying determinants of the health status of the individual. The policy instruments implicitly associated with each of these fields are, respectively, the scientific method (regarding health research and delivery), legislation, persuasion, and re-organization (Legowski and McKay 2000). The release of the Lalonde report gave rise to

changes in the development of public policies that began to focus not only on issues related to individual lifestyles (such as smoking, diet, and physical activity), but also on the development of healthy public policies, e.g., legislation regarding the wearing of seat belts (Health Canada 1998). Another important feature of the report is the conceptual change with respect to the determinants of health. The identification and labelling of the four fields indicated that improvement of human health does not depend on increased investment in the health care system alone. However, Evans and Stoddart (1994:43) point out that because of its focus on individual level risk factors and specific diseases, the report "has tended to lead not away from but back to the health care system itself". Therefore, although the four-field health concept broadened the range of determinants of health, it did little to shift the perspective on health away from the positivist, biomedical focus.

The second report is *Achieving Health for All: A Framework for Health Promotion* (Epp 1986). The release of this document was in response to two key developments in the health sector in the 1980s. The first was the expansion of the roles of provincial and territorial health departments to include health promotion by tackling structural problems such as poverty, unemployment, and powerlessness. The other development was the realization that Canada's health promotion strategy appeared to be narrowly focused on individual lifestyle and inconsistent with that of other developed countries, particularly in Europe, which was based on social and environmental factors. This indicated a need to re-focus health promotion in the country, and the policy response was the Epp report. This report further broadened the conceptualisation of the

determinants of health because it shifted away from concentrating on individual lifestyle (as did the Lalonde report) to an expanded viewpoint on health that includes social and environmental factors. As Faresjo (1992) notes, the important social factors to be considered from this expanded viewpoint on health are social stratification, social network, social support and life-style factors.

The third report is the *Strategies for Population Health: Investing in the Health of Canadians* (Federal Provincial and Territorial Advisory Committee on Population Health 1994). This report, which summarized the existing knowledge on the broad determinants of health, represented an official endorsement of the population health approach by the Federal/Provincial/Territorial Ministers of Health (Health Canada 1998). It contained a framework to guide the development of policies and strategies to improve population health.

Developed by the Canadian Institute for Advanced Research (CIAR), the population health framework evolved during a time of government budgetary constraints and a demand for the justification of government spending on all programs, including health (Legowski and McKay 2000). The development of the framework was influenced by the now popular, broader view of health, which includes the influence of socio-economic factors, e.g., income, social status, social support networks, education, employment and working conditions, gender, and culture. During the last decade, population health became a prominent concept in public health programming (Edwards 1999) and in research on the determinants of health. It pertains to "... meeting the basic needs for all, achieving adequate levels of economic and social development, nurturing

social relationships that are mutually supportive and respectful, and ensuring the quality and sustainability of the environment" (Hancock et al. 1999: S22). The Federal, Provincial, and Territorial Advisory Committee on Population Health (1994) defines it as the health of a population as measured by health status indicators and as influenced by social, economic, and physical environments, personal health practices, individual health capacity and coping skills, human biology, early childhood development, and health services. Thus, the population health concept is based on an expanded model of health, which includes the influence of social, cultural, and economic factors (Curtis and Taket 1996), and builds on work in the areas of public health, community health, and health promotion (Health Canada 1998). As Frankish and others (1999) note, population health research is concerned with whole communities, not just individuals or groups, and has the intent to explain differences in health at the population rather than individual level (also McGrail et al. 1998). The shift in the focus on individual health status, and hence, individual level determinants of health, to that of the entire society indicates a conceptual shift towards interactions between humans and their living and working environments.

The work of Marmot and Mustard (1994) illustrates a population-based approach to studying the determinants of health. Analyzing various hypotheses about the particular characteristics of populations that set off the biological responses leading to coronary disease in individuals, the authors argue that the large differences in the rate of occurrence of CHD both between and within countries suggest the influence of the social, cultural, and economic features of those societies. They maintain that examining the problem of CHD from a population base provides a perspective that makes for

incorporating a wide range of sources and types of knowledge, and provides insights different from the biomedical perspective. In his support for a societal approach to health, Rose (1992), for example, argues that many individual level health risks require intervention at the societal level because behaviour is socially determined, and individuals can only be changed by changing society. Rose reviews the distribution of health risk factors in thirty-two different countries at all levels of economic development, and comes to the conclusion that the proportion of a particular country's population that is at high risk depends upon the average risk factor level in the country. Based on this evidence, he concludes that in order to reduce the proportion of a country's population at high risk, the whole country's exposure to that risk has to be reduced. Syme (1996), for example, draws attention to the limited success that attempts to change individual risk behaviours have had. He reviews the problem in the context of the disappointing results of the Multiple Risk Factor Intervention Trial (MRFIT) in the United States. After six years of persuasion, men in the highest 10 per cent of risk for coronary heart disease made very little change in their eating and smoking habits. Syme makes the following comments on the ineffectiveness of pursuing individual level determinants of health, as revealed by the MRFIT project:

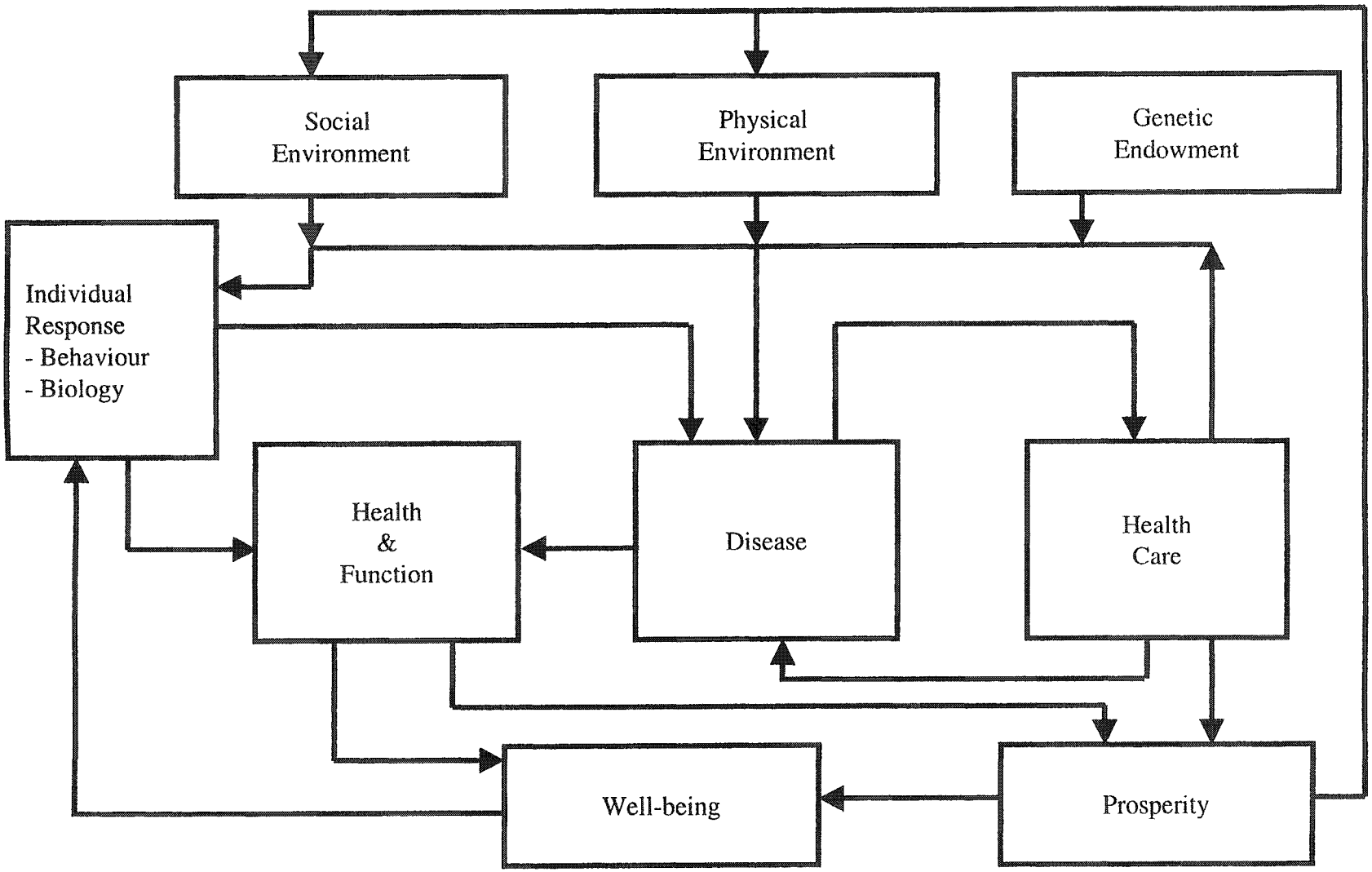
> even when people do successfully change their high risk behaviors, new people continue to enter the at-risk population to take their place. For example, every time we finally helped a man in the MRFIT project to stop smoking, it is probable that, on that day, one or two children in a school yard somewhere were for the first time taking their first tentative puffs on a cigarette. So, even when we do help high risk people to lower their risk, we do nothing to change the distribution of disease in the population because ... we have done nothing to influence those forces in the society that caused the problem in the first place. (Syme 1996:22)

As pointed out above, it would be more effective to address the structural determinants of health, such as the socio-economic, political, and psycho-social environments, from a population perspective rather than from an individual level perspective.

2.2.2 CONCEPTUALISING POPULATION HEALTH

Evans and Stoddart propose an analytic framework (Figure 2.1) within which to organize the evidence on the determinants of health. The framework shows the links between four key determinants of health - social environment, physical environment, genetic endowment, and individual response - and various notions of health. It illustrates the direct impact of these determinants on disease and individual response to the incidence of disease. It also illustrates their indirect impact on health and function, health care, well-being, and prosperity. Thus within the framework, a distinction can be made among disease (as perceived and responded to from a biomedical perspective), health and functioning (from the individual's point of view), and well-being (which depends on health and functioning) (Evans and Stoddart 1990). Their framework extends the *health fields* concept by permitting a more complex and subtle consideration of social and physical environmental influences on both behaviour and biological constitution. This framework represents the socio-ecological perspective on health, offering an expanded scope for research on the health of both individuals and population groups. The authors note, however, that its utility will be tested by the extent to which it provides meaningful categories for organizing the diverse determinants of health.

Figure 2.1 Population Health Framework



SOURCE: Evans and Stoddart (1990)

Various authors have critiqued the population health perspective and frameworks as articulated by the CIAR team. Coburn and Poland (1996) note that the CIAR vision of the social determinants of health lacks a social theory grounding within which to understand the social nature of the determinants of health (see, also, Poland et al. 1998). They point out that the CIAR analysis is implicitly based on an empiricist quantitative approach, which is objective and value-free. Coburn and Poland counter that it is impossible to approach any topic without a prior perspective, or theoretical assumption, both of which go a long way to shape scientific inquiry (see, also, Raphael and Bryant 2000). The argument here, it appears, is that the call for a population approach to health rather than committing more resources to an already excessively large health care system (Evans and Stoddard 1994) does draw (at least implicitly) from some social theory. Evans and Stoddart attribute the large size of the health care system to the activities of powerful providers who benefit from it. The counter-argument is that these powerful providers "... operate ... in a social, political and historical context in relation to the state and to social movements that created universal health care in the first place" (Coburn and Poland 1996:309). Raphael and Bryant (2000) point out that the population health analysis does not take into account how health determinants are created and maintained by powerful economic and social forces. Insights into these theoretical underpinnings of the evolution of the health care system and the associated activities of its key players would inform any recommendations for change. To illustrate the absence of social explanations in the CIAR population health analysis, Coburn and Poland argue, for

instance, that the analysis is completely silent on capitalism although we live in a capitalist society with a particular structure and logic.

Labonte (1995:167) points out that if this health perspective predominates (to the exclusion, for example, of health promotion) in bureaucratic circles, "... its focus on a critique of health care expenditures in a context of fiscal restraint, its emphasis on epidemiological methods, its economic conservatism and its silence on ecological questions of overall economic scale ..." (also Eyles 1999) could inadvertently compromise the delicate "... legitimacy for empowerment, qualitative research, and political advocacy ..." Thus, while on one hand the argument is that health care costs should be curtailed to free up financial resources for economic investment that would generate more wealth and impact health favourably, the argument on the other hand, is that the economic environment engenders inequalities in opportunities, which have adverse health impacts. These counteracting claims appear to indicate that the real causes of health inequalities are of a much more complex origin than the economic system *per se*, possibly the socio-political environment within which both the health and economic systems operate.

Poland et al. (1998) take this debate further by critiquing the conceptual basis of the population health perspective. A basic argument for population health is that the fundamental determinants of the health of a population (or a subgroup of the population) include but extend beyond the formal health care sector (Evans and Stoddart 1994), so that further investment in the health care system is unlikely to result in an improvement in the health of the population. Therefore, attention should be turned to the wealth-

generating sectors of the economy, presumably in the belief that the resultant wealth and prosperity will have a positive impact on population health. Poland and others regard this viewpoint as an under-theorization and oversimplification of the link between economic prosperity and health. In their view, this "wealth-ensures-population health" theory does not adequately address the social forces that produce poverty and inequality, distributive justice, and environmental sustainability. This links back to Coburn and Poland's (1996) criticism that the CIAR analysis of population health lacks a theoretical grounding (see above). It is cautioned that the call made by proponents of the population health perspective for a re-direction of resources away from health care into wealth-generating sectors of the economy "... may be used politically to justify further cutbacks to health care without concomitant reinvestment in or reallocation to other sectors which might produce health ... " (Poland et al. 1998:786). The crux of the critique here is that the social influences cited as determining population health are themselves engendered by modern industrial capitalism. This viewpoint is shared by Coburn (2000) who contends that there is a particular affinity between neo-liberal political doctrines, income inequality, and lowered social cohesion – neo-liberalism produces both of these. Neo-liberalism gives rise to greater income inequality and lowered social cohesion, which are both detrimental to population health (Kawachi et al. 1997; Lomas 1998). Coburn observes that by undermining the welfare state, neo-liberalism has a negative (though indirect) impact on health status.

The above critiques of the theoretical basis of the population health perspective appear to indicate that "immediate" determinants of population health, such as income

inequality, have been given disproportionate attention, and not been linked to "underlying" causes, such as political economy. It must also be noted, however, that from the point of view of empirical research, for example, it might be less problematic to quantify income inequality, lowered social cohesion, poverty, reduced environmental sustainability, etc, than the *advanced industrial capitalism* that arguably engenders them. Further, to the extent that such variables are outcomes of the political economy environment, it is theoretically sound to use them as surrogate determinants to capture the impacts of advanced industrial capitalism on population health.

Eyles (1999) also points out that while the importance of the environment as a determinant of individual and population health has been recognized in the CIAR frameworks, the role of the biophysical environment remains to be fully articulated. He notes, however, that such articulation remains a difficult task because the relationship between the biophysical environment and health are "fraught with scientific uncertainty and dissension" (p. S31).

Another criticism related to the population health perspective is that gender differences in health are not given explicit consideration. Gender is an important basis of inequality because in many societies, the assignment of gender roles has resulted in a situation where, historically, more men than women are found on the higher rungs of the socio-economic ladder. Gender differences have been linked to health status in recent empirical studies. For example, Elliott and Dean's (1998) ecological analysis of psychosocial stress and heart disease in British Columbia reveals a difference between males and females in the number and type of individual stress indicators that are found to

be significant. They find that while only age and language spoken at home are significantly associated with self-reported stress in the case of females, these and four other socio-economic and demographic factors – education, occupation, family status, and income – are significantly related to self-reported stress for males. These results, the authors conclude, appear to support the theory of excessive stress associated with multiple roles to a greater extent for males than females. Upon conducting a multiple regression analysis of data from the 1994 Canadian National Population Health Survey, Denton and Walters (1999) find that structures of social inequality (age, family structure, main activity, education, occupation, income and social support) are the most important determinants of health acting both independently and through their influence on the behavioural determinants of health. In an international comparison of gender differences in adult health (using data from the United States, Jamaica, Malaysia, and Bangladesh), Rahman et al (1994) show that women fare worse than men across a variety of self-reported health measures in the four countries; with these disparities persisting even after adjustments for the impact of differential mortality selection by gender and socio-demographic factors. The data from Jamaica indicate that gender disparities in adult health arise early and persist throughout the life cycle. However, in spite of the evidence that gender partly accounts for differences in health status, women's health, for example, remains under-researched (Rosenberg and Wilson 2000). In investigating the degree and causes of the social gradient in morbidity in a cohort of 10314 British civil servants in the Whitehall II study (Marmot et al. 1991), for instance, the focus of attention is on employment grade, leaving out gender differences in health impact. Kawachi et al.

(1997) explore the extent of social capital in each of the fifty states of the United States to both mortality and the extent of income inequality, and conclude from their study that social capital appears to be one of the pathways through which income inequality exerts itself on the population. This study also does not explore gender specificity as a potential determinant of the inequalities in the health outcomes investigated. Gender differences might play a role in the generation of social capital (e.g., some states might have more or less of social capital depending on the balance of gender roles), and gender differences in access to social capital might play a role in how this capital mediates the health impacts of income inequality. There are some exceptions, however. Walters and others (1996) found gender inequalities in health in a proportional random sample of 2285 male and female nurses registered in the Province of Ontario on the basis of demands of paid work (overload, exposure to hazards), unpaid work (time pressures, caring for a dependent adult) and overall stress in life. For example, they found that young female nurses who report time pressures and caring for a dependent adult are more likely than their male counterparts to report health problems; and that male nurses who dislike housework are more likely to experience health problems. Their analyses point to the need for further investigation of the social production of health outcomes with respect to gender.

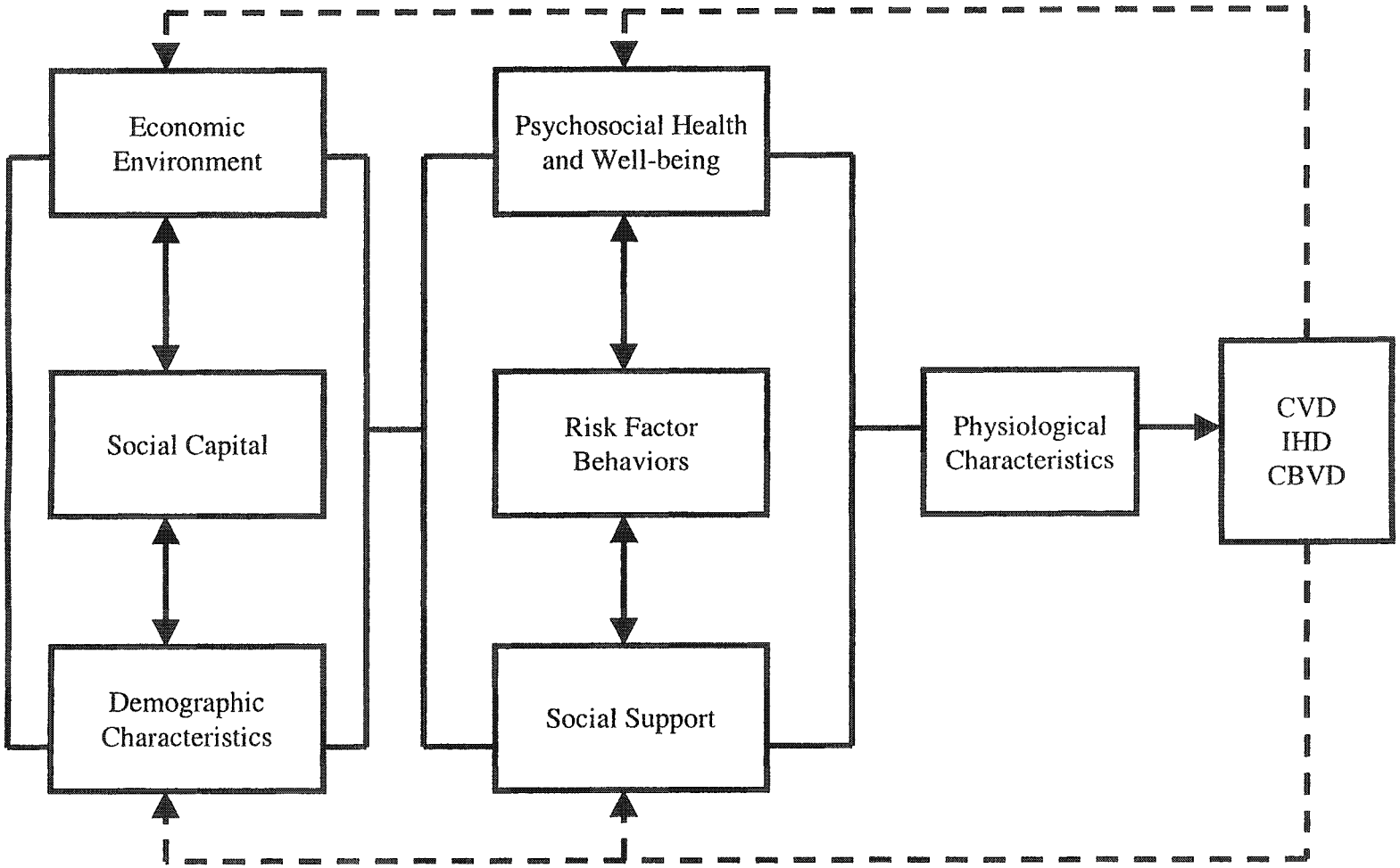
In spite of criticisms of the population health perspective, "it has become ... a persuasive and pervasive element in policy discourse in Canada" (Eyles et al. 2001, p.1612). It informs much research in the areas of population health and health promotion (Elliott et al. 1998), and it has become the guiding framework for much Canadian and U.S. health and health promotion policy and research (Elliott 1999).

Informed by Evans and Stoddart's (1990) framework for studying the determinants of population health (Figure 2.1), and using public health units as the units of analysis, this research is aimed ultimately at understanding the geographic variation in cardiovascular disease outcomes in Ontario. Drawing from Evans and Stoddart's framework, a conceptual model of potential determinants of cardiovascular disease is developed (Figure 2.2) to guide the investigation. In this model, risk factors for cardiovascular events in the study area are categorized into seven main constructs that concomitantly interact. At the same time, these constructs define categories of factors that share a common characteristic. Ultimately, the health impacts of the variables categorized under the various constructs are manifested in physiological characteristics, such as hypertension, diabetes and obesity, which are important risk factors for cardiovascular disease. Conceptually, cardiovascular disease can link back to the constructs to complete a potential reciprocal relationship with its potential risk factors.

2.3 EXPLAINING CARDIOVASCULAR DISEASE OUTCOMES

The relevant empirical literature for this research is summarized under two main headings: ecological studies of regional variations in CVD mortality and morbidity, and individual level studies of CVD risk factors. The literature on ecological level research on CVD risk factors, as well as on the regional variations in CVD outcome serve to identify a broad range of risk factors for possible inclusion as predictor variables, and to identify the methods that might be used to investigate them effectively.

Figure 2.2 Conceptual Model for Studying Geographic Variation in CVD Outcome



As highlighted in Section 2.2.1, the works of Rose (1992), Marmot and Mustard 1994), Syme (1996), and Wilkinson (1996) show that it is more effective to address the determinants of health at the societal (i.e., ecological) level than at the level of the individual (see also Raphael and Farrell 2002; O'Loughlin et al. 1999; Fitzpatrick 2001). Several reasons have been suggested for this. Wilkinson (1996), for example, notes that only a small part of social class variations in mortality can be explained in terms of individual differences in health. He argues that social class gradients in all-cause mortality, for instance, remain nearly as steep even after adjusting for the effects of major individual risk factors. Another reason is that risk exposures are largely sociologically patterned, and many of the determinants of health identified as important at the individual require a societal level intervention (Rose 1992). This is because health risk behaviours, such as smoking and excessive alcohol consumption, are socially determined and can only be modified by changing society (Wilkinson 1996). After reviewing the distribution of health risk factors in a large number of countries at various levels of economic development, Rose came to the conclusion that the proportion of individuals at high health risk in any population is a function of the mean risk level in that population. He argues, therefore, that it is not possible to reduce the proportion of the population at high risk without first reducing the society's exposure to that risk. As Wilkinson (1996:21) points out, taking a societal (or ecological) approach to health "has implications for policy, as well as for the research needed to guide it ..." In the light of the above, the ecological approach is adopted in this research because undertaking an individual level analyses with the view to understanding the regional variations in CVD mortality and

morbidity in the study area would result leaving out the underlying determinants. Wilkinson (1996:21) says of the public policy responses to individual level health research:

> What happens is that the original source of the problem in society is left unchanged (and probably unknown) while expensive new services are proposed to cater for the individuals most affected. Each new problem leads to a demand for additional resources for services to try to put right the damage which continues to be done. Because the underlying flaw in the system is not put right, it gives rise to a continuous flow, both of people who have suffered as a result, and of demands for special services to meet their needs.

2.3.1 ECOLOGICAL LEVEL STUDIES OF REGIONAL VARIATIONS IN CVD OUTCOMES

Compared to individual level analyses, fewer studies have systematically examined regional variations in CVD mortality and morbidity. Statistics compiled by the WHO show that Canada ranks tenth among 20 selected countries in IHD mortality for men and women, while for stroke mortality it ranks as the lowest for men and the third lowest for women (WHO 1995). Within Canada, there appears to be an east-west gradient in mortality rates such that Atlantic Canada has higher rates than the western provinces (HSFC 1997a). At a more local scale, the evidence points to urban-rural differences (Wing et al. 1992), with lower and more rapidly declining rates in urban areas, suggesting a combination of socio-demographic factors and differential access to acute care services.

There is evidence of an inverse gradient between socio-economic status and CVD mortality in Canada based on an ecological analysis of 1986 and 1991 census and

mortality data (Sheth et al. 1997). This study also shows that the gradient varies by ethnic group, being stronger for whites than for South Asians. A recent ecologic analysis of IHD mortality in British Columbia by Elliott and Dean (1998) provides support for an association with psychosocial stress factors such as education, employment status, and multiple roles. Their analysis provides an important insight into the ways in which psychosocial factors are linked to heart disease, and suggests that stressful experiences may not always be detrimental to heart health.

There is empirical evidence of geographic variation in CVD. In Ontario, Alter et al. (1999) illustrate geographic variation in access to health care by analysing inter-neighbourhood variations in mortality from heart disease within one year of treatment for a heart attack. They find that up to one year after a heart attack, 80.5 per cent of patients from neighbourhoods in the highest median income quintile were still alive compared to 75.9 per cent of patients from neighbourhoods in the lowest median income quintile.

In a recent monograph developed by the Institute for Clinical Evaluative Sciences, Bondy et al. (1999) also describe the area variation in heart disease mortality rates in Ontario. They document a wide variation in mortality rates by county: between 1994 and 1997, the highest age- and sex-adjusted mortality rate for CVD by county was 75 per cent higher than the lowest rate. They find that many of the counties with high heart disease mortality rates are rural and agricultural. This is consistent with the HSFC (1997a) finding that the county-specific rate differences for 1991 to 1993 were related to geographic area, with big cities having lower rates than smaller communities.

Rith-Najarian et al. (2002) also compare by region the cardiovascular risk factors among American Indian populations with diabetes. In their study, selected measures of CVD risk (blood pressure, total cholesterol, obesity, tobacco use, average blood glucose level, and proteinuria) were aggregated by region and adjusted to calculate regional rates for 10,889 subjects (2,595 aged less than 45 years and 8,294 aged 45 years and over). Their study shows that among the younger group of subjects, the rates of the selected risks vary significantly among the regions (Alaska, Colorado Plateau, Great Lakes, Great Plains, Southern Plains, Pacific, and Southwest), with the exception of proteinuria and total cholesterol. For example, obesity rates are highest in the Southwest and Pacific regions (78 per cent each) and lowest on the Colorado Plateau (67 per cent). Current tobacco use also varied markedly between the regions, with the lowest rates in the Southwest (29 per cent) and Colorado Plateau regions (12 per cent) and the highest in the Great Plains (59 per cent). They find that among patients aged 45 years and over, there is significant regional variation for all the selected measures of CVD risk. They conclude for their analysis that American Indians and Alaska Natives with diabetes carry a substantial burden of modifiable CVD risk factors, but there are differences in patterns of risk factors in regions of the U.S.

In Europe, too, ecological analyses of observed regional variations in CVD risks and outcome have been undertaken. For example, in the Scottish Heart Health Study, Crombie and others (1990) analyze data for 10,359 men and women from 22 districts of Scotland to try to explain the geographical variation of coronary heart disease mortality. The district level analysis shows that of the "classic" CHD risk factors, only cigarette

smoking is strongly associated with heart disease mortality among both men and women. They find that mean diastolic blood pressure is weakly associated with rates of CHD mortality among men, and high-density lipoprotein cholesterol shows a strong negative association with CHD mortality among women. Total cholesterol shows a weak negative association with heart disease mortality, but the authors explain that a strong association with CHD mortality would not be expected because serum concentration of cholesterol is uniformly high in all the districts. Their study shows that clustering of CHD risk factors (including smoking, alcohol, and diet among men, and smoking, diet, and obesity among women) is associated with much of the regional variation in heart disease mortality in Scotland.

In Sweden, substantial regional differences have been observed in coronary heart disease mortality. A higher mortality rate has been found in the north than in the south, and in the mid-west than in the mid-east (Hammar et al. 1992). The authors set out to extend the analyses of these variations by investigating whether there are regional variations in myocardial infarction (MI) incidence, and whether these variations could be explained in terms of regional variations in smoking, hypertension, and serum cholesterol. Their study involves eight Swedish counties and covers the period 1976-1981. It comprises about half of the total population of Sweden in the age range 30-64 years. The study shows that, taking age differences into account, and compared with Stockholm County in the southern part of the country, there is a 30-40% increased incidence of myocardial infarction in the two northernmost counties and in two other counties for females (rate ratio [RR]=1.40-1.44) and for males {RR=1.22-1.37). The

authors conclude from the results of this study that the regional differences in MI incidence are not related to differences in cigarette smoking (their study revealed no tendency of the proportion of current smokers to be greater in the regions with the highest MI incidence), but are, in part, related to differences in serum cholesterol and, to some extent, to differences in blood pressure.

Kruger and others (1990) also observe regional differences in mortality time trends for ischemic heart disease/sudden unexpected deaths (IHD/SUD) among males in Norway during the period 1966-1985. Their study reveals that there are low rates of IHD/SUD mortality in Western Norway and a correspondingly high mortality in the North. Between the 1966-70 and 1981-85 periods, mortality decreased by 12 per cent in 11 of the country's 19 counties. This contrasted with one county, Oppland, where the IHD/SUD mortality increased by 12 per cent over the two periods. This was the only county in which there was a statistically significant increase in IHD/SUD mortality between the two periods. The IHD/SUD mortality increase in Oppland took place almost solely in the northwestern, rural part of the county. The rate increased by 24.8 per cent in rural areas, but decreased by 15.8 per cent in the urban municipalities. This means that there was spatial variation in the mortality outcome even at the local level. The authors note that although this study is not designed to explore the etiological mechanisms underlying the regional variations, it does reveal that there are significant geographical differences in IHD/SUD mortality within a country with a largely homogeneous medical system.

As illustrated by the above studies, the focus of ecological studies of regional

variation in CVD outcomes have been largely on "classical" risk factors – smoking, high blood pressure (or hypertension), serum cholesterol level, diabetes, obesity (Gensini et al. 1998) – to the exclusion of socio-economic, demographic, and psychosocial variables, such as income inequality, unemployment, employment grade, education, social capital, social support, and psychosocial stress, which have been shown to have an impact on cardiovascular health.

2.3.2 INDIVIDUAL LEVEL STUDIES OF CVD RISK FACTORS

Although individual level CVD risk factors have been the subject of extensive health research, the list of risk factors in Ontario is constrained by the availability and quality of the relevant data for the constituent public health units – the principal geographic units for analysis in this research. The risk factors for ischemic heart disease and stroke have been summarized in a recent Heart and Stroke Foundation report (HSFC 1997a) which draws upon a series of papers published in the *Canadian Medical Association Journal* in relation to the Canadian heart health surveys (Canadian Heart Health Surveys Research Group 1992; MacDonald et al. 1992). The primary focus in the HSFC report is on modifiable risk factors for the purpose of developing risk reduction interventions as part of the Canadian Heart Health Initiative. The result of such limited focus is that the full range of the determinants of CVD is yet to be identified. In view of increasing evidence of the role played by the psychosocial and socio-economic environments in the health status of individuals as well as populations (Wilkinson 1997; Kawachi et al. 1997; Elliott and Dean 1998; Coelho et al. 2000), this study places

emphasis on the so-called non-traditional cardiovascular risk factors. To this end, this research explores a number of socio-economic, psychosocial, and demographic variables that have been linked to cardiovascular health outcome. The following sub-sections contain reviews of the literature relating to individual level studies of these determinants of CVD (which are grouped under the constructs in the conceptual model for this research [Figure 2.2]).

2.3.2.1 ECONOMIC CHARACTERISTICS

The economic variables explored in this study (Table 2.1) include income and income-related variables (e.g., average income and incidence of low-income), unemployment, and dwelling characteristics. Income-related variables, particularly income inequality, are among the most widely investigated economic variables in health research. Studies of the health impact of income inequality, however, are conducted at the ecological level (Section 2.2.1).

Unemployment (and, in some cases, the threat of unemployment) has been linked to health outcomes. For example, Mattiasson and others (1990) conducted a longitudinal study of quality of sleep and serum cholesterol concentrations in men threatened with redundancy. The objective is to assess whether the threat of unemployment affects risk factors for cardiovascular disease. The subjects of the study are a cohort of 715 middle-aged male shipyard workers and 261 age- and sex-matched controls who are followed up for a mean of 6.2 (SD 1.9) years in Malmo, Sweden. The first investigation took place during a period of relative economic stability for the shipyard and the second during the

TABLE 2.1 ECONOMIC VARIABLES AS DETERMINANTS OF CARDIOVASCULAR DISEASE (CVD)		
Variable	Link to CVD	References
Income inequality	Hypothesized to lead to increased levels of disenchantment, which may have deleterious behavioural and health effects	Kawachi et al. (1994); Wilkinson (1994)
	Income inequality exerts a large indirect effect on overall mortality through disinvestments in social capital	Kawachi et al. (1997)
Unemployment	Research over the last two decades has indicated that, changes in national economic indicators, including unemployment rates have influenced those in cardiovascular disease mortality rates.	Brenner (1997)
	The threat of unemployment increases serum cholesterol concentration in middle-aged men. The increase in serum cholesterol is related to changes in other risk factors for CVD. These findings might partly explain the excessive CVD mortality recorded among the unemployed.	Mattiasson et al. (1990)
Housing tenure	Housing tenure is found to be the most discriminatory measure of social status in relation to coronary heart disease	Woodward et al. (1992); Shewry et al. (1992)
	Housing tenure mediates coronary heart disease, diabetes mellitus, and circulatory disease	Tunstall-Pedoe et al. (1995); Sundquist and Johansson (1997); Nilsson et al. (1998)

phase of its closure. They find that there is a greater increase in serum cholesterol concentrations among the shipyard workers threatened with unemployment. However, the evidence for an association between imminent job loss and health is limited (Lavis et al. 2001).

In France Saurel-Cubizolles et al. (2000) explore the relation between unemployment and the psychological distress of 632 mothers one year after childbirth. After adjusting for a number of variables, they find that unemployed mothers have an excess of psychological distress compared with employed mothers (odds ratio = 1.87; 95% confidence interval = 1.12, 3.13). An excess of psychological distress among unemployed compared with employed women was observed in all social groups. So, this study provides an empirical linkage between unemployment and psychosocial health, which, in turn, is linked to cardiovascular events (Rozanski et al. 1999; Black and Garbutt 2002).

Studies linking dwelling value with population health status are rare. However, housing tenure, a related social status variable, is implicated in cardiovascular disease outcomes (see, e.g., Woodward et al. 1992; Shewry et al. 1992), and is explored as a mediating factor for coronary heart disease (Tunstall-Pedoe 1995), for diabetes mellitus (Nilsson et al. 1998), and for coronary and circulatory disease (Sundquist and Johansson 1997). In their investigation of the link between social status and coronary heart disease among 10,359 men and women from the Scottish population, for example, Woodward and his colleagues find that for each of four measures of social status – level of education, years of education, housing tenure, and occupation – the least advantaged have

a significantly higher coronary heart disease prevalence ($p< 0.01$). Their study shows that the highest odds ratios are associated with housing tenure; being 1.63 and 1.55 for men and women respectively, comparing those who live in rented accommodation with owner-occupiers. After adjustment for a number of coronary heart disease risk factors, housing tenure is still highly significant ($p< 0.001$), with odds ratio of 1.48 for men and for 1.45 women (Woodward et al. 1992). Upon investigating the relationships among the social factors themselves, the authors find that housing tenure removes the significant effects of education and occupation in men, and of education in women. No other social factor removes the significant effect of housing tenure ($p< 0.001$). They conclude, therefore, that housing tenure in Scotland is the most discriminatory measure of social status in relation to coronary heart disease. Housing tenure, however, may just be an indicator of relative position in society, which has been shown in the health inequalities literature to be an important determinant of morbidity and mortality (Wilkinson 1996; Marmot et al. 1997). Owner-occupier status is linked to dwelling value, which can be regarded as an indicator of many things: wealth, worth, success and achievement in life, a sense of personal as well as financial security, peace of mind, and social and neighbourhood status. Jerrett et al. (1997) have stated that average dwelling value is sometimes regarded as an index of permanent average income, representing a person's long term ability to pay, and that "the ability to pay for capital assets is usually based more on permanent income, as opposed to the more transitory annual income" (p.1794).

2.3.2.2 SOCIAL CAPITAL

In recent years, social capital has become an important theme in the literature on determinants of health. This notwithstanding, there is an apparent lack of consistent theoretical or empirical justification for the different conceptualisations of this variable, particularly in studies of health inequalities (Macinko and Starfield 2001). Social capital is defined as the features of social structure, such as levels of interpersonal trust, norms of reciprocity, and mutual aid, that act as resources for such collective action (Coleman 1988; Putnam et al. 1993; Kawachi 1999). This research adopts Putnam's (1995:66) definition of social capital as the "features of social organization, such as networks, norms and trust, that facilitate coordination and cooperation for mutual benefit". In his view, social capital consists of social networks, such as networks of civic engagement, and associated norms that have an effect on the productivity of the community. Recent research suggests that social capital has an impact on personal safety and security (Sampson et al. 1997), general health status (Kawachi et al. 1997, 1999; Gorski 2000), cardiovascular health (Lomas 1998), and economic development (Grootaert 1998; Gorski 2000). It also has a moderating effect on the health impacts of other factors, such as poverty (Kawachi et al. 1997; Cattell 2001) and income inequality (Kawachi et al. 1997); and it is a primary contributor to the decision to take certain kinds of action around environmental health hazards (Wakefield et al. 2001).

According to Gorski (2000), greater connection to family (social support) and to community (social capital) correlates with better health and developmental outcomes. He argues that the benefits that accrue from social relationships and affiliations include

improved resistance to infectious diseases (also Cohen et al. 1997), higher resilience to the damaging effects of poverty and maltreatment (also Runyan et al. 1998), and reduced exposure to violence (Sampson et al. 1997). In Gorski's view, "how we live together, the quality and meaning of our relationships in family, community and society, seems to directly influence our individual and population health outcomes" (2000:147).

Research has shown that the health impact of social capital is contextual in nature. For example, in an individual-level analysis of social capital, socio-economic status and health, Veenstra (2000) finds that there is no compositional effect of social capital on health. Using survey data collected in Saskatchewan, Canada (n = 534), Veenstra describes the relationships between individual-level elements of social capital (trust, commitment, and identity in the social-psychological dimension; participation in clubs and associations, and civic participation in the action dimension) and self-rated health status before and after controlling for human capital (socioeconomic status measured by income and education). His study shows that social engagement (frequency of socialization with work-mates, attendance at religious services, and participation in clubs and associations) is positively related to health, but commitment to personal happiness, trust, and civic participation is not related to health. Veenstra observes that:

> ... the contextual nature of social capital leads one to suspect that social capital rich communities may have influences upon individual's [sic] health through pathways other than networking and receiving support ... the challenge, therefore, of social capital research is to identify contextual influences upon health – effects that are, unfortunately, less easily discerned empirically (2000, p.620).

Kawachi and others have also illustrated the contextual effect of social capital in their analysis of social capital and individual self-rated health, with adjustment for

individual household income, health behaviours, and other covariates in 39 US states. They assessed self-rated health (using the question, "Is your overall health excellent, very good, good, fair, or poor?") among 167,259 individuals residing in 39 US states, sampled by the Behavioural Risk Factor Surveillance System. Their results show that residents of states that have low social capital are at an increased risk of poor self-rated health even after adjustment for proximal variables such as low income, low education, and smoking. For example, the odds ratio for fair or poor health associated with living in areas with the lowest levels of social trust is 1.41 compared with living in high-trust states (Kawachi et al. 1999).

Putnam (1995) argues that parental involvement in the educational process through participation in parent-teacher associations is an important form of civic engagement, which represents a particularly productive form of social capital. Licensed day care facilities are important rallying points for such civic engagement. They constitute valuable local resources that can play a key role in developing relationships of trust. They afford a sense of neighbourhood safety and represent a tangible long-term investment in human capital. Therefore, they are used in this study as components of social capital. Other variables selected as indicators of social capital are: number of voluntary organizations (to represent level of civic engagement), charitable donations (to represent reciprocity), and municipal per capita expenditure on environmental defence, social assistance, culture and libraries, and parks and recreation. Social capital is investigated in this study in view of its possible contextual effect on the geographic variation in CVD outcome in Ontario. Table 2.2 details how social capital has been

linked to cardiovascular disease in the literature.

TABLE 2.2 SOCIAL CAPITAL VARIABLES AS DETERMINANTS OF CARDIOVASCULAR DISEASE

Variables	Link to CVD	Reference
Voluntary organizations Charitable donations Licensed day care facilities Municipal per capita expenditure on: Environmental defense, Social assistance, Culture and libraries, Parks and recreation	Income inequality is strongly correlated with both per capita group membership and lack of social trust. In turn, both social trust and group membership are associated with total mortality, as well as rates of death from coronary heart disease. Thus, income inequality leads to increased mortality via disinvestment in social capital.	Kawachi et al. (1997)
	Social capital, measured as social networks and social support, appears to be protective in developing some heart disease and mental illnesses.	McCarthy (2000)

2.3.2.3 DEMOGRAPHIC CHARACTERISTICS

Table 2.3 shows the demographic variables explored in this study and how they are linked to cardiovascular disease. Education mediates the negative health impacts of other variables. To illustrate, Mittleman and others (1997) studied the influence of educational attainment on the relative risk of myocardial infarction (MI) onset following episodes of anger among 1623 patients (including 501 women)

TABLE 2.3 DEMOGRAPHIC VARIABLES AS DETERMINANTS OF CARDIOVASCULAR DISEASE		
Variable	Link to CVD	Reference:
Less than high school education	Incidence of congestive heart failure is positively and significantly associated with less than a high school education (e.g., relative risk [RR]= 1.22 compared to those with high school or higher education)	He et al. (2001)
	In a twelve-year follow-up study, coronary heart disease (CHD) mortality was analyzed in 6431 men fasting and free of prevalent CHD based on risk factor levels and was further divided into rapid and non-rapid deaths. A J-shaped cholesterol-CHD risk function was present for both total and low-density lipoprotein cholesterol. When education level was considered, the J-shaped risk function was present only among men with less than a high school education.	Shestov et al. (1993)
Marital status	Upon examining the effect of marital status on mortality in a cohort of 281,460 men and women, aged 45+ years, of black and white races, each of the non-married groups generally showed statistically significant increased risk compared to their married counterparts. For CVD mortality, widowed and never-married white males aged 45-64 showed statistically significant increased RRs of 1.25 and 1.32, respectively, whereas each non-married group – widowed, divorced/separated, and never-married – of white females showed statistically significant increased RRs from 1.50 to 1.60.	Johnson et al. (2000)

interviewed 4 days (on average) following a myocardial infarction. They categorize educational attainment into three levels: 1) less than high school, 2) completion of high school, and 3) at least some college. Their study shows that the risk of having a MI triggered by isolated episodes of anger decline significantly with increasing levels of educational attainment. They find that the relative risk of MI is twice as high among those with less than high school education (RR = 3.3; 95% CI: 2.0-5.4) compared with patients with at least some college education (RR = 1.6; 95% CI: 0.9-2.9).

In their analysis of the link between psychosocial stress and heart disease in British Columbia, Elliott and Dean (1998) find that the most important explanatory factor for mortality from ischemic heart disease is education. Specifically, they find that variables that measure failure to complete a particular level of education are the most significant recurring variables in their models, and that with only one exception, *university degree*, all the education variables are positively related to heart disease outcomes. As the above studies illustrate, education is inversely associated with incidence of cardiovascular disease (see, also, Gupta et al. 1994; Colhoun 1998). The above studies are evidential of education as a determinant of heart health. In this study, therefore, it is expected that less than high school education will be positively associated with the CVD outcomes studied.

Presumably, being married indicates that an individual has social support. Burnley (1998) demonstrates that the lack of such support by unmarried persons is detrimental to their health in general and their cardiovascular health in particular. He finds that in New South Wales (Australia) between 1969 and 1994, although mortality

from ischemic heart disease declined in all marital status as well as occupational status groups, and in all geographic areas, it declined more slowly among never married and divorced males. Similarly, Johnson et al. (2000) find that in a cohort of 281,460 men and women (grouped as married, widowed, divorced/separated, and never-married) who were part of the National Longitudinal Mortality Study in the USA, each of the non-married categories show elevated relative risks of death compared to married persons, and these effects continue to be strong after adjustment for other socio-economic factors. The study shows that for persons aged 45-64, each of the non-married groups generally shows statistically significant increased risk compared to their married counterparts. The study also shows that for cardiovascular disease mortality, widowed and never-married white males aged 45-64 showed statistically significant increased relative risks of 1.25 and 1.32, respectively, whereas each non-married group of white females showed statistically significant increased relative risks from 1.50 to 1.60.

Other studies have also shown that there is a direct relationship between being unmarried and poorer health. Using bed occupancy in health and social care facilities as a proxy for ill health, Prior and Hayes (2001) test the hypothesis that being married and physical health are positively related. They use census data on all individuals aged 15 years and over occupying beds in general health and social care facilities in England and Wales, Scotland and Northern Ireland in 1971, 1981, and 1991. The results of their analyses suggest that married men and women are healthier than non-married men and women, as reflected in their much lower use of health and social care beds. The results also show that this positive relationship between marriage and health has increased

steadily since the 1970s. Thirdly, they find that within the non-married population, whereas the single are most at risk among men, the widowed are most at risk among women. They conclude from their findings that throughout the United Kingdom not only are married people healthier than non-married people, as reflected in their much lower use of health and social care beds, but also this relationship holds irrespective of gender. It has also been shown in a number of population studies that a higher mortality is experienced by unmarried individuals, people who live alone, and people who are divorced or never married (Sorlie et al. 1995; Joung et al. 1996; Tucker et al. 1996; Sundquist and Johansson 1997; Nakanishi et al. 1998). Therefore, the current literature on the health effects of marital status appears to indicate that being unmarried is a risk factor for cardiovascular disease.

However, some studies have not demonstrated this association. For example, in a longitudinal study using a nationally representative sample of British women aged 35 years and over drawn from the National Health Service Central Register, Cheung (2000) finds that although being single (HR = 1.45) is significantly associated with higher all-cause mortality, being divorced and being widowed are not associated with excess mortality risk (each HR = 1.09). Avlund et al. (1998) also show in a recent Danish study that men living alone experienced an increased mortality, but find no such association among women. In constrast to this, Moritz and Satariano (1993) find that married women and those living with their spouse showed increased mortality.

2.3.2.4 PSYCHOSOCIAL HEALTH AND WELL-BEING

The role of psychosocial factors in health has received attention in both the population and occupational health literatures (e.g., Marmot and Mustard 1994; Elliott 1995; Wilkinson 1996; Marmot 1997; Orth-Gomer 1997). Psychosocial factors include both psychological characteristics such as personality, and life experiences such as long-term stress and social isolation (Steptoe 1999). It has been suggested that the role of psychosocial factors may be particularly important in understanding gender-related differences in health risk (Elliott 1995; HSFC 1997b). The discourse has been taken further by linking the cardiovascular effects of psychosocial well-being to social and community structures, particularly socio-economic position, social cohesion and social capital (Marmot and Mustard 1994; Wilkinson 1996; Lomas 1998). Various models are postulated to explain how psychosocial variables impact on cardiovascular health, but the multiplicity of possible explanations suggests that the mechanisms involved are probably complex and multidimensional, rather than a simple, direct causal relationship (King 1997). Psychosocial factors might contribute to a fuller understanding of the geographic variations in heart disease outcome in Ontario in the light of the fact that these variations cannot be explained fully in terms of the more conventional risk factors alone (Chapter One). Table 2.4 contains examples of possible pathways by which psychosocial variables impact on cardiovascular health.

TABLE 2.4 PSYCHOSOCIAL HEALTH AND WELLBEING AS DETERMINANTS OF CARDIOVASCULAR DISEASE		
VARIABLE	LINK TO CVD	REFERENCES
Psychosocial stress	Arteriosclerosis (a CVD) is now considered to be the result of a chronic inflammatory process. Repeated episodes of acute psychological stress, or chronic psychological stress, may induce a chronic inflammatory process culminating in arteriosclerosis. Stress, by activating the sympathetic nervous system, the hypothalamic-pituitary axis, and the renin-angiotensin system, causes the release of various stress hormones such as catecholamines, corticosteroids, glucagon, growth hormone, and renin, and elevated levels of homocysteine, which induce a heightened state of cardiovascular activity. The argument is made that in reacting to stressors, humans mount stress/inflammatory responses in the arteries, which, if repetitive or chronic, may culminate in arteriosclerosis.	Black and Garbutt (2002)
	A number of newer, "nontraditional" cardiovascular risk factors have been identified based on recent studies of the pathogenesis of arteriosclerosis and ateriothrombotic cardiovascular events. These include chronic inflammation and its markers, such as psychosocial factors, such as environmental stress and responsiveness to stress.	Oparil and Oberman (1999)

Table 2.4 continued		
Psychosocial stress (cont'd)	Possible pathways by which SES affects cardiovascular disease include effects of chronic stress mediated by the brain, differences in lifestyles and behaviour patterns, and access to health care. At the present time, the second of these is the strongest candidate.	Pickering (1999)
	The evidence that psychosocial factors contribute significantly to the pathogenesis and expression of coronary artery disease (CAD) is composed largely of data relating CAD risk to 5 specific psychosocial domains: (1) depression, (2) anxiety, (3) personality factors and character traits, (4) social isolation, and (5) chronic life stress. Pathophysiological mechanisms underlying the relationship between these entities and CAD can be divided into behavioral mechanisms, whereby psychosocial conditions contribute to a higher frequency of adverse health behaviors, such as poor diet and smoking, and direct pathophysiological mechanisms, such as neuroendocrine and platelet activation.	Rozanski et al. (1999)

2.3.2.5 RISK FACTOR BEHAVIOURS

The risk factor behaviours included in this research are smoking, excess fat in diet, physical inactivity, and excess alcohol consumption. Table 2.5 shows how these risk factors are linked to cardiovascular disease. Smoking is an important risk factor that

TABLE 2.5 RISK FACTOR BEHAVIOURS AS DETERMINANTS OF CARDIOVASCULAR DISEASE		
VARIABLE	LINK TO CVD	REFERENCES
Smoking	Hypertension and smoking interact to increase the incidence of cardiovascular disease. Smoking increases the cardiovascular risk of hypertension for any level of systolic or diastolic blood pressure possibly by effects on hemostatic function lipoproteins, peroxidation and oxidative damage.	Oncken (1996)
	Smoking acts synergistically with high cholesterol levels and hypertension to significantly increase the risk profile of coronary artery disease, so that the risk profile of smokers with both of these factors is worse than the sum of the independent risk levels related to each individual risk factor.	Gensini et al. (1998)
	The mechanism of the increase in coronary heart disease risk associated with smoking may partly be due to smoking-related changes in intermediate risk factors such as lipid levels, fibrinogen and blood pressure.	Cullen et al. (1998)

Table 2.5 continued		
Excess fat in diet	Higher intakes of saturated and trans fats are associated with an increased risk of CHD while higher intakes of monounsaturated and polyunsaturated fats are associated with reduced risk. This is because saturated and trans fatty acids raise serum low-density lipoprotein cholesterol and lower high-density lipoprotein cholesterol in humans. It has been determined that consumption of more than 30 per cent fat in diet is a risk factor for cardiovascular disease.	Hu et al. (1999); Toeller et al. (1999) Zock and Katan (1997); Klor et al. (1997); Grundy (1997) CCSCC (1998)
Physical inactivity	Cardiovascular disease incidence and mortality, and specifically ischemic heart disease, are causally related to physical activity in an inverse, dose-response fashion.	Kohl (2001); Whaley and Blair (1995)
	An average of 3.0+ kcal/kg/day of energy expenditure is approximately the amount of exercise that is required for cardio-vascular benefit	Ministry of Health, Ontario (1990)
Excessive alcohol consumption	There is an inverse relation between alcohol consumption and fasting insulin concentrations. Some studies have found a U shaped relation, and this is probably due to the inclusion of diabetic subjects. As hyperinsulinemia has been shown to be positively associated with cardiovascular disease, it may be one of the variables that explain the protective effect of moderate alcohol consumption on cardiovascular disease.	Konrat et al. (2002)

accounts for a significant proportion of deaths from cardiovascular disease, coronary artery disease, and ischemic stroke (Gensini et al. 1998). The risk of CVD events is directly proportional to the number of cigarettes smoked per day (Negri et al. 1993), and it has been found that smoking cessation is associated with a significant reduction in cardiovascular risk (Gensini et al. 1998). It is therefore expected that the smoking variable used in this study – current daily smoking – will be positively associated with the CVD outcome rates.

The literature on the link between nutrition and CVD focuses mainly on fat intake, i.e., saturated and trans fats vs. unsaturated (monounsaturated and polyunsaturated) fats, and its effects on plasma cholesterol and fetal nutrition (which is linked to birth weight). Higher intakes of saturated and trans fats are associated with increased an increased risk of CHD while higher intakes of monounsaturated and polyunsaturated fats are associated with reduced risk (Hu et al. 1999; Toeller et al. 1999). This is because saturated and trans fatty acids raise serum low-density lipoprotein cholesterol and lower high-density lipoprotein cholesterol in humans (Zock and Katan 1997; Klor et al. 1997; Maryniuk and Peterson 1997; Grundy 1997). It has been determined that consumption of more than 30 per cent fat in diet is a risk factor for cardiovascular disease (Canadian Cardiovascular Society Consensus Conference 1998). The nutrition variable explored in this study, therefore, is the prevalence of consumption of greater than 30 per cent fat in diet, which is expected to be positively associated with CVD.

Much work has been done on the possible connection between physical exercise

and cardiovascular health. Observational, population-based studies have consistently shown an inverse dose-response gradient between physical activity or fitness and CHD (Whaley and Blair 1995). The concern for physical inactivity as a CVD risk factor is heightened by the fact that modern societies are increasingly characterized by sedentary lifestyles resulting from increasing automation of hitherto manual functions. For example, Wenger (1995), reports that over a quarter of US women aged 20 to 74 years are sedentary. In Canada, the proportion of adults who are inactive in their leisure time is as high as 38 per cent. At the provincial level, this varies from 29 per cent in British Columbia to 40 per cent in Newfoundland (Canadian Cardiovascular Society Consensus Conference 1998). Meanwhile, epidemiological data show that sedentary subjects have, on average, double the risk of CVD as active individuals (Jennings 1995).

Some studies have shown that alcohol, when consumed in moderation, decreases mortality from cardiovascular disease (Kannel and Ellison 1996). However, Rayo and Marin (1998) point out that although moderate alcohol intake – between 10 and 30 grams of ethanol a day – decreases cardiovascular mortality, this beneficial effect may well be outweighed because excessive alcohol consumption raises mortality due to other causes, especially injury, cirrhosis of the liver, and some types of cancer. Moreover, problem drinking (well beyond two drinks per day) is associated with increased cardiovascular mortality (Kannel and Ellison 1996). The combination of protective and harmful influences of alcohol consumption results in a U-shaped mortality curve, such that non-drinkers have a higher risk than moderate drinkers, but the risk increases with increasing consumption Gensini et al. (1998). In a recent report, Jaglal et al. (1999) state that most

of the protective effect of alcohol against heart disease is found with as little as 5 grams of ethanol per day. They note, however, that the consequences of inappropriate alcohol use are so significant that no major agency has recommended its use for the prevention of heart disease. Thus, guidelines on whether or not to start, to continue, to modify or to stop alcohol consumption must be given on an individual basis, taking into account the relative risks and benefits for each patient (Rayo and Marin 1998). This study uses the definition of excessive alcohol consumption provided by the Canadian Cardiovascular Disease Consensus Conference (1998) – greater than 9 drinks (females) and 14 drinks (males) per seven-day week – and it is expected that this variable will be positively associated with CVD outcome.

2.3.2.6 SOCIAL SUPPORT

Table 2.6 describes the possible links between social support and CVD outcome and the directions of these associations. Social support is one of the structural determinants of health (Denton and Walters 1999) on which recent research has focused. Although the exact mechanism by which it impacts on heart health is not fully known (Tennant 1999), recent empirical studies suggest that lack of social support has a detrimental impact on cardiovascular health. For example, in the Stockholm Female Coronary Risk Study, Orth-Gomer and others (1998) investigate the role of social support in the severity and extent of coronary artery disease in one hundred and thirty-one women hospitalized for an acute coronary event. They find that after adjustment for age, lack of social support was associated with measures of coronary artery disease (CAD). With

further adjustment for smoking, education, menopausal status, hypertension, high density lipoprotein and body mass index, the risk ratio for stenosis greater than 50% in a coronary artery (a measure of CAD) in women with poor as compared to those with strong social support was 2.5 (95% CI = 1.2 to 5.3; p=0.003). This means that women with poor social support are 2.5 times more likely to have the risk for CAD, and indicates that lack of social support is positively associated with cardiovascular disease outcome.

TABLE 2.6 SOCIAL SUPPORT AS DETERMINANT OF CARDIOVASCULAR DISEASE

VARIABLE	LINK TO CVD	REFERENCES
Social support	One situational factor that impacts cardiovascular responses to stress is the presence of other people and their behavior. Cardiovascular reactivity is greater for low-efficacy speakers and for those receiving positive feedback.	Hilmert et al. (2002)
	The presence of an ally, especially a female, markedly reduces cardiovascular responses compared both to the presence of a non-supportive other, and to experiencing the stress alone.	Christenfeld and Gerin (2000)
	Lack of social support contributes to the severity of coronary artery disease in women, independent of standard risk factors.	Orth-Gomer et al. (1998)

2.3.2.7 PHYSIOLOGICAL CHARACTERISTICS

Table 2.7 describes how the physiological characteristics explored in this study – obesity, hypertension, and diabetes – are linked with CVD. Bjorntorp (1997) observes that obesity has now become a world-wide epidemic and is associated with large economic costs and prevalent diseases, particularly with central body fat distribution. Defined as weight/height^2 above a certain cut point, its prevalence varies from 7% in France to 32.8% in Brazil (Saw and Rajan 1997). In Canada, 31 per cent of adults are considered to be obese, i.e., with body mass index (BMI) greater than 27.0 (Canadian Cardiovascular Society Consensus Conference 1998). The prevalence of obesity increases with age and is greater in men (35%) than in women (27%) (Reeder et al.1992). Obesity appears to be associated with a particularly high risk of coronary artery disease (Gensini et al. 1998). In adults obesity is associated with greater left ventricular mass, an important predictor of cardiovascular morbidity (Sasson 1993). Severe obesity (BMI >= 35.0) is associated with approximately a two-fold increase in total mortality and with a several-fold increase in mortality due to diabetes, cardiovascular disease, cerebrovascular disease, and certain forms of cancer. Sjostrom (1992) asserts that studies that have not been able to confirm this have been small and/or short term, failed to control for smoking or early mortality, controlled for intermediate risk factors in an inappropriate way, or have a reduced internal validity due to misclassification biases.

There is evidence that high blood pressure (or hypertension) is an independent risk factor for coronary artery disease mortality for both men and women in all age and

TABLE 2.7 PHYSIOLOGICAL CHARACTERISTICS AS DETERMINANTS OF CARDIOVASCULAR DISEASE		
VARIABLE	LINK TO CVD	REFERENCES
Obesity	Severe obesity (BMI => 35) is associated with approximately a two-fold increase in total mortality and with a several-fold increase in mortality due to diabetes, cardiovascular disease, cerebrovascular disease, and certain forms of cancer.	Sjostrom (1992)
	Obesity is associated with an increase in all-cause mortality and cardiovascular mortality, with a particularly high risk for subjects with central obesity. Central obesity is also part of the so-called 'metabolic X syndrome' including insulin resistance, which appears to be associated with a particularly high risk of coronary artery disease.	Gensini et al. (1998)
	In adults obesity is associated with greater left ventricular mass, an important predictor of cardiovascular morbidity.	Sasson et al. (1993)
Hypertension	Individuals with high blood pressure tend to have high heart rates. Fast heart rate precedes the development of high blood pressure and serves as an early indicator of coronary heart disease.	Julius et al. (1998)

Table 2.7 continued		
	Hypertension is a significant, strong and independent risk factor for coronary artery disease morbidity and mortality, and the reduction of events and mortality by anti-hypertensive treatment is well documented.	Gensini et al. (1998)
Diabetes	Type 1 and type 2 diabetes mellitus are associated with an increased risk of cardiovascular disease, especially in women.	Gensini et al. (1998)
	The risk of diabetes for cardiovascular morbidity and mortality is mainly related to disorders of glucose intolerance - particularly type 2 diabetes, and pre-diabetic conditions, including insulin resistance.	Henry (1998)

ethnic groups (Gensini et al. 1998). It has been found that blood pressure and heart rate are positively correlated. Fast heart rate precedes the development of high blood pressure; hence it serves as an early indicator of CHD (Julius et al. 1998). Hypertension, which afflicts 10 per cent of the population of Ontario (Kirk-Gardner and Steven 1994), is associated with an increased risk of developing coronary heart disease, stroke, congestive heart failure, and peripheral vascular disease (McAbee 1995). It is therefore included in this study as a potential physiological risk factor for CVD, and is expected to be positively associated with the outcome variables.

Diabetes has been identified as an important risk factor for cardiovascular disease. Diabetics have a 2-3 times higher absolute risk of cardiovascular events than non-diabetics, and the risk of death is also related to obesity, smoking, hypertension, and glycaemic control (Gensini et al. 1998). The risk of diabetes for cardiovascular

morbidity and mortality is mainly related to disorders of glucose intolerance - particularly type 2 diabetes, and pre-diabetic conditions, including insulin resistance (Henry 1998). In some situations the risk of cardiovascular disease might be reduced by the prevention of diabetes. In a review of the literature on the association between diabetes and cardiovascular conditions Laakso (1998) notes that diabetes mellitus, particularly non-insulin-dependent diabetes mellitus, increases the risk for all manifestations of atherosclerotic vascular disease, coronary heart disease, cerebrovascular disease, and peripheral vascular disease. He finds that non-insulin-dependent diabetes mellitus is associated with several adverse cardiovascular risk factors, including hypertension, hyperinsulinemia, serum lipid and lipoprotein abnormalities, obesity, and central obesity.

2.4 SUMMARY

This chapter explored relevant literature for informing our understanding of geographic variation in cardiovascular disease mortality and morbidity. The literature review spanned the geographies of health, trends in the geographies of population health, and population health concepts. It also included a review of studies explaining regional variations in CVD outcomes and individual level studies of CVD risk factors.

The review of the literature on the geographies of health revealed that while a particular research question (such as what drives spatial variation in CVD outcomes) can be addressed either quantitatively or qualitatively (or, indeed, using a combination of these two approaches) depending on the research objective(s), there is a wide variation in

the perspective from which this can be pursued. In sum, it is the nature of the research question that determines the method or set of methods to be used.

The trends in the geographies of population health indicate that, generally, there has been a shift from biomedical perspectives on health towards socio-ecological perspectives, with emphasis on understanding health at the level of the entire population rather than addressing individual cases. This is based on the idea that individual level health is fundamentally determined by societal level factors.

This chapter has proposed a conceptual model for understanding geographic variation in CVD outcome. The model groups the risk factors for CVD into seven risk constructs that ultimately give rise to cardiovascular diseases, which, in turn, are linked back to the constructs in a somewhat cyclical relationship.

While the review of individual level studies of CVD risk factors shows that socio-economic and psychosocial variables play key roles in cardiovascular outcomes, these have been little explored in ecological level studies of regional variations in CVD. Therefore, in order to understand the geographic variation in CVD outcome in Ontario, this research extends the range of determinants beyond physiological and behavioural risk factors to include non-traditional risk factors such as socio-economic, demographic, and psychosocial variables.

The next chapter outlines the methodology used in this research, which comprises of the design of the research, the definition and derivation of variables, and the specification of the analytical techniques used.

CHAPTER THREE

METHODOLOGY

3.1 INTRODUCTION

This chapter is divided into three main sections. Section 3.1 describes the design of the study, including the sources of data used. Section 3.2 describes how the variables included in the analysis are defined and/or derived. The techniques used to analyse the geographic variations in CVD, as well as the determinants of these variations, are described in the final section.

3.2 STUDY DESIGN

3.2.1 STUDY AREA AND UNITS OF ANALYSYS

The study area – the Province of Ontario – is located approximately between Latitudes 42.5^0 North and 52^0 North, and it extends approximately from Longitude 74^0 W to 95^0 W. It is bounded on the west by the Province of Manitoba, on the south by United States, and on the east by the Province of Quebec (Figure 3.1). The Province is divided into 42 public health units (Ministry of Health, Ontario 1990). There is an expected lack of specificity in the incidence of CVD in Metropolitan Toronto. So, following Walter et al. (1999), the public health units in this area, namely York, East York, North York, Etobicoke, Scarborough, and Toronto-City were combined into the City of Toronto public health unit. This resulted in 37 spatial areas (Figure 3.2) that formed the primary units for analysis in this study.

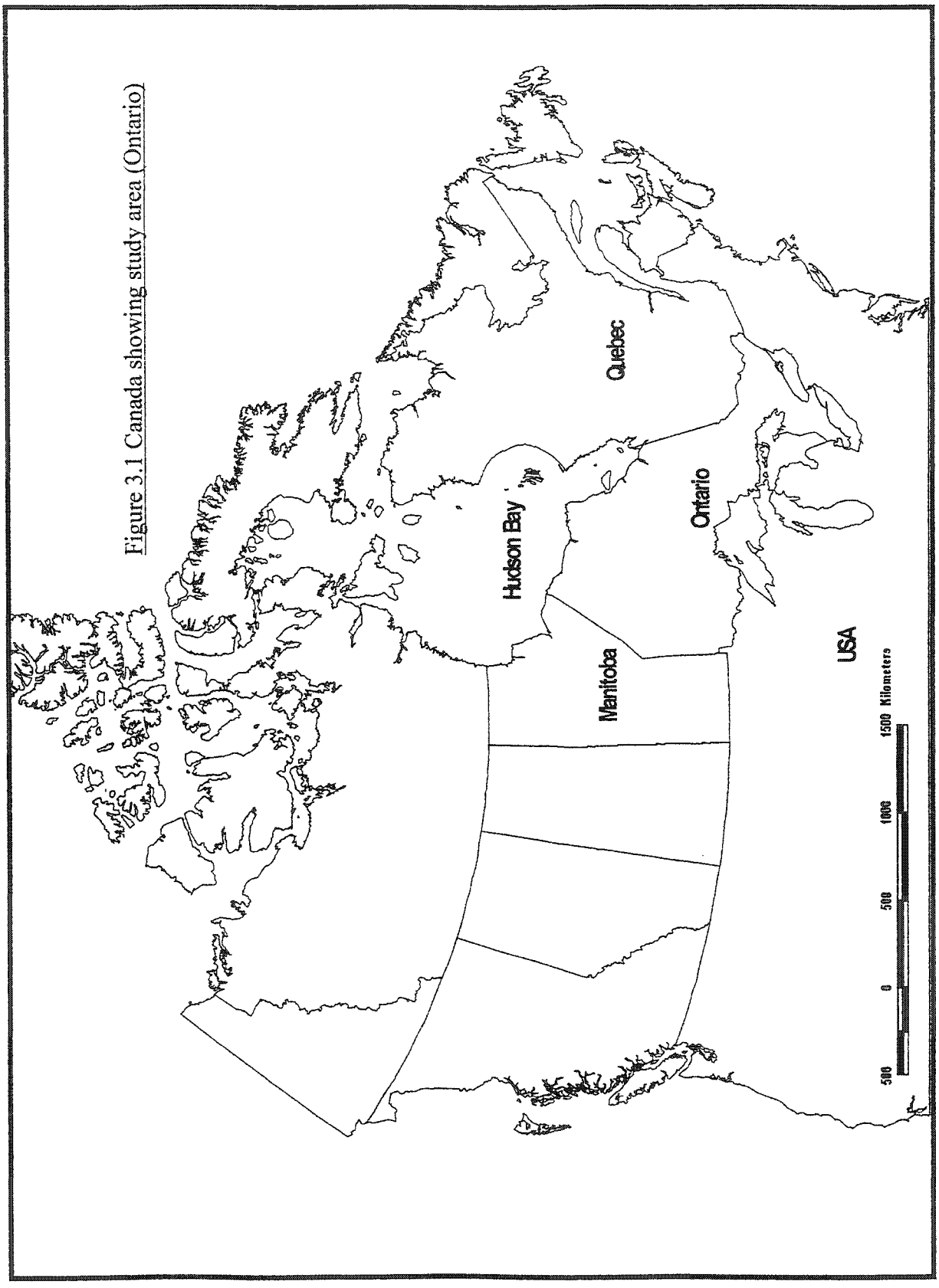
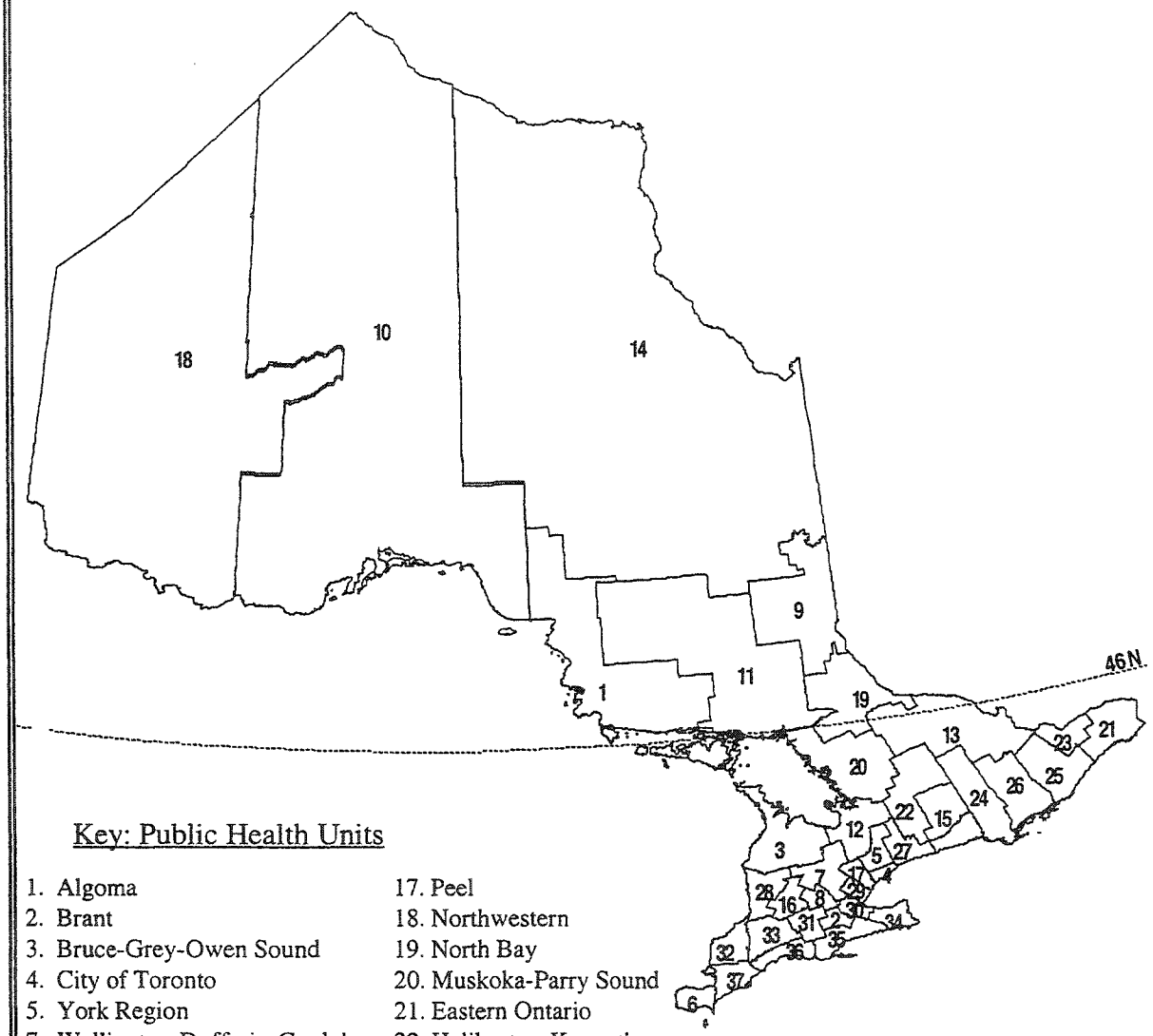


Figure 3.1 Canada showing study area (Ontario)

Figure 3.2 Public Health Units of the Province of Ontario



Key: Public Health Units

1. Algoma
2. Brant
3. Bruce-Grey-Owen Sound
4. City of Toronto
5. York Region
7. Wellington-Dufferin-Guelph
6. Windsor-Essex
8. Waterloo
9. Timmiskaming
10. Thunder Bay
11. Sudbury
12. Simcoe
13. Renfrew
14. Porcupine
15. Peterborough
16. Perth
17. Peel
18. Northwestern
19. North Bay
20. Muskoka-Parry Sound
21. Eastern Ontario
22. Haliburton-Kawartha
23. Ottawa-Carleton
24. Hastings-Prince Edward
25. Leeds-Grenville-Lanark
26. Kingston-Frontenac-Lennox & Addington
27. Durham
28. Huron
29. Halton
30. Hamilton-Wentworth
31. Oxford
32. Lambton
33. Middlesex-London
34. Niagara
35. Haldimand-Norfolk
36. Elgin-St Thomas
37. Kent-Chattam

3.2.2 DATA SOURCES

3.2.2.1 OUTCOME MEASURES

The outcome measures used in this study were mortality and morbidity from all cardiovascular diseases (CVD), ischemic heart disease (IHD), and cerebrovascular disease (CBVD), for both sexes combined, and separately for females and males. CVD mortality data for the period 1986-1994 were obtained from the death statistics collected by the Registrar General of Ontario. They included data on mortality due to all circulatory diseases (ICD-9 codes 390-459), ischemic heart disease (ICD-9 codes 410-414), and cerebrovascular disease (ICD-9 codes 430-438).

The information recorded on death certificates is a useful source of population-based mortality data. A major strength of these data is that law requires completing and forwarding of the information on the death certificate. However, questions about the reliability of coding the underlying cause of death raise doubts about the accuracy and completeness of the information provided. Myers and Farquhar (1998) suggest that inadequate or misinterpreted clinical information can result in erroneous ante mortem diagnoses, which are then recorded on the death certificate. They note that the rate of major discrepancy between autopsy results and death certificate information has been found to be as high as 30 per cent. This exemplifies the difficulty of establishing appropriate numerators for ecological level analyses (Walter 1991b). However, death certificate data are the most complete mortality data available.

Data on CVD, IHD, and CBVD morbidity are obtained from hospital separation statistics collected by the Canadian Institute for Health Information (CIHI). These data

are one of the best available databases on morbidity information for the population of Ontario because it represents the use of in-patient services by the entire population and is not dependent on self-reporting of conditions by survey respondents. The data are collected and coded in a systematic way at the hospital level and then checked for coding errors and omissions by CIHI before being made available for analyses. A limitation of the CIHI data is that they reflect the number of separations and not the number of people with at least one admission for a given cause or group of causes. Re-admission rates are likely to be greater in urban public health units because of the proximity to health care facilities such as acute care centres and ambulatory services. Another potential limitation in the use of the hospital separation data is that the reliability of coding clinical information varies across conditions. For example, conditions such as acute myocardial infarction are coded more reliably than others such as cerebrovascular disease (Williams and Young 1996). However, there is evidence that disease coding reliability increases as diagnostic specificity decreases (e.g., ICD 4 digit codes to ICD 3 digit codes [Williams and Young, 1996]). Therefore, the use of broad disease groupings such as CVD (ICD-9 codes 390-459), IHD (ICD-9 codes 410-414), and CBVD (ICD-9 codes 430-438) helps to minimize the impact of coding reliability. Another limitation of using in-patient service data to estimate morbidity is the assumption that service use accurately represents morbidity in the population, without accounting for the influences of differential access to services on the data (Eyles et al. 1991). Regarding the difficulty of establishing appropriate numerators, the CIHI database from which the morbidity data for this study were drawn can be expected to be comprehensive and complete for the residents of

Ontario because access to care is universal and does not change over time (Institute for Clinical Evaluative Studies 1996).

3.2.2.2 EXPLANATORY VARIABLES

The data on CVD risk factors were drawn from a variety of sources. Socio-economic and demographic data come from the Statistics Canada 1991 census profiles 2A and 2B for counties. The census is carried out every five years and is a reliable source of socio-demographic information for the population of Ontario. Socio-economic information is collected from 20% of the households. The 20% samples surpass the sample size of any available population-based surveys. The more recent 1996 census data was not used because is falls outside the period considered in this study – 1986-1994.

Data on the physiological, behavioural, and psychosocial risk factors investigated in this study come from the 1990 Ontario Health Survey (OHS) - a comprehensive health survey of the population of Ontario. A total of 61,239 individuals in 35,479 dwellings representative of the provincial population completed the survey. The survey used a multi-stage stratified sample design in which the population of the province was first stratified into public health units (PHU), and then into urban and rural strata. The urban stratum of the PHU consisted of the urban core and fringe components of any Census Metropolitan Areas or Census Agglomeration Areas present in the PHU, and the remainder of the PHU constituted the rural sub-stratum. The questionnaire for the survey comprised an interviewer-completed and a self-completed portion. The target population

for the first portion consisted of all residents of private dwellings in Ontario during the survey period, excluding residents of Indian reserves, inmates of institutions, Foreign Service personnel, and residents of remote areas. The target population for the second portion was the same as above, but restricted to persons aged 12 years and over at the time of the survey. The overall response rate for the entire province and for the whole survey was 87.5% to the interviewer-completed portion, and 77.2% to the self-completed questionnaire (Ministry of Health, Ontario 1990).

The OHS data set included responses to single questions as well as variables derived from responses to several related questions. Both types of variables were used in this analysis. The sample design requires that statistical weights be applied to individual level data prior to aggregation at the PHU level. The weight is proportional to the number of persons each respondent represented in the survey. Each household and individual in the target population had a different probability of being included in the sample. Further, adjustments were made to the weights derived from the probability of selecting the dwellings to account for non-responses at the individual and household levels. Following Walter et al. (1999), the individual statistical weights were re-scaled to analytical weights before being applied in the aggregation of CVD risk factors within the PHUs. The re-scaled weights added up to the total number of respondents in the sample for the PHU. Each re-scaled weight was then multiplied by a factor of 100 to make for convenient computation. In order to eliminate the confounding effects of differing age distribution in the calculation of rates across PHUs, the risk factor data were directly age-standardized to the provincial population distribution prior to aggregation for ecological

analysis at the PHU level. The OHS data were stratified into 37 public health units after combining the data for six Metropolitan Toronto PHUs (Section 3.2), and for the Bruce and Grey-Owen Sound PHUs, which were merged into one public health unit – Bruce-Grey-Owen Sound – after the survey was designed (Ministry of Health, Ontario 1990). The OHS provided a large data set from which indices of risk factors for CVD (including tobacco use, excessive fat consumption, physical inactivity, and psychosocial factors) were derived. While the OHS data were compiled for 42 PHUs (which were subsequently regrouped to 37 PHUs as described above), some of the CVD risk factor data used in this study come from the 1991 census were compiled for 49 census divisions in the Province. Therefore, the risk factor data needed to be re-worked for the 37 spatial units for analysis.*

Other sources were accessed for data on cardiovascular risk factors. The names and postal addresses (including the postal codes) of a total of 27,272 voluntary organizations in Ontario were obtained from the Revenue Canada web site (as of December, 2000). The coordinates of the centroid of each postal code area were derived using the 1991 postal code conversion file. Coordinates could not be assigned to about 1.03 per cent (n = 281) of the postal codes. This suggests that the organizations in whose addresses these postal codes occurred were either non-existent in 1991 or they existed but have since changed their postal addresses. Using ArcView, the postal codes were then assigned to the PHUs to determine the number of voluntary organizations in each PHU. The assignment of the postal codes was based on the following criterion: the coordinates

* Patrick DeLuca of McMaster University GIS Laboratory did these computations.

of the centroid of the postal code should fall within the boundary of the PHU.

A list of licensed day care facilities and their postal addresses was also obtained from the Children's Services Division of the Ontario Ministry of Community and Social Service. These data were processed in the same manner as the data on voluntary organizations. The data on per capita municipal expenditure on environmental defence, social assistance, parks and recreation, culture and libraries, and location quotient of doctors were available by county only. They had been prepared and used in a previous study that investigated the socio-economic and environmental covariates of premature mortality in Ontario, and were supplied by the author (Jerrett 1998, personal communication). The above county level potential CVD risk factor data were used in a sensitivity analysis, which is described in Chapter Five.

3.2.3 STUDY PERIOD

The period covered by the study, 1986-1994, was divided into two parts: 1986-1989 and 1990-1994. The division of the study period into two parts would make it possible to analyse any temporal changes in the combination of variables that predicted the geographic variations in CVD mortality and morbidity. The division of the period into two parts was also done in order to investigate any impact on the geographic variation in CVD outcomes of a mandatory public health programme embarked upon by Ontario public health departments in 1989 (Ministry of Health, Ontario 1989). The goal of the programme is to increase the length and quality of life by reducing the mortality and morbidity associated with chronic diseases, injuries and substance abuse. Chronic

diseases covered by the program include heart disease, stroke, cancer, osteoporosis, diabetes, and chronic lung diseases such as emphysema. The surveillance and prevention programmes for heart disease, which formed part of the mandatory public health programmes, included the prevention of tobacco use and the promotion of physical activity and nutrition. Therefore, the year 1989 appears to be an appropriate temporal divide for analysing and comparing spatial variation in the CVD outcomes in the Province before and after the initiation of the programs.

3.2.4 LEVEL OF ANALYSIS

The observed geographic variations in cardiovascular disease outcome in Ontario (Chapter One) have been documented both by county (e.g., Heart and Stroke Foundation of Ontario 1997) and by public health unit (e.g., Jaglal et al. 1999). This research aims to understand these regional variations using the multi-group comparison ecologic study design. In this design, data on a health outcome (such as CVD mortality or morbidity) and on exposure variables (such as CVD risk factors) are obtained for each areal unit and these are analysed statistically to determine if any significant associations exist between the two sets of variables (Walter 1991a). In the multi-group comparison ecologic study design, the focus is on a group (such as the population of a county or public health unit) rather than the individual in the population. This study adopts public health units as the units of analysis because they are the main units of local level health administration and decision-making in Ontario.

Walter (1991a) describes the advantages and difficulties associated with the

ecologic method of study. The main merit of the ecologic method is that it facilitates the study of very large populations, as, for example, in studies that involve comparisons between different countries (with populations in the order of millions). This contrasts with alternative designs such as case-control methods that typically involve a few hundred or thousand respondents. The ecologic method also relies on existing databases, which implies that both exposure and outcome data are used directly without having to interview individual survey respondents. Therefore, coupled with the ability to study large populations, the use of existing data enhances cost efficiency (in terms of time and money) in epidemiological research. For example, there is no need to wait for incident cases of disease to occur, as required in a cohort study, or to wait for a large number of case series, as required in a case-control approach. The ecologic method can also be used to investigate suspicious clusters of disease in relatively small geographic areas, e.g., an elevated rate of occurrence of a water-borne disease around a locally contaminated source of water supply.

Walter (1991a) points out that the most important methodological difficulty associated with ecologic studies is ecologic fallacy, which arises from making a causal inference about an individual phenomenon based on group data. Also called ecologic bias (Greenland and Morgenstern 1989), it refers, generally, to the failure of ecologic estimates of effect to reflect the true effect at the level of the individual (Morgenstern and Thomas 1993). Walter (1991b) points out that the ecologic study design uses aggregated data and so the paired distribution of exposure and health outcome at the individual level remains unknown. Due to this, it is possible that there would be a distortion of the

association between determinants and health outcome. Some methodological steps have been suggested to reduce ecologic bias. One is to select the areal units of analysis that minimize within region exposure variation and maximize between-region variation (Morgenstern and Thomas 1989). This means that the units of analysis must be as homogeneous as possible. Morgenstern and Thomas suggest that one way of achieving homogeneity is to choose the smallest unit of analysis for which required data are available, for example, census divisions. In this respect, the adoption of public health units as the units of analysis in the present study appears to be suitable, given that for most of the province of Ontario, the county (i.e., census division) lines approximate the geographical boundaries of most public health units, the exceptions being the health units of Algoma, Muskoka-Parry Sound, North Bay and District, Porcupine, Renfrew and Timiskaming. For the most part, these discrepancies will not greatly affect the OHS results for these areas (Ministry of Health, Ontario 1990). Morgenstern and Thomas (1989) warn against using smaller units, which might increase the problem of migration between groups (also Walter 1991b). Another strategy suggested to help reduce the possibility of ecologic bias is the use of regression techniques to assess the relationship between exposure variables and health outcomes. Walter (1991b) argues that this is entirely appropriate because if the ecologic subgroups are homogeneous with respect to determinants, regression yields estimates of coefficients that are not biased. As discussed above, using smaller units of analysis enhances the homogeneity of subgroups.

Another difficulty associated with the ecologic approach is that the extent of the existing database used has a limiting effect on the analyses. Walter (1991a) notes, for

example, that disease registries may not include disease events relevant to the research question, or they may even use an inappropriate coding scheme with respect to the research question. Furthermore, a researcher might be interested in the contributory causes of death, as opposed to the underlying cause of death, but it may be difficult to obtain the relevant data from routine vital statistics. Also, it may be difficult to draw causal conclusions from ecologic analysis because of confounding.

As Morgenstern and Thomas (1989) point out, an ecologic design, despite its associated methodological limitations, might be the appropriate option at a given time. This study aims at understanding the regional variations in CVD in Ontario. The investigation is informed by the population health perspective, which emphasizes a societal level approach to addressing the health of the entire population or sub-groups rather than the health of the individual member of the population. In this respect, the practical option is to adopt an ecological study design.

3.3 OPERATIONAL DEFINITION AND DERIVATION OF VARIABLES

Given that a large proportion of the regional variation in cardiovascular outcomes in Ontario remains unexplained, this research investigates a wide range of potential determinants identified based on a review of the CVD risk factor literature (Chapter 2). These variables are categorized into the broad areas represented by the constructs in the conceptual model developed to guide this study (Figure 2.2). Tables 3.1 – 3.7 summarize how these variables were defined and/or derived for the research.

Table 3.1 Definition of CVD Risk Factors – Economic Characteristics		
Risk factor	Definition/derivation	Data source
Living in a rental unit	Persons (per 1000 population aged 12 years and over) living in a rental unit as opposed to owning their own home	1990 OHS
Dwelling needs major repairs	Persons (per 1000 population aged 12 years and over) whose dwelling requires major repairs for reasons such as sagging floors, bulging walls or damaged electrical wiring.	
Average dwelling value	Mean dollar value of dwelling units in the PHU	1991 census of Canada
Average income	Average household income in the PHU	
Income inequality	Gini coefficient, an index (ranging from 0 to 1) of income inequality. A higher coefficient indicates greater income inequality	
Unemployment rate	The unemployed labour force expressed as a percentage of the total labour force - this includes only persons 15 years of age and over, but excludes institutional residents. The rates come from the 1991 Census and are available by county only	
Incidence of low-income family	The proportion of economic families or unattached individuals (aged 15 years and over in private households) below the low-income cut-offs	

Table 3.2 Definition of CVD Risk Factors – Social Capital		
Risk factor	Definition/derivation	Data source
Voluntary organizations	The number of voluntary organizations per 1000 people who are members of voluntary associations	Customs and Revenue Canada
Charitable donations	Charitable donations per 1000 population aged 12 years and over	

Table 3.2 (continued)		
Licensed day care facilities	Number of day care facilities in the PHU per 1000 population aged 0-5 years	Ontario Ministry of Community and Social Welfare
Environmental defence expenditure per capita	Municipal expenditure per capita to defend against the adverse effects of environmental change	Jerrett (1998)
Expenditure on social assistance	Municipal expenditure per capita on social assistance (county level)	
Expenditure on recreation	Municipal expenditure per capita on recreation (county level)	
Expenditure on culture	Municipal expenditure per capita on culture and libraries (county level)	

Table 3.3 Definition of CVD Risk Factors – Demographic Characteristics		
Risk factors	Definition/derivation	Data source
Less than high school education	persons aged 18 years and over per 1000 population in the same age category that have less than high school education.	1991 Census of Canada
Marital status – unmarried	persons aged 15 years and over per 1000 population in the same age category who are not married. It includes separated, divorced, and widowed persons.	
Doctors' location quotient	Ratio of a county's share of medical doctors to that of the Province. If this ratio is greater than 1.0, it means the particular county has more than a proportionate share of doctors, if less than 1.0, the county has less than a proportionate share of medical doctors, implying diminished access to health care. A ratio of 1.0 means the county has a proportionate share of doctors	Jerrett (1998)

Table 3.4 Definition of CVD Risk Factors – Psychosocial Health and Well-being		
Risk factors	Definition/derivation	Data source
Experiencing stressful life	Persons aged 12 years and over (per 1000 population of the same age category) who reported experiencing stressful life. (combined affirmative responses to the question, "as a whole, would you describe your life as 'very stressful' or 'fairly stressful'?")	1990 OHS
Dissatisfied with social life	Persons aged 12 years and over (per 1000 population of the same age category) not satisfied with their social life. (The responses, 'somewhat unsatisfied' and 'very unsatisfied', to the question "how satisfied are you with your social life", were combined to derive this variable.)	
Unhappiness in life	Persons aged 12 years and over (per 1000 population of the same age category) who reported usually feeling unhappy in life. (This variable was derived by combining the responses, 'somewhat unhappy', 'unhappy with little interest in life', and 'so unhappy that life is not worthwhile'.)	
Experiencing poor health	Persons aged 12 years and over (per 1000 population of the same age category) who rate their health as poor compared to the health of other persons their own age	
Dissatisfied with health	Persons aged 12 years and over (per 1000 population of the same age category) not satisfied with their health. (Combined responses: 'not too satisfied' and 'not at all satisfied' to the question, "how satisfied are you with your health")	
Low state of well- being	Persons aged 12 years and over (per 1000 population of the same age category) whose state of well-being is low. (This variable is a combination of two categories of Well-Being Scale scores from 1 to 4. The Well-Being Scale assessed seven indicators: energy, control of emotions, state of morale, interest in life, perceived stress, perceived health status, and satisfaction about relationships. In the OHS Regional Reports, categories 1 and 2 are combined to indicate a "low" state of well-being). (see Ontario Ministry of Health, 1990:26-27)	
Physical activity limitation	Persons aged 12 years and over (per 1000 population of the same age category) who, compared to other people of the same age in good health, are limited in the kind or amount of activity they can do because of a long-term physical or mental condition or health problem.	

Table 3.5 Definition of CVD Risk Factor Behaviours		
Risk factors	Definition/derivation	Data source
Physical inactivity	Persons aged 12 years and over (per 1000 population of the same age category) whose physical activity index is less than 1.5 (i.e., physical exercise results in an energy expenditure below 1.5 kcal/kg/day). An average of 3.0+ kcal/kg/day of energy expenditure is approximately the amount of exercise that is required for cardiovascular health benefit. Those averaging 1.5-2.9 kcal/kg/day of energy expenditure might experience some health benefits but probably little cardiovascular benefit (Ontario Ministry of Health, 1990:31). Energy expenditure (EE) values were calculated as: EE = sum of (N x D/60 minutes per hour x METS/30 days per month), where N = the number of time of activity, D = the average duration in minutes of that activity, and METS = the energy cost of the activity expressed as kilocalories expended per kilogram of body weight per hour of activity (Ontario Ministry of Health, 1990).	1990 OHS
Smoking	Persons aged 12 years and over (per 1000 population in the same age category) who currently smoke daily	
Excessive alcohol consumption	Drinking in excess of 14 drinks per seven-day week for men, or drinking in excess of 9 drinks per seven-day week for women	
Excess fat in diet	Persons whose total caloric intake from dietary fat is greater than 30 per cent	

Table 3.6 Definition of CVD Risk Factors – Social Support		
Risk factors	Definition/derivation	Data source
Low social participation (ages 16-59 years)	Participation in a social support system by persons aged 16-59 years. The social support index examines the number of friends and relatives the respondent felt close to, the amount of leisure time spent alone versus with others, satisfaction with social life, and the availability of a confidant and a helper. A higher index score indicates a higher degree of participation in a social support system.	1990 OHS

Table 3.6 (continued)		
Low social participation (seniors aged 60 and over)	The index incorporates the number of close relatives and friends, the amount of leisure time spent alone, satisfaction with social life, and membership in voluntary associations. A higher score indicates more active social participation.	1990 OHS
No help in time of need	Person (per 1000 population aged 12 years and over) who answered "NO" to the question, "among your friends or in your family, is there someone who can help you in a time of need?"	
Dysfunctional family	Persons (per 1000 population aged 12 years and over) who had a score >= 2.17 (range: 1.00 - 4.00) on the General Functioning scale (a subset of the McMaster Family Assessment Device [Byles et al, 1988:97-104]) which measured the overall health or pathology of the family on the OHS. Scores less than 2.17 indicate a healthy family while scores >= 2.17 indicate a dysfunctional family	
No one to confide in	Persons (per 1000 population aged 12 years and over) who answered "NO" to the question "among your friends or in your family, is there someone you confide in or talk to freely about your problems?"	
Seniors living alone	Non-family persons aged 65 years and over living alone per 1000 population of persons in the same age group	
Membership in associations	Persons (per 1000 population aged 12 years and over) who answered "YES" to the question "are you a member of any voluntary organizations or associations, such as church and school groups, labour unions, or social, civic and fraternal clubs".	

Table 3.7 Definition of CVD Risk Factors – Physiological Characteristics		
Risk factors	Definition/derivation	Data source
Hypertension	persons aged 12 years and over (per 1000 population of the same age category) who answered "YES" to the question, "do you have hypertension?"	1990 OHS
Diabetes	persons aged 12 years and over (per 1000 population of the same age category) who answered "YES" to the question, "do you have diabetes?"	
Obesity	persons aged 20-64 years per 1000 population in the same age category whose body mass index (BMI) is greater than 27.0. A body mass index greater than 27.0 is associated with an increased risk of developing health problems, particularly hypertension, hyperlipidemia and coronary heart disease (see Ontario Ministry of Health, 1990:33). Only persons aged 20-64 years, and females in this age category who are not pregnant are included as BMI is not a suitable measure of obesity for infants, children, adolescents, pregnant women or adults aged 65 years and over (Ostbye et al, 1995).	

3.4 ANALYTICAL METHODS

Data from various sources were aggregated at the public health unit level to address the objectives of this study. The first objective of the research was to examine the temporal and spatial variation in CVD mortality and morbidity in Ontario. Using the outcome data obtained from CIHI (see above), comparative mortality and morbidity figures (CMF) were computed for each public health unit, for both sexes combined, and for females and males separately. This was done for two time periods - 1986-89 and 1990-94. The comparative mortality (or morbidity) figure is a summary of the incidence rate ratios between an observed population (such as the population of a public health unit)

and a standard population (such as the population of Ontario). The CMF was obtained by dividing the directly standardized rate for the observed population by the rate for the standard population*.

To illustrate the spatial variation in the outcome rates, choropleth maps of the CMFs were created using the ArcView Version 3.2 software. Spatial autocorrelation tests were performed on the outcome data in order to ascertain if there was spatial clustering. Spatial autocorrelation, if significant, indicates that a variable exhibits a regular pattern in space such that its value at a location depends on, and is similar to, values of the same variable in neighbouring locations. Spatial autocorrelation can be measured in different ways, depending on the type of data. If the data are on a nominal scale, then a join-count statistic – incorporating a binary distribution of the Rook's case, Bishop's case, or Queen's case – is appropriate (Robinson 1998). If, however, the data being analysed are on an interval or ratio scale (as in this study), then the Moran's *I* or Geary's *c* statistic can be used. In this study, the more commonly used global Moran's *I* statistic is chosen over Geary's *c* statistic because its interpretation is simpler. Moran's *I* is based on the covariation of juxtaposed map values, and it operates in a similar fashion as the Pearson's product moment correlation coefficient.

Moran's *I* statistic is estimated as:

$$I = \frac{n\sum_{i=1}^{n}\sum_{j=1}^{n} w_{ij}\left(y_i-\bar{y}\right)\left(y_j-\bar{y}\right)}{\left(\sum_{i=1}^{n}\left(y_i-\bar{y}\right)^2\right)\left(\sum_{i\neq j}\sum w_{ij}\right)}.$$

* Patrick DeLuca of McMaster University GIS Laboratory did these computations.

In the above equation, the coefficients w_{ij} represent the geographic contiguity of areas (i.e., public health units) i and j such that $w_{ij} = 1$ if areas i and j share a common boundary, and $w_{ij} = 0$ otherwise, and y_i represents attribute values (in this case, the CMFs). The coefficient, w_{ij}, is a generalized weight that represents the hypothesized influence of areal unit i on areal unit j (Robinson 1998). This study used the approximate sampling distribution approach (see Bailey and Gatrell 1995) to test the significance of the Moran's I statistic. This method is based on the assumption that if y_i are observations on random variables Y_i whose distribution is normal, and if Y_i and Y_j are spatially independent when $i \neq j$, then Moran's I has a sampling distribution which is approximately normal. The theoretical mean (or expected value), $E(I)$, of this distribution is defined as $E(I) = -\frac{1}{(n-1)}$, where n is the number of observations, and the theoretical standard deviation is obtained by computing the square root of its variance. The variance of I is defined as

$$VAR(I) = \frac{n^2(n-1)S_1 - n(n-1)S_2 - 2(n-2)S_0^2}{(n+1)(n-1)^2 S_0^2},$$

where $$S_0 = \sum_{i \neq j} \sum w_{ij},$$

$$S_1 = \frac{1}{2} \sum_{i \neq j} \sum \left(w_{ij} + w_{ji}\right)^2, \text{ and}$$

$$S_2 = \sum_k \left(\sum_j w_{kj} + \sum_i w_{ik} \right)^2.$$

From the above, z-scores can be computed for the *I* values and their significance can be judged by means of a standard normal table. The z-score is obtained by subtracting the expected value of *I* from the statistic and then dividing the result by its theoretical standard deviation. The critical z-score used to determine significance is 1.96, based on a two-tailed significance level of $p = 0.05$.

The estimation of the Moran's *I* statistic makes use of a row-standardized weight matrix based on Rook's case adjacency, which defines polygons that share a common boundary as neighbours. Tiefelsdorf et al. (1999) discuss three types of neighbour weight coding schemes. The C-coding scheme de-emphasizes the leverage of spatial entities, such as the interior sub-regions of a study area, which have a relatively large number of neighbours. In Ontario, an example of this is Wellington-Dufferin-Guelph public health unit, which shares a boundary with eight other public health units. W-coding, on the other hand, emphasizes the leverage of spatial units that are located on the periphery of study regions, such that they have few neighbours. For example, Northwestern, Windsor-Essex, Niagara, and Eastern Ontario have up to only two neighbours each. The third type of coding scheme, S-coding, is designed to address topology-induced heterogeneity (Tiefelsdorf et al. 1999). This coding scheme addresses the local variances of the spatial units, which depend on the local linkage degree. This study adopts the W-coding scheme to take into account the worse case scenario where the limited interaction between peripheral public health units and their (usually fewer) first order neighbours would have been discounted.

Anselin (1992) suggests that in computing spatial statistics, the spatial weight matrix should be row-standardized so as to yield a meaningful interpretation of the results. In order to row-standardize, each element in a row is divided by the corresponding row sum, such that each element, w_{ij}, becomes

$$w_{ij} \Big/ \sum_j w_{ij},$$

where i represents the rows and j represents the columns.

Further statistical analyses are needed in order to determine whether statistically significant clusters existed that represented areas of elevated rates of CVD outcome (i.e., whether or not there were hot spots). The statistic of choice is referred to as local indicators of spatial association (LISA). The LISA indicator allows for the decomposition of the more traditional global indicators of spatial association – the global Moran's I – into the contribution of each individual observation (i.e., the CMF). Anselin (1995) suggests that as an operational definition, the LISA statistic satisfies two requirements: 1) the LISA for each observation indicates the degree of significant spatial clustering of similar values around that observation, and, 2) the sum of LISAs for all observations is proportional to a global indicator of spatial association. Thus, LISA statistics can be interpreted as indicators of local pockets of non-stationarity, or hot spots. Anselin (1995) defines the local I_i statistic as:

$$I_i = \left(z_i / s^2\right) \sum_j w_{ij} z_j, \; j \neq i,$$

where the z_i and z_j are deviations, $\left(x_i - \bar{x}\right)$ and $\left(x_j - \bar{x}\right)$, from the mean. The summation is over just those j values within a distance d of i, and $w_{ij} = 0$. The spatial weight matrix w_{ij}

is in row-standardized form. In order to assess the statistical significance of the local *I* statistic, z-scores were computed for each observation by subtracting the expected value and then dividing the result by the theoretical standard deviation. Thus:

$$Z(I_i) = (I_i - E[I_i]) / \sqrt{Var[I_i]}.$$

The expected value of the local statistic is defined as:

$$E[I_i] = \frac{-\sum_j w_{ij}}{(n-1)}.$$

The theoretical standard deviation can be obtained by computing the square root of the variance of local Moran's *I*. The variance is defined as follows:

$$Var[I_i] = \frac{w_{i(2)}(n-b_2)}{(n-1)} + \frac{2w_{i(kh)}(2b_2-n)}{(n-1)(n-2)} + \frac{w_i^2}{(n-1)^2}.$$

In the above equation,

$b_2 = \frac{m_4}{m_2^2}$, i.e., the fourth moment around the mean divided by the square of the second moment around the mean,

$$m_4 = \frac{\sum_i z_i^4}{n},$$

$$w_{i(2)} = \sum_j w_{ij}^2, j \neq i, \text{ and}$$

$$2w_{i(kh)} = \sum_k \sum_h w_{ik} w_{ih}, k \neq i \text{ and } h \neq i.$$

The term $2w_{i(kh)}$ is twice the sum of the cross products for all weights for i with themselves, using k and h to distinguish between the subscripts. Since each pair of

observations, i and j, has its own specific weight, a cross product of weights are two weights multiplied by each other (where $i \neq j$), and the sum of these cross products is twice the sum of all possible interactions (Levine 1999). In the case of the LISA analyses, too, the critical z-score used to determine significance is 1.96. The results of the hot spots analyses are described in Chapter four.

The second objective was to examine the prevalence of CVD risk factors. The risk factor data were used to derive the prevalence rates of the risk factors in the public health units. The risk factor data were directly age-standardized to the mid-1990 Ontario population in order to adjust for differences over time in the population composition of the PHUs. The prevalence of the risk factors will be described with respect to sex, age, education, type of public health unit (i.e., urban or rural), and the relative location of the public health unit in Ontario (i.e., northern or southern Ontario). For the purpose of this analysis, age is categorized into five groups. These are 12-19 years to represent teenage and adolescent years, 20-44 years to represent young adulthood, 45-64 years for middle ages, 65-74 years for "young" seniors, and 75 years and over to represent older seniors. These cut-points conform to the age group cut-points used in the Statistics Canada 1991 census. Level of education was dichotomized as follows: less than high school education, and at least high school education. In order to examine the spatial variation in the prevalence of risk factors, the public health units were classified as urban or rural, and by relative location in Ontario. If a public health unit had a census metropolitan area (CMA) – as defined in the 1991 census – located either wholly or partly within its boundary, it was classified as urban. Based on this definition, the City of Toronto,

Durham, Halton, Hamilton-Wentworth, Middlesex-London, Niagara, Ottawa-Carlton, Peel, Waterloo, Windsor-Essex, and York Region were classified as urban public health units. The remaining public health units (Figure 3.2) were classified as rural. In defining the relative location of the public health unit in Ontario, reference was made to the grouping of public health units (by the Ontario Ministry of Health and Long Term Care) into health intelligence units in five health planning regions. These are southwest, central west, central east, east, and north. Southern Ontario was defined as comprising the public health units in the first four planning regions, while northern Ontario consists of the public health units in the last planning region. The boundary between southern and northern Ontario coincides approximately with Latitude 46° N (Figure 3.2).

The first step in modelling the geographic variation in CVD mortality and morbidity (i.e., objective 3) was to examine the relationship (i.e., bivariate correlation) between each outcome and each of the variables identified in the literature (Section 3.3). This concluded the process of selecting potential covariates to be included in the multivariate regression models. Variables were selected for each of the constructs in the conceptual model (Figure 2.2) developed for this study. The selection was based on the following criteria: (1) the association between the potential risk factor and the particular CVD outcome bears the expected sign, i.e., a positive sign for an expected direct relationship and a negative sign for an expected inverse relationship; and (2) the significance level (i.e., *p*-value) of the relationship is not greater than 0.2. The p = 0.2 cut point is adopted in this research to allow for the retention in the models of important covariates, which could otherwise be excluded if the conventional p-value of 0.05 were

used (Mickey and Greenland 1989). This research follows the example of Walter et al. (1994) who used a less conservative significance level ($p \leq 0.1$) in their study of geographic variations in cancer incidence in Ontario. Although an even less conservative cut-point at $p = 0.2$ is used in this study, Walter (personal communication) supports this level in exploratory analyses.

3.5 SUMMARY

This chapter has described the types and sources of data used in the study. The data come from five main sources namely, the 1990 Ontario Health Survey, the 1991 census of Canada, the Child Care Services Division of the Ontario Ministry of Community and Social Welfare, Customs and Revenue Canada, and data compiled from the Ontario Ministry of Municipal Affairs' Municipal Financial Information Returns and used in a study of the determinants of municipal environmental defense expenditures in Ontario (Jerrett 1999).

It has also described/defined the variables that are explored as potential CVD risk factors to be included in the modeling of regional variations in CVD outcomes. These are categorized into seven CVD risk constructs (Figure 2.2). The descriptions/definitions of the potential risk factors are presented in Tables 3.1 – 3.7, along with the data source for each risk factor.

This chapter also described the analytical methods adopted for the study. Rates were calculated for risk factors and used to assess their prevalence at the individual level within each PHU. Choropleth maps are used to illustrate spatial variations in the CMFs.

The spatial autocorrelation statistic (global Moran's *I*) is used to ascertain the extent of spatial clustering in the outcome data, while the local indicator of spatial association (LISA, i.e., local Moran's *I*) is used to determine if statistically significant local clusters exist that constitute hot spots of CVD outcome. The criteria for selecting potential covariates for modeling the geographic variation in CVD mortality and morbidity are described in this chapter. To model the spatial variations, a forward, stepwise multivariate regression technique is adopted. The regression models also require a significance level of $p \leq 0.2$ for a covariate to be retained.

The next chapter, Chapter Four, describes the spatial and temporal variations in the outcome variables, as well as the prevalence of the CVD risk factors identified from the literature. Chapter Four also contains a description of the bivariate relationships between the CVD outcomes and risk factors. The results of the standard multivariate regression of the geographic variations in cardiovascular disease mortality and morbidity on risk factors are presented in Chapter Five.

CHAPTER FOUR

VARIATIONS IN CARDIOVASCULAR DISEASE OUTCOME AND RISK FACTORS IN ONTARIO

4.1 INTRODUCTION

This chapter addresses the first two objectives of the research, that is, to describe the spatial and temporal variations in CVD mortality and morbidity, and to examine the prevalence of a broad range of potential CVD risk factors in Ontario. The data and methods used, including the criteria for selecting the potential determinants of geographic variation in CVD mortality and morbidity in Ontario, were described in the previous chapter. Section 4.2 describes the spatial and temporal variations in CVD mortality and morbidity in Ontario during the two time periods considered – 1986-1989 and 1990-1994 (see Chapter 3). These analyses were done for both sexes combined, and separately for females and males. Section 4.3 contains a description of spatial autocorrelation analyses done to determine if significant spatial clustering of the CVD outcome rates occurred in the study area during the time period studied. Section 4.4 describes the results of a GIS analysis done (using GIS and S-PLUS spatial analysis tools) to identify local clusters (or 'hot spots') of CVD mortality and morbidity. The analyses of 'hot spots' were also done for both sexes combined, and separately for females and males. Section 4.5 explores the prevalence of the potential risk factors of CVD in Ontario and the spatial variations in these rates. The rates of the risk

factors were analyzed with respect to sex, age, education, type of public health unit (i.e., urban or rural), and the relative location of the public health unit in Ontario (i.e., northern or southern Ontario). Section 4.5 also describes the relationship (i.e., bivariate correlation) between the risk factors and the various CVD outcomes. The results of the bivariate correlation analyses (Appendices 4.33 – 4.74) show the variables subsequently included in the multivariate models (Chapter 5) based on the selection criteria described in Chapter 3 (Section 3.3). The summary and discussion of the results are presented in Section 4.6.

4.2 SPATIAL AND TEMPORAL VARIATIONS IN CARDIOVASCULAR DISEASE OUTCOME

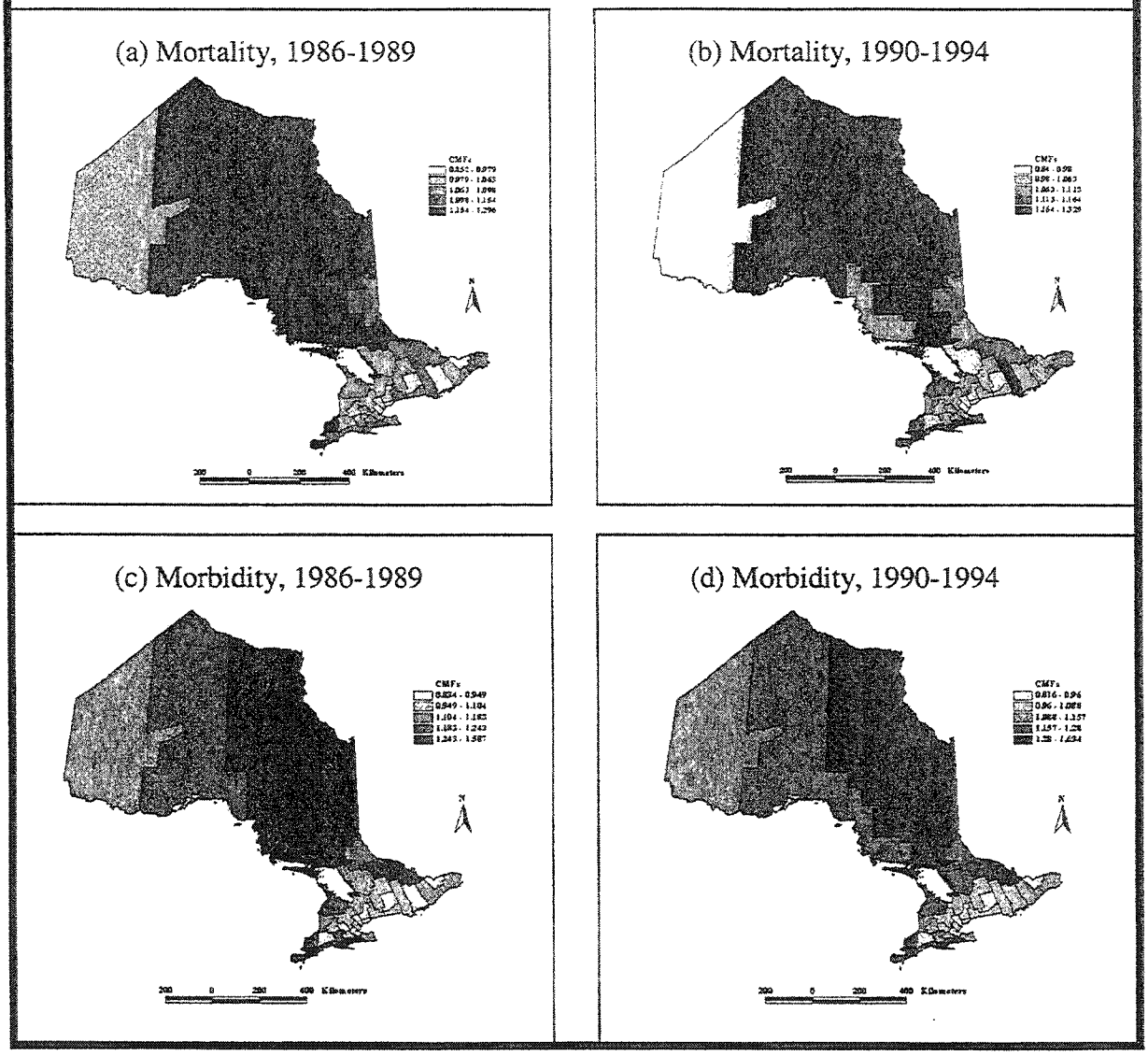
There were significant geographic and temporal variations in CVD outcomes (i.e., in the CMFs) across public health units in the Province. These are described in this section for both sexes combined and separately for females and males.

4.2.1 COMBINED FEMALES AND MALES

Figures 4.1(a) and 4.1(b) show the spatial patterns of combined female and male CVD mortality during the first and second time periods respectively. Overall, the spatial pattern of the CMFs varied little between the two periods. The figures show that the public health units that had the highest rates (i.e., CMFs) of CVD mortality during both periods were mainly located in northern Ontario, but some public health units in southern Ontario, such as Windsor-Essex, Lambton, Kent-Chatham, Haldimand-Norfolk, Hastings-Prince Edward, and Eastern Ontario also had high rates of CVD mortality. In the case of combined

female and male CVD morbidity, too, the public health units that had the highest CMFs during the two periods were mainly in northern Ontario (Figures 4.1(c) and 4.1(d)). These

Figure 4.1 Combined Female and Male CVD in Ontario



suggest that during both periods, the CVD mortality and morbidity impacts of the associated risk factors were generally greater in northern Ontario than in southern Ontario. There was minimal variation in the spatial pattern of the rates depicted when Figures 4.1(a) and 4.1(b)

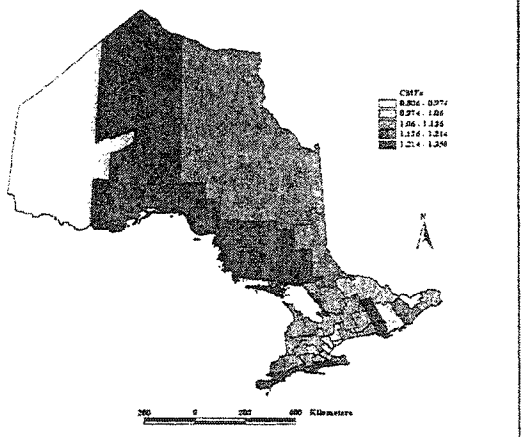
are compared. Similarly, there is little difference in the spatial patterns depicted in Figures 4.1(c) and 4.1(d). In a report produced for the Heart and Stroke Foundation of Ontario on the temporal variation in CVD outcome rates for Ontario counties (which, to a large extent, have the same boundaries as the public health units) during the same period covered by this study (Elliott 2003), only the Toronto Metropolitan Municipality was found to experience a significant change in the rates of CVD outcome over time. Figure 4.1(c) appears to depict a spatial gradient in the CMFs for combined female and male CVD morbidity across northern Ontario such that the CMFs increased eastwards. This suggests that the morbidity impact of the associated risk factors was elevated in the eastern part of northern Ontario. The gradient remained largely unaltered during the second period (Figure 4.1(d)), which suggests that the underlying processes persisted over time.

Figures 4.2(a) and 4.2(b) show the spatial pattern of combined female and male ischemic heart disease mortality during the first and second periods respectively. There was little variation in this pattern over time, and, unlike the CVD outcomes, there was a more even geographic distribution of high and low CMFs. In the case of combined female and male IHD morbidity, however, the higher rates tended to be concentrated in northern Ontario (Figures 4.2(c) and 4.2(d)). Within southern Ontario, higher rates of combined female and male IHD morbidity occurred in the eastern and western part, with lower rates occurring in the central part of this region.

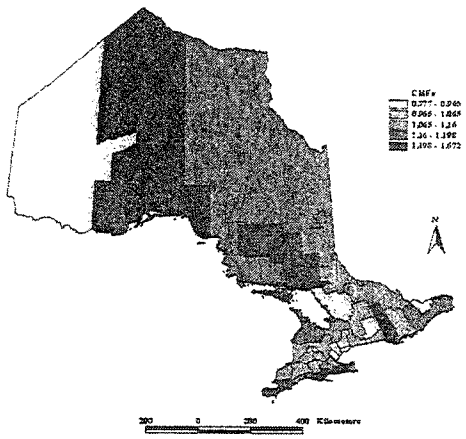
The spatial pattern of combined female and male cerebrovascular disease mortality differs from that of CVD and IHD. During both periods, there was a juxtaposition of high and low CMFs in the Province (Figures 4.3(a) and 4.3(b)).

Figure 4.2 Combined Female and Male IHD in Ontario

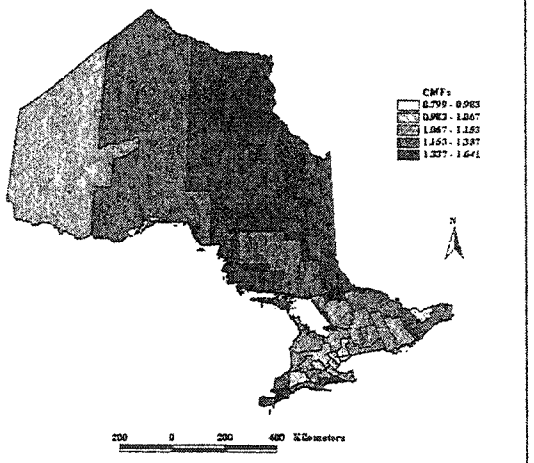
(a) Mortality, 1986-1989



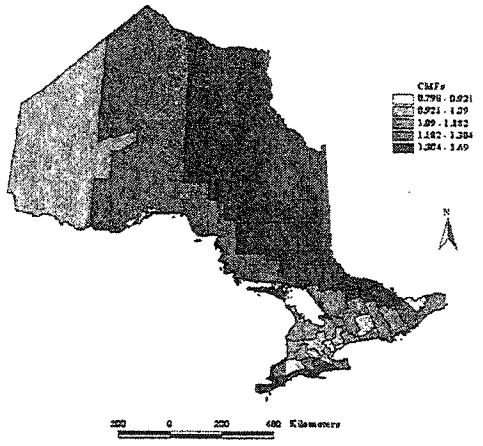
(b) Mortality, 1990-1994



(c) Morbidity, 1986-1989

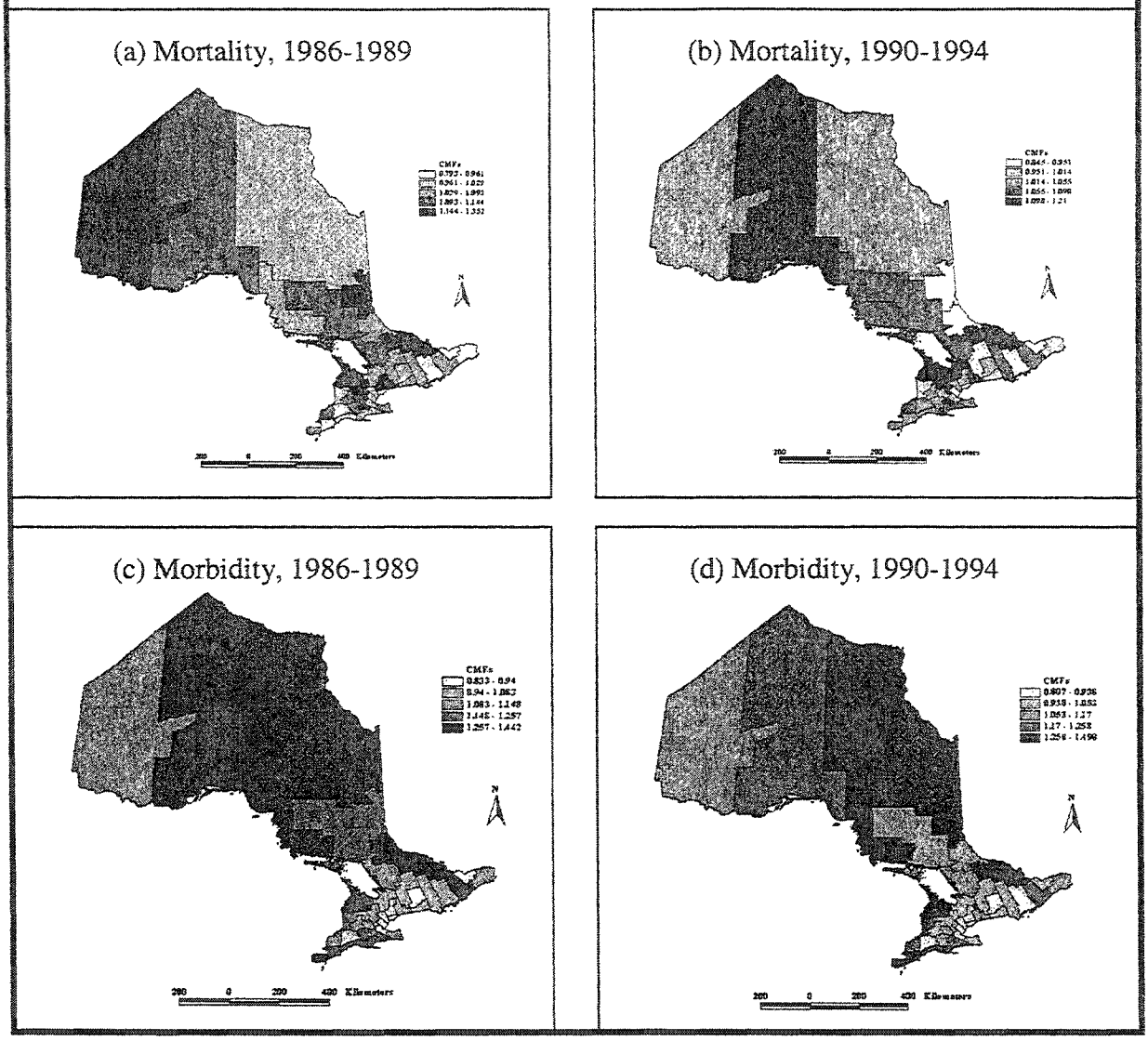


(d) Morbidity, 1990-1994



There was a similar juxtaposition of the CMFs for combined female and male CBVD morbidity during both periods (Figures 4.3(c) and 4.3(d)). Generally, however, the public health units in southern Ontario tended to experience lower rates of CBVD for both sexes combined.

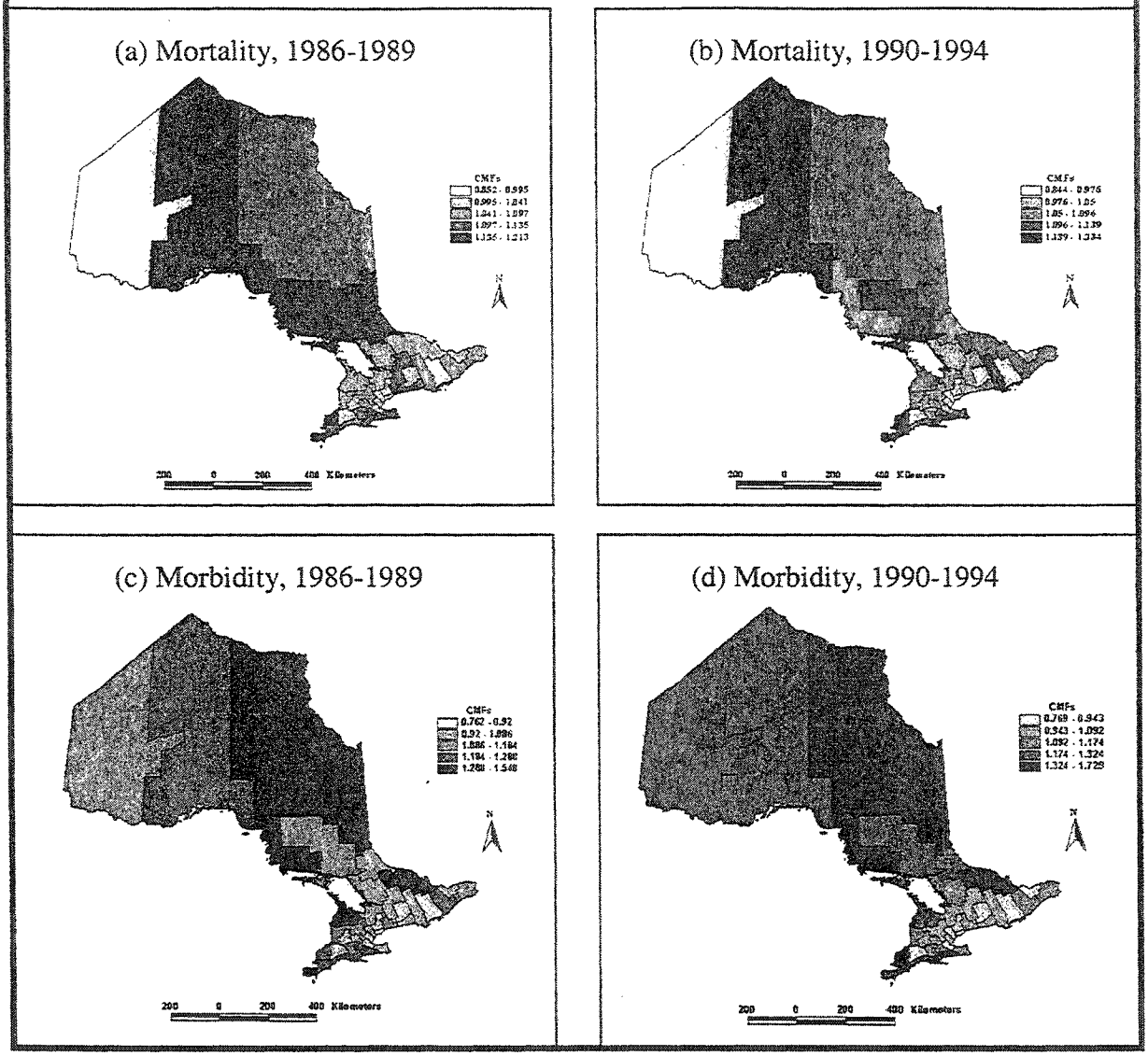
Figure 4.3 Combined Female and Male CBVD in Ontario



4.2.2 FEMALES

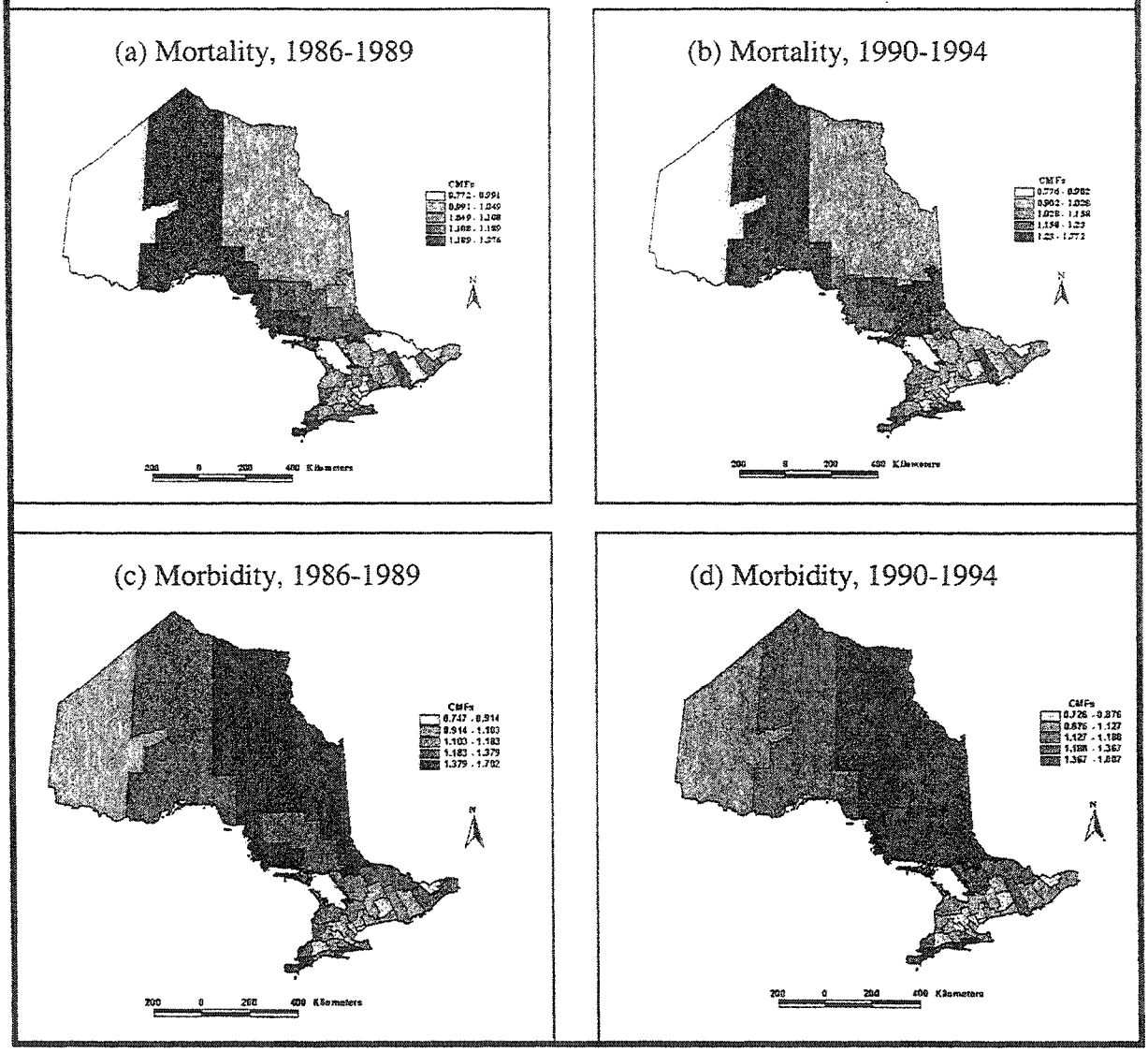
The spatial patterns of the CMFs for female CVD, IHD, and CBVD mortality and morbidity during the two time periods are shown in Figures 4.4(a) – 4.6(d). There were groups of contiguous PHUs with high as well as low rates of CVD mortality and morbidity in both northern and southern Ontario during the two time periods (Figures 4.4(a) – 4.4(d)).

Figure 4.4 Female CVD in Ontario



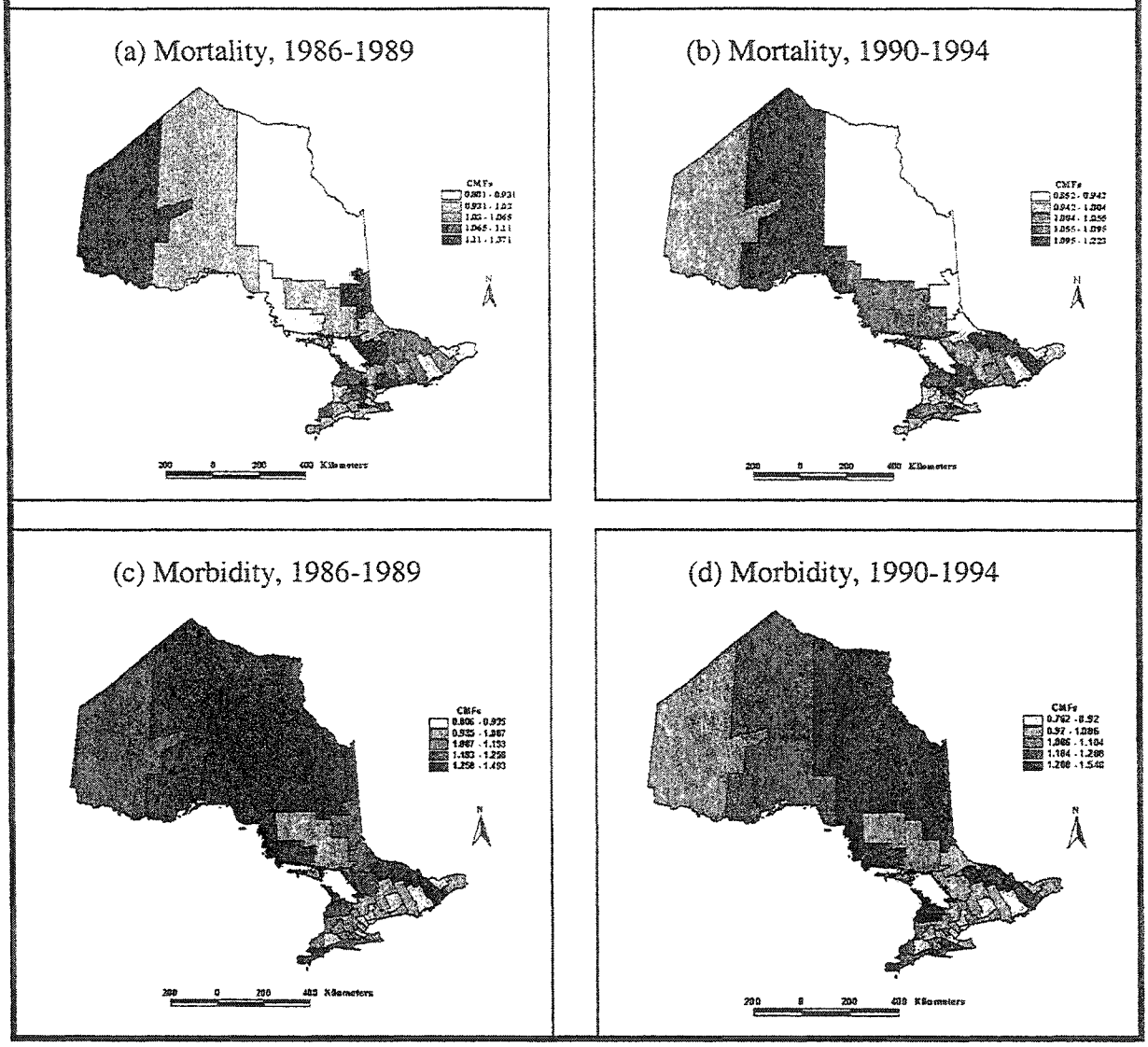
second period. For example, the spatial pattern of the CMFs for ischemic heart disease morbidity during the period 1986-1989 (Figure 4.5(c)) is similar to the pattern for the second period (Figure 4.5(d)). In the case of female CBVD mortality, particularly in the first time period, the higher rates occurred mainly in southern Ontario. In northern Ontario, Northwestern and Timiskaming (Figure 4.6(a)), and Thunder Bay, Algoma, and Sudbury

Figure 4.5 Female IHD in Ontario



District (Figure 4.6(b)) also had high rates. For female CBVD morbidity, however, there were groups of contiguous PHUs with high CMFs interspersed with similar groups of PHUs with low CMFs.

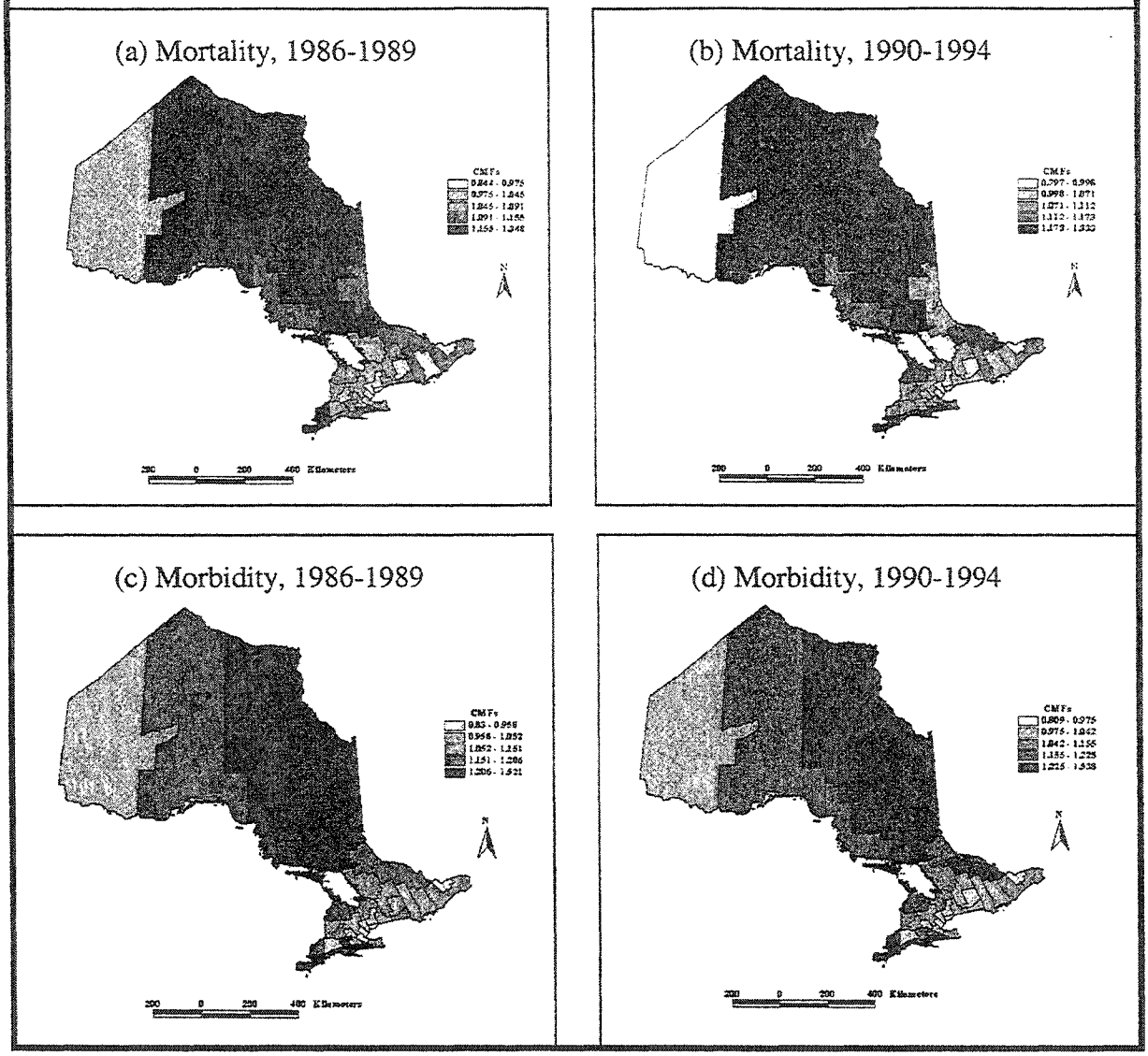
Figure 4.6 Female CBVD in Ontario



4.2.3 MALES

Figures 4.7(a) – 4.9(b) depict the spatial patterns in the male CVD outcomes during the time period studied. All the male outcomes exhibit what appear to be clusters of high rates interspersed with clusters of low rates. A striking feature of the male outcome rates, too, is the similarity in the spatial pattern of the outcome rates from the first time period to

Figure 4.7 Male CVD in Ontario

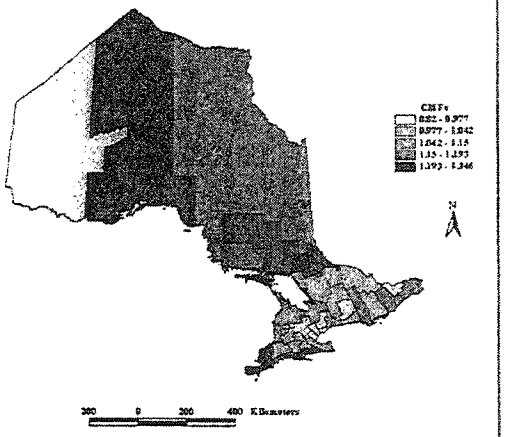


the second period. Some public health units appeared to have consistently high rates of most of the outcomes. For example, Windsor-Essex, Kent-*Chatham*, Lambton, Elgin-St. Thomas, and Haldimand-Norfolk had mostly high rates of CVD and IHD mortality and morbidity during the two time periods (Figures 4.7(a) – 4.8(d)). With the exception of male CBVD mortality (1990-1994) and CBVD morbidity (1990-1994), Sudbury District had high rates of

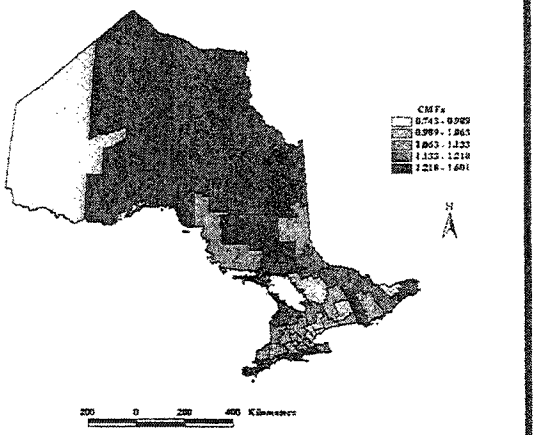
all the cardiovascular disease outcomes. Other PHUs, e.g., Ottawa-Carlton, Peterborough, City of Toronto, Peel, and Halton had consistently low rates of all the outcomes, while Northwestern and Kingston-Frontenac-Lennox & Addington had low rates of most of the outcomes.

Figure 4.8 Male IHD in Ontario

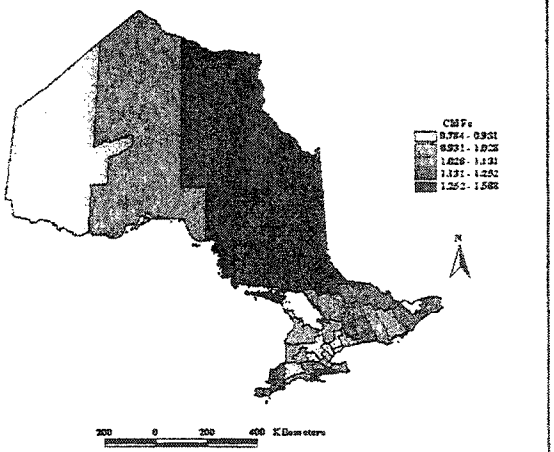
(a) Mortality, 1986-1989



(b) Mortality, 1990-1994



(c) Morbidity, 1986-1989



(d) Morbidity, 1990-1994

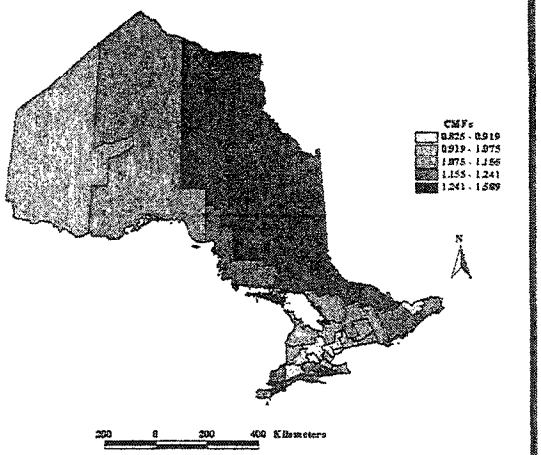
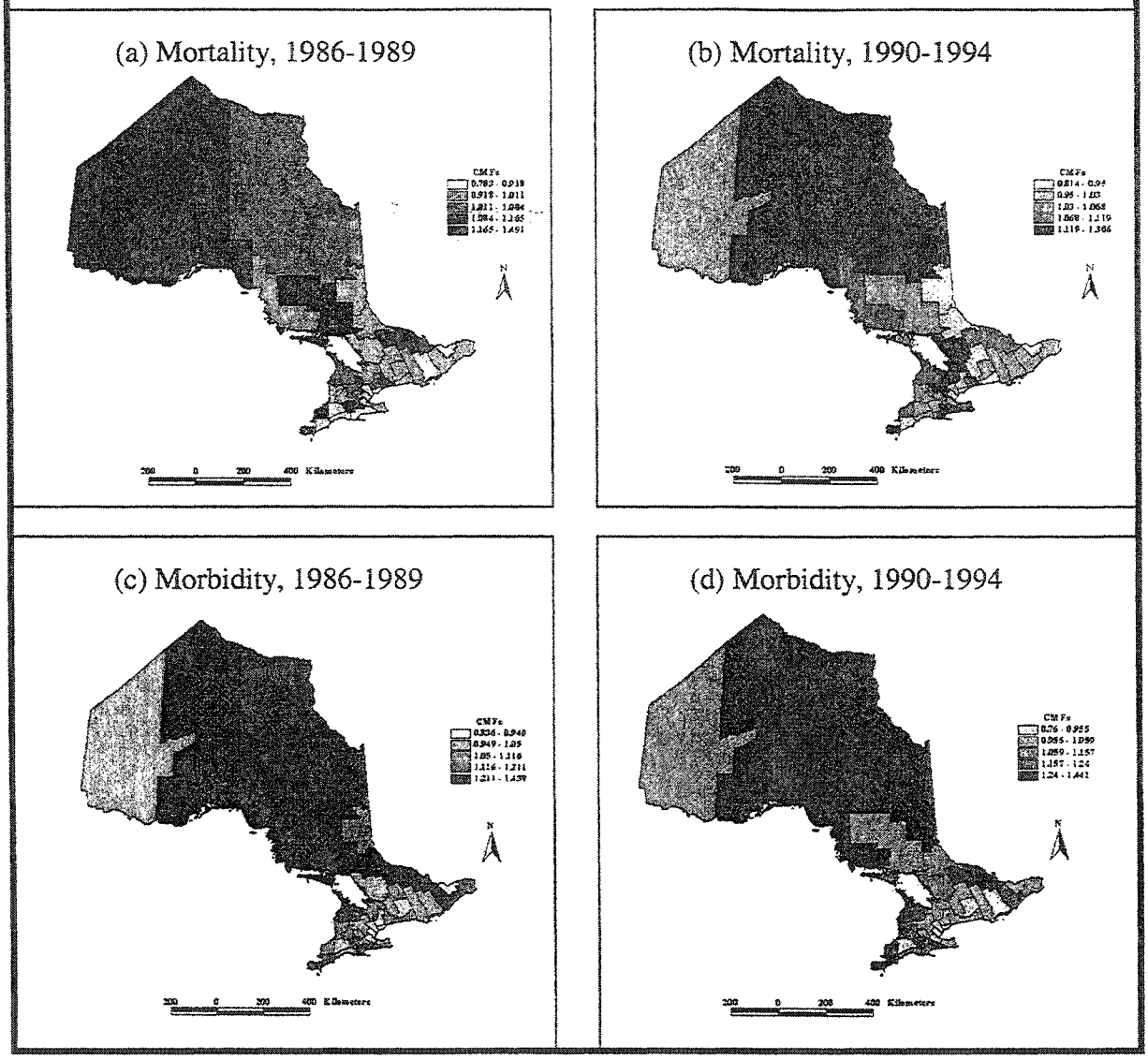


Figure 4.9 Male CBVD in Ontario



4.2.4 SUMMARY

The spatial patterns in the outcome variables described above indicate that there were geographic variations in all the CVD outcomes. This is indicated by the checkerboard arrangement of high and low outcome rates throughout the Province. However, some public

health units or groups of public health units tended to have consistently high or low rates of certain CVD outcomes. Overall, the spatial patterns described indicate that while high rates of CVD outcome tended to be concentrated in northern Ontario and in the western and eastern part of southern Ontario, low outcome rates tended to be concentrated in the central part of southern Ontario. There was minimal change in the rates between the two time periods, which indicates that the underlying processes for these outcomes persisted over time in the Province. The next section describes spatial autocorrelation analyses undertaken to determine if significant spatial clustering of the CVD outcome rates occurred in the Province during the time period studied.

4.3 SPATIAL AUTOCORRELATION ANALYSIS OF CVD RATES

The above descriptions of the outcome variables show that northern Ontario appeared to have clusters of higher CMFs while southern Ontario mostly had clusters of lower CMFs. Spatial autocorrelation (SAC) tests were performed on the outcome data in order to ascertain if clustering actually occurred. Spatial autocorrelation, if significant, is an indication that an observed phenomenon exhibits a regular spatial pattern such that its value at a location depends on, and is similar to, values of the same phenomenon at neighbouring locations or in neighbouring areas. The results of the SAC tests are presented in Tables 4.1 and 4.2. In Tables 4.1 and 4.2, a z-value of 1.96 or greater indicates that there was significant spatial clustering of the corresponding CVD outcome rates during the time period studied. Clustering of rates suggests spatial aggregation of the associated risk factors. It also suggests that the health impacts of those risk factors are spatially concentrated.

Table 4.1 Spatial autocorrelation (SAC) of CVD mortality rates in the PHUs of Ontario: Both sexes combined, Females, and Males

Outcome	Year	Sex	SAC value (Moran's *I*)	Z-value
CVD	1986-1989	Both	0.3765	3.75632
		Female	0.2658	2.72791
		Male	0.4060	4.03063
	1990-1994	Both	0.3103	3.14139
		Female	0.2305	2.39990
		Male	0.3525	3.53351
IHD	1986-1989	Both	0.3100	3.13861
		Female	0.1444	1.59987
		Male	0.4069	4.03899
	1990-1994	Both	0.2599	2.67308
		Female	0.2150	2.25588
		Male	0.2997	3.04290
CBVD	1986-1989	Both	-0.0342	-0.05965
		Female	-0.0724	-0.41460
		Male	-0.0090	0.17450
	1990-1994	Both	-0.1152	-0.81229
		Female	-0.1494	-1.13008
		Male	0.0503	0.72551

4.3.1 MORTALITY

Although the SAC of the CVD mortality rates was generally moderate during the first and second time periods (i.e., 0.3765 and 0.3103 respectively for both sexes combined [Table 4.1]), it was lower for females (1986-1989: 0.2658 and 1990-1994: 0.2305) than for males

(1986-1989: 0.4060 and 1990-1994: 0.3525). Therefore, the degree of spatial aggregation of the associated risk factors for male CVD mortality was greater than that of females.

Similar to CVD, the SAC in the IHD mortality was lower for females (1986-1989: 0.1444, and 1990-1994: 0.2150) than for males (1986-1989: 0.4069, and 1990-1994: 0.2997), which suggests greater spatial aggregation of the risk factors for male IHD mortality than those for female IHD mortality. The SAC value for female ischemic heart disease mortality during the first time period was not significant. This indicates that the associated risk factors for female IHD mortality became more spatially concentrated over time. For males, however, the reduction in the SAC from 0.4069 during the period 1986-1989 to 0.2997during the period 1990-1994 implied that the associated risk factors for male IHD mortality became less spatially concentrated over time. The data in Table 4.1 show that none of the SAC values for CBVD mortality was significant during both time periods considered.

4.3.2 MORBIDITY

The SAC values for CVD, IHD, and CBVD morbidity were all significant (Table 4.2). The SAC values for the CVD morbidity rates for both sexes combined during the first and second time periods were high – 0.4584 and 0.4488 respectively (Table 4.2). This indicates strong spatial aggregation of the associated risk factors. There was a minimal drop of 0.01 in the SAC value from the first time period to the second, which suggests that the degree of spatial aggregation of the associated risk factors remained more or less unchanged over time. During both time periods, the SAC of the female CVD morbidity rates (0.40 during 1986-1989 and 0.43 during 1990-1994) was lower than that of male CVD morbidity

Table 4.2 Spatial autocorrelation (SAC) of CVD morbidity rates in the PHUs of Ontario: Both sexes combined, Females, and Males

Outcome	Year	Sex	SAC value (Moran's *I*)	Z-value
CVD	1986-1989	Both	0.4584	4.51752
		Female	0.4039	4.01111
		Male	0.4935	4.84366
	1990-1994	Both	0.4488	4.42832
		Female	0.4252	4.20903
		Male	0.4682	4.60858
IHD	1986-1989	Both	0.4723	4.64668
		Female	0.4378	4.32611
		Male	0.4891	4.80278
	1990-1994	Both	0.4787	4.70615
		Female	0.4916	4.82601
		Male	0.4560	4.49522
CBVD	1986-1989	Both	0.3119	3.15626
		Female	0.2434	2.51977
		Male	0.3018	3.06241
	1990-1994	Both	0.2619	2.69167
		Female	0.1881	2.00593
		Male	0.3243	3.27148

(0.49 and 0.47 respectively [Table 4.2]). As in the case of CVD mortality, this indicates that there was greater spatial aggregation of the associated risk factors for male CVD morbidity.

While the SAC of female CVD morbidity increased from 0.4252 in the first time period to 0.4378 in the second time period, that of male CVD morbidity decreased from 0.4935 to 0.4682. This suggests that while the risk factors associated with female CVD

morbidity became more spatially concentrated, those associated with male CVD morbidity became less so over time.

Table 4.2 shows that the SAC of the IHD morbidity rates were also high. During the first time period, the SAC of the IHD morbidity rates for both sexes combined was 0.4723 and it was 0.4787 during the second period. Thus, the high spatial aggregation of the risk factors for IHD morbidity for both sexes combined remained more or less unchanged over time. The data in Table 4.2 indicate that while the SAC value of the female IHD morbidity rates increased from 0.4378 during the first period to 0.4916 during the second period, the SAC value of the male IHD morbidity rates decreased from 0.4891 to 0.4560 (i.e., the rates became less spatially concentrated over time). These changes in the SAC values reflect changes in the spatial patterns of the underlying risk factors. They could also be reflective of changes in the importance of particular risk factors.

Compared to CVD and IHD morbidity, the SAC of the combined female and male CBVD morbidity, which was 0.3119 during the first period and 0.2619 during the second period, indicated moderate spatial aggregation of the associated risk factors. During both time periods, the SAC of female CBVD morbidity was much lower than that of males – 0.2434 compared to 0.3018 during the first period, and 0.1881 compared to 0.3243 during the second period (Table 4.2). This suggests that there was a greater degree of spatial aggregation of the male CBVD morbidity rates in the Province.

4.3.3 SUMMARY

The above SAC analyses has revealed that with the exception of CBVD mortality (for

which the SAC values were not statistically significant [Table 4.1]) and female IHD morbidity during the second period (Table 4.2), the cardiovascular disease outcome rates for females had higher SAC values than the rates for males. This suggests that generally, the associated risk factors for male cardiovascular disease were spatially aggregated to a greater degree than those for female cardiovascular disease. The analyses also show that the SAC values for the CBVD morbidity rates were much lower than those for CVD and IHD morbidity, which indicates that there was a lesser degree of spatial clustering in the CBVD rates. The analyses also revealed that, generally, the male cardiovascular disease outcomes were more spatially clustered than the female outcomes.

It has been pointed out that population size and age structure may have an impact on spatial autocorrelation statistics. Walter (1992a) notes that because the global Moran's *I*, for example, can be considered normally distributed if there are more than 20 spatial units of analysis, its statistical significance can be tested using approximate z-tests with theoretical means and variances. He points out, however, that this approach can be inaccurate because it does not take population effects into account. An example is cited of Ontario where the population of metropolitan Toronto is about 200 times the population of Kenora District. When populations of spatial units are different, areas with small populations tend to have more variable rates, and are therefore more likely to have an extreme value. On the other hand, an area that has a large population, such as metropolitan Toronto, would tend to have less variation in the rates, so that the likelihood that an extreme rate would occur is small. Therefore the population scenario in Ontario represents a violation of the assumption that the rates are random variables with the same normal distribution. However, in a report prepared

for the Heart and Stroke Foundation of Ontario on the spatial autocorrelation of cardiovascular disease mortality and morbidity in Ontario at the county level (for the same time period as this research), Elliott (2003) has determined through simulation that the total population and age structure of the Province had little effect on the Moran's *I* statistic. Since the public health units of Ontario approximate the counties, it is expected that the population and age structure would have limited, if any, effect on the PHU level SAC results.

4.4 ANALYSIS OF HOT SPOTS OF CVD MORTALITY AND MORBIDITY

Although the SAC analyses showed that there was significant spatial clustering in the cardiovascular disease outcomes, further analyses were undertaken to determine if there were significant local clusters that represented 'hot spots' of CVD outcome. Hence, the local Moran's *I* statistic was computed for the CMFs for each public health unit, and a z-score was computed for each local *I* value (see Chapter Three for a full explanation of this method). The local Moran's *I* statistic indicates the degree of local spatial clustering of similar or dissimilar observations of an attribute. The *z*-scores and their corresponding local Moran's *I* statistics and CMFs were examined to determine significant local spatial clusters or ''hot spots'' of CVD outcome. Anselin (1995) states that for the local Moran's *I* static, a positive value indicates spatial clustering of similarly high or low values, and a negative value indicates a clustering of dissimilar values (e.g., a public health unit with a high CMF surrounded by public health units with low values, or a public health unit with a low CMF surrounded by public health units with high values). To be significant, the *z*-score of the local Moran's *I* must be either $\geq +1.96$ or ≤ -1.96. So, to determine if a local cluster is a hot

spot, it is necessary to examine the local Moran's *I* static, the z-score, and the corresponding CMF. If the local Moran's *I* statistic is positive, it means that the particular public health unit had a CMF that was similar to that of its neighbours. If the CMFs of the public health units in question are high, then a hot spot (or cluster of elevated CVD outcome rates) exists. On the other hand, if the local *I* statistic is negative; this indicates dissimilarity between the observed CMF and its neighbouring values. The significant CMFs (and the associated public health units) that did not constitute 'hot spots' of CVD outcome are reported in Appendices 4.1 – 4.32, along with indications of the similarity or dissimilarity between these CMFs and their neighbouring values.

4.4.1 ALL CARDIOVASCULAR DISEASES (ICD-9 390-459.9)

Tables 4.3 and 4.4 show public health units with significant positive *z*-scores and their respective neighbours for CVD mortality for both sexes combined. In the first period, four 'hot spots' of CVD mortality occurred in northern Ontario (Table 4.3). Each of these public health units had a high CMF surrounded by first order neighbours (i.e., neighbouring public health units that share a common boundary with the public health unit in question) that had similarly high CMFs. For all CVD, public health units that had CMFs that were dissimilar to the CMFs of their neighbours are shown in Appendices 4.1 – 4.12. All of these public health units are located in southern Ontario. The CMF of Durham was higher than the CMFs of its neighbours. While this public health unit cannot be described as a hot spot, the checkerboard arrangement of high and low CMFs suggests that there was spatial discontinuity in the underlying factors for CVD mortality in the local area. In the second

Table 4.3 'Hot spots' of combined female and male CVD mortality in Ontario, 1986-1989					
'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Sudbury District	1.92	2.23	1.296	Algoma Porcupine Timiskaming North Bay Muskoka-Parry Sound	1.166 1.189 1.130 1.175 1.063
Timiskaming	1.05	2.84	1.130	Porcupine Sudbury District North Bay	1.189 1.296 1.175
Porcupine	1.75	2.99	1.189	Thunder Bay Algoma Sudbury District Timiskaming	1.208 1.166 1.296 1.130
Algoma	1.76	3.09	1.166	Thunder Bay Porcupine Sudbury District	1.208 1.189 1.296

period, only Porcupine and Algoma (in the northern part of the Province) emerged as 'hot spots' of combined female and male CVD mortality (Table 4.4). The sex-specific data showed almost similar spatial patterns.

Table 4.4 'Hot spots' of combined female and male CVD mortality in Ontario, 1990-94					
'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Windsor-Essex	1.66	2.19	1.164	Kent-*Chatham*	1.329
Porcupine	1.19	2.22	1.210	Thunder Bay Algoma Sudbury Timiskaming	1.260 1.113 1.276 1.128
Algoma	0.49	2.64	1.113	Thunder Bay Porcupine Sudbury	1.260 1.210 1.276

The analysis of the female CVD mortality data revealed that during the first time period, there were four 'hot spots' of CVD mortality (Table 4.5), all of which occurred in northern Ontario. But during the second period, there were only three 'hot spots', including Windsor-Essex in southern Ontario (Table 4.6). In the case of males, there were three 'hot spots' of CVD mortality, namely Algoma, Timiskaming, and North Bay, during the first period (Table 4.7). In the second period, the 'hot spots' of male CVD mortality were Porcupine, Algoma, and Windsor-Essex (Table 4.8). Comparing Table 4.6 and Table 4.8, it can be seen that during the second time period, the 'hot spots' of CVD mortality for female and males were the same. On the whole, the mortality data showed that there were minimal differences between females and males with respect to the location of 'hot spots' of CVD mortality in the Province during the time period studied.

Table 4.5 'Hot spots' of female CVD mortality in Ontario, 1986-1989

'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Porcupine	1.13	2.88	1.130	Thunder Bay Algoma Sudbury District Timiskaming	1.173 1.168 1.213 1.147
Algoma	1.69	2.44	1.168	Thunder Bay Porcupine Sudbury District	1.173 1.130 1.213
Sudbury District	1.57	2.26	1.213	Muskoka-Parry Sound Algoma Porcupine Timiskaming North Bay	1.066 1.168 1.130 1.147 1.154
Timiskaming	1.29	2.28	1.147	North Bay Sudbury District Porcupine	1.154 1.213 1.130

Table 4.6 'Hot spots' of female CVD mortality in Ontario, 1990-1994					
'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Algoma	0.32	1.99	1.096	Thunder Bay Porcupine Sudbury District	1.207 1.707 1.250
Porcupine	0.38	2.06	1.707	Thunder Bay Algoma Sudbury District Timiskaming	1.207 1.096 1.250 1.140
Windsor-Essex	2.12	2.47	1.159	Kent-*Chatham*	1.334

Table 4.7 'Hot spots' of male CVD mortality in Ontario, 1986-1989					
'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Algoma	1.37	3.18	1.148	Thunder Bay Porcupine Sudbury District	1.214 1.202 1.348
Timiskaming	0.69	2.94	1.110	Porcupine Sudbury District North Bay	1.202 1.348 1.777
North Bay	0.96	1.98	1.777	Timiskaming Sudbury District Muskoka-Parry Sound Renfrew	1.110 1.348 1.041 1.155
Porcupine	1.66	2.83	1.202	Thunder Bay Algoma Sudbury District Timiskaming	1.214 1.148 1.348 1.110

Table 4.8 'Hot spots' of male CVD mortality in Ontario, 1990-1994					
'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Algoma	0.48	2.86	1.116	Thunder Bay Porcupine Sudbury District	1.291 1.278 1.283
Porcupine	1.50	2.14	1.278	Thunder Bay Algoma Sudbury District Timiskaming	1.291 1.116 1.283 1.112
Windsor-Essex	1.56	2.01	1.177	Kent-*Chatham*	1.333

The CVD morbidity data represented a slightly different scenario. During the first time period, there were six 'hot spots' of combined female and male CVD morbidity (Table 4.9). These public health units, which are all located in northern Ontario, are contiguous; and so this cluster can be regarded as one hot spot. No hot spot of combined female and male CVD morbidity occurred in southern Ontario during this period. All six 'hot spots' observed

Table 4.9 'Hot spots' of combined female and male CVD morbidity in Ontario, 1986-89

'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
North Bay District	0.65	2.20	1.236	Timiskaming District Sudbury District Muskoka-Parry Sound Renfrew	1.504 1.281 1.183 1.272
Thunder Bay	0.84	2.51	1.230	Algoma Porcupine Northwestern	1.389 1.587 1.150
Algoma	2.00	2.53	1.389	Thunder Bay Porcupine Sudbury District	1.230 1.587 1.281
Timiskaming	2.89	2.68	1.504	Porcupine Sudbury District North Bay District	1.587 1.281 1.236
Porcupine	3.28	3.07	1.587	Thunder Bay Algoma Sudbury District Timiskaming	1.230 1.389 1.281 1.504
Sudbury District	1.26	3.45	1.281	Algoma Porcupine Timiskaming North Bay District Muskoka-Parry Sound	1.389 1.587 1.504 1.236 1.183

in the first period persisted in the second period (Table 4.10). Five public health units – Thunder Bay, Porcupine, Algoma, Timiskaming, and North Bay – were 'hot spots' of female CVD morbidity in the first period (Table 4.11). These public health units persisted as 'hot

Table 4.10 'Hot spots' of combined female and male CVD morbidity in Ontario, 1990-94

'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Thunder Bay	0.73	2.15	1.235	Northwestern	1.123
				Porcupine	1.634
				Algoma	1.280
North Bay District	0.77	2.27	1.255	Timiskaming	1.506
				Sudbury District	1.283
				Muskoka-Parry Sound	1.169
				Renfrew	1.331
Algoma	1.19	2.53	1.280	Thunder Bay	1.235
				Porcupine	1.634
				Sudbury District	1.283
Porcupine	2.99	2.71	1.634	Thunder Bay	1.235
				Algoma	1.280
				Sudbury District	1.283
				Timiskaming	1.506
Timiskaming	2.95	2.80	1.506	Porcupine	1.634
				Sudbury District	1.283
				North Bay District	1.255
Sudbury District	1.14	3.19	1.283	Algoma	1.280
				Porcupine	1.634
				Timiskaming District	1.506
				North Bay District	1.255
				Muskoka-Parry Sound	1.169

spots' of female CVD morbidity in the second period, in addition to Sudbury District (Table 4.12). The public health units that were 'hot spots' of male CVD morbidity during both time periods (Tables 4.13 and 4.14) were almost the same as those for females. However, when the data in Tables 4.11 and 4.12 are compared to the data in Tables 4.13 and 4.14 respectively, it can be seen that the CMF of female CVD morbidity was greater than the

CMF of male CVD morbidity in each corresponding case. The higher CMFs for females implies that there was a relatively higher incidence of female CVD morbidity, and suggests that there was a greater impact of the associated risk factors on females.

Table 4.11 'Hot spots' of female CVD morbidity in Ontario, 1986-1989

'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Thunder Bay	0.85	2.72	1.260	Northwestern Porcupine Algoma	1.304 1.630 1.451
Porcupine	2.72	2.76	1.630	Thunder Bay Algoma Sudbury District Timiskaming	1.260 1.451 1.275 1.563
Algoma	1.73	2.20	1.451	Thunder Bay Porcupine Sudbury District	1.260 1.630 1.275
Timiskaming	2.39	2.32	1.563	Porcupine Sudbury District North Bay	1.630 1.275 1.269
North Bay	0.59	2.08	1.269	Renfrew Muskoka-Parry Sound Sudbury District Timiskaming	1.341 1.218 1.275 1.563
Sudbury District	0.86	3.23	1.275	Algoma Porcupine Timiskaming North Bay Muskoka-Parry Sound	1.451 1.630 1.563 1.269 1.218

Table 4.12 'Hot spots' of female CVD morbidity in Ontario, 1990-1994

'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Sudbury District	1.10	3.17	1.324	Algoma Porcupine Timiskaming North Bay Muskoka-Parry Sound	1.334 1.729 1.625 1.289 1.171

Table 4.12 (continued)

Porcupine	3.02	2.79	1.729	Thunder Bay Algoma Sudbury District Timiskaming	1.278 1.334 1.324 1.625
Timiskaming	3.05	2.75	1.625	Porcupine Sudbury District North Bay	1.729 1.324 1.289
Algoma	1.22	2.49	1.334	Thunder Bay Porcupine Sudbury District	1.278 1.729 1.324
North Bay	0.76	2.34	1.289	Renfrew Muskoka-Parry Sound Sudbury District Timiskaming	1.414 1.171 1.324 1.625
Thunder Bay	0.81	2.30	1.278	Northweatern Porcupine Algoma	1.206 1.729 1.334

Table 4.13 'Hot spots' of male CVD morbidity in Ontario, 1986-1989

'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Thunder Bay	0.67	2.11	1.190	Northwestern Porcupine Algoma	1.007 1.521 1.324
Porcupine	3.58	3.23	1.521	Thunder Bay Algoma Sudbury District Timiskaming	1.190 1.324 1.271 1.440
Algoma	2.10	2.72	1.324	Thunder Bay Porcupine Sudbury District	1.190 1.521 1.271
Sudbury District	1.59	3.53	1.271	Algoma Porcupine Timiskaming North Bay Muskoka-Parry Sound	1.324 1.521 1.440 1.200 1.145

Table 4.13 (continued)					
Timiskaming	3.23	2.91	1.440	Porcupine Sudbury District North Bay	1.521 1.271 1.200
North Bay	0.67	2.23	1.200	Timiskaming Sudbury District Muskoka-Parry Sound Renfrew	1.440 1.271 1.145 1.206

Table 4.14 'Hot spots' of male CVD morbidity in Ontario, 1990-1994					
'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Porcupine	2.73	2.49	1.538	Thunder Bay Algoma Sudbury District Timiskaming	1.187 1.225 1.239 1.396
Algoma	1.05	2.47	1.225	Thunder Bay Porcupine Sudbury District	1.187 1.538 1.239
Sudbury District	1.10	3.10	1.239	Algoma Porcupine Timiskaming North Bay Muskoka-Parry Sound	1.225 1.538 1.396 1.223 1.154
Timiskaming	2.66	2.77	1.396	Porcupine Sudbury District North Bay	1.538 1.239 1.223
North Bay	0.75	2.12	1.223	Timiskaming Sudbury District Muskoka-Parry Sound Renfrew	1.396 1.239 1.154 1.257

4.4.2 ISCHEMIC HEART DISEASE (ICD-9 430-438.8)

The analyses showed that in the first time period, Windsor-Essex, Algoma, and Porcupine were 'hot spots' of IHD mortality for both sexes combined (Table 4.15). During the second period, only Windsor-Essex remained as a hot spot of IHD mortality for both sexes combined (Table 4.16). Windsor-Essex was also the only hot spot of female IHD

mortality in the first and second periods (Tables 4.17 and 4.18). However, there was an increase in the value of its CMF from the first period to the second period. This suggests that

Table 4.15 'Hot spots' of combined female and male IHD mortality, 1986-1989					
'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Windsor-Essex	1.84	2.07	1.202	Kent-*Chatham*	1.358
Algoma	1.54	2.13	1.264	Sudbury District Porcupine Thunder Bay	1.272 1.159 1.276
Porcupine	0.67	2.44	1.159	Thunder Bay Timiskaming Sudbury District Algoma	1.276 1.142 1.272 1.264

Table 4.16 'Hot spots' of combined female and male IHD mortality, 1990-1994					
'Hot spot'	Moran's I	Z-score	CMF	First order neighbour	CMF
Windsor-Essex	1.73	3.13	1.201	Kent-*Chatham*	1.672

Table 4.17 'Hot spots' of female IHD mortality in Ontario, 1986-1989					
'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Windsor-Essex	2.10	2.14	1.218	Kent-*Chatham*	1.376

Table 4.18 'Hot spots' of female IHD mortality in Ontario, 1990-1994					
'Hot spot'	Moran's I	Z-score	CMF	First order neighbours	CMF
Windsor-Essex	2.15	3.32	1.230	Kent-*Chatham*	1.792

while the spatial extent of the female IHD mortality impact remained unchanged over time, the burden of mortality in Windsor-Essex became heavier. Compared to females, there were more 'hot spots' of male IHD mortality during both time periods (Tables 4.19 and 4.20).

Table 4.19 'Hot spots' of male IHD mortality in Ontario, 1986-1989

'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Porcupine	0.86	2.41	1.175	Thunder Bay Algoma Sudbury District Timiskaming	1.236 1.193 1.309 1.165
Algoma	1.11	2.26	1.193	Thunder Bay Porcupine Sudbury District	1.236 1.175 1.309
Timiskaming	0.78	2.07	1.165	Porcupine Sudbury District North Bay	1.175 1.309 1.197
Windsor-Essex	1.93	2.08	1.200	Kent-*Chatham*	1.346

Table 4.20 'Hot spots' of male IHD mortality in Ontario, 1990-1994

'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Algoma	0.27	2.22	1.133	Thunder Bay Porcupine Sudbury District	1.348 1.244 1.337
Windsor-Essex	1.59	2.95	1.188	Kent-*Chatham*	1.601
Elgin-St. Thomas	0.51	1.98	1.190	Kent-*Chatham* Middlesex-London Oxford Haldimand-Norfolk	1.601 1.006 1.053 1.369

The data show that the pattern of 'hot spots' of IHD morbidity was different from that of IHD mortality. A striking feature of the 'hot spots' of IHD morbidity is that none occurred in southern Ontario, implying that the IHD morbidity burden was concentrated largely in northern Ontario. During the first period considered, five public health units emerged as 'hot

spots' of combined female and male IHD morbidity (Table 4.21). These public health units persisted as 'hot spots' of combined female and male IHD morbidity during the second period (Table 4.22). Three of these public health units, North Bay, Porcupine, and Sudbury District, experienced increases in their CMFs during the second time period, indicating an increase in the health impact of the associated risk factors over time. The other two public health units, Timiskaming and Algoma, experienced decreases in their CMFs, which suggests that there was a decline in the cardiovascular health impact of the associated risk factors.

Table 4.21 'Hot spots' of combined female and male IHD morbidity in Ontario, 1986-1989

'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Algoma	1.73	2.08	1.449	Thunder Bay Porcupine Sudbury District	1.234 1.480 1.337
Sudbury District	1.49	3.51	1.337	Algoma Porcupine Timiskaming North Bay Muskoka-Parry Sound	1.449 1.480 1.641 1.389 1.149
North Bay	1.36	2.30	1.389	Renfrew Muskoka-Parry Sound Sudbury District Timiskaming	1.240 1.149 1.337 1.641
Timiskaming	3.41	2.75	1.641	North Bay Sudbury District Porcupine	1.389 1.337 1.480
Porcupine	2.45	3.15	1.480	Timiskaming Sudbury District Algoma Thunder Bay	1.641 1.337 1.449 1.234

Table 4.22 'Hot spots' of combined female and male IHD morbidity in Ontario, 1990-94					
'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
North Bay	1.50	2.27	1.466	Renfrew Muskoka-Parry Sound Sudbury District Timiskaming	1.328 1.164 1.454 1.530
Timiskaming	3.09	3.35	1.530	North Bay Sudbury District Porcupine	1.466 1.454 1.690
Porcupine	2.56	2.52	1.690	Timiskaming Sudbury District Algoma Thunder Bay	1.530 1.454 1.304 1.263
Sudbury District	1.83	3.23	1.454	Algoma Porcupine Timiskaming North Bay Muskoka-Parry Sound	1.304 1.690 1.530 1.466 1.164
Algoma	1.02	2.49	1.304	Thunder Bay Porcupine Sudbury District	1.203 1.690 1.454

Although the 'hot spots' of female IHD morbidity consisted of the same public health units during both periods (Tables 4.23 and 4.24), the CMFs for these public health units, with the exception of Algoma, were higher during the second time period. These data suggest that the female IHD morbidity burden in the 'hot spots' in question increased over time. The female IHD morbidity burden decreased over time in Algoma. With the exception of Algoma, which was not a hot spot during the first period, the 'hot spots' of male IHD morbidity consisted of the same public health units during both time periods (Tables 4.25 and 4.26). Generally, the CMFs were higher in the second period, with the exception of Timiskaming where the CMF decreased from 1.588 to 1.391. Thus, similar to female IHD morbidity, the male IHD morbidity burden at these 'hot spots' generally increased over time.

Table 4.23 'Hot spots' of female IHD morbidity in Ontario, 1986-1989					
'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Sudbury District	1.13	3.35	1.354	Algoma Porcupine Timiskaming North Bay Muskoka-Parry Sound	1.554 1.592 1.702 1.477 1.179
Porcupine	2.41	3.07	1.592	Timiskaming Sudbury District Algoma Thunder Bay	1.702 1.354 1.554 1.371
Timiskaming	2.83	2.55	1.702	North Bay Sudbury District Porcupine	1.477 1.354 1.592
Algoma	1.84	2.18	1.554	Sudbury District Porcupine Thunder Bay	1.354 1.592 1.371
North Bay	1.19	1.97	1.477	Timiskaming Sudbury District Muskoka-Parry Sound Renfrew	1.702 1.354 1.179 1.268

4.4.3 CEREBROVASCULAR DISEASE (ICD-9 450-459.9)

There was only one hot spot of combined female and male CBVD mortality during each time period. During the first period, North Bay was the only hot spot of combined female and male CBVD mortality, while Northwestern was the only hot spot of combined female and male CBVD mortality during the second period. The analyses also show that there was no hot spot of female CBVD mortality during the two time periods. However, one public health unit – Sudbury District – had a rate that was dissimilar to, and comparatively higher than, the CMF of its neighbours (Appendix 4.26). There was no hot spot of male CBVD mortality during the first period. During the second period, however, two 'hot spots'

of male CBVD mortality – Northwestern and Algoma – emerged in northern Ontario (Table 4.28), indicating that the male CBVD mortality situation had worsened over time.

Table 4.24 'Hot spots' of female IHD morbidity in Ontario, 1990-1994

'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Sudbury District	2.10	3.38	1.612	Algoma	1.406
				Porcupine	1.807
				Timiskaming	1.733
				North Bay	1.631
				Muskoka-Parry Sound	1.190
Porcupine	2.65	2.77	1.807	Thunder Bay	1.272
				Algoma	1.406
				Sudbury District	1.612
				Timiskaming	1.733
Timiskaming	3.57	3.47	1.733	North Bay	1.631
				Sudbury District	1.612
				Porcupine	1.807
Algoma	1.15	2.50	1.406	Thunder Bay	1.272
				Porcupine	1.807
				Sudbury District	1.612
North Bay	1.75	2.40	1.631	Renfrew	1.367
				Muskoka-Parry Sound	1.190
				Sudbury District	1.612
				Timiskaming	1.733

There were a larger number of 'hot spots' of combined female and male CBVD morbidity than that of mortality during both time periods. During the period 1986-1989, there were five 'hot spots' of CBVD morbidity for both sexes combined (Table 4.29), all of which were located in northern Ontario. During the second period, the number of 'hot spots' of combined female and male CBVD morbidity decreased to four (Table 4.30). However, all of them were still located in northern Ontario. These suggest that the CBVD morbidity burden tended to concentrate in this region.

Table 4.25 'Hot spots' of male IHD morbidity in Ontario, 1986-1989					
'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Porcupine	2.12	3.02	1.379	Thunder Bay	1.129
				Algoma	1.363
				Sudbury District	1.309
				Timiskaming	1.588
Sudbury District	1.63	3.45	1.309	Algoma	1.363
				Porcupine	1.379
				Timiskaming	1.588
				North Bay	1.325
				Muskoka-Parry Sound	1.119
Timiskaming	3.59	2.74	1.588	Porcupine	1.379
				Sudbury District	1.309
				North Bay	1.324
North Bay	1.42	2.48	1.324	Renfrew	1.214
				Muskoka-Parry Sound	1.119
				Sudbury District	1.309
				Timiskaming	1.588

Table 4.26 'Hot spots' of male IHD morbidity in Ontario, 1990-1994					
'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Porcupine	2.17	2.12	1.589	Thunder Bay	1.145
				Algoma	1.228
				Sudbury District	1.345
				Timiskaming	1.391
Sudbury District	1.46	2.95	1.345	Algoma	1.228
				Porcupine	1.589
				Timiskaming	1.391
				North Bay	1.361
				Muskoka-Parry Sound	1.134
Timiskaming	2.46	3.12	1.391	Porcupine	1.589
				Sudbury District	1.345
				North Bay	1.361
North Bay	1.22	2.06	1.361	Timiskaming	1.391
				Sudbury District	1.345
				Muskoka-Parry Sound	1.134
				Renfrew	1.295
Algoma	0.78	2.33	1.228	Thunder Bay	1.145
				Porcupine	1.589
				Sudbury District	1.345

Table 4.27 'Hot spots' of combined female and male CBVD mortality in Ontario, 1990-94					
'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Northwestern	0.29	2.03	0.927	Thunder Bay	1.350

Table 4.28 'Hot spots' of male CBVD mortality in Ontario, 1990-1994					
'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Northwestern	0.05	2.45	1.034	Thunder Bay	1.306
Algoma	0.93	2.50	1.105	Thunder Bay	1.306
				Porcupine	1.190
				Sudbury District	1.060

Table 4.29'Hot spots' of combined female and male CBVD morbidity in Ontario, 1986-89					
'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
North Bay	1.10	2.03	1.292	Renfrew	1.442
				Muskoka-Parry Sound	1.128
				Sudbury District	1.257
				Timiskaming	1.179
Porcupine	1.76	2.23	1.387	Thunder Bay	1.320
				Algoma	1.284
				Sudbury District	1.257
				Timiskaming	1.179
Timiskaming	0.61	2.34	1.179	North Bay	1.292
				Sudbury District	1.257
				Porcupine	1.387
Algoma	1.56	2.52	1.284	Thunder Bay	1.320
				Porcupine	1.387
				Sudbury District	1.257
Sudbury District	0.91	2.31	1.257	Algoma	1.284
				Porcupine	1.387
				Timiskaming	1.179
				North Bay	1.292
				Muskoka-Parry Sound	1.128

Table 4.30 'Hot spots' of combined female and male CBVD morbidity in Ontario, 1990-94

'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Thunder Bay	1.00	2.01	1.258	Northwestern Porcupine Algoma	1.130 1.443 1.300
Porcupine	1.77	2.06	1.443	Thunder Bay Algoma Sudbury District Timiskaming	1.258 1.300 1.166 1.294
Algoma	1.25	2.02	1.300	Thunder Bay Porcupine Sudbury District	1.258 1.443 1.166
Sudbury District	0.31	1.98	1.166	Algoma Porcupine Timiskaming North Bay Muskoka-Parry Sound	1.300 1.443 1.294 1.083 1.103

There were two 'hot spots' of female CBVD morbidity in Ontario during the first period (Table 4.31), both of which were located in northern Ontario. During the second period, the number of 'hot spots' of female CBVD morbidity increased to six (Table 4.32), indicating an extension in the geographic area covered by this local cluster of elevated rates. Although the size of the cluster of 'hot spots' of female CBVD morbidity increased during the second period, it was still entirely located in northern Ontario. The data suggest that the CBVD morbidity burden in Ontario was greater during the second period than during the first period.

There were four 'hot spots' of male CBVD morbidity during the period 1986-1989 (Table 4.33). By the second time period only two of them – Sudbury District and Porcupine – remained as 'hot spots' of male CBVD morbidity (Table 4.34). During both time periods,

none of these 'hot spots' occurred in southern Ontario. The reduction in the number of public health units that emerged as 'hot spots' suggests an improvement in the male CBVD

Table 4.31 'Hot spots' of female CBVD morbidity in Ontario, 1986-1989

'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Sudbury District	0.34	2.07	1.182	Algoma	1.308
				Porcupine	1.301
				Timiskaming	1.216
				North Bay	1.258
				Muskoka-Parry Sound	1.225
North Bay	0.83	2.08	1.258	Timiskaming	1.216
				Sudbury District	1.182
				Muskoka-Parry Sound	1.225
				Renfrew	1.493

Table 4.32 'Hot spots' of female CBVD morbidity in Ontario, 1990-1994

'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Porcupine	3.02	2.79	1.512	Thunder Bay	1.241
				Algoma	1.296
				Sudbury District	1.173
				Timiskaming	1.298
Sudbury District	1.10	3.17	1.173	Algoma	1.296
				Porcupine	1.512
				Timiskaming	1.298
				North Bay	1.004
				Muskoka-Parry Sound	1.086
Algoma	1.22	2.49	1.296	Thunder Bay	1.241
				Porcupine	1.512
				Sudbury District	1.173
Thunder Bay	0.81	2.30	1.241	Northwestern	1.127
				Porcupine	1.512
				Algoma	1.296
Timiskaming	3.05	2.75	1.298	Porcupine	1.512
				Sudbury District	1.173
				North Bay	1.004
North Bay	0.76	2.34	1.004	Timiskaming	1.298
				Sudbury District	1.173
				Muskoka-Parry Sound	1.086
				Renfrew	1.548

morbidity situation over time in northern Ontario. In the south, Durham had an elevated rate of male CBVD morbidity compared to its neighbours (Appendix 4.32).

Table 4.33 'Hot spots' of male CBVD morbidity in Ontario, 1986-1989

'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Sudbury District	1.32	2.31	1.311	Algoma	1.248
				Porcupine	1.439
				Timiskaming	1.128
				North Bay	1.311
				Muskoka-Parry Sound	1.027
Algoma	1.79	3.15	1.248	Porcupine	1.439
				Thunder Bay	1.323
				Sudbury District	1.311
Timiskaming	0.44	3.02	1.128	Porcupine	1.439
				Sudbury District	1.311
				North Bay	1.311
Porcupine	2.39	2.50	1.439	Thunder Bay	1.323
				Algoma	1.248
				Sudbury District	1.311
				Timiskaming	1.128

Table 4.34 'Hot spots' of male CBVD morbidity in Ontario, 1990-1994

'Hot spot'	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Sudbury District	0.26	2.04	1.149	Algoma	1.288
				Porcupine	1.369
				Timiskaming	1.278
				North Bay	1.157
				Muskoka-Parry Sound	1.108
Porcupine	1.47	2.02	1.369	Thunder Bay	1.266
				Algoma	1.288
				Sudbury District	1.149
				Timiskaming	1.278

4.4.4 SUMMARY

The analysis of 'hot spots' of CVD has shown that with the exception of few outcomes, each of the cardiovascular disease outcomes investigated in this research had at least one hot spot of occurrence. Overall, the public health units that most frequently emerged as 'hot spots' of cardiovascular disease outcome are Thunder Bay, Porcupine, Algoma District, Sudbury District, Timiskaming, and North Bay. This cluster of 'hot spots' was located in the northern region of the Province. The number of constituent public health units in this cluster (and for that matter, its geographic extent as well as its spatial configuration) varied between the two time periods. It also varied by sex, and by the specific CVD outcome considered. 'Hot spots' of CVD mortality and morbidity occurred in southern Ontario, too, although with lesser frequency compared to the scenario in northern Ontario. In southern Ontario, 'hot spots' of CVD outcome occurred only in Windsor-Essex and Elgin-St. Thomas. However, Durham had an elevated rate for a few CVD outcomes compared to its neighbours. The existence of 'hot spots' of CVD outcome, as revealed by the above analyses, further illustrates the geographic variations in the CVD outcomes investigated.

4.5 PREVALENCE OF POTENTIAL RISK FACTORS OF CARDIOVASCULAR DISEASE IN ONTARIO

This section describes the prevalence of the potential risk factors of CVD in the Province, and the spatial variations in the prevalence rates. The rates were analysed with respect to sex, age, education, type of public health unit (i.e., urban or rural), and the relative location of the public health unit. This section also describes the bivariate relationship

between the risk factors explored and the various CVD outcomes. The variables subsequently included in the multivariate models (based on the selection criteria described in Chapter 3) are shown in Appendices 4.33 – 4.74

4.5.1 ECONOMIC CHARACTERISTICS

The proportion of the general population of Ontario that lived in dwellings that needed major repairs during the period studied tended to decrease with age (Table 4.35). It was highest for those aged 12-19 years (9.0 %) and lowest for those aged 65-74 years (5.9%).

Table 4.35 Prevalence (%) of CVD risk factors – economic characteristics

Risk Factor		Living in a rental unit	Dwelling needs major repairs
Age Group	12-19	19.2	9.0
	20-44	28.6	8.4
	45-64	13.5	6.4
	65-74	17.7	5.9
	75+	27.2	6.5
Sex	Females	23.6	7.7
	Males	21.4	7.6
Education	< High school	25.3	9.4
	≥ High school	21.9	6.4
Type of PHU	Urban PHU	26.5	6.1
	Rural PHU	20.2	8.6
Relative location	Northern Ontario	22.9	11.2
	Southern Ontario	22.5	7.0

This proportion was nearly the same for females and males – 7.7 per cent and 7.6 per cent respectively – but it was greater among those who had less than high school education

(9.4 %) than among those who had at least high school education (6.4 %). The rate of persons living in dwellings that needed major repairs was higher in rural public health units (8.6 %) than in urban public health units (6.1 %), and it was higher in northern Ontario compared to southern Ontario (Table 4.35). With the exception of female IHD mortality during the period 1986-1989 (Appendix 4.35) and female CBVD mortality during the period 1990-1994 (Appendix 4.37), this variable was positively associated with CVD outcomes; and all these associations were statistically significant (Appendices 4.33 – 4.38).

There were substantial geographic variations in average dwelling value. Province-wide, it ranged from $74,704 in Timiskaming in northern Ontario (Figure 3.1) to $323,531 in York Region in southern Ontario. It varied more widely in southern Ontario (range = $222,493) than in northern Ontario (range = $48,982). Average dwelling value was inversely associated with CVD mortality for both sexes combined during the two periods: $r = -0.697$, $p < .001$ during the first period, and $r = -0.743$, $p < .001$ during the second period. It was also inversely correlated with female and male CVD mortality during both periods (Appendix 4.33) as well as CVD morbidity for both sexes combined, and for females and males separately (Appendix 4.34). With the exception of CBVD mortality for both sexes combined and for females, average dwelling value was also inversely associated with the outcomes of ischemic heart disease (Appendices 4.35 and 4.36) and cerebrovascular disease (Appendices 4.37 and 4.38).

Average household income also varied widely in the Province. It ranged from $38,042 in Timiskaming, a rural public health unit in northern Ontario to $74,289 in York Region, an urban public health unit in southern Ontario. Average household income varied

more widely among urban public health units (from $45,578 in Niagara to $74,289 in York Region, range = $28,711) than among rural public health units (from $38,042 in Timiskaming to $52,007 in Wellington-Dufferin-Guelph, range = $13,965). It also varied more widely in southern Ontario (range = $33,784) than in northern Ontario (range = $10,404). However, average household incomes in southern Ontario were generally higher than those in northern Ontario. Average household income was significantly associated with the CVD outcomes in the expected direction (Appendices 4.33 to 4.38), with the exception of CBVD mortality for both sexes combined, females and males during the period 1986-1989 (Appendix 4.37) and CBVD morbidity for both sexes combined and for females during the period 1990-1994 (Appendix 4.38).

In this research, income inequality was measured using the gini coefficient. The gini coefficient ranges between zero, which means an absence of any inequality in income distribution and one, indicating complete inequality in income distribution. A larger coefficient, therefore, indicates a greater degree of income inequality. During the period under study, income inequality at the public health unit level in Ontario ranged from 0.237 in York Region, an urban public health unit located in southern Ontario (which had the highest average household income of $74,289) to 0.373 in Timiskaming, a rural public health unit located in northern Ontario (which had the lowest average household income of $38,342). Income inequality was positively associated with the CVD mortality and morbidity outcomes (Appendices 4.33 to 4.38) with the exception of CBVD mortality for both sexes combined and for females and males separately during the time period 1986-1989, and CBVD mortality for males during the period 1990-1994 (Appendix 4.37).

The data on unemployment rates were available by county only. The rate of unemployment in the Province ranged between 5 per cent (Perth County) and 12 per cent (Haliburton County). Unemployment rate was positively associated with some CVD outcomes (Appendices 4.33 to 4.38). The only outcomes with which it was not significantly related are male IHD mortality during the period 1990-1994, all the CBVD mortality outcomes during both time periods, and male CBVD morbidity during the period 1986-1989.

The data on incidence of low-income families were also available by county only. Halton County had the lowest incidence of low-income family, 5.3 per cent, while the highest incidence, 16.3 per cent, occurred in the Toronto Regional Municipality. Both counties are in southern Ontario. The outcome variables with which incidence of low-income family was significantly associated are male CVD mortality (1986-1989); male CVD morbidity (1986-1989); combined female and male CVD morbidity (1990-1994); and male CVD morbidity (1990-1994) (Appendices 4.33 and 4.34). Other outcomes with which incidence of low-income family was significantly associated were male IHD mortality (1986-1989) and all the IHD morbidity outcomes (Appendices 4.35 and 4.36). The analyses revealed that the correlations between incidence of low-income family and the rest of the CVD outcomes were not significant.

4.5.2 SOCIAL CAPITAL

Based on the bivariate correlation analyses, the social capital variables selected for inclusion in the multivariate regression analyses are number of voluntary organizations, average charitable donations, licensed day care facilities, and the per capita municipal

expenditure on environmental defence, social assistance, recreation, and culture. As the data in Table 4.36 indicate, these factors varied widely in the Province during the period studied. The number of voluntary organizations per 1000 members ranged from 4 each in Kent-*Chatham* and Timiskaming to 60 in Northwestern. These are all rural public health units. This variable was inversely correlated with the CVD outcomes (Appendices 4.39 to 4.44), with the exception of combined female and male CVD mortality (1986-1989), combined female and male CVD morbidity (1986-1989), all the CBVD mortality outcomes during both time periods, female CBVD morbidity (1986-1989), and all the CBVD morbidity outcomes during the period 1990-1994, indicating that as membership in voluntary organizations increased, CVD outcome rates decreased.

Table 4.36 Analysis of social capital variables

Variable	Average	Minimum	Maximum	Range
Number of voluntary organizations per 1000 members	15	4	60	57
Average charitable donations ($)	227	156	497	342
Number of licensed day care facilities per 1000 pre-school children	5	1	24	23
Per capita municipal expenditure ($) on:				
environmental defense	534	276	957	682
social assistance	284	76	550	474
recreation	84	46	143	97
culture	32	15	87	72

Average charitable donation was lowest in North Bay ($156) and highest in Niagara ($497). It varied more widely in southern Ontario (range = $324) than in northern Ontario (range = $60). When urban and rural PHUs are compared, average charitable donation varied more widely in urban PHUs (range = $314) than in rural PHUs (range = $111).

Average charitable donation was inversely correlated with most of the outcome variables (Appendices 4.39 to 4.44), indicating that as average charitable donations increased, CVD mortality and morbidity rates decreased.

The number of licensed day care facilities per 1000 pre-school children also varied geographically in Ontario. On average, there were more facilities per 1000 children in southern Ontario (mean = 6) than in northern Ontario (mean = 2). There were five public health units – Timiskaming, Muskoka-Parry Sound, Kent-Chatham, Perth, and Huron – where there was only one licensed day care facility per 1000 pre-school children. With the exception of Timiskaming, all these public health units are in southern Ontario. The Ottawa-Carleton public health unit had the highest number of licensed day care facilities – 24 per 1000 pre-school children – in the Province. This variable was inversely associated with all the cardiovascular disease outcomes investigated, and the associations were all significant (Appendices 4.39 to 4.44). This indicates that as average dwelling value increased, CVD outcome rates in the Province of Ontario decreased during the period studied.

The data on per capita municipal expenditure on environmental defense (such as flood and storm water control, fire prevention, sewer works, water treatment, and waste collection and disposal), social assistance (such as payments to the elderly to help offset accommodation costs, and payments to assist mothers with dependent children, persons temporarily or permanently unable to work, and persons who are blind), recreation (such as the maintenance of public parks), and culture (such as historic preservation) were available at the county level only. These are summarized in Table 4.36. The lowest per capita municipal expenditure on environmental defense, $276, was in Sudbury District in northern Ontario

while the highest amount, $957, was in Ottawa-Carlton Regional Municipality in southern Ontario. The lowest amount for social assistance, $76, was also in Sudbury District, but the highest per capita municipal expenditure on social assistance was in Toronto Metropolitan Municipality. There was less variation in the per capita municipal expenditures on recreation and culture than in the case of environmental defense and social assistance. Nevertheless, there were geographic differences in these factors. The highest per capita amount spent on recreation, $143, was in Thunder Bay District, while the lowest amount, $46, was in Prince Edward County. The per capita expenditure on culture ranged from $15 in Prescott and Russell United County to $87 in Toronto Metropolitan Municipality, resulting in a range of $72. With some exceptions, these variables were inversely associated with the CVD mortality and morbidity outcomes (Appendices 4.39-4.44).

4.5.3 DEMOGRAPHIC CHARACTERISTICS

The demographic characteristics considered in this research are age, sex, marital status, education, and location quotient of medical doctors. (The location quotient of doctors of a region, such as a county, is the its share of doctors relative to that of a larger region, such as a province, of which the county is part. If this ratio is greater than 1.0, it means the particular sub region has more than proportionate share of the larger region's total number of doctors. If the ratio is 1.0, it means the sub region has a proportionate share of doctors. But if the ratio is less than 1.0, it means the sub region has less than proportionate share of medical doctors, indicating diminished access to health care compared to other sub regions.) Age and sex were taken into account in the derivation of the variables and in the computation

of the outcome rates. The bivariate correlation analyses revealed that being unmarried was inversely correlated with all the outcome variables, implying that as the rate of unmarried persons increased, the rate of CVD mortality and morbidity decreased. These results are inconsistent with the findings of earlier studies (see Chapter 2, Section 2.3.2.3), which suggest that being unmarried is detrimental to health in general and cardiovascular health in particular. Therefore, the current data do not support the expected relationship between cardiovascular disease outcome and being unmarried.

Overall, about 37 per cent of the population of Ontario aged 18 years and over had less than high school education. Among persons aged 18-19 years, it was 48.5 per cent, but this decreased to 23.6 per cent among those aged 20-44 years (Table 4.37). The high prevalence of this risk factor among teenagers may probably be due to the fact that at the time of the OHS survey, many of them had not yet completed high school by reason of their age. From young adult years, the prevalence of having less than high school education increased with age. The rate also differed by sex. It was lower for females (36.5 %) than for males (37.8 %). Rural public health units had a higher rate (40.7 %) of persons with less than high school education than urban public health units (31.0 %). Similarly, northern Ontario had a higher rate (43.2 %) than southern Ontario (36 %). With the exception of female cerebrovascular disease mortality during the period 1990-1994 (Appendix 4.49), less than high school education was positively associated with all the CVD outcomes (Appendices 4.45 - 4.50). This means that as the rate of persons with less than high school education increased, the rate of the CVD outcomes also increased. This association was stronger for CVD morbidity than mortality.

Table 4.37 Prevalence (%) of CVD risk factors – demographic characteristics			
Risk Factor		Less than high school education	Being unmarried
Age Group	12-19	48.5	62.8
	20-44	23.6	28.1
	45-64	46.7	14.2
	65-74	59.6	28.1
	75+	62.8	51.0
Sex	Females	36.5	32.8
	Males	37.8	29.3
Education	< High school	-	28.8
	≥ High school	-	28.3
Type of PHU	Urban PHU	31.0	33.8
	Rural PHU	40.7	29.5
Relative location	Northern Ontario	43.2	31.4
	Southern Ontario	36.0	31.0

The data on doctors' location quotient was available by county only. Prescott and Russell County had the lowest doctors' location quotient, 0.597. The highest doctors' location quotient, 1.53, was associated with Frontenac County. This variable was significantly associated with few CVD mortality and morbidity outcomes (Appendices 4.45 – 4.50), but is still included in the analysis in order to explore the possible impact of differential access to health care on the regional variations in cardiovascular disease outcomes.

4.5.4 PSYCHOSOCIAL HEALTH AND WELL-BEING

The psychosocial health and well-being variables explored in this research are:

experiencing stressful life, dissatisfaction with social life, unhappiness in life, dissatisfaction with health, feeling of low well being, self-reported poor health status, and physical activity limitation. The correlation analyses of the data showed that the first two variables were not significantly associated with any of the CVD outcomes during the two time periods. The data shows that the prevalence of perceived dissatisfaction with health increased steadily with age during the period under study (Table 4.38). It was more prevalent among females (11.3 %) than among males (10.5 %), and it was more prevalent among persons who have less than high school education (15.6 per cent compared to 8.8 per cent for those completing high school). The rate of perceived dissatisfaction with health was higher in urban public health units (11.4 %) than rural public health units (10.6 %), and the rate in northern Ontario (12.5 %) was higher than in southern Ontario (10.6 %). Perceived dissatisfaction with health was significantly associated only with the female and combined female and male CVD outcomes; it was not significantly associated with any of the male outcomes (Appendices 4.51 – 4.56).

The rate of perceived low well-being fluctuated with increasing age (Table 4.38). Generally, however, it decreased with age, being highest (15.4 %) among those aged 20-44 years and lowest (9.4 %) among those aged 65-74 years. Females had a higher rate of perceived low well-being than males. It was also more prevalent (15.4 %) among those with less than high school education than among those with high school or higher education (12.7 %). The rate of perceived low well-being was higher among urban public health units than among rural public health units, and it was higher in northern Ontario than in southern Ontario (Table 4.38). Perceived low well-being was significantly correlated with few CVD

outcomes (Appendices 4.51 – 4.56). The relationships were positive for these significant correlations, which means that as the rate of perceived low well-being increased, the rate of the associated CVD outcomes also increased.

The rate of self-reported poor health status was low (2 %) in the population aged 12 years and over in Ontario during the time period studied. Generally, the prevalence of self-reported poor health status increased with age, being highest among seniors aged 65-74 years (Table 4.38). It was significantly higher among persons with less than high school education (4 %) than among those who had at least high school (1.2 %), ($\chi^2 = 45702.8$, $p < .001$). Although the variation in the rate of self-reported poor health status by type of public health unit was minimal, i.e., 2.1 per cent in rural PHUs and 2.0 per cent in urban PHUs (Table 4.38), the difference was significant ($\chi^2 = 491.08$, $p < .001$). The prevalence of this variable was the same for females and males, but it was much higher (4 %) among those with less than high school education than among those who had high school or higher education (1.2 %). Urban and rural public health units had nearly the same rates of self-reported poor health status (2 per cent and 2.1 per cent respectively), while northern Ontario had a higher rate (3 %) than southern Ontario (1.9 %). Self-reported poor health status was significantly positively correlated with most of the outcome variables (Appendices 4.51 – 4.56). Female CVD mortality (1990-1994), female IHD mortality (1986-1989 and 1990-1994), and combined female and male CBVD mortality (1990-1994) were not significantly associated with self-reported poor health status. Similarly, female CBVD mortality was not associated

Table 4.38 Prevalence (%) of CVD risk factors – psychosocial health and well being								
Risk Factor		Experiencing stressful life	Dissatisfied with social life	Unhappiness in life	Dissatis-faction with health	Feeling of low well being	Self-reported poor health status	Physical activity limitation
Age Group	12-19	38.6	6.5	4.6	8.1	13.6	0.6	2.7
	20-44	57.7	15.2	4.3	8.4	15.3	1.3	6.4
	45-64	49.9	12.2	6.5	13.5	12.7	3.4	12.7
	65-74	26.1	9.1	6.4	16.3	9.4	4.0	15.8
	75+	24.9	9.6	8.4	19.2	10.4	3.7	18.9
Sex	Females	46.5	13.4	5.1	11.3	14.8	2.1	9.2
	Males	50.0	11.3	5.5	10.5	12.4	2.1	8.7
Education	< High sch	42.2	11.4	7.3	15.6	15.4	4.0	14.5
	≥ High sch	54.6	14.4	4.2	8.8	12.7	1.2	6.9
Type of PHU	Urban PHU	51.2	13.3	5.9	11.4	14.2	2.0	7.7
	Rural PHU	46.5	11.8	4.9	10.6	13.3	2.1	9.6
Relative location	Northern Ontario	45.7	12.1	5.3	12.5	14.4	3.0	11.1
	Southern Ontario	48.7	12.4	5.3	10.6	13.5	1.9	8.5

with this variable during the two time periods studied.

Overall, 8.9 per cent of Ontario residents aged 12 years and over reported that they were limited in their daily activities due to a health or other condition. The prevalence of physical activity limitation increased with age from 2.7 per cent for teenagers to 18.9 per cent among those aged 75 years and over. The rate for females (9.2 %) was higher than for males (8.7 %). It was more than twice as prevalent (14.5 %) among persons with less than high school education as among those with at least high school education (6.9 %). Urban public health units had a lower rate of daily activity limitation (7.7 %) than rural public health units (9.6 %), and the rate was higher in northern Ontario than in southern Ontario (Table 4.38). This variable was positively correlated with all the CVD morbidity outcomes. It was, however, not significantly associated with some of the CVD mortality outcomes (Appendices 4.51 – 4.56).

4.5.5 RISK FACTOR BEHAVIOURS

The CVD risk factor behaviours explored in this research are: current daily smoking, physical inactivity, excess fat in diet, and excessive drinking. The correlation analyses revealed that excess alcohol consumption was not significantly associated with any of the CVD outcomes investigated (Appendices 4.57 – 4.62). The data show that overall, about a quarter of Ontario's population smoked daily during the period 1986-1989. The prevalence of daily smoking among teenagers was about 13 per cent. This rate jumped to 31.9 per cent among young adults, and then, it decreased with age (Table 4.39). The prevalence of smoking differed between males and females. While almost 23 per cent of females aged 12

years and over smoked daily, the rate was significantly higher among males – 27.7 per cent (χ^2 = 15312.54, p< .001). Daily smoking was more prevalent among persons with less than high school education (32.2 %) than among those with at least high school education (24.2 %). The data showed that generally, smoking was more prevalent in rural PHUs than urban PHUs, and that it was more prevalent in northern Ontario than in southern Ontario (Table 4.39). The correlation analysis showed that during both periods, smoking was significantly related to almost all of the cardiovascular outcomes in the expected direction (i.e., a positive relationship). These associations were stronger for morbidity than for mortality in all cases (Appendices 4.57 – 4.62). The associations of smoking with female cerebrovascular disease mortality during both time periods were not significant.

The prevalence of physical inactivity was one of the highest rates of CVD risk factors in Ontario (55.4 %) during the time period considered in this research. Generally, it was more prevalent among younger persons of both sexes. Similar to daily smoking, there was a sharp increase in the prevalence of physical inactivity from teenage years (30.1 %) to young adulthood (61.1 %). It was highest (61.2 %) among adults aged 45-64 years (Table 4.39). It was significantly higher among females (59.1 %) than among males (50.7 %), (χ^2 = 36206.93, p< .001). The rate of physical inactivity was higher among those who had at least high school education than among those who had less than high school education. However, the prevalence of physical inactivity was almost similar with respect to the type of public health unit (i.e., urban or rural) and the relative location of the public health unit in Ontario (Table 4.39). The results of the correlation analyses showed that this variable was significantly associated with few CVD outcomes (Appendices 4.57 – 4.62).

Table 4.39 Prevalence (%) of CVD risk factors – risk factor behaviours						
Risk Factor		Daily smoking	Excess alcohol consumption (females)	Excess alcohol consumption (males)	Physical inactivity	Excess fat in diet
Age Group	12-19	13.1	4.8	7.6	30.1	78.5
	20-44	31.9	9.2	18.3	61.1	82.2
	45-64	26.1	7.0	14.8	61.2	75.4
	65-74	16.8	4.2	8.2	52.2	64.7
	75+	11.3	2.0	3.1	46.5	54.6
Sex	Females	22.9	7.1	-	59.1	74.9
	Males	27.7	-	14.2	50.7	78.9
Education	< High school	32.2	5.4	15.2	56.1	70.2
	≥ High school	24.2	9.0	15.9	59.8	80.7
Type of PHU	Urban PHU	22.7	7.7	14.5	55.2	72.7
	Rural PHU	26.7	6.7	14.0	54.8	79.2
Relative location	Northern Ontario	29.7	6.9	14.8	54.7	78.5
	Southern Ontario	24.4	7.1	14.1	55.0	76.5

Excess fat in diet was the most prevalent risk factor for cardiovascular disease in Ontario. Its overall prevalence in the population aged 12 years and over was almost 77 per cent. The age distribution of the rate of this risk factor shows that it was generally high for all age groups, although it was higher for younger persons (Table 4.39). There was a peak prevalence of 82.2 per cent associated with the age group 20-44 years; and thereafter, the prevalence decreased with age. The prevalence among females (74.9 %) was lower than among males (78.9 %). Excess fat consumption was more prevalent among those who had high school or higher education than among those with less than high school education. The prevalence of excess fat in diet was also significantly higher in rural public health units compared to urban public health units, but it was only slightly higher in northern Ontario than in southern Ontario (Table 4.39). The correlation analyses showed that excess fat in diet was significantly positively associated with almost all CVD outcomes. The only exceptions are: male CVD morbidity (1990-1994), male CBVD morbidity (1990-1994), and all the CBVD mortality outcomes during both time periods.

4.5.6 SOCIAL SUPPORT

Low social participation, incidence of dysfunctional family, no help from family or friends in time of need, no friend or family member to confide in, membership in voluntary associations, and living alone were the social support variables examined in this research. The correlation analyses revealed that with the exception of membership in voluntary associations, each of these was significantly associated with at least one CVD outcome during the time period considered in this research. The data available for this research does

not seem to support the hypothesis that membership in voluntary associations enhances cardiovascular health. The correlation results in Appendices 4.63 – 4.68 show that membership in voluntary associations was positively correlated with all the CVD outcomes, which means that as the rate of membership in voluntary organizations increased, the rate of CVD mortality and morbidity also increased.

The data on low social participation was separated for those aged 16-59 years and those aged 60 years and over. Generally, low social participation was more prevalent among younger persons than among seniors. The rate of low social participation was highest among persons aged 20-44 years (25.1 %), and decreased more or less gradually, to 16.2 per cent among seniors aged 75 years and over. Considering teenagers, young persons, and adults, the prevalence of low social participation among females (23.8 %) differed minimally, but significantly (χ^2=30.35, p< .001), from the prevalence among males (23.6 %) (Table 4.40). Considering seniors aged 60 years and over, however, the prevalence of low social participation among females (17.3 %) was not significantly different from the prevalence of this risk factor among males (17.2 per cent, χ^2=0.003, p = .955). Variations were also modest with respect to level of education and type of public health unit. For those aged 16-59 years, low social participation was more prevalent among those with less than high school education (24.5 %) than among those with high school or higher education (23.8 %). For seniors aged 60 years and over, the rate of low social participation among persons with less than high school education was 17.1 per cent, compared to 17.8 per cent for those with higher education. The prevalence of low social participation among those aged 16-59 years was significantly higher for urban public health units (19 %) than for rural public health units

Table 4.40 Prevalence (%) of CVD risk factors – social support						
Risk Factor		Low social participation (ages 16-59)	Low social participation (ages 60 and over)	Dysfunctional family	No help from family or friends in times of need	No friend or family member to confide in
Age Group	12-19	19.3	-	28.0	1.7	4.6
	20-44	25.1	-	16.0	2.8	9.1
	45-64	22.0	-	20.1	2.8	9.4
	65-74	-	17.6	22.2	2.1	8.3
	75+	-	16.2	22.4	2.9	7.5
Sex	Females	23.8	17.3	19.3	2.1	5.9
	Males	23.6	17.2	20.0	3.2	10.9
Education	< High school	24.5	17.1	23.5	3.2	9.7
	≥ High school	23.8	17.8	15.9	2.5	8.4
Type of PHU	Urban PHU	23.5	19.0	18.9	2.8	8.2
	Rural PHU	23.8	16.5	20.0	2.5	8.3
Relative location	Northern Ontario	26.5	19.1	20.4	2.6	8.6
	Southern Ontario	23.2	17.0	19.5	2.6	8.2

(16.5 per cent, χ^2=12.44, p < .001). For those aged 60 years and over, however, there was no significant difference between the rate for urban and rural public health units – 23.5 per cent and 23.8 per cent respectively (χ^2=1.61, p = .205). For both age groups, the prevalence of low social participation was higher in northern Ontario than in southern Ontario (Table 4.40). Appendices 4.63 to 4.68 show the CVD outcomes with which low social participation for those aged 16-59 years was associated. Low social participation for seniors aged 60 years and over was not significantly associated with any of the outcome variables.

The incidence of dysfunctional families in Ontario was 19.6 per cent. The rate was highest (28 %) among teenagers and lowest (16 %) among young adults aged 20-44 years. Those who had less than high school education experienced a greater rate of dysfunctional family than those who had high school or higher education. Urban public health units had a lower rate than rural public health units, and northern Ontario had a higher rate than southern Ontario (Table 4.40). The correlation analyses showed that the incidence of dysfunctional family was significantly positively associated with most of the CVD outcomes (Appendices 4.63 – 4.68).

The rate of persons who had no help from family or friends in times of need was low in Ontario during the period studied. Overall, 2.6 per cent of the population aged 12 years and over reported that they had no help from family members or friends in times of need. Although the rate was generally low, it varied by age; it was lowest (1.6 %) among teenagers, and highest (2.9 %) among seniors aged 75 years and over. The rate also varied by sex: 2.1 per cent among females and 3.2 per cent among males. There was a greater incidence of lack of help from family or friends among those who has less than high school education (3.2 %)

than among those with higher education (2.5 %). The prevalence of this variable for urban public health units was minimally higher (2.8 %) than that of rural public health units (2.5 %), but the difference was significant (χ^2=7.701, p = .021). There was no difference between northern and southern Ontario with respect to the prevalence of this variable (Table 4.40). The correlation analyses showed that this variable was positively associated with few CVD outcomes (Appendices 4.63 – 4.68).

On average, 8.3 per cent of the population of Ontario aged 12 years and over reported that they had no friends or family members to confide in. The prevalence of this variable was highest (9.4 %) among adults aged 45-64 years and lowest (4.6 %) among teenagers. The prevalence of the variable among males was almost twice as high as among females (Table 4.40). Persons with less than high school education had a higher rate of this variable than those with high school or higher education. The rate of the variable for urban public health units (8.2 %) was not significantly different (χ^2=0.189, p = .910) from the rate for rural public health units (8.3 %). Similarly, the rate in northern Ontario (8.6 %) was not significantly different (χ^2=3.005, p = .223) from the rate in southern Ontario (8.2 %). The analyses show that this variable was significantly positively correlated with many CVD outcomes, particularly the female and combined female and male outcomes (Appendices 4.63 – 4.68).

There was geographic variation in the prevalence of seniors living alone in the Province. The rate of seniors living alone ranged from 17.6 per cent in York Region, an urban public health unit in southern Ontario, to 37 per cent in Timiskaming, a rural public health unit in northern Ontario. It varied more widely in southern Ontario – from 17.6 per

cent in York Region to 31.3 per cent in Kent-*Chatham* – than in northern Ontario, where it ranged from 28.5 per cent in North Bay to 37 per cent in Timiskaming. The correlation analyses revealed that the incidence of seniors living alone was significantly associated with most of the CVD outcome variables in the expected direction (i.e., a positive relationship). The only exceptions are CBVD mortality for both sexes combined and for females during both time periods.

4.5.7 PHYSIOLOGICAL CHARACTERISTICS

The physiological CVD risk factors screened were hypertension, diabetes, and obesity. Hypertension was one of the least prevalent of the cardiovascular disease risk factors considered in the research. On average, about 10 per cent of the population aged 12 years and over had hypertension. It was least prevalent among teenagers (0.3 %), and it increased with age (Table 4.41). Hypertension was more prevalent among females (10.6 %) than among males (8.4 %). The rate of hypertension was more than twice as high (15.6 %) among persons with less than high school education as among those with at least high school education (7.7 %). It was significantly higher (9.9 %) in rural public health units than in urban public health units (9 %) ($\chi^2 = 47.86$, $p < .001$). It was also significantly higher in northern Ontario than in southern Ontario (Table 4.41). The correlation analyses show that hypertension was positively associated with few CVD outcomes (Appendices 4.69 – 4.74)

The rate of diabetes increased with age (Table 4.41), although it was generally low (2.8 %) in the Province. Seniors aged 65-74 years had the highest rate of diabetes (8.7 %) during the period studied. Females and males differed significantly with respect to the rate of

Table 4.41Prevalence (%) of CVD risk factors – physiological characteristics				
Risk Factor		Hypertension	Diabetes	Obesity
Age Group	12-19	0.3	0.4	–
	20-44	2.9	0.9	22.5
	45-64	15.5	4.4	33.3
	65-74	28.9	8.7	–
	75+	30.4	8.1	–
Sex	Females	10.6	3.1	21.8
	Males	8.4	2.5	31.3
Education	< High school	15.6	4.9	32.4
	≥ High school	7.7	2.0	23.5
Type of PHU	Urban PHU	9.0	2.5	23.6
	Rural PHU	9.9	3.0	27.9
Relative location	Northern Ontario	9.9	3.1	29.7
	Southern Ontario	9.5	2.7	25.6

diabetes – 3.1 per cent and 2.5 per cent respectively (Table 4.41). The rate was more than twice as high for persons with less than high school education (4.9 %) as for those who had high school or higher education (2.0 %). It was higher in rural public health units than in urban public health units, and it was also higher in northern Ontario (2.8 %) than in southern Ontario (Table 4.41). As expected, diabetes was positively associated with most of the outcome variables (Appendices 4.69 – 4.74).

The overall prevalence of obesity (i.e., body mass index greater than 27.0) in the Ontario population aged 20-64 years was 26.3 per cent. Obesity was more prevalent among persons aged 45-64 years (33.3 %) than among young adults aged 20-44 years (22.5 %). It was less prevalent among females (21.8 %) than among males (31.3 %). The analysis showed that obesity was more prevalent among persons with less than high school education (32.4 %) than those with higher education (23.5 %). The rate was higher in rural public health units than urban public health units, and it was higher in northern Ontario (Table 4.41). With the exception of a few outcomes, obesity was significantly associated to the CVD outcomes considered, and these associations were in the expected direction (Appendices 4.69 – 4.74). For all the CVD outcomes, the relationships that were significant were stronger for morbidity than mortality. For example, while the coefficient of the association between obesity and combined female and male CVD mortality for the periods 1986-1989 and 1990-1994 were 0.440 ($p = 003$) and 0.446 ($p = 0.003$) respectively (Appendix 4.69), the corresponding coefficients for combined female and male CVD morbidity (Appendix 4.70) were 0.641 ($p < 0.001$) and 0.598 ($p < 0.001$).

4.5.8 SUMMARY

The analyses of the prevalence of CVD risk factors show that within each risk construct, a number of factors were significantly associated with the CVD outcomes studied. The results show that with few exceptions, the economic characteristics explored were significantly associated with the CVD outcomes in the expected directions. Income inequality, for example, was positively associated with most of the CVD mortality and morbidity variables. This is consistent with the evidence that relative income has a powerful influence on health (Wilkinson 1997). Kawachi and others (1997), for instance, find that after adjustment for poverty, a 1% rise in the index of income inequality (the Robin Hood Index) was associated with an increase in age-adjusted total mortality rate of 21.7 deaths per 10,000 persons, while Kaplan et al. (1997) found that the degree of income inequality in each of the states of the United States in 1980 was a strong predictor of levels of total mortality a decade later.

As expected, variables used in this study to represent social capital were associated with many of the outcome variables in the expected direction. The correlation coefficients for the relationships between licensed day care facilities and the CVD outcomes were high (Appendices 4.39 – 4.44). The inverse relationship between social capital and the CVD outcomes is consistent with the literature on the relationship between social capital and health, which indicates that this variable has a positive influence on health (Kawachi et al. 1997; Lomas 1998; Veenstra 2000).

Of the demographic variables explored in the bivariate correlation analysis, the education variable – less than high school education – was positively associated with CVD

mortality and morbidity as expected. However, the marital status variable – being unmarried was inversely associated with CVD. This runs counter to what is expected based on the evidence in the literature on marital status and health (Chapter 2). Scarinci et al. (2002), for example, report that in a sample of 1,407 Black women recruited through the National Black Women's Health Project in the United States, never-married women exhibited significantly higher levels of depression compared to women who were married or living together with an intimate partner. Lund et al. (2002) also analyze how mortality is associated with cohabitation status (living alone/not living alone), living with/without a partner, and marital status respectively, in a random sample of 1265 Danish women and men aged between 50 and 70 years. They find that individuals living alone experienced a significantly increased mortality (hazard ratio (HR) = 1.42, CI: 1.04-1.95) compared to individuals not living alone. Similarly, individuals living without a partner experienced increased mortality (HR = 1.38, CI: 1.01-1.88). These analyses are adjusted for functional ability, self-rated health, having children, smoking, diet and physical activity. They find no evidence of an indirect effect of health behaviours on the association between living arrangements and mortality.

Surprisingly, there was no association between experiencing stressful life and any of the CVD outcomes, although psychosocial stress has been linked to cardiovascular disease. For example, Black and Garbutt (2002) argue that stress causes the release of various stress hormones such as catecholamines, corticosteroids, glucagon, growth hormones, renin, and elevated levels of homocysteine, which induce a heightened state of cardiovascular activity. Pickering (1999) maintains that the possible pathways by which SES affects cardiovascular disease include effects of chronic stress mediated by the brain. Rozanski et al. (1999) also

note that the evidence that psychosocial factors contribute significantly to the pathogenesis and manifestation of coronary artery disease is composed largely of data relating it to the following specific psychosocial domains: depression, anxiety, personality factors and character traits, social isolation, and chronic life stress. It is therefore interesting that the current data does not support a link between stress and CVD. Self-reported poor health status was one of the least prevalent of the risk factors during the period studied. Nevertheless, it was significantly positively correlated with most of the CVD outcomes. This is interesting because general self-reported health is used widely in health research (van Doorslaer et al. 1997) and has been shown to be highly correlated with other measures of health, such as mortality, diagnosed morbidity, symptom reporting (Hoeymans et al. 1997; Miilunpalo et al. 1997) and also functional status (Gold et al. 1996).

Four CVD risk factor behaviours, namely excessive alcohol consumption, smoking, physical inactivity, and excess fat in diet, were investigated for their association with CVD outcomes during the period studied. The data show that excess alcohol consumption was not related to CVD outcome during the period studied. This, however, is inconsistent with the findings of earlier studies that excess alcohol consumption is associated with increased risk of cardiovascular disease mortality (Kannel and Ellison 1996; Gensini et al. 1998; Jaglal et al. 1999). Smoking was one of the most prevalent risk factors during the period. Overall, about 25 per cent of Ontario's population smoked daily. Smoking was significantly associated in the expected direction with most of the outcome variables. Although smoking remains a well-known risk factor for cerebrovascular disease (i.e., stroke) in both women and men (HSFC 1997), the data show that it is not significantly associated with female CBVD

mortality during both time periods. The rates of physical inactivity and excess fat in diet were also high. However, the correlation analyses show that physical inactivity was significantly associated with few of the CVD outcomes. On the other hand, excess fat in diet, which emerged as the most prevalent of the risk factors during the period studied, was significantly associated with most CVD outcomes.

All but one of the social support variables used – membership in voluntary associations – were significantly associated with at least one CVD outcome variable in the expected direction. The analyses show that membership in voluntary associations was positively associated with all the CVD outcome variables (Appendices 4.63 – 4.68). Thus, the data does not seem to support the hypothesis of an inverse relationship between this variable and health. This is consistent with the findings of Veenstra (2000) that socialization with neighbours, volunteering, communicating on the internet, and the number of clubs and associations that respondents belonged to were unrelated to health. However, Veenstra found that attendance at religious services was related to health overall, although the relationship did not appear to be linear.

Of the physiological risk factors screened, hypertension was the least prevalent. The prevalence of diabetes was also low, while obesity afflicted about 26 per cent of Ontarians aged 12 years and over. While hypertension was significantly associated with few CVD outcomes, diabetes and obesity were significantly associated with most of them. These relationships are positive, and are consistent with the CVD literature in which they are identified as risk factors (e.g., Gensini et al. 1998; Wenger 1995; Negri et al. 1993; Hu et al. 1999; Whaley and Blair 1995; Canadian Cardiovascular Society Consensus Conference

1998).

Overall, the analyses show that there was variation in the prevalence of the risk factors significantly associated with CVD outcomes. Hence, they constitute potential determinants of the observed geographic variations in these CVD outcomes. The relevant potential determinants were included in a multivariate modelling process for each of the CVD outcome variables to determine the underlying factors for the geographic variations in these outcomes (i.e., objective 3). The results of the multivariate modelling process are presented and discussed in Chapter Five.

4.6 SUMMARY AND CONCLUSION

The analyses have shown that the pattern of the spatial variation in the cardiovascular disease outcome rates remained largely unchanged between the two time periods studied. For example, during both periods, higher CMFs occurred mainly in northern Ontario while lower CMFs occurred mainly in southern Ontario. The GIS analyses of the outcome data have shown that there was significant clustering in the outcome rates and that there were local clusters of similarly high rates (or 'hot spots') of CVD mortality and morbidity. The main hot spot of CVD outcome was located in the northern part of the Province, and consisted mainly of the public health units of Sudbury District, Algoma, Thunder Bay, Porcupine, Timiskaming, and North Bay. Occasionally, Northwestern and Muskoka-Parry Sound were included in this northern cluster. The clustering of outcome rates that occurred in the southern part of the Province was, however, of a different kind. In most cases, the neighbours of the significant public health unit did not have consistently higher or lower

CMFs, but rather, a juxtaposition of higher and lower CMFs. While these clusters of dissimilar CMFs cannot be defined as 'hot spots' within the context of the local Moran's *I* statistic, it may be conjectured that they indicated discontinuities in an underlying process, such as environmental effect, or the availability of, and proximity to acute care facilities and services. They may also be indications of the differential impacts of heart health promotion in the neighbouring public health units.

The analyses presented in this chapter have also shown that there were variations within the province of Ontario of the prevalence of potential cardiovascular disease risk factors with respect to age, sex, education, type of public health unit, and relative location of the public health unit in the Province. The prevalence of many of the potential risk factors tended to be higher among persons who had less than high school education than among those with high school or higher education, suggesting that education affects the impacts of many CVD risk factors. Risk factor prevalence tended to be higher in rural public health units than in urban public health units. Another dimension of the geographic variation in the prevalence of the potential risk factors explored is the comparison between northern and southern Ontario. Consistent with the findings of earlier studies (e.g., Jaglal et al. 1999), the prevalence of the potential risk factors tended to be higher among northern Ontario residents than among southern Ontario residents. One reason that could explain the spatial variations in the prevalence of the risk factors is the variability in the levels of implementation of community-based heart health activities in the Province. In their assessment of public health capacity to support community-based heart health promotion, Elliott et al. (1998) find that while there is little variation in overall implementation across public health units when all

activities are taken into account, there is variability in the level of implementation of heart health promotion by risk factor and by risk factor/setting combinations. Variability in the level of implementation is highest for tobacco activities, followed by nutrition, physical activity, and general heart health in that order. They find that variability is highest for physical activity in health care settings and lowest for tobacco activities in the community setting. The authors warn, however, that allowance has to be made for the artefact introduced by the small number of items used to arrive at this finding. With few exceptions, the potential CVD risk factors identified in this study were significantly associated with the CVD outcomes in the expected directions.

Given the regional variations in the outcomes and the geographic variation in the prevalence of the potential risk factors, the stage is set for a multivariate modelling of the outcomes, which is described in the next chapter.

CHAPTER FIVE

DETERMINANTS OF GEOGRAPHIC VARIATION IN CARDIOVASCULAR DISEASE MORTALITY AND MORBIDITY IN ONTARIO

5.1 INTRODUCTION

This chapter describes the results of multivariate modeling of cardiovascular disease mortality and morbidity in Ontario. The modeling consisted of fitting ordinary least squares regressions. Spatial autocorrelation analyses of the regression residuals were done to ascertain the appropriateness of incorporating spatial dependency structure in the models. The outcome variables in the regressions were the comparative mortality and morbidity figures (CMF) for all cardiovascular disease, ischemic heart disease, and celebrovascular disease for females, males, and both sexes combined during two time periods, 1986-1989 and 1990-1994. The CMF is a ratio of the actual mortality or morbidity rate of each public health unit to the expected rate, i.e., if the outcome had occurred at the Provincial rate. Section 5.2 describes the standard regression models and results. Section 5.3 contains a separate analysis that involved variables explored in this study for which data were available only at the county level. This was done as a sensitivity analysis to gain insight into the predictive value of these variables, as they were not included in the public health unit level analyses. The last section contains discussions of the statistical analyses and conclusions.

5.2 MODELLING GEOGRAPHIC VARIATION IN CARDIOVASCULAR DISEASE

5.3.1 STATISTICAL ANALYSES

For each outcome, the selected independent variables were included in a weighted ordinary least squares regression, which is stated as

$$Y = X\beta + \epsilon,$$

where Y is a (n X 1) vector of the random variables, Y_i, (in this case, the CMFs) in each of the PHUs; X is a (n X p) matrix of the values of p explanatory variables, with β being the corresponding (p X 1) vector of coefficients, and ϵ is a (n X 1) vector of random variables representing disturbances. In standard regression analysis, these errors are assumed to have constant variance and to be independent, so that their covariance is zero. Hence, it is assumed that there is no spatial dependence in the observed CMF values, and the ordinary least squares model can be fit to the observed data.

Exploratory tests were carried out to check the assumptions of the ordinary least squares regression. Histograms and normal probability plots were used to examine the normality of the distribution of both the outcome and risk factor data, and scatter plots were used to check for linearity in the expected relationship between the outcome variables and the selected risk factors. In order to reduce the effect of possible multicollinearity of the predictors to a minimum, only variables with a condition index of 10 or less (Gujarati 1995) were included in the final models. The modeling was done for all cardiovascular diseases combined, ischemic heart disease, and celebrovascular disease mortality and morbidity during the two time periods (1986-1989 and 1990-1994). It was

done for both sexes combined, and for females and males separately. In all thirty-eight predictor variables* were explored. Each model contained 37 public health units (i.e., data points) and used up to four predictor variables, resulting in a minimum cases-to-independent variable ratio of 9.25 to 1. This meets the minimum standard of 5 cases to 1 independent variable that is required for stable regression results (Tabachnick and Fidell 1989).

Tests were also performed on the regression results to check for other potential assumption violations. The assumption of homoscedasticiy means that the regression residuals have approximately equal variance for all predicted scores of the outcome variable. The weighted least squares regression method was adopted to forestall the possible effects of non-constant variance on the models. In this study, each data point was weighted by the inverse of the variance* (Kleinbaum 1988; Chatterjee and Price 1991) of the CMF. The use of weights helps to adjust the amount of influence each data point has on the estimates of the model parameters to an appropriate level. Thus, the more precise observations (that is, those with less variability) are given greater weight in determining the regression coefficients.

A forward stepwise regression procedure was used, with a significance level of $p \leq 0.2$ required in the partial test for a variable to be retained in the final model. The fits of

---

* One problem associated with multiple regression models is that of multiple comparisons, whereby the more tests done, the higher the likelihood of falsely rejecting the null hypothesis. In this study, this issue is minimized as the predictors were selected based on evidence in the risk factor literature indicating their link to CVD.

* Johnson and Dinardo (1997) suggest using the inverse of the standard deviation of the dependent variable as the weight. In this study, however, the inverse of the variance is used because it gives minimum variance (and hence shortest confidence interval) properties to the final estimates of the parameters being investigated.

alternative models were compared using F-tests, and the absolute residuals from the regressions were also mapped. The distributions of the standard regression residuals were checked for normality using the Kolmogorov-Smirnov test.

The data were also examined for spatial patterns by calculating the first-order spatial autocorrelation (SAC) in the residuals. ("First-order spatial autocorrelation" refers to the autocorrelation of the values of an attribute in geographically contiguous areas.) The calculation of first-order SAC was an exploratory analysis to determine whether spatial modeling should be used. First-order spatial autocorrelation was assessed using the Moran's *I*-statistic (i.e., global Moran's *I*). It is defined as

$$I = \frac{n\sum_{i=1}^{n}\sum_{j=1}^{n} w_{ij}\left(y_i-\bar{y}\right)\left(y_j-\bar{y}\right)}{\left(\sum_{i=1}^{n}\left(y_i-\bar{y}\right)^2\right)\left(\sum_{i\neq j}\sum w_{ij}\right)},$$

where the coefficients w_{ij} represent the geographic contiguity of areas (i.e., public health units) i and j, and y_i represents attribute values (in this case, the standard regression residuals). The coefficients, w_{ij}, are elements of a spatial proximity matrix such that w_{ij} = 1 if areas i and j share a common boundary, and $w_{ij} = 0$ otherwise. A p-value of 0.05 was used to determine the significance of the SAC values. The value of the SAC, if significant, would indicate the extent of spatial dependency in the data that is not taken into account in the standard regression models.

With the exception of the SAC values of the residuals from the standard regression models for six outcomes – male IHD mortality (1986-1989), female CVD morbidity (1986-1989), female IHD morbidity (1986-1989 and 1990-1994), male IHD

morbidity (1990-1994), and male CBVD morbidity (1986-1989) – the SAC values were not statistically significant (Tables 5.1 and 5.2). The lack of statistical significance* of the SAC values indicates the absence of spatial autocorrelation in the data, and suggests that nothing would be gained by fitting any model that builds in spatial dependence (Bailey and Gatrell 1995).

Table 5.1 Results from SAC analysis of regression residuals for CVD, IHD, and CBVD mortality

		1986-1989		1990-1994	
Outcome	Sex	SAC of residual	p-value of SAC	SAC of residual	p-value of SAC
All cardio-vascular diseases (CVD)	Both sexes	-0.032	0.965	-0.096	0.518
	Females	0.039	0.526	-0.066	0.718
	Males	-0.095	0.523	0.074	0.332
Ischemic heart disease (IHD)	Both sexes	0.004	0.761	-0.034	0.935
	Females	-0.072	0.678	-0.055	0.794
	Males	0.491	0.045	0.104	0.212
Cerebro-vascular disease (CBVD)	Both sexes	-0.063	0.739	-0.127	0.347
	Females	-0.039	0.909	-0.092	0.540
	Males	0.058	0.413	-0.096	0.514

5.2.2 RESULTS OF THE MODELLING PROCESS

The following sub-sections describe the results of the statistical analyses. The potential CVD risk factors entered into the various models are shown in Appendices 5.1 – 5.18. The associations of these risk factors with particular CVD outcomes were

* Although the SAC values of six out of the thirty-six models (Tables 5.1 and 5.2) are significant, no spatial models are fitted for these because they are not expected to have any significant impact on the overall findings.

discussed in the preceding chapter. The zero-order Pearson's product moment correlation coefficients of these associations are presented in Appendices 4.1 – 4.42.

Table 5.2 Results from SAC analysis of regression residuals for CVD, IHD, and CBVD morbidity

		1986-1989		1990-1994	
Outcome	Sex	SAC of residual	p-value of SAC	SAC of residual	p-value of SAC
All cardio-vascular diseases (CVD)	Both sexes	0.022	0.639	0.146	0.099
	Females	0.318	0.001	0.059	0.411
	Males	0.110	0.191	0.138	0.116
Ischemic heart disease (IHD)	Both sexes	0.089	0.267	0.120	0.159
	Females	0.250	0.008	0.205	0.027
	Males	0.085	0.284	0.240	0.011
Cerebro-vascular disease (CBVD)	Both sexes	0.012	0.706	-0.074	0.660
	Females	0.584	0.413	-0.096	0.514
	Males	0.263	0.005	-0.015	0.899

The covariates in the final models consisted of different combinations of the following variables: average dwelling value, number of licensed day care facilities per 1000 population of children aged 0-5 years, less than high school education, self-reported poor health status, daily smoking, low social participation, diabetes, and obesity. Thus, all the risk factor constructs in the conceptual model proposed in Chapter 2 for studying the geographic variation in cardiovascular disease outcomes (Figure 2.2) are represented in the final models. The value of the coefficient, *b*, of each predictor variable retained in the final models (Tables 5.3 – 5.11) represents the proportionate change in the outcome variable per unit change in the particular predictor variable, while the other factors

remain constant. The coefficients were significant at $p \leq 0.2$, and they take on the expected sign in all the models. The value of r^2 change associated with each predictor retained in the model is the proportion of the total geographic variation in the outcome variable explained by that predictor adjusted for the other factors at that stage. In other words, it is the contribution of that predictor to the explained variation in the outcome. The "Model R^2" represents the total explained variation.

Table 5.3 Results of multivariate regression for outcome: combined female and male CVD

Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0016	0.0001	0.740	-0.0018	0.0003	0.661
Poor health status	0.0029	0.0014	0.029	0.0057	0.0023	0.046
Obesity	-	-	-	0.0014	0.0007	0.052
Model R^2	-	-	0.769	-	-	0.759
Adjusted model R^2	-	-	0.756	-	-	0.737
	1990-1994					
Average dwelling value	-0.0018	0.0002	0.793	-0.0016	0.0003	0.641
Licensed day care facilities	-	-	-	-0.0098	0.0034	0.096
Poor health status	-	-		0.0056	0.0022	0.044
Model R^2	-	-	0.793	-	-	0.782
Adjusted model R^2	-	-	0.788	-	-	0.762

5.2.2.1 COMBINED MODELS - FEMALES AND MALES

Table 5.3 summarizes the results of regressing female and male CVD mortality and morbidity (1986-1989 and 1990-1994) on the variables included in the final models. The proportion of the total variations in the outcomes explained by the CVD models tended to be greater for the period 1990-1994 (79 per cent) than for 1986-1989 (77 per cent). The corresponding figures for CVD morbidity are 76 per cent and 78 per cent (Table 5.3). These represent an improvement in the fit of the respective models over time. While this is so, this total explained variation was almost the same for CVD mortality as for morbidity during both time periods (Table 5.3). Average dwelling value dominated the CVD models. During the first period, 1986-1989, it explained about 74 per cent of the geographic variation in CVD mortality. The only other predictor retained in the model, self-reported poor health status, explained only about 3 per cent of the variation partially. During the second period, average dwelling value was the only predictor of CVD mortality for both sexes combined, explaining 79 per cent of the variation in the outcome (Table 5.3).

Average dwelling value and self-reported poor health status were the only variables retained in the models for IHD morbidity during the two time periods. In the case of IHD mortality, average dwelling value was the only significant explanatory variable during both time periods (Table 5.4). With the exception of CBVD morbidity during the first time period, the CBVD models for both sexes combined (Table 5.5) had lower predictive power compared to the models for CVD and IHD. For example, during the first time period, the significant predictors for CBVD mortality – smoking and having

Table 5.4 Results of multivariate regression for outcome: combined female and male IHD						
Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0020	0.0002	0.718	-0.0025	0.0003	0.667
Poor health status	-	-	-	0.0077	0.0024	0.076
Model R^2	-	-	0.718	-	-	0.743
Adjusted model R^2	-	-	0.710	-	-	0.727
	1990-1994					
Average dwelling value	-0.0026	0.0003	0.697	-0.0027	0.0003	0.633
Poor health status	-	-	-	0.0094	0.0027	0.098
Model R^2	-	-	0.697	-	-	0.731
Adjusted model R^2	-	-	0.688	-	-	0.715

less than high school education – explained a total of only about 33 per cent of the geographic variation in this outcome. This notwithstanding, it was the only model that retained daily smoking as a significant predictor of variations in cardiovascular disease in the Province during the period studied – 1986-1994. During the period 1990-1994, only "licensed day care facilities" was significant, explaining about 39 per cent of the variation in CBVD mortality in the standard regression model.

5.2.2.2 SEX-SPECIFIC MODELS – FEMALES

The regression results for female CVD outcome are summarized in Table 5.6. Average dwelling value was again the most influential covariate in the models. Self-reported poor health status was less dominant. For instance, while it was absent from both the mortality and morbidity models during the first period, it explained only about 8

per cent of the variation in female CVD morbidity during the second period compared to about 59 per cent for average dwelling value. This indirectly illustrates the role that

Table 5.5 Results of multivariate regression for outcome: combined female and male CBVD

Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-	-	-	-0.0017	0.0003	0.682
Licensed day care facilities	-	-	-	-0.0074	0.0031	0.071
Less than high school education	0.0007	0.0003	0.125	-	-	-
Poor health status	-	-	-	0.0044	0.0020	0.032
Smoking	0.0008	0.0005	0.207	-	-	-
Model R^2	-	-	0.332	-	-	0.785
Adjusted model R^2	-	-	0.292	-	-	0.765
	1990-1994					
Licensed day care facilities	-0.0084	0.0018	0.392	-0.0125	0.0045	0.437
Smoking	-	-	-	0.0015	0.0008	0.054
Model R^2	-	-	0.392	-	-	0.491
Adjusted model R^2	-	-	0.375	-	-	0.460

housing characteristics, particularly housing tenure, play in health. For example, McIntyre et al. (2001) have shown that there is significant association between housing tenure and health measures such as limiting long-term illness, depression, and general health. The models for female CVD mortality and morbidity during the period 1986-1989 were two of the few models that retained diabetes as a significant predictor of the

geographic variation in CVD in Ontario. Diabetes explained about 4 per cent of the variation in CVD mortality and 3 per cent of the variation in CVD morbidity.

Table 5.6 Results of multivariate regression for outcome: female CVD						
Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0013	0.0002	0.655	-0.0014	0.0004	0.624
Licensed day care facilities	-	-	-	-0.0148	0.0038	0.123
Diabetes	0.0030	0.0015	0.038	0.0052	0.0024	0.030
Model R^2	-	-	0.693	-	-	0.778
Adjusted model R^2	-	-	0.675	-	-	0.757
	1990-1994					
Average dwelling value	-0.0016	0.0002	0.730	-0.0027	0.0003	0.597
Poor health status	-	-	-	0.0070	0.0025	0.075
Model R^2	-	-	0.730	-	-	0.672
Adjusted model R^2	-	-	0.722	-	-	0.653

Table 5.7 shows the models for female IHD outcomes. Average dwelling value was retained in all four models, as was diabetes. Diabetes appears to be important as a predictor only in respect of female cardiovascular disease outcomes because it was not retained in any of the combined or male models. None of the four models contained self-reported poor health status as a significant predictor. This suggests that it did not have a significant cardiovascular health impact on females in Ontario during the period studied.

This is interesting because self-rated health has been found to be an important predictor of mortality and morbidity. For example, in investigating the validity of various self-reported health assessments in predicting physician contacts and all-cause mortality in a

Table 5.7 Results of multivariate regression for outcome: female IHD						
Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0017	0.0003	0.596	-0.0028	0.0004	0.599
Diabetes	0.0053	0.0023	0.053	0.0088	0.0035	0.064
Model R^2	-	-	0.649	-	-	0.663
Adjusted model R^2	-	-	0.649	-	-	0.643
	1990-1994					
Average dwelling value	-0.0025	0.0003	0.604	-0.0033	0.0043	0.536
Low social participation	-	-	-	0.0037	0.0009	0.139
Diabetes	0.0084	0.0031	0.073	0.0064	0.0036	0.034
Model R^2	-	-	0.676	-	-	0.709
Adjusted model R^2	-	-	0.658	-	-	0.683

prospective study in Finland, Miilunpalo et al. (1997) find a consistent inverse association, standardized by age, sex and social status, between perceived health status and perceived physical fitness and mortality. Also, Crighton et al. (2000, in press) find that there is consistency between a high rate of 'poor' self-rated health and high mortality rate in Karakalpakstan, Central Asia. Low social participation explained almost 14 per

cent of the variation in female IHD morbidity in the second period. This was the only model that retained the variable in question as a significant predictor of cardiovascular disease in the Ontario during the period studied. Therefore, although low social participation was retained as a significant predictor of female IHD morbidity, its cardiovascular health impact appears to be limited.

The results for female CBVD are shown in Table 5.8. While average dwelling value was significant for morbidity in both periods, it was absent from the mortality models. Having less than high school education was the only significant variable retained in the model for CBVD mortality during the first period. During the second period, it was licensed day care facilities. The respective proportions of the variation in CBVD mortality explained by these variables were 23 per cent and 26 per cent. During both periods, the explained variation in female CBVD morbidity was much greater than that of mortality. For example, the explained variation in female CBVD morbidity during the first period about 74 per cent compared to about 23 per cent for female CBVD mortality. Average dwelling value and licensed day care facilities, which explained substantial proportions (65 per cent and 9 per cent respectively) of the variation in female CBVD morbidity during the first period were not retained in the corresponding mortality model. Therefore, the low explained variation of female CBVD mortality compared to morbidity, and the absence of average dwelling value and licensed day care facilities from the latter model appear to suggest that during the first period, other factors not included in this study may have mediated female CBVD mortality.

Table 5.8 Results of multivariate regression for outcome: female CBVD						
Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-	-	-	-0.0014	0.0004	0.650
Licensed day care facilities	-0.0105	0.0024	0.351	-0.0112	0.0033	0.089
Model R^2	-	-	0.351	-	-	0.739
Adjusted model R^2	-	-	0.332	-	-	0.723
	1990-1994					
Average dwelling value	-	-	-	-0.0021	0.0003	0.503
Charitable donations	-0.0003	0.0001	0.219	-	-	-
Physical inactivity	0.0006	0.0004	0.040	-	-	-
Poor health status	-	-	-	0.0048	0.0026	0.046
Model R^2	-	-	0.259	-	-	0.549
Adjusted model R^2	-	-	0.216	-	-	0.523

5.2.2.3 SEX-SPECIFIC MODELS – MALES

The results from the regression of male cardiovascular disease on the risk factors are shown in Table 5.9. Average dwelling value and self-reported poor health status were still the dominant covariates in these male models. For example, during the period 1986-1989, over 80 per cent of the variation in male CVD mortality and over 70 per cent of the variation in morbidity was explained by average dwelling value and self-reported poor health status. Social capital, represented by licensed day care facilities, was also an important covariate, particularly in the morbidity models. During both periods, it

explained about 8 per cent of the variation in male CVD morbidity. Self-reported poor health status, which was retained in all the four male CVD models, was also important. During the first period, it explained 7 per cent of the variation in male CVD mortality and 5 per cent of the corresponding morbidity outcome. The predictive power of self-reported poor health status in the male CVD models somewhat decreased over time. For example, during the second period, it explained only about 4 per cent of the variation in male CVD mortality (Table 5.9).

Table 5.9 Results of multivariate regression for outcome: male CVD

Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0016	0.0001	0.768	-0.0013	0.0002	0.696
Licensed day care facilities	-	-	-	-0.0081	0.0025	0.079
Poor health status	0.0043	0.0011	0.073	0.0046	0.0014	0.053
Model R^2	-	-	0.841	-	-	0.828
Adjusted model R^2	-	-	0.832	-	-	0.812
	1990-1994					
Average dwelling value	-0.0019	0.0002	0.805	-0.0013	0.0002	0.682
Licensed day care facilities	-	-	-	-0.0081	0.0026	0.077
Poor health status	0.0036	0.0013	0.037	0.0047	0.0015	0.054
Model R^2	-	-	0.842	-	-	0.813
Adjusted model R^2	-	-	0.833	-	-	0.796

Table 5.10 shows the modeling results for male IHD mortality and morbidity. Average dwelling value and self-reported poor health status were retained as significant covariates in all the four models for male IHD morbidity. However, their predictive power appears to have diminished over time. For example, while average dwelling value explained 78 per cent and 70 per cent of the variation in male IHD mortality and morbidity respectively during the first period, it explained only about 76 per cent and 65 per cent of the variations in these outcomes respectively during the second period. In the case of IHD mortality, this resulted in a decrease in the variation explained from about 82 per cent during the first period to 78 per cent during the second period. In the case of

Table 5.10 Results of multivariate regression for outcome: male IHD

Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0020	0.0002	0.780	-0.0020	0.0002	0.701
Poor health status	0.0038	0.0014	0.039	0.0061	0.0017	0.081
Model R^2	-	-	0.819	-	-	0.782
Adjusted model R^2	-	-	0.808	-	-	0.770
	1990-1994					
Average dwelling value	-0.0025	0.0002	0.756	-0.0015	0.0003	0.646
Licensed day care facilities	-	-	-	-0.0084	0.0032	0.068
Poor health status	0.0040	0.0020	0.027	0.0069	0.0019	0.087
Model R^2	-	-	0.783	-	-	0.800
Adjusted model R^2	-	-	0.770	-	-	0.782

male IHD morbidity, however, the entry of licensed day care facilities into the model as an additional significant covariate during the second period resulted in an increase in the variation explained to 80 per cent from 78 per cent during the previous period.

As in the case of the female CBVD models, the variation explained of male CBVD mortality during both periods is much less than those of morbidity (Table 5.11). For instance, during the second period, the model explained about 66 per cent of the variation in male CBVD morbidity but only about 43 per cent of the variation in

Table 5.11 Results of multivariate regression for outcome: male CBVD

Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-	-	-	-0.0020	0.0002	0.671
Licensed day care facilities	-0.0150	0.0030	0.416	-	-	-
Excessive alcohol consumption	0.0012	0.0009	0.031	-	-	-
Model R^2	-	-	0.447	-	-	0.671
Adjusted Model R^2	-	-	0.414	-	-	0.662
	1990-1994					
Average dwelling value	-	-	-	-0.0021	0.0003	0.656
Licensed day care facilities	-0.0092	0.0023	0.353	-	-	-
Seniors living alone	0.0009	0.0004	0.076	-	-	-
Model R^2	-	-	0.429	-	-	0.656
Adjusted model R^2	-	-	0.396	-	-	0.646

mortality. The comparative weakness of the male CBVD mortality models suggests that certain factors that mediated male CBVD outcome during the period studied may have been excluded from this research, and offers scope for future studies of, for example, the determinants of variations in the rates of cerebrovascular disease outcomes.

5.3 COUNTY LEVEL SENSITIVITY ANALYSIS

The data on some of the potential CVD risk factors explored in this research were available only at the county level, and therefore a sensitivity analysis was done using these data to get an idea of the effect they would have had on the outcome variables. The risk factors in question are unemployment rate, incidence of low-income family, access to health care, and per capita municipal expenditure on environmental defense, social assistance, parks and recreations, and culture and libraries. This analysis was based on the proposition that since the county boundaries largely coincide with the public health unit boundaries, except in northern Ontario, the results of the county-level analyses would approximate those obtained in the PHU-level analyses. Average dwelling value – the most significant variable in the PHU-level regressions – was also included in this sensitivity analysis. This was done in order to ascertain whether the predictive power of this variable would remain high regardless of the spatial configuration of the relevant data. Table 5.12 shows the results of the regression of combined female and male CVD mortality and morbidity on the above predictors during the two periods. During both periods and for both mortality and morbidity, average dwelling value explained the largest proportion of the total variation in the outcomes. For example, while average

dwelling value explained almost 65 per cent of the variation in combined female and male CVD morbidity during the first period, doctors' location quotient and unemployment rate explained 4 per cent and 3 per cent respectively of this variation. During the second period, average dwelling value explained about 63 per cent of the variation in combined female and male CVD compared to municipal per capita

Table 5.12 Results of multivariate regression for outcome: combined female and male CVD

Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0015	0.0003	0.416	-0.0018	0.0003	0.648
Doctors location quotient	-	-	-	-0.2314	0.0815	0.041
Unemployment rate	-	-	-	0.0214	0.0103	0.027
Model R^2	-	-	0.416	-	-	0.716
Adjusted Model R^2	-	-	0.404	-	-	0.697
	1990-1994					
Average dwelling value	-0.0020	0.0003	0.489	-0.0021	0.0002	0.629
Per capita expenditure on environmental defense	-	-	-	-0.0003	0.0001	0.034
Model R^2	-	-	0.489	-	-	0.663
Adjusted Model R^2	-	-	0.478	-	-	0.648

expenditure on environmental defense, which explained 3 per cent of the variation. In the case of CVD mortality, average dwelling value was the only significant predictor during both periods.

The pattern for ischemic heart disease was not much different. Average dwelling value still explained the largest proportion of the variation in the combined female and male IHD outcomes – about 65 per cent and 62 per cent respectively of the variation in morbidity during the first and second periods. Compared to this, doctors' location quotient explained 4 per cent of the variation in combined female and male IHD morbidity during the first period. During the second period per capita expenditure on environmental defense replaced doctors' location quotient in the morbidity model, and it explained about 5 per cent of the variation (Table 5.13).

Table 5.13 Results of multivariate regression for outcome: combined female and male IHD

Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0021	0.0004	0.359	-0.0021	0.0003	0.648
Doctors location quotient	-	-	-	-0.2116	0.0945	0.035
Model R^2	-	-	0.359	-	-	0.683
Adjusted Model R^2	-	-	0.345	-	-	0.669
	1990-1994					
Average dwelling value	-0.0027	0.0005	0.388	-0.0025	0.0003	0.619
Per capita expenditure on environmental defense	-	-	-	-0.0005	0.0002	0.052
Model R^2	-	-	0.388	-	-	0.671
Adjusted Model R^2	-	-	0.375	-	-	0.656

The regression results for combined female and male CBVD mortality and morbidity are shown in Table 5.14. Average dwelling value dominated the morbidity models for the two periods. During the first period, it explained 64 per cent out of the total explained variation, which is about 68 per cent. It was the only significant variable

Table 5.14 Results of multivariate regression for outcome: combined female and male CBVD

Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-	-	-	-0.0018	0.0003	0.641
Doctors location quotient	-	-	-	-0.1793	0.0815	0.034
Unemployment rate	0.0606	0.0129	0.114	-	-	-
Incidence of low income family	-0.0350	0.0091	0.215	-	-	-
Model R^2	-	-	0.329	-	-	0.675
Adjusted Model R^2	-	-	0.299	-	-	0.661
	1990-1994					
Average dwelling value	-	-	-	-0.0020	0.0002	0.582
Model R^2	-	-	-	-	-	0.582
Adjusted Model R^2	-	-	-	-	-	0.573

retained in the morbidity model during the second period. The model for combined female and male CBVD mortality for the first period appears to be weak. Incidence of low-income family appears to explain a greater part of the variation – about 22 per cent out of 33 per cent total explained variation (Table 5.14). But, contrary to what might be

expected, its negative coefficient (-0.0349) implies that as the incidence of low-income family increases, the rate of combined female and male CBVD mortality decreases. During the second time period, none of the variables investigated was a significant explanatory factor for the regional variation in combined female and male CBVD mortality (Table 5.14).

The sex-specific outcome data were also modeled at the county level and the results are shown in Tables 5.15 – 5.17 for females and Tables 5.18 – 5.20 for males. The evidence presented in these tables suggests that average dwelling value was still the

Table 5.15 Results of multivariate regression for outcome: female CVD

Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0012	0.0003	0.240	-0.0020	0.0003	0.604
Doctors location quotient	-	-	-	-0.2439	0.0133	0.043
Model R^2	-	-	0.240	-	-	0.647
Adjusted Model R^2	-	-	0.224	-	-	0.632
	1990-1994					
Average dwelling value	-0.0017	0.0003	0.367	-0.0025	0.0003	0.582
Model R^2	-	-	0.367	-	-	0.582
Adjusted Model R^2	-	-	0.353	-	-	0.573

predictor that had the greatest explanatory power in the models. For instance, during both periods, average dwelling value was the only variable retained in the mortality models for female CVD (Table 5.15), female IHD (Table 5.16), male CVD (Table 5.18),

and male IHD (Table 5.19). It also accounted for the largest proportion of the variation explained by each of the morbidity models for these outcomes.

For male CBVD morbidity during both time periods, however, average dwelling value was the only significant predictor (Table 5.20). Other significant variables are per capita municipal expenditure on environmental defense (Table 5.16) unemployment rate and per capita municipal expenditure on social assistance (Table 5.17), and doctors' location quotient (Table 5.18).

Table 5.16 Results of multivariate regression for outcome: female IHD

Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0017	0.0005	0.169	-0.0023	0.0004	0.585
Doctors location quotient	-	-	-	-0.3334	0.1244	0.040
Unemployment rate	-	-	-	0.0392	0.0156	0.046
Model R^2	-	-	0.169	-	-	0.671
Adjusted Model R^2	-	-	0.152	-	-	0.649
	1990-1994					
Average dwelling value	-0.0028	0.0006	0.299	-0.0031	0.0004	0.589
Unemployment rate	-	-	-	0.0372	0.0173	0.036
Environmental defense	-	-	-	-0.0005	0.0001	0.034
Model R^2	-	-	0.299	-	-	0.659
Adjusted Model R^2	-	-	0.284	-	-	0.636

Table 5.17 Results of multivariate regression for outcome: female CBVD						
Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-	-	-	-0.0022	0.0003	0.613
Unemployment rate	0.0450	0.0113	0.141	-	-	-
Social assistance	-0.0006	0.0002	0.133	-	-	-
Model R^2	-	-	0.274	-	-	0.613
Adjusted Model R^2	-	-	0.243	-	-	0.605
Table 5.17 (continued)						
	1990-1994					
Average dwelling value	-	-	-	-0.0020	0.0003	0.501
Social assistance	-0.0005	0.0002	0.097	-	-	-
Model R^2	-	-	0.097	-	-	0.501
Adjusted Model R^2	-	-	0.078	-	-	0.491

Table 5.18 Results of multivariate regression for outcome: male CVD						
Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0018	0.0003	0.451	-0.0061	0.0002	0.682
Doctors location quotient	-	-	-	-0.1750	0.0661	0.030
Unemployment rate	-	-	-	0.0201	0.0084	0.032
Model R^2	-	-	0.451	-	-	0.745
Adjusted Model R^2	-	-	0.440	-	-	0.728

Table 5.18 continued						
	1990-1994					
Average dwelling value	-0.0022	0.0003	0.516	-0.0019	0.0002	0.668
Environmental defense	-	-	-	-0.0003	0.0001	0.033
Model R^2	-	-	0.516	-	-	0.701
Adjusted Model R^2	-	-	0.505	-	-	0.688

Table 5.19 Results of multivariate regression for outcome: male IHD						
Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-0.0023	0.0004	0.455	-0.0022	0.0002	0.671
Model R^2	-	-	0.455	-	-	0.671
Adjusted Model R^2	-	-	0.444	-	-	0.664
	1990-1994					
Average dwelling value	-0.0026	0.0004	0.412	-0.0021	0.0002	0.628
Environmental defense	-	-	-	-0.0004	0.0001	0.058
Model R^2	-	-	0.412	-	-	0.686
Adjusted Model R^2	-	-	0.399	-	-	0.672

Table 5.20 Results of multivariate regression for outcome: male CBVD						
Predictors	Mortality			Morbidity		
	1986-1989					
	b	S.E. of *b*	R^2 change	*b*	S.E. of *b*	R^2 change
Average dwelling value	-	-	-	-0.0020	0.0002	0.626
Model R^2	-	-	-	-	-	0.626
Adjusted Model R^2	-	-	-	-	-	0.618
	1990-1994					
Average dwelling value	-	-	-	-0.0021	0.0002	0.632
Unemployment rate	.0294	0.0104	0.144	-	-	-
Model R^2	-	-	0.144	-	-	0.632
Adjusted Model R^2	-	-	0.126	-	-	0.624

The above analyses show that regardless of the spatial configuration of the units of analysis used, average dwelling value exerted the largest influence on the geographic variations in cardiovascular disease mortality and morbidity in the Province during the time period considered in this research.

5.4 SUMMARY AND DISCUSSION

The statistical analyses described in this chapter suggest that, in general, the models explain substantial proportions of the geographic variations in cardiovascular disease in the study area. A number of issues arise from the results. First, the variables retained in these models span all the risk constructs in the conceptual model developed to guide this study (Figure 2.2), and thus provide some validation to the explanatory models.

Of these variables (Table 5.21), the ones representing the socio-economic and psychosocial environments accounted for the largest contribution (mostly over 90 per cent of the explained variation) in the models in which they occurred. This supports the suggestion that psychosocial risk factors could contribute to our explanation of the etiology of heart disease (Elliott 1995), and bears out the emphasis (in this study) on the importance of socio-economic and psychosocial risk factors in understanding the geographic variation in CVD in Ontario. The analyses illustrate the utility of an ecological level conceptual model in investigating the determinants of spatial variation in health outcomes. The challenge associated with such a model, however, is how to derive meaningful ecological level variables to represent its component constructs. In this study, for example, this difficulty was addressed by aggregating the data on individual level characteristics at the public health unit level.

Table 5.21 Variables retained in final models

Risk construct	Variables retained in models
Economic characteristics	Average dwelling value
Social capital	Licensed day care facilities Average charitable donations
Demographic characteristics	Less than high school education
Psychosocial health and well-being	Self-reported poor health status
Risk factor behaviours	Smoking Physical inactivity Excessive alcohol consumption
Social support	Low social participation Seniors living alone
Physiological characteristics	Obesity Diabetes

Second, the percentage of the geographic variations in CVD and IHD explained by the combined and sex-specific models in the two periods ranged between 64 per cent and 84 per cent, which is much greater than the 30 per cent explainable in terms of conventional risk factors (Chapter One). Thus, by including socio-economic and psychosocial variables in the explanatory models, not only has this research expanded the range of potential determinants of CVD outcomes in Ontario, it has also provided a basis for rethinking the emphasis on individual level, physiological and behavioural characteristics in CVD risk factor research and heart health programming. It is noteworthy that only two physiological risk factors – obesity and diabetes – were retained in the final models. Obesity was a significant predictor in the model for combined female and male CVD morbidity during the first period. It accounted for 5.2 per cent of the variation compared to average dwelling value, which accounted for 66.1 per cent. Diabetes was retained in five models, but its greatest contribution to the explained variation in a CVD outcome was 7.3 per cent for female IHD mortality during the second period. The only behavioural risk factors retained in the final models are smoking and excessive alcohol consumption. Smoking was a significant predictor only for combined female and male CBVD mortality and morbidity in the first period, explaining, respectively, 20.7 per cent and 5.4 per cent of these variations. Excessive alcohol consumption was a significant predictor for only male CBVD mortality in the first period, explaining 3 per cent of the variation (Table 5.11). When considering potential determinants of CVD, the importance of shifting emphasis towards the socio-economic and psychosocial environments is underscored in this study by the limited number of

these conventional risk factors retained in the final models, the limited number of models (for different CVD outcomes) in which these were significant, and their limited explanatory power (i.e., their contribution to the total explained variation). The limited explanatory power of these conventional risk factors could be due to the fact that there is little dispersion in their prevalence across public health units in the province (Tables 5.22 – 5.24). For example, the mean rates of combined female and male daily smoking, physical inactivity, and excess fat in diet are 26.5% (standard deviation [SD] = 3.4%), 55.5% (SD = 3.9%), and 78.3% (SD = 4.7%) respectively. Thus, these variables are not picked up as significant predictors in most of the multivariate regression models.

Third, the regression results show that average dwelling value was the most influential covariate in the models. This is not altogether surprising, as dwelling value is an indicator of both economic characteristics, such as permanent wealth, and psychosocial characteristics, such as personal worth, success and achievement in life, a sense of financial security, and social and neighbourhood status. For example, Jerrett, et al. (1997) suggest that average dwelling value is sometimes regarded as an index of permanent average income, representing a person's long term ability to pay, and that "the ability to pay for capital assets is usually based more on permanent income, as opposed to the more transitory annual income" (p.1794). In this research, the proportion of variation in outcome explained by average dwelling value remained high after controlling for sex. In the case of males, for example, the proportion explained increased for all CVD and IHD outcomes compared to when both sexes were combined.

Table 5.22 Age-standardized Prevalence Rates (%) in Ontario, 1986-1994 – Combined Females and Males			
Public health unit	Smoking	Physical inactivity	Excess fat in diet
Algoma	26.2	54.8	77.2
Brant	28.6	60.0	79.4
Bruce-Grey-Owen Sound	24.9	54.2	83.3
City of Toronto	20.3	55.1	65.8
Durham	29.2	54.6	77.7
Eastern Ontario	29.0	56.1	80.2
Elgin-St. Thomas	30.2	59.2	80.4
Haldimand-Norfolk	28.8	57.8	80.9
Haliburton-Kawartha	28.2	55.0	82.9
Halton	22.2	56.5	81.5
Hamilton-Wentworth	24.7	56.1	76.7
Hastings-Prince Edward	27.9	54.7	81.1
Huron	21.7	57.4	84.1
Kent-Chatham	25.8	56.7	80.4
Kingston-Frontenac-Lennox & Addington	25.1	58.1	79.8
Lambton	25.3	56.0	80.6
Leeds-Grenville-Lanark	28.4	53.4	79.8
Middlesex-London	25.3	54.9	75.7
Muskoka-Parry Sound	27.9	54.4	75.8
Niagara	23.3	59.2	80.2
North Bay	31.6	53.0	78.6
Northwestern	31.7	53.3	78.3
Ottawa-Carlton	24.1	48.6	77.1
Oxford	26.4	59.5	80.8
Peel	23.8	56.8	71.3
Perth	24.4	57.1	81.1
Peterborough	27.3	49.8	79.5
Porcupine	33.4	54.8	79.0
Renfrew	27.4	54.5	80.1
Simcoe	25.0	48.7	74.1
Sudbury	30.9	59.1	79.8
Thunder Bay	28.1	54.9	79.2
Timiskaming	30.4	58.4	80.0
Waterloo	22.8	56.0	79.0
Wellington-Dufferin-Guelph	27.7	56.4	82.2
Windsor-Essex	23.7	49.3	59.9
York Region	17.4	57.3	72.9

Table 5.23 Age-standardized Prevalence Rates (%) in Ontario, 1986-1994 – Females			
Public health unit	Smoking	Physical inactivity	Excess fat in diet
Algoma	22.7	61.9	76.2
Brant	24.2	65.4	78.4
Bruce-Grey-Owen Sound	21.7	58.8	81.4
City of Toronto	17.7	59.3	63.9
Durham	27.5	61.1	74.5
Eastern Ontario	27.2	58.7	79.0
Elgin-St. Thomas	26.1	60.9	76.5
Haldimand-Norfolk	25.9	63.1	80.0
Haliburton-Kawartha	23.9	60.3	83.6
Halton	20.3	61.0	80.0
Hamilton-Wentworth	21.4	60.3	74.3
Hastings-Prince Edward	24.6	55.8	76.8
Huron	19.0	61.9	83.2
Kent-Chatham	24.8	60.9	80.7
Kingston-Frontenac-Lennox & Addington	24.7	60.8	77.3
Lambton	24.2	61.3	77.3
Leeds-Grenville-Lanark	26.2	56.0	76.8
Middlesex-London	21.5	58.5	73.2
Muskoka-Parry Sound	27.6	58.7	76.8
Niagara	20.4	62.9	78.2
North Bay	30.9	58.2	77.1
Northwestern	30.6	58.3	75.5
Ottawa-Carlton	22.8	52.8	73.3
Oxford	22.6	61.7	80.4
Peel	20.1	60.6	69.3
Perth	20.2	61.3	78.2
Peterborough	28.6	53.6	78.7
Porcupine	31.9	58.2	77.7
Renfrew	25.7	60.1	79.6
Simcoe	23.7	52.7	73.9
Sudbury	29.5	62.1	78.0
Thunder Bay	25.9	61.0	75.6
Timiskaming	28.1	60.6	77.3
Waterloo	18.4	57.4	75.2
Wellington-Dufferin-Guelph	25.8	60.6	81.8
Windsor-Essex	22.0	57.3	59.5
York Region	15.8	63.4	71.3

Table 5.24 Age-standardized Prevalence Rates (%) in Ontario, 1986-1994 – Males			
Public health unit	Smoking	Physical inactivity	Excess fat in diet
Algoma	29.4	48.4	78.0
Brant	33.1	54.1	80.6
Bruce-Grey-Owen Sound	28.1	49.4	85.2
City of Toronto	23.2	50.7	67.9
Durham	30.9	47.9	80.7
Eastern Ontario	31.0	53.0	81.7
Elgin-St. Thomas	34.3	57.8	84.5
Haldimand-Norfolk	31.8	52.5	82.0
Haliburton-Kawartha	32.6	49.6	82.4
Halton	24.3	51.2	82.7
Hamilton-Wentworth	28.1	51.7	79.6
Hastings-Prince Edward	31.7	53.7	85.7
Huron	24.5	52.8	85.0
Kent-Chatham	26.9	52.5	80.0
Kingston-Frontenac-Lennox & Addington	25.8	55.5	82.8
Lambton	26.4	50.5	84.4
Leeds-Grenville-Lanark	30.6	50.6	83.3
Middlesex-London	29.6	50.4	78.2
Muskoka-Parry Sound	27.9	49.3	74.7
Niagara	26.4	55.4	82.4
North Bay	32.3	47.5	80.3
Northwestern	32.7	48.6	81.3
Ottawa-Carlton	25.1	44.5	81.5
Oxford	30.4	57.0	81.1
Peel	27.6	53.4	73.8
Perth	28.6	52.8	84.4
Peterborough	26.0	45.7	80.7
Porcupine	35.4	51.2	80.0
Renfrew	29.1	49.3	80.7
Simcoe	26.5	45.0	74.3
Sudbury	31.9	55.4	81.7
Thunder Bay	30.3	48.7	83.0
Timiskaming	33.2	56.3	83.1
Waterloo	27.4	54.7	83.1
Wellington-Dufferin-Guelph	30.0	52.1	82.5
Windsor-Essex	25.5	40.8	60.3
York Region	19.0	50.9	74.0

Lastly, although self-reported poor health status was less influential than average dwelling value, it was significant in several of the models. It was, however, significant in only two female-only models – CVD morbidity (1990-1994) and CBVD morbidity (1990-1994). Thus, self-reported poor health status appears to have had little or no impact on female cardiovascular outcome, particularly mortality, in Ontario during the period studied. For males, self-reported poor health status was significant in all the CVD and IHD models, but not the CBVD models. This probably reflects the pattern of the prevalence of self-reported poor health among older adults and seniors in Ontario. For the age groups 45-64 years, 65-74 years, and 75+ years, the prevalence of self-reported poor health is higher among males (3.9%, 4.4%, and 3.9% respectively) than among females (2.9%, 3.7%, and 3.5% respectively).

The analyses in this chapter show that a combination of socio-economic, psychosocial, behavioural, and physiological factors are associated with spatial variations in cardiovascular disease outcomes in Ontario. Generally, the proportion of explained variance for both mortality and morbidity is lower in the female models than in the male models. The mortality scenario is consistent with the findings of Jerrett et al. (1998) from an investigation of the socio-economic and environmental covariates of premature mortality in Ontario. As Jerrett et al. (1998) suggest, the lower proportion of explained variance is suggestive of the absence of important determinants of female cardiovascular mortality from the current models.

The results from the ecological level analyses suggest that the socio-economic and psychosocial environments play a larger role in determining the geographic variation

in cardiovascular events between PHUs than the so-called traditional risk factors – smoking, excessive fat intake, and lack of exercise. This finding is also consistent with the results of previous studies. For example, Raphael and Farrell (2002) note that biomedical and lifestyle factors account for rather small proportions of variance in CVD rates among populations (see also Lantz et al. 1998; Roux et al. 2001). As explained above, the diminished significance or absence of these risk factors in the explanatory models for the geographic variation in CVD outcomes in Ontario is largely due to the fact that there is little variation in their prevalence across the public health units. Since the socio-economic and psychosocial environments appear to play a significant role in determining the geographic variation in cardiovascular outcomes, public health policies and programmes aimed at reducing the rate of cardiovascular disease mortality and morbidity in the Province need to begin to focus on these factors in addition to the traditional risk factors. For example, the inverse social gradient in mortality from coronary heart disease observed among British civil servants in the first Whitehall study has been attributed to differences in the psychosocial work environment (Marmot et al. 1997); and it has been shown that attention to psychosocial factors can lighten the burden of ill health for working people and to make the social gradient in mortality and morbidity less steep (Marmot 1999). The regression results also suggest that a population health approach to addressing the risk factors for cardiovascular disease in the Province has a great potential for explaining the geographic variation in morbidity and mortality due to cardiovascular disease. These results are further addressed in the final chapter.

CHAPTER SIX

CONCLUSIONS

6.1 INTRODUCTION

This thesis described a statistical analysis of geographic variations in heart disease mortality and morbidity in Ontario during the period 1986-1994. The specific outcome measures were: all cardiovascular diseases (ICD-9 codes 390-459), ischemic heart disease (ICD-9 codes 410-414), and cerebrovascular disease (ICD-9 codes 430-438). Several issues defined the scope of the research. First, CVD constitutes an enormous mortality, morbidity, and economic burden at both the provincial and national levels (Chan and Young 1999; Heart and Stroke Foundation of Canada 1999), and it persists as the leading cause of death and disability in Canada (HSFC 1997a). Second, while there has been a rather modest decline in recent years in the proportion of total deaths attributable to CVD, deaths from all cause CVD in Ontario and Canada are projected to double by the year 2018 due to population growth and aging (Gallop and Naylor 1999; Heart and Stroke Foundation of Canada 1999). This is likely to translate into an increased burden on both the national and provincial economies. A related substantive issue is that while geographic variation in CVD mortality and morbidity rates have been observed across Ontario (Bondy et al. 1999), there is limited knowledge on the range of the determinants of these variations (Jaglal et al. 1999). In light of the above issues, there

is the need to gain more insight into the factors that drove the regional variations. In response to this need, the following objectives were addressed:

1. To describe the temporal and spatial variations in CVD mortality and morbidity rates in Ontario.
2. To examine the prevalence and distribution of a broad range of potential CVD risk factors in Ontario.
3. To model the geographic variation in CVD mortality and morbidity in Ontario.

6.2 SUMMARY OF FINDINGS

With respect to the first objective, the study found that there was little or no variation in the CVD mortality and morbidity rates over time (Section 4.3). A comparison of choropleth maps of the rates for the two time periods studied shows that the spatial pattern of rates remained virtually the same over time. It was not possible to test the statistical significance of change in the rates over time because the required data – year-by-year rates within each period – were not available at the PHU level. However, a parallel, county level analysis of the CVD outcome data showed that there was virtually no change in the rates between the two time periods (Inter-office Memorandum, Elliott and DeLuca to Heart & Stroke Project Team, 1999). The lack of temporal variation in the CVD outcome rates at the public health unit level could be due to the fact that there was little or no temporal change in the associated explanatory variables (Table 5.3 – 5.11).

The study found marked geographic variations in the CVD mortality and morbidity rates. The rates tended to be higher in northern Ontario than in southern Ontario. The spatial autocorrelation tests (Section 4.4) showed that there was spatial clustering in the data. Consistent with this general spatial pattern, the GIS analyses showed that there were hot spots of CVD mortality and morbidity. One dominant cluster of CVD hot spots was found in northern Ontario. The spatial extent of this cluster (in terms of the number of public health units that formed it) changed between the two time periods, and varied depending on the specific CVD outcome considered. These notwithstanding, it consisted mostly of Sudbury District, Algoma District, Thunder Bay, Porcupine, Timiskaming, and North Bay District (Figure 3.1). Occasionally, Muskoka-Parry Sound and Renfrew emerged as part of this cluster. In southern Ontario, the clusters identified consisted mainly of public health units with CVD outcome rates that were statistically dissimilar. Here, statistically significant public health units typically had neighbours with higher CMFs as well as neighbours with lower outcome rates. By interpretation (Schabenberger 1999, 2000) of the definition adopted in this study for the local Moran's *I* statistic (Anselin 1995), those significant PHUs did not constitute hot spots of CVD mortality or morbidity.

Regarding objective 2, this study found marked variations in the prevalence of the CVD risk factors investigated, particularly the non-traditional risk factors. The analyses revealed variations in the rates by sex, age, level of education, whether the public health unit is urban or rural, and whether it is in northern or southern Ontario. Generally, the prevalence of risk factors was higher among males than among females (Tables 4.35 –

4.41), which suggests more exposure to health risks. A few of the risk factors showed generally decreasing prevalence rates with age, e.g., dwelling needs major repairs (Table 4.35), experiencing stressful life (Table 4.38), daily smoking, excess alcohol consumption, physical inactivity, and excess fat in diet (Table 4.39). On the other hand, less than high school education (Table 4.37), dissatisfaction with health and physical activity limitation (Table 4.38), and hypertension and diabetes (Table 4.41) became more prevalent with increasing age. Most of the risk factors were more prevalent among those with less than high school education than among those with high school or higher education. This appears to support the view that education – a gateway to fundamental social resources such as knowledge and influence (Jerrett et al. 1998) – has a moderating effect on these risk factors. The education variable was retained in a few of the models as a significant predictor of regional variation in CVD outcome during the study period. This is consistent with the CVD risk factor literature, which indicates that less than a high school education is positively associated with congestive heart failure (He et al. 2001), coronary heart disease (Shestov et al. 1993), and the risk of having a myocardial infarction triggered by isolated episodes of anger (Mittleman et al. 1997). The results here are also consistent with the findings of a previous study that estimated the prevalence and distribution patterns of CVD and selected risk factors in the adult population of Ontario (Kirk-Gardner and Steven 1994). They found that prevalence of smoking in the Province was higher among males than among females; physical inactivity was more prevalent among females than among males; and obesity was more prevalent among males in each age group than among their female counterparts. The

prevalence of smoking, physical inactivity, and excess fat in diet, however, varied little across public health units. The present analysis revealed that about 2.8 per cent of the adult population in Ontario was diabetic. This compares well with the 3.0 per cent prevalence reported by Kirk-Gardner and Steven. The prevalence of many of the CVD risk factors was higher in rural public health units than in urban public health units. Similarly, the rates were higher in northern Ontario than in southern Ontario for most of the risk factors (Tables 4.35 – 4.41).

The variables retained in the multivariate regression models (Table 5.21) span all the risk constructs in the conceptual model developed in this study (Figure 2.2) thereby providing some validation. Of these variables, those representing the socio-economic and psychosocial environments accounted for the largest contribution in the models in which they occurred, indicating the role of psychosocial risk factors in the etiology of heart disease (Elliott 1995). Compared to using only smoking, excess fat in diet, and physical inactivity as explanatory variables, the inclusion of socio-economic and psychosocial variables in the explanatory models resulted in a large increase in the proportion of the geographic variations in cardiovascular disease outcomes explained. Smoking was retained in only two models (Table 5.5) while physical inactivity was retained in only one model (Table 5.8). Excess fat in diet was not retained in any of the models. The limited explanatory power of these conventional risk factors could be due to the fact that there is little dispersion in their prevalence across the public health units (Tables 5.22 – 5.24). Thus, there is insufficient evidence in this study to indicate the impact the mandatory public health programmes of Ontario public health departments

may have had on the geographic variations in CVD outcomes. Since the mandatory programmes for heart disease included the prevention of tobacco use and the promotion of physical activity and nutrition, the lack of variation in the prevalence of these risk factors suggests that any impacts of the mandatory surveillance and prevention programmes on them were similar for the public health units.

Self-reported poor health status was retained in several of the models. However, these included only two female-only models – CVD morbidity (1990-1994) and CBVD morbidity (1990-1994), reflecting the prevalence of self-reported poor health status among older adults and seniors in Ontario. For those aged 45 years and over, the prevalence of self-reported poor health is higher among males than among females (Table 4.46). Overall, the retention of this variable in some of the models in this study highlights the role of the psychosocial environment as a potential determinant of variations in heart health outcome.

Average dwelling value emerged as the most influential covariate in the models, being retained in over 80 per cent of the models and accounting for between 50 per cent (Table 5.8) and 81 per cent (Table 5.9) of the variation in the models in which it was retained. The proportion of variation in CVD outcome explained by average dwelling value remained high after controlling for sex (Tables 5.9 and 5.10), indicating the robustness of this variable in the models.

6.3 CONTRIBUTIONS OF THE STUDY

This research is one of few ecological level studies on regional variations in cardiovascular disease outcomes undertaken at the level of local health administrative units within a province. It marks a beginning in understanding the factors that underlie the differing levels of cardiovascular outcomes across local areas. In realizing its objectives, this study has made theoretical, methodological, and substantive contributions.

6.3.1 THEORETICAL CONTRIBUTIONS

An important theoretical contribution of this study is the development of a conceptual model (Figure 2.2) to guide the investigation of the underlying causes of geographic variation in cardiovascular disease outcomes. The conceptual utility of this model, which is informed by the population health perspective (Evans et al. 1994), is that it allows for the organization of potential risk factors for CVD into constructs within which a range of variables can be conceptualized as determinants of spatial variation in cardiovascular disease outcomes. The effectiveness of the model is borne out by the large proportion of the geographic variation in cardiovascular disease in Ontario that it has helped to explain (Chapter Five).

However, there are challenges to be addressed in operationalizing this framework. While for some constructs in the model, such as the economic environment, it is relatively easy to conceptualize representative risk factors such as average income (Diez-Roux et al. 2000), income inequality (Kawachi et al. 1997; Bruce et al. 1998; Wilkinson

1997; Roux et al. 2000; Ross et al. 2000), and unemployment rate (Mattiasson et al. 1990; Brenner 1997; Weber and Lehnert 1997), it is more difficult to do so for other constructs in the model, such as social capital (Kawachi et al 1997; Lomas 1998; McCarthy 2000). Various individual level components of social capital have been suggested, e.g., interpersonal trust, reciprocity, and mutual aid (Coleman 1988; Putnam et al. 1993; Kawachi 1999); and norms and networks of civic engagement (Putnam 1995). Some studies have used social support variables such as frequency of socialization with family members and with friends, volunteering, and membership in clubs and associations as components of social capital (Veenstra 2000). The conceptual challenge in using these component variables of social capital in the model is that their contextual influences upon health are less easily discerned empirically (Veenstra 2000).

There is also a challenge pertaining to the substantive implications of the model. It would be a relatively straightforward endeavour to formulate policies aimed at increasing average incomes, and reducing income inequality and unemployment. But, this may not be the case with social capital, for example, the definition of which varies greatly (Grootaert 1998). In this study, the representative variable for social capital retained in the final models is licensed day care facilities. Although it has been suggested that the availability of licensed day care facilities enhances social capital (Wilkinson and Marmot 1998), it is not immediately obvious, for instance, how providing more licensed day care facilities can augment social capital. Part of the reason lies in the fact that there is a paucity of good theoretical accounts of how to build social capital (Kawachi et al.

1997), although there are many accounts of how it can be diminished by various socio-economic forces (Putnam 1995).

Although all the constructs in the conceptual model are represented in the final models, the analyses show that variables representing the socio-economic and psychosocial environments are the most dominant explanatory variables in the models. On the other hand, the analyses revealed that variables representing behavioural and physiological characteristics, though significant, were among the weakest predictors in the models in which they were retained. Therefore, these findings highlight the need, when searching for the determinants of geographic variations in heart health outcome, to begin to shift emphasis away from the so-called traditional risk factors (or classical risk factors [Gensini et al. 1998]), toward non-traditional risk factors, such as the socio-economic environment (Marmot and Mustard 1994; Wilkinson 1997; Jerrett et al. 1998; Pickering 1999; Bartley et al. 2000) and the psychosocial environment (Elliott 1992, 1995; Eyles et al. 1993; Elliott and Dean 1998; Rozanski 1999; Tennant 1999; Black and Garbutt 2002; Cole et al. 2002). The results of this study constitute additional empirical evidence for the importance of considering context when investigating variations in health outcomes (Eyles and Donovan 1990; Wilkinson 1996; Syme 1996; Diez-Roux 1998).

6.3.2 METHODOLOGICAL CONTRIBUTION

The methodological contribution of this research is mainly in the illustration of how GIS and spatial analytical techniques can be applied in studying variations in

chronic disease outcomes, particularly when the research question has a spatial dimension to it. Specifically, this study used the S-PLUS (spatial statistics) extension of the ArcView GIS program, first, to test for the presence of spatial clustering in the CVD outcome data, and second, to identify significant local clusters of elevated CVD mortality and morbidity rates (or hot spots). The application of the GIS/spatial statistics technique also made it possible to map the results of these analyses. These analyses would not have been possible using standard statistical techniques alone. To date, the two only other ecological level studies of the spatial variations in cardiovascular disease outcome at this level of geography – Wing et al (1990) and Pickle and Gillum (1999) – did not incorporate GIS analytical techniques. Wing and colleagues investigated geographic variation of CVD mortality within the United States. They addressed the question of whether relative geographic inequality in the mortality rates (as measured by the weighted coefficient of variation of State Economic Area rates) increased or decreased during the period 1962-1982. Their results suggest that factors influencing the per cent decline of CVD mortality were not reaching communities of the U.S. equally. The application of GIS and spatial analysis techniques could have enhanced the understanding of the geographic variations in the health outcomes they investigated (Gatrell and Loytonen 1998; Rushton 1998; Loytonen 1998), particularly within the context of a systematic approach to the formulation and testing of hypotheses (Jacquez 1998). For example, their analysis would have revealed whether or not there was significant clustering of communities that experienced relative decline of CVD mortality, those that experienced stable rates, and those communities where the rate increased, which would

lead to hypotheses about the underlying dynamics of such patterns. Similarly, Pickle and Gillum's description of variations in the geographic patterns of both coronary heart disease and stroke mortality in the United States between 1988 and 1992 could have been greatly enhanced by incorporating GIS analytical techniques.

The ecological approach adopted in this study facilitated the investigation of the regional variation in cardiovascular disease outcomes in the Province by allowing the use of geographic areas (the public health units) as the units of statistical analyses. This methodology facilitated the identification of clusters of elevated CVD outcome rates (or 'hot spots'). Therefore, the adoption of the ecological level approach had substantive implications, which are described in the next section.

6.3.3 SUBSTANTIVE CONTRIBUTIONS

The findings of this study have several implications. First, the absence of temporal change in the CVD rates, as mentioned above, suggests that the rates more or less stabilized during the time period studied. This is important information that could be useful for heart health programming in the Province. Second, the concentration of the hot spots of CVD mortality and morbidity in northern Ontario suggests that greater attention needs to be paid to this region when addressing the underlying determinants of the geographic variations in the CVD outcomes in the Province. Third, since the prevalence of the CVD risk factors identified in this study vary by age, sex, level of education, type of public health unit, and relative location in Ontario, these will have substantive

implications to the extent they are factored into heart health-related public health policies and programmes in the Province.

As noted above, the limited explanatory power of the traditional risk factors suggests the need to consider the potential role of the socio-economic and psychosocial environments when looking for the determinants of CVD outcomes. This is important for policy considerations (or reconsiderations) because efforts to reduce CVD mortality through lifestyle modification and cholesterol reduction, for example, have rather limited efficacy (O'Loughlin et al. 1999; Fitzpatrick 2001; Raphael and Farrell 2002). In contrast, Raphael and Farrell (2002) maintain that CVD can be seen as emanating from processes of material deprivation, excessive psychosocial stress, and societal features that lead to unhealthy behaviours. By using the ecological level model in Figure 2.2, this research has expanded the range of potential determinants of CVD outcomes in Ontario. It has also provided a basis for rethinking the emphasis on individual level, physiological and behavioural characteristics in CVD risk factor research and heart health programming.

6.4 FUTURE RESEARCH DIRECTIONS

Although this study enhances an understanding of some of the factors that underlie the spatial variations in cardiovascular disease mortality and morbidity in Ontario during the time period studied, a number of questions remain to be addressed that pertain to theory, substantive issues, and methodology. With respect to theory, it would be useful to ascertain how well the conceptual model developed and used in this study

would perform given a different geographic setting, such as another province of Canada; or given a different spatial scale, such as the provincial level (with the provinces as the units of analysis). It would also be useful to know how well the model would perform given a different chronic disease, say, diabetes or cancer. An affirmative finding would further buttress the suggestion that contextual influences play a major role in determining health status (Wilkinson 1996; Syme 1996). This study was conducted at the ecological level, using data on potential determinants that were aggregated at the public health unit level. A key question, then, is: will the models explain the same or similarly high proportions of the geographic variations in cardiovascular disease outcomes at the individual level of analysis? Further, will the same set of significant predictors emerge if the investigation is repeated at the individual level, and, particularly, will average dwelling value still be the single most influential variable to be associated with geographic variation in CVD outcome in the Province?

There are a number of substantive issues arising from this research that warrant further investigation. The first concerns the time period covered by the study – 1986-1994. Although the analyses revealed that the CVD outcome rates appeared to stabilize over the period studied, the question remains as to whether this trend has remained the same or changed since 1994. If the latter has occurred, what has been the direction of the change? A related question would need to be addressed: was this new trend in the outcomes associated with the same sets of factors as pertained during the 1986-1994 period?

A second issue concerns the role of ethnicity in the observed geographic variations in CVD outcomes in the Province. It has been reported, for instance, that there are higher rates of mortality from diabetes and cardiovascular disease in First Nations populations than in non-indigenous populations in Canada (Johnson et al. 2002; Jim et al 2002; Pioro et al. 1996). Also, a north-south gradient has been observed in the prevalence of diabetes among First Nations in Canada with the lowest rates in the north (Martin and Yidegiligne 1998). Due to lack of good data, however, it was not possible to include an ethnicity variable in the current investigation. If good quality ethnicity data become available, it would be useful to explore the extent to which ethnicity impacts the observed geographic variation in cardiovascular mortality and morbidity in Ontario.

The third substantive issue arises from the fact that, generally, the morbidity models in this study appeared to perform better than the mortality models. This suggests that there was an additional variable that was not included in the mortality analyses, which had a significant influence on the CVD mortality levels. As explained in Chapter Five, the absence of a variable on access to health care in the public health unit level analyses is probably not sufficient explanation. This is because, although the county-level analyses provided useful insight into the impact access to health care might have on spatial variations in the outcome rates at the public health unit level, the question still remains as to how big such impact would be. Therefore, further work is needed on the range of potential determinants of CVD mortality in the Province. It will be important to investigate any moderating effects of access to curative health interventions on such potential determinants of CVD mortality. As well, it will be important to explore the

impact of access to preventive health care and health promotion intervention on the regional variation in CVD morbidity.

Finally, the dominance of average dwelling value in the explanatory models raises an important substantive question deserving of further research: which of the variables indexed by dwelling value can be manipulated for cardiovascular benefit? As stated in Chapter Two, dwelling value is sometimes regarded as indicative of permanent average income, representing a person's long-term ability to pay. So, its dominance in the models suggests that it is a sensitive indicator of 'wealth', and flags the need for further work to establish conceptual and empirical links to CVD outcomes. This would provide a basis for its manipulation with the view to improving upon the cardiovascular scenario in the Province.

An important methodological challenge associated with the conceptual model developed in this study was how to quantify the variables that represent the social capital, psychosocial health, and social support constructs. While variables such as income inequality and average dwelling value were fairly easily measured quantitatively, others such as health status and social participation were not easily quantifiable. The latter lend more easily to qualitative methods of measurement. Given that this study was conducted at the ecological level using exclusively quantitative methods, proxies had to be used, such as the proportion of the target population who were in a particular category. One solution that can be incorporated in similar research in future is to adopt a mixed method of analysis, which accommodates the use of both quantitative and qualitative methods of analysis (Scherer and Lane 1997; Sandelowski 2001; Fawcett et al. 2001).

This research illuminates the regional variation in cardiovascular disease mortality and morbidity in Ontario. By adopting a socio-ecological approach to analysis, it has identified social system variables as the key factors driving these regional variations. As part of the current debate on the etiology of cardiovascular disease, it indicates that in Ontario the potential determinants are, to a large extent, located "beyond biomedicine and lifestyle" (Raphael and Farrell 2002: iii). The evidence here is suggestive of, as well as supports, the call made at the Fourth International Heart Health Conference in Japan in 2001 – The Osaka Declaration – for the need to take the necessary actions on the social, economic, and political factors that contribute to the epidemic of cardiovascular disease.

BIBLIOGRAPHY

Alter, D.A., C.D. Naylor, P. Austin, and J.V. Tu 1999. Effects of socioeconomic status on access to invasive cardiac procedures and on mortality after acute myocardial infarction. *New England Journal of Medicine* 341(18):1359-1367.

Amick, B.C., S. Levine, A.R. Tarlov, and D.C. Walsh (eds). 1995. *Society and Health.* New York: Oxford University Press.

Anderton, D.L., A.B. Aderson, J.M. Oakes, and M.R. Fraser. 1994. Environmental equity: the demographics of dumping. *Demography* 31:229-248.

Anselin, L. 1992. *SpaceStat Tutorial: A Workbook for Using SpaceStat in the Analysis of Spatial Data.*

Anselin, L. 1995. Local indicators of spatial association – LISA. *Geographical Analysis* 27(2):93-115

Avlund, K., M.T. Damsgaard, and B.E. Holstein. 1998. Social relations and mortality. An eleven year follow-up study of 70-year-old men and women in Denmark. *SSM* 47:635-43.

Bailey, T.C., and A.C. Gatrell. 1995. *Interactive spatial data analysis.* Edinburgh Gate, Harlow, England: Longman.

Barrett, F. A. 1986. Medical Geography: Concept and Definition. In *Medical Geography: Progress and Prospect*, ed, Pacione, M. 1-34, London: Croom Helm.

Bartley, M., R. Fitzpatrick, D. Firth, and M. Marmot. 2000. Social distribution of cardiovascular disease risk factors: change among men in England 1984-1993. *Journal of Epidemiology & Community Health* 54(11):806-814.

Basinski, A.S.H. 1999. Hospitalization for cardiovascular medical diagnosis. In *Cardiovascular Health & Services in Ontario: An ICES Atlas,* eds, David C. Naylor and Pamela M. Slaughter, 15-49. Toronto: Institute for Clinical Evaluative Sciences.

Beegom, R., and R.B. Singh. 1997. Association of higher saturated fat intake with higher risk of hypertension in an urban population of Trivandrum in south India. *International Journal of Cardiology.* 58(1):63-70

Bendel, R.B. and A.A. Afifi. 1977. Comparison of stopping rules in forward regression. *J. Amer. Statistical Association* 72:46-53.

Bjorntorp, P. 1997. Body fat distribution, insulin resistance, and metabolic diseases. *Nutrition*. 13(9):795-803.

Black, D., J.N. Morris, C. Smith, P. Townsend, and M. Whitehead. 1988. *Inequalities in Health: The Black Report. The Health Divide*. London: Penguin.

Black, P.H. and Garbutt, L.D. 2002. Stress, inflammation and cardiovascular disease. *Journal of Psychosomatic Research*. 52(1):1-23.

Blair, S. N. and S. Brodney. 1999. Effects of physical inactivity and obesity on morbidity and mortality: current evidence and research issues. *Medicine & Science in Sports & Exercise* 31(11 Suppl):S646-62.

Bolinder, G. 1997. Overview of knowledge of health effects of smokeless tobacco. Increased risk of cardiovascular diseases and mortality because of snuff. *Lakartidningen*. 94(42):3725-31.

Bondy, S.J., S. Jaglal, and P. Slaughter. 1999. Area Variation in Heart Disease Mortality Rates. In *Cardiovascular Health & Services in Ontario: An ICES Atlas*, eds, David C. Naylor and Pamela M. Slaughter, 51-62. Toronto: Institute for Clinical Evaluative Sciences.

Braunwald E, Pfeffer M, Sacks F. CARE Trial results. 1996. Reported at the American College of Cardiology 45th Annual Scientific Session, Orlando, Fla.

Brenner, M.H. 1997. Heart disease mortality and economic changes; including unemployment; in Western Germany 1951-1989. *Acta Physiologica Scandinavica. Supplementum* 640:149-52.

Bruce, P.K., I. Kawachi, D. Prothrow-Smith, K. Louchner, and V. Gupta. 1998. Social capital, income inequality, and firearm violent crime. *SSM* 47(1):7-17.

Brunner, E.J. 1997. Stress and the biology of inequality. *British Medical Journal* 314: 1272-76.

Burnley, I.H. 1998. Inequalities in the transition of ischaemic heart disease mortality in New South Wales, Australia, 1969-1994. *Soc Sci Med* 47(9): 1202-1222.

Butchart, A. 1998. *The Anatomy of Power. European Constructions of the African Body*. New York: Zed Books.

Byles, J., C. Byrne, M. H. Boyle, and D. R. Offord. 1988. Ontario Child Health Study: Reliability and Validity of the General Functioning Subscale of the McMaster Family Assessment Device. *Family Process* 27(1):97-104.

Canadian Cardiovascular Society Consensus Conference, National Cardiovascular Disease Surveillance System. Risk indicators worksheet R-1. 13-14 November, 1998.

Canadian Heart Health Surveys Research Group. 1992. The Federal-Provincial Canadian Heart Health Initiative. *Canadian Medical Association Journal* 146(6): 1-2.

Cannon, R.O. 1998. Role of nitric oxide in cardiovascular disease: focus on the endothelium. *Clinical Chemistry.* 44(8 Pt 2):1809-19.

Case, R.B., A.J. Joss, N. Case, M. Mcdermott, and S. Eberly. 1992. Living alone after myocardial infarction. *Journal of the American Medical Association* 267:515-19.

Castelli WP, Garrison RJ, Wilson PW, Abbott RD, Kalousdian S, Kannel SB. 1986. Incidence of coronary heart disease and lipoprotein cholesterol levels: Framingham Study. *JAMA* 256:2835-8.

Cattell, V. 2001. Poor people, poor places, and poor health: the mediating role of social networks and social capital. *SSM* 52(10):1501-1516.

Chan, B., and W. Young. 1999. Burden of cardiac disease. In *Cardiovascular Health & Services in Ontario: An ICES Atlas*, eds, David C. Naylor and Pamela M. Slaughter, 1-13. Toronto: Institute for Clinical Evaluative Sciences.

Chatterjee, S. and B. Price. 1991. *Regression analysis by example.* 2nd edition. New York: Wiley.

Cheung, Y.B. 2000. Marital status and mortality in British women: a longitudinal study. *International Journal of Epidemiology* 29(1):93-9.

Christenfeld, N. and Gerin, W. 2000. Social support and cardiovascular reactivity. *Biomedicine & Pharmacotherapy* 54(5):251-7

Cliff, A. and J.K. Ord. 1981. Spatial Processes: Models and Applications. London, England: Pion Limited.

Cliff, A.D. and M.R. Smallman-Raynor. 1992. The AIDS pandemic: global geographical patterns and local spatial processes. *Geographical Journal* 158:182-98.

Coburn, D. 2000. Income inequality, social cohesion and the health status of populations: the role of neo-liberalism. *Soc Sci Med* 51:135-146.

Coburn, D. and Poland, B. 1996. The CIAR Vision of the Determinants of Health: A Critique. *Can J. Public Health* 87(5):308-10.

Coelho, R., E. Ramos, J. Prata, and H. Barros. 2000. Psychosocial indexes and cardiovascular risk factors in a community sample. *Psychotherapy & Psychosomatics* 69(5):261-74.

Coleman, J.S. 1988. Social capital in the creation of human capital. *American Journal of Sociology* 94:S95-121.

Colhoun, H.M., H. Hemingway, and N.R. Poulter. 1998. Socio-economic status and blood pressure: an overview analysis. *Journal of Human Hypertension* 12(2):91-110.

Colombel, A. and Charbonnel, B. 1997. Weight gain and cardiovascular risk factors in the post-menopausal women. *Human Reproduction.* 12 Suppl 1:134-45.

Craddock, S. 2000. Disease, social identity, and risk: rethinking the geography of AIDS. *Trans Inst Br Geogr* NS 25:153-168.

Crighton, E J., S.J. Elliott, R, Upshur, J. van der Meer, and I. Small. (In press). The Aral Sea disaster and self-rated health. *Health and Place.*

Crombie, I.K., W.C. Smith, R. Tavendale, and H. Tunstall-Pedoe. 1990. Geographical clustering of risk factors and lifestyle for coronary heart disease in the Scottish Heart Health Study. *British Heart Journal* 64(3):199-203.

Cullen, P., H. Schulte, and G. Assman. 1998. Smoking, lipoproteins and coronary heart disease risk. Data from the Munster Heart Study (PROCAM). *European Heart Journal* 19(11):1632-41.

Curtis, S. and A. Taket. 1996. *Health and Society: changing perspectives.* London: Holder Headline Group.

Davidson, R. and J. G. MacKinnon. 1993. *Estimation and Inference in Econometrics.* New York, NY: Oxford University Press.

Dennis BH, Zhukovsky GS, Shestov DB, Davis CE, Deev AD, Kim H, *et al.* 1993. The association of education with coronary heart disease mortality in the USSR Lipit Research Clinics Study. *Int J Epidemiol* 22:420-7.

Denton, M. and V. Walters. 1999. Gender differences in structural and behavioral determinants of health: an analysis of the social production of health. *SSM.* 48(9):1221-1235.

DeSouza et al., 1997. Plasma fibrinogen levels in healthy postmenopausal women: physical activity and hormone replacement status. *Journals of Gerontology. Series A, Biological Sciences & Medical Sciences* 52(5):M294-8.

Diez-Roux, A.V. 1998. Bringing context back into epidemiology: variables and fallacies in multilevel analysis. American Journal of Public Health 88(2): 216-22.

Diez-Roux, A.V., M.E Northridge, A. Morabia, M.T. Bassett, and S. Shea. 1999. Prevalence and Social Correlates of Cardiovascular Disease Risk Factors in Harlem. *Am J Public Health* 89:302-307.

Diez-Roux, A.V., B.G. Link, and M.E. Northridge. 2000. A multilevel analysis of income inequality and cardiovascular disease risk factors. *SSM* 50(5):673-87.

Doll, R., and R. Peto. 1976. Mortality in relation to smoking: 20 years' observation in male British doctors. *Br Med J* 2:1525-36.

Donnan, G.A., et al, 1993. Smoking as a risk factor for stroke. *Cerebrovascular Disease* 3:129-138.

Dunn, J.R. 2002. Housing and inequalities in health: a study of socioeconomic dimensions of housing and self reported health from a survey of Vancouver residents. *Journal of Epidemiology & Community Health* 56(9):671-81.

Dunn, J.R. and M.V. Hayes. 1999. Identifying social pathways for health inequalities. The role of housing. *Annals of the New York Academy of Sciences* 896:399-402.

Dunn, J.R., and M.V. Hayes. 1999. Toward a lexicon of population health. *Canadian Journal of Public Health* 90 Suppl 1:S7-10.

Edwards, N. 1999. Population health: determinants and interventions. *Can J. Public Health* 90(1):10-1

Elliott, S.J. 1992. *Psychosocial impacts in populations exposed to solid waste facilities.* A Thesis Submitted to the School of Graduate Studies in partial fulfillment of the requirements for the Degree of Doctor of Philosophy. McMaster University.

Elliott, S.J. 1995. Psychosocial stress, women and heart health: a critical review. *Soc Sci Med* 40(1): 75-89.

Elliott, S.J., and A. Dean. 1998. An ecologic analysis of psychosocial stress and heart disease in British Columbia. *Canadian Journal of Public Health* 89(2): 137-41.

Elliott, S.J., S.M. Taylor, R. Cameron, and R. Schabas. 1998. Assessing public health capacity to support community-based heart health promotion: the Canadian Heart Health Initiative, Ontario Project (CHHIOP). *Health Education Research.* 13(4):607-622.

Elliott, S.J. 1999. And the Question Shall Determine the Method. *The Professional Geographer* 51(2): 240-3

Elliott, S.J. and DeLuca, P. (Unpublished). Analysis of change over time. Inter-office Memorandum to Heart & Stroke Project Team, July 27, 1999. McMaster University.

Elliott, S.J., S.D. Walter, T. Abernathy, S. Martin Taylor, P.F. DeLuca, and G.A. Djietror. 2003. Final Report to Heart and Stroke Foundation of Ontario: The 'Hot Spots' Project. Hamilton, Ontario: McMaster University

Epp, J. 1986. Achieving Health for all: A Framework for Health Promotion. Ottawa, ON: Health and Welfare Canada.

Evans, R.G. 1994. Introduction. In *Why are some people healthy and others not*, eds. Evans *et al. 3-26.* Hawthorne, N.Y: ALDINE DE GRUYTER.

Evans, R.G., M. Barer, and T.R. Marmor. 1994. *Why are some people healthy and others not*?: *The Determinants of Health of Populations.* Hawthorne, N.Y: ALDINE DE GRUYTER.

Evans, R.G., and G.L. Stoddart. 1990. Producing Health, Consuming Health Care. *Soc. Sci. Med.* 31(12): 1347-1363.

Evans, R.G., and G.L. Stoddart. 1994. Producing Health, Consuming Health Care. In *Why are some people healthy and others not*, eds. Evans *et al.*27-64. Hawthorne, N.Y: ALDINE DE GRUYTER.

Eyles, J. 1990. How significant are the spatial configurations of health care systems? *SSM* 30 (1): 157-64.

Eyles, J. and J. Donovan. 1990. "I couldn't afford to be ill": Experiences of Health and Illness in Contemporary Britain. Aldershot: Avebury.

Eyles, J., S. Birch, S. Chambers, J. Hurley, and B. Hutchison. 1991. A needs-based methodology for allocating health care resources in Ontario, Canada: development and an application. *Soc Sci Med* 33: 489-500.

Eyles, J.D. 1999. Health, environmental assessments and population health: tools for a complex process. *Canadian Journal of Public Health* 90 Suppl 1:S31-4

Eyles, J., S. Martin Taylor, N. Johnson, and J. Baxter. 1993. Worrying about waste: living close to solid waste disposal facilities in Southern Ontario. *SSM* 37 (6): 805-812.

Eyles, J., M. Brimacombe, P. Chaulk, G. Stoddart, T. Pranger, and O. Mouse. 2001. What determines health? To where should we shift resources? Attitudes towards the determinants of health among multiple stakeholder groups in Prince Edward Island, Canada. *SSM* 53: 1611-1619.

Fagard, R.H. 1993. Physical fitness and blood pressure. *Journal of Hypertension* - Supplement. 11 Suppl 5:S47-52.

Faresjo, T. 1992. Social environment and health--a social epidemiological frame of reference. *Scandinavian Journal of Primary Health Care*. 10(2):105-10.

Farley et al. 1998. Hormonal contraception and risk of cardiovascular disease. An international perspective. *Contraception* 57(3):211-30.

Fawcett, J., Watson, J., Neuman, B., Walker, P. H., Fitzpatrick, J. 2001. An invitation for dialogue about theory-guided, evidenced-based holistic nursing practice. Journal of Nursing Scholarship, 33, 115-119.

Federal Provincial and Territorial Advisory Committee on Population Health. 1994. *Strategies for Population Health: Investing in the Health of Canadians*. Halifax: Health Canada.

Ferrie, J.E., M.J. Shipley, et al. 1995. Health effects of anticipation of job change and non-employment: longitudinal data from the Whitehall II study. *BMJ*. 311(7015):1264-9

Foster, L. And M. Edgell. 1992. The Geography of Death: Mortality Atlas of British Columbia, 1985-89. Western Geographical Series. Vol. 26. Victoria, B.C: University of Victoria.

Frank, J.W. 1995. Why population health? *Canadian Journal of Public Health* 86(3):162-164.

Frankish, J., G. Veenstra, and G. Moulton. 1998. Population Health in Canada: Issues and Challenges for Policy, Practice and Research. *Can J. Public Health* 90 Suppl. 1:S71.

Fitzpatrick, M. 2001. *The Tyranny of Health: Doctors and the Regulation of Lifestyle.* London: Routledge.

Frohlich, E.D., et al. 1992. The heart in hypertension. *New England Journal of Medicine* 327: 998-1008.

Fuller-Thomson, E., J.D Hulchanski., and S. Hwang. 2000. The housing/health relationship: what do we know? *Reviews on Environmental Health* 15(1-2):109-33.

Gallop, R.K., and C.D. Naylor. 1999. Foreword. In *Cardiovascular Health & Services in Ontario: An ICES Atlas*, eds, David C. Naylor and Pamela M. Slaughter, xiii-ix. Toronto: Institute for Clinical Evaluative Sciences.

Gatrell, A.C. 2002. *Geographies of Health: An Introduction.* Malden, MA: Blackwell Publishers Inc.

Gatrell, A. C. and M. Löytönen (Eds). 1998. *GIS and Health.* London ; Philadelphia, PA : Taylor & Francis, c1998.

Gatrell, A.C. and M. Loytonen 1998. GIS and health research: An introduction. In *GIS and Health,* eds, Gatrell, A. C. and M. Löytönen, 3-16. London ; Philadelphia, PA : Taylor & Francis.

Gensini, G.F., M. Comeglio, and A. Colella. 1998. Classical risk factors and emerging elements in the risk profile for coronary artery disease. *European Heart Journal* 19 Suppl A:A53-61.

Getis, A., and J.K. Ord. 1996. Local spatial statistics: and overview. In *Spatial Analysis: Modelling in a GIS Environment*, eds, P. Longley and M. Batty, 261-279, GeoInformation International: Cambridge, England.

Gheorghiade et al., 1997. Benatar D. Konstam MA. Stoukides CA. Bonow *RO.* Pharmacotherapy for systolic dysfunction: a review of randomized clinical. *American Journal of Cardiology.* 80(8B):14H-27H.

Ginter, E. 1998. Cardiovascular disease prevention in eastern Europe. *Nutrition.* 14(5):452-7.

Glantz SA, Parmley WW. Passive smoking and heart disease. Mechanisms and risk. *JAMA* 1995; 273:1047-53.

Gold M, Franks P, Erickson P. 1996. Assessing the health of a nation: the predictive validity of a preference-based measure and self-rated health. *Med Care* 34:163–77.

Gorski, P.A. 2000. Caring relationships: an investment in health? Public Health Reports. 115(2-3):144-150.

Greenland, S. and H. Morgenstern. 1989. Ecological bias, confounding, and effect modification. *Int. J Epidemiol* 18:269-274.

Griffith, D. 1987. *Spatial Autocorrelation: A Primer.* Washington, D.C: Association of American Geographers.

Grootaert, C. 1998. *Social Capital: The Missing Link?* Social Capital Initiative, Working Paper No. 3, The World Bank.

Grootendorst, P. V., D. H. Feeny, and W. Furlong. 1997. Does it matter whom and how you ask? Inter- and intra-rater agreement in the Ontario Health Survey. *J. Clin Epidemiol* 50(2):127-135.

Grundy, S.M. 1997. Cholesterol and coronary heart disease. The 21st century. *Archives of Internal Medicine* 157(11):1177-1184.

Gujarati, D.N. 1995. *Basic Econometrics.* New York, N.Y: McGraw-Hill, Inc.

Gupta R, V.P. Gupta, and N.S. Ahluwalia. 1994. Educational status, coronary heart disease, and coronary risk factor prevalence in a rural population of India. *Br Med J* 1332-6.

Halle, M., A. Berg, and J. Keul. 1997. Reducing cholesterol in cardiovascular rehabilitation - exercise versus drug therapy . *Wiener Klinische Wochenschrift. Supplementum.* 2:29-32.

Hammar, N., A. Ahlbom, and T. Theorell. 1992. Geographical differences in myocardial infarction incidence in eight Swedish counties, 1976-1981. *Epidemiology* 3(4):348-55.

Hancock, T. 1999. Future directions in population health. *Canadian Journal of Public Health.* 90 Suppl 1:S68-70.

Hancock, T., R. Labonte, and R. Edwards. 1999. Indicators that count: Measuring population health at the community level. *Canadian Journal of Public Health.* 90 Suppl 1:S22-6.

Handy, F. 1977. Income and air pollution in Hamilton Ontario. *Alternatives* 6:18-24.

Hayes M.V. et al, eds, 1994. *The Determinants of Population Health: A Critical Assessment.* Western Geographic Series, Vol. 29, University of Victoria.

Hayes, M.V., and J.R. Dunn. 1998. *Population Health in Canada: a Systematic Review.* Ottawa: Canadian Policy Research Networks.

Haynes, S.G., et al. 1980. The relationship of psychosocial factors to coronary heart disease in the Framingham Study: III. Eight year incidence of coronary heart disease. *American Journal of Epidemiology* 111: 37.

Hayreh, S.S. 1999. The role of age and cardiovascular disease in glaucomatous optic neuropathy. *Survey of Ophthalmology* 43 Suppl 1:S27-42.

He, J., Ogden, L.G., Bazzano, L.A., Vupputuri, S., Loria, C., Whelton, P.K. 2001. Risk factors for congestive heart failure in US men and women: NHANES I epidemiologic follow-up study. *Archives of Internal Medicine* 161(7):996-002.

Health Canada. 1993. *Promoting Heart Health in Canada: A focus on heart health inequalities.* Ottawa: Health Canada.

Health Canada. 1996. *Towards a Common Understanding: Clarifying the Core Concepts of Population Health.* A Discussion Paper. Ottawa, ON: Health Canada.

Health Canada. 1998. *In the Name of Health - Shifting Paradigms, Shifting Perspectives. Canada's Experience in Adopting and Implementing a Population Health Perspective. An Overview and Upgrade.* Ottawa, ON: Health Canada.

Health Canada. 1998. *Taking Action on Population Health: A Position Paper for Health Promotion and Programs Branch Staff.* Ottawa, ON: Health Canada.

Health Canada. 1999. *Leading Causes of Death and Hospitalization in Canada.* Population and Public Health Branch. Health Canada. http://www.hc-sc.gc.ca/pphb-dgspsp/publicat/lcd-pcd97/

Health Canada. Centers for Disease Control and Prevention. World Heart Federation. 2001. *The Osaka Declaration. Health, economics and political action: stemming the global tide of cardiovascular disease.* Declaration of the Fourth International Heart Health Conference, Osaka, Japan, May 2001.

Heart and Stroke Foundation of Canada. 1997a. *Heart Disease and Stroke in Canada*, Ottawa: Heart and Stroke Foundation of Canada.

Heart and Stroke Foundation of Canada, 1997b. *Women, Heart Disease and Stroke in Canada: Issues and Options*, Ottawa: Heart and Stroke Foundation of Canada.

Heart and Stroke Foundation of Canada. 1997c. *Differences in the distribution of mortality due to different forms of cardiovascular disease within a Canadian province*. Ottawa: Heart and Stroke Foundation of Canada.

Heart and Stroke Foundation of Canada. 1999. *The Changing Face of Heart Disease and Stroke in Canada.* Ottawa, Canada: Heart and Stroke Foundation of Canada.

Heart and Stroke Foundation of Ontario. 1997. *Differences in the distribution of mortality due to different forms of cardiovascular diseases within a Canadian province.* Heart and Stroke Foundation of Ontario.

Helminen A., et al. 1997. Social network in relation to plasma fibrinogen. *Journal of Biosocial Science* 29(2):129-39.

Hemingway, H., Shipley, M., Macfarlane, P., Marmot, M. 2000 Impact of socioeconomic status on coronary mortality in people with symptoms, electrocardiographic abnormalities, both or neither: the original Whitehall study 25 year follow up. *Journal of Epidemiology & Community Health* 54(7):510-6.

Hennekens, C.H., D. Evans, and R. Peto. 1979. Oral contraceptive use, cigarette smoking and myocardial infarction. *Br J Fam Plann* 5:66-7.

Henry, R.R. 1998. Type 2 diabetes care: the role of insulin-sensitizing agents and practical implications for cardiovascular disease prevention. *American Journal of Medicine* 105 (1A):20S-26S

Hertzman, C. 1995. *Environment and Health in Central and Eastern Europe.* Washington: World Bank.

Hertzman, C., J. Frank, and R.G. Evans. 1994. Heterogeneities in Health Status and the Determinants of Population Health. In *Why are some people healthy and others not*, eds. Evans *et al.* 67-92. Hawthorne, N.Y: ALDINE DE GRUYTER.

Hibbard, J., and C. Pope. 1991. Effect of domestic and occupational roles on morbidity and mortality. *Soc Sci Med* 32(7):805-811.

Hilmert, C.J., Christenfeld, N., Kulik, J.A. 2002. Audience status moderates the effects of social support and self-efficacy on cardiovascular reactivity during public speaking. *Annals of Behavioral Medicine* 24(2):122-31.

Hodgson, C. and E. Jamieson. 1997. Self-reported cardiovascular disease and risk factors. Prevalence in Ontario among women 50 and older. *Canadian Family Physician* 43:1747-1752.

Hoeymans N, Feskens E, Kromhout D, *et al.* 1997. Ageing and the relationship between functional status and self-rated health in elderly men. *Soc Sci Med* 45:1527–36.

Howard, G., Anderson, R.T., Russell, G., Howard, V.J., Burke, G.L. 2000. Race, socioeconomic status, and cause-specific mortality. *Annals of Epidemiology.* 10(4):214-23.

Howden-Chapman, P. 2002. Housing and inequalities in health. *Journal of Epidemiology & Community Health* 56(9):645-6

Hu, F.B., M.J. Stampfer, E. Rimm, A. Ascherio, B.A. Rosner, D. Spiegelman, and W.C. Willett. 1999. Dietary fat and coronary heart disease: a comparison of approaches for adjusting for total energy intake and modeling repeated dietary measurements. *American Journal of Epidemiology* 149(6):531-40.

Iannantuono, A. 2002. *Manitoba, Ontario and Prince Edward Island: A Policy Analysis of the Canadian Heart Health Initiative – Dissemination Phase.* Technical Report. Prepared for the Canadian Heart Health Dissemination Project Investigative Team. August, 2002.

Institute for Clinical Evaluative Studies. 1996. *Inventory of Studies on The Accuracy of Canadian Health Administrative Databases.* Technical Report. Pub. No. 96-03-TR. Toronto, Ontario: ICES.

Jacquez, G.M. 1998. GIS as an Enabling Technology. In *GIS and Health,* eds, Gatrell, A. C. and M. Löytönen, 17-28. London ; Philadelphia, PA : Taylor & Francis.

Jaglal, S., S.J. Bondy, and P. Slaughter. 1999. Risk factors for cardiovascular disease. In *Cardiovascular Health & Services in Ontario: An ICES Atlas,* eds, David C. Naylor and Pamela M. Slaughter, 63-82. Toronto: Institute for Clinical Evaluative Sciences.

Jenei, Z., D. Pall, E. Katona, P. Polgar, Z. Karanyi, M. Bodor, and G. Kakuk. 2000. Prevalence of cardiovascular risk factors of the smokers and non-smokers in the city of Debrecen, Hungary. *Public Health* 114(4):295-259.

Jennings GL. 1995. Mechanisms for reduction of cardiovascular risk by regular exercise. *Clinical & Experimental Pharmacology & Physiology*. 22(3):209-11.

Jerrett, M., J. Eyles, D. Cole, and S. Reader. 1997. Environmental equity in Canada: an empirical investigation into the income distribution of pollution in Ontario. *Environment and Planning A* 29:1777-1800.

Jerrett, M., J. Eyles, and D. Cole. 1998. Socioeconomic and environmental covariates of premature mortality in Ontario. *SSM* 47(1):33-49.

Jialal, I. 1998. Evolving lipoprotein risk factors: lipoprotein(a) and oxidized low-density lipoprotein. *Clinical Chemistry*. 44(8 Pt 2):1827-32.

Jim, A., J.D. Martin, and C. Sarin. 2002. A diabetes mellitus in the First Nations population of British Columbia, Canada. Part 1. Mortality. *International Journal of Circumpolar Health*. 61(3):251-253.

Johnson, N.J., Backlund, E., Sorlie, P.D., Loveless, C.A. 2000. Marital status and mortality: the national longitudinal mortality study. *Annals of Epidemiology* 10(4):224-38.

Johnson, J. and J. DiNardo. 1997. *Econometric methods*. 4th edition. New York: McGraw-Hill.

Johnson, S., D. Martin, and C. Sarin. 2002. Diabetes mellitus in the First Nations population of British Columbia, Canada. Part 3. Prevalence of diagnosed cases. International Journal of Circumpolar Health. 61(3):260-264.

Jones, K., and G. Moon. 1987. *Health, disease and society: An introduction to medical geography*. London: Routledge.

Jones, K., Moon, G. and Clegg, A. 1991. Ecological and individual effects in childhood immunization uptake: a multi-level approach. *Soc. Sci. Med.* 33(4):501-8.

Jones, D.W., C.T. Sempos, T.J. Thom, A.M. Harrington, H.A. Taylor Jr, B.W. Fletcher, B.D. Mehrotra, S.B. Wyatt, and C.E. Davis. 2000. Rising Levels of Cardiovascular Mortality in Mississippi, 1979-1995. *The American Journal of Medical Sciences* 319(3):131-137.

Joung, I.M.A., J.J. Glerum, F.W.A.v. Poppel, J.W.P.F. Kardaun, and J.P. Mackenbach. 1996. The contribution of specific causes of death to mortality differences by matiral status in the Netherlands. *European Journal of Public Health* 6:142-49.

Julius, S., P. Palatini, and S.D. Nesbitt. 1998. Tachycardia: an important determinant of coronary risk in hypertension. *Journal of Hypertension - Supplement.* 16(1):S9-15.

Kanaroglou, P. 1996. On spurious correlation in geographical problems. *The Canadian Geographer* 40(3):194-202.

Kannel, W.B. 1998. Overview of atherosclerosis. *Clinical Therapeutics.* 20 Suppl B:B2-17.

Kannel, W.B., and R.C. Ellison. 1996. Alcohol and coronary heart disease: the evidence for a protective effect. *Clinica Chimica Acta.* 246(1-2):59-76.

Kaplan, G.A., E. Pamuk, J.W. Lynch, R.D. Cohen, and J.L. Balfour. 1996. income inequality and mortality in the United States. *BMJ* 31:999-1003.

Karasek, R.A., and T. Theorell. 1990. *Health work: stress, productivity and the reconstruction of working life.* New York: Basic Books.

Kawachi, I. 1999. Social capital and community effects on population and individual health. *Annals of the New York Academy of Sciences* 896:120-130.

Kawachi, I., B.P. Kennedy, K. Lochner, and D. Prothrow-Smith. 1997. Social Capital, Income Inequality, and Mortality. *American Journal of Public Health* 87:1491-8.

Kawachi, I., B.P. Kennedy, and R. Glass. 1999. Social capital and self-rated health: a contextual analysis. *American Journal of Public Health* 89(8):1187-93.

Kawachi, I., S. Levine, S.M. Miller, K. Lasch, and B. Amick. 1994. *Income Inequality and Life Expectancy: Theory, Research, and Plocy.* Boston, M.A: Health Institute, New England Medical Center,1994. Society and Health Working Paper 94-2

Kawachi, I., S. Marshall, and N. Pearce. 1991. Social class inequalities in the decline of coronary heart disease among New Zealand men, 1975-1977 to 1985-1987. *International Journal of Epidemiology.* 20(2):393-8.

Kearns, R.A. 1991. The place of health in the health of place: The case of the Hokianga Special Medical Area. *SSM* 33:519-530.

Kennedy, B.P., I. Kawachi, D. Prothrow-Stith, K. Lochner, and V. Gupta. 1998. Social capital, income inequality, and firearm violent crime. *SSM* 47(1):7-17.

Kilander, L., H. Nyman, M. Boberg, and H. Lithell. 1997. Cognitive function, vascular risk factors and education. A cross-sectional study based on a cohort of 70-year-old men. *Journal of Internal Medicine*. 242(4):313-21.

King, K.B. 1997. Psychologic and social aspects of cardiovascular disease. *Ann Behav Med* 19(3):264-70.

Kirk-Gardner, R. and D. Steven. 1994. An analysis of the Ontario Health Survey from a cardiovascular perspective. *Canadian Journal of Cardiovascular Nursing* 5(3):7-14.

Klor, et al., 1997. Nutrition and cardiovascular disease. *European Journal of Medical Research*. 2(6):243-57.

Kohl, H.W. 3rd. 2001. Physical activity and cardiovascular disease: evidence for a dose response. *Medicine & Science in Sports & Exercise* 33(6 Suppl):S472-83.

Konrat, C., L.I. Mennen, E. Caces, P. Lepinay, F. Rakotozafy, A. Forhan, and B. Balkau. 2002. Alcohol intake and fasting insulin in French men and women. The D.E.S.I.R. Study. *Diabetes & Metabolism* 28(2):116-23.

Kramer, L.M. 1995. Implementing new dietary guidelines of the National Cholesterol Education Program. AACN *Clinical Issues* 6(3):418-31; quiz 495-6.

Kritz H. Schmid P. Sinzinger H. Passive smoking and cardiovascular risk. *Archives of Internal Medicine*. 155(18):1942-8, 1995 Oct 9.

Kruger, O. 1991. Risk factor changes and mortality changes: a regional perspective on ischemic heart disease in Norway 196601985. *Soc Sci Med* 33(4): 423-8.

Kruger, O., S. Westin, and E.H. Nymoen. 1990. Ischaemic heart disease among men in Norway during the period 1966-1985: two counties with different mortality time trends. *Journal of Internal Medicine* 228(5):483-91.

Laakso, M. 1998. Hypertension and macrovascular disease--the killing fields of NIDDM. *Diabetes Research & Clinical Practice* 39 Suppl:S27-33.

Labonte, R. 1995. Population health and health promotion: what do they have to say to each other? *Can J of Public Health* 86(3):165-168.

Lakka, H.M., T.A. Lakka, J. Tuomilehto, J. Sivenius, and J.T. Salonen. 2000. Hyperinsulinemia and the risk of cardiovascular death and acute coronary and cerebrovascular events in men: the Kuopio Ischaemic Heart Disease Risk Factor Study. *Archives of Internal Medicine* 160(8): 1160-8.

Lalonde, M. 1974. A New Perspective on the Health of Canadians. Ottawa, Health Canada.

Lantz, P.M., J.S. House, J.M. Lepkowski, D.R. Williams, R.P. Mero, and J.J. Chen. 1998. Socioeconomic factors, health behaviours, and mortality. *Journal of the American Medical Association* 279(21):1703-8.

Lavis, J.N., C.A. Mustard, J.I. Payne, and M.S. Farrant. 2001. Work-related population health indicators. *Can J of Public Health* 92(1): 72-8.

Legowski, B. and L. McKay. 2000. Health beyond health care: twenty-five years of federal health policy development. *Canadian Policy Research Network Discussion Paper* No. H/04, Ottawa, ON: CPRN.

LeGrand, J. 1987. Inequalities in health. Some international comparisons. *Eur Econ Rev.* 31:182-191.

Leong, K.S. and J.P. Wilding. 1999. Obesity and diabetes. *Best Practice & Research Clinical Endocrinology & Metabolism.* 13(2):221-37.

Levine, N. 1999. *CrimeStat Version 1.0 Users Manual.* Washington, D.C: National Institute of Justice

Liao, Y. and R.S. Cooper. 1995. Continued adverse trends in coronary heart disease mortality among blacks, 1980-91. *Public Health Rep*110: 572-579.

Licata, G., R. Scaglione, and L.J. Dominguez. 1999. Early markers of cardiovascular damage in obese subjects. *Nutrition Metabolism & Cardiovascular Diseases* 9(2):78-86.

Lindenstrom, E. 1995. Influence of systolic and diastolic blood pressure on stroke risk: a prospective observational study. *American Journal of Epidemiology* 15: 142(12):1279-90.

Lomas, J. 1998. Social capital and health: implications for public health and epidemiology. *Soc. Sci. Med.* 47(9): 1181-8.

Loytonen, M. 1998. GIS, Time Geography and Health. In *GIS and Health,* eds, Gatrell, A. C. and M. Löytönen, 97-110. London ; Philadelphia, PA : Taylor & Francis.

Luepker, R.V., W.D. Rosamond, R. Murphy, J.M. Sprafka, A.R. Folsom, P.G. McGovern, and H. Blackburn. 1993. Socioeconomic status and coronary heart disease risk factor trends. The Minnesota Heart Survey. *Circulation* 88(5 Pt 1):2172-2179.

Lund, R., P. Due, J. Modvig, B.E. Holstein, M.T. Damsgaard, and P.K. Andersen. 2002. Cohabitation and marital status as predictors of mortality – an eight year follow-up study. *SSM* 55(4):673-9.

MacDonald, S. 1992. Multiple cardiovascular risk factors in Canadian adults. *Canadian Medical Association Journal* 146(11, suppl.):2021-29.

Macinko, J. and B. Starfield. 2001. The utility of social capital in research on health determinants. Milbank Quarterly. 79(3):387-427.

MacIntyre, S., S. MacIver, and A. Sooman. 1993. Area, Class and Health: Should we be Focusing on Places or People? *Jnl Soc. Pol.* 22 (2): 213-234.

Mackenbach, J.P., A.E. Cavelaars, A.E. Kunst, and F. Groenhof. 2000. Socioeconomic inequalities in cardiovascular disease mortality; an international study. *European Heart Journal* 21(14):1141-51.

Marks, J.B. and P. Raskin. 2000. Cardiovascular risk in diabetes: a brief review. *Journal of Diabetes & its Complications* 14(2):108-15.

Marmot, M.G. 1986. Does stress cause heart attacks? *Postgraduate Medical Journal* 62: 683-86.

Marmot, M.G. 1996. Socio-economic factors in cardiovascular disease. *Journal of Hypertension* - Supplement. 14(5):S201-5.

Marmot, M. 1997. The socio-economic environment and cardiovascular disease, Plenary paper, 4th International Conference on Preventive Cardiology. Montreal, Quebec, June 29-July 3, 1997.

Marmot, M. 1999. Importance of the psychosocial environment in epidemiologic studies. *Journal of Work, Environment & Health* 25 Suppl 4: 49-55.

Marmot, M. 2001. Sustainable development and the social gradient in coronary heart disease. *European Heart Journal* 22(9):740-750.

Marmot, M.G. and J.F. Mustard. 1994. Coronary heart disease from a population perspective. In *Why are some people healthy and others not*, eds. Evans et al., 189-214. *Hawthorne,* N.Y: ALDINE DE GRUYTER.

Marmot, M.G., and G.D. Smith. 1989. "Why Are the Japanese Living Longer?" *British Medical Journal* 299:1547-51.

Marmot, M.G., Bosma, H., Hemingway, H., Brunner, E., and S. Stansfeld. 1997. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. *Lancet* 350(9073):235-9.

Marmot, M.G., G. Davey Smith, et al. 1991. Health inequalities among British civil servants: the Whitehall II study. *Lancet* 337: 1387-93.

Marmot, MG., Fuhrer, R., Ettner, SL., Marks, NF., Bumpass, LL., and C.D. Ryff. 1998. Contribution of psychosocial factors to socioeconomic differences in health. *The Milbank Quarterly* 76(3):403

Marmot, M.G., M. Kogevinas, and M.A. Elston. 1987. Social/Economic Status and Disease. *Annual Review of Public Health* 8:111-35.

Marmot, M.G., Shipley, M.J., and G. Rose. 1984. Inequalities in death – specific explanations of a general pattern? *Lancet* 1(8384):1003-6.

Martin, J.D. and H.M. Yidegiligne. 1998. Diabetes mellitus in the First Nations population of British Columbia, Canada. *International Journal of Circumpolar Health* 57 Suppl 1: 335-339.

Maryniuk, M.D. and A.E. Peterson. 1997. Optimizing lipid levels through diet. *Lippincott's Primary Care Practice.* 1(3):285-294.

Mattiasson, I., Lindgarde, F., Nilsson, J.A., and Theorell, T. 1990. Threat of unemployment and cardiovascular risk factors: longitudinal study of quality of sleep and serum cholesterol concentrations in men threatened with redundancy. *BMJ* 301(6750): 461-6.

McAbee, R. 1995. Primary prevention of hypertension: a challenge for occupational health nurses. *AAOHN Journal* 43(6):306-12.

McCarthy, M. 2000. Social determinants and inequalities in urban health. *Reviews on Environmental Health* 15(1-2):97-108.

McIntyre, S., R. Hiscock, A. Kearns, and A. Ellaway. 2001. Housing tenure and car access: further exploration of the nature of their relations with health in a UK setting. *J Epidemiol Community Health* 55:330–1.

McGrail, K., Ostry, A., Thomas, V., et al. 1998. Determinants of Population Health: A Synthesis of the Literature. Ottawa: Report to Health Canada, December 1998.

Mickey, J. and S. Greenland. 1989. The impact of confounder selection criteria on effect estimation. *Am. J. Epidemiol* (129):125-37.

Miilunpalo, S., I. Vuori, P. Oja, M. Pasanen, and H. Urponen, 1997. Self-rated health status as a health measure: the predictive value of self-reported health status on the use of physician services and on mortality in the working-age population. *Journal of Clinical Epidemiology* 50(5):517-28.

Ministry of Health, Ontario. 1989. *Mandatory Health Programs and Services Guidelines*. Ministry of Health, Ontario.

Ministry of Health, Ontario. 1990. *Ontario Health Survey 1990. User's Guide Vol. 1. Documentation.* Toronto: Ministry of Health, Ontario.

Mittleman, M.A., M. Maclure, M. Nachnani, J.B. Sherwood, and J.E. Muller. 1997. Educational attainment, anger, and the risk of triggering myocardial infarction onset. The Determinants of Myocardial Infarction Onset Study Investigators. *Archives of Internal Medicine* 157(7):769-75.

Molarius, A., J.C. Seidell, S. Sans, J. Tuomilehto, and K. Kuulasmaa. 2000. Educational level, relative body weight, and changes in their association over 10 years: an international perspective from the WHO MONICA Project. *American Journal of Public Health* 90(8):1260-8.

Montague, T., *et al.* 1994. Prevention and regression of coronary atherosclerosis. Is it safe and efficacious therapy? *Chest* 105(3):718-26.

Morgenstern, H. and D. Thomas. 1993. Principles of study design in environmental epidemiology. *Environmental Health Perspectives* Volume 101, S4:23-38.

Moritz, D.J. and W.A. Satariano. 1993. Factors predicting stage of breast cancer at diagnosis in middle aged and elderly women: the role of living arrangements. *Journal of Clinical Epidemiology* 46:443-54.

Moustapha, A., and K. Robinson. 1999. Homocysteine: an emerging age-related cardiovascular risk factor. *Geriatrics* 54(4):41, 44-6, 49-51.

Mustard, J.F., and J. Frank. 1991. *The Determinants of Health.* CIAR paper #5. Tonronto. Reprinted in Hayes M.V. et al, eds, (1994). *The Determinants of Population Health: A Critical Assessment.* Western Geographic Series, Vol. 29, University of Victoria.

Myers, K.A., and D.R.E. Farquhar. 1998. Improving the accuracy of death certification. *CMAJ* 158:1317-23.

Nair, C., H. Colburn, D. McLean, and A. Petrasovits 1989. Cardiovascular disease in Canada. *Health Reports*. 1(1): 1-22.

Nakanishi, N., I. Nakura, K. Nagano, H. Yoneda, T. Takatorige, F. Shinsho, and K. Tatara. 1998. Mortality in relation to the type of household among elderly people living in a community. *Journal of Epidemiology* 8:65-72.

Negri, L., M. G. Franzosi, C. La Vecchia, I. Santoro, A. Nobili, and G. Tognoni, on behalf of GISSI-EPRIM Investigators: 1993. Tar yield of cigarettes and risk of acute myocardial infarction. *Br Med J* 306:1567-70.

Nilsson, P.M., Johansson, S.E., and Sundquist, J. 1998. Low educational status is a risk factor for mortality among diabetic people. *Diabetic Medicine* 15(3):213-9.

Nishi, N., S. Nanto, S. Shimai, Y. Matsushima, K. Otake, A. Ando, K. Yamasaki, S. Soga, and K. Tatara. 2001. Effects of hostility and lifestyle on coronary heart disease among middle-aged urban Japanese. *Journal of Epidemiology* 11(6): 243-8.

O'Loughlin, J.L., G. Paradis, K. Gray-Donald, and L. Renaud. 1999. The impact of a community-based heart disease prevention program in a low-income, inner city neighbourhood. *American Journal of Public Health* 89:1819-26.

Oncken, C.A. 1996. Medical perspective: does smoking cessation affect casual or ambulatory blood pressure? *Blood Pressure Monitoring* 1(6):443-446.

Ontario Ministry of Health. 1993. *Opportunities for Health: Promoting Heart Health.* Report of the Chief Medical Officer of Health: Ontario Ministry of Health.

Oparil, S. and Oberman. A. 1999. Nontraditional cardiovascular risk factors. *American Journal of the Medical Sciences* 317(3):193-207.

Orth-Gomer, K. 1997. Social stress, isolation and socio-economic status, Plenary paper, 4th International Conference on Preventive Cardiology. Montreal, Quebec, June 29-July 3, 1997.

Orth-Gomer, K., M. Horsten, S.P. Wamala, M.A. Mittleman, R. Kirkeeide, B. Svane, L. Ryden, and K. Schenck-Gustafsson. 1998. Social relations and extent and severity of coronary artery disease. The Stockholm Female Coronary Risk Study. *European Heart Journal* 19(11):1648-56.

Orth-Gomer, K., Rosengen, A., Wilhelmsen, L. 1993. Lack of social support and incidence of coronary heart disease in middle-aged Swedish men. Psychosocial *Medicine* (55):37-43.

Petersen, A, and Lupton, D. 1996. *The New Public Health: Health and Self in the Age of Risk,* Sage, London.

Pekkanen J, Linn S, Heiss G *et al.* Ten-year mortality from cardiovascular disease in relation to cholesterol level among men with and without preexisting cardiovascular disease. *N Eng J Med* 1990; 322:1700-7.

Petersen, P., et al. 1989. Placebo-controlled randomized trial of warfarin and aspirin for prevention of thromboembolic complications in atrial fibrillation. *The Lancet* 1: 175-179.

Pfeffer MA, Sacks FM, Moye LA *et al.* 1995. Cholesterol and recurrent events: a secondarry prevention trial for normolipidemic patients. *Am J Cardiol* 76:98C-106C.

Phillips D, Kawachi I, Marshall S. And Purdie G. 1991. No evidence for social class inequalities in intervention for coronary heart disease in Otago 1987-1989. *New Zealand Medical Journal* 104: 507-510.

Philo, C. 1996. Staying in? Invited comments on "Coming out: exposing social theory in medical geography", *Health and Place* 2:35-40.

Pickering, T. 1999. Cardiovascular pathways: socioeconomic status and stress effects on hypertension and cardiovascular function. *Annals of the New York Academy of Sciences* 896:262-77.

Pickle, L.W. and R.F. Gillum. 1999. Geographic variation in cardiovascular disease mortality in US blacks and whites. *Journal of the National Medical Association* 91 (10): 545-56.

Pioro, M.P., R.F. Dyck, and D.C. Gillis. 1996. Diabetes prevalence rates among First Nations adults on Saskatchewan reserves in 1990: comparison by tribal grouping, geography and with non-First Nations people. *Canadian Journal of Public Health.* 87(5):325-328.

Pocock, S.J., et al. 1982. Analysing geographic variations in cardiovascular mortality. *Journal of the Royal Statistical Society, Series A,* 145:313-341.

Poland, B., D. Coburn, A. Robertson, and J. Eakin. 1998. Wealth, equity and health care: a critique of a "population health" perspective on the determinants of health. *SSM* 46(7):785-98.

Prior, P.M. and B.C. Hayes. 2001. Marital status and bed occupancy in health and social care facilities in the United Kingdom. *Public Health* 115(6):401-6.

Private Communication from Dr. Mike Jerrett: School of Geography and Geology, McMaster University.

Private Communication from Dr. Stephen Walter: Department of Clinical Epidemiology and Biostatistics, McMaster University.

Putnam, R.D., R. Leonardi, and R.Y. Nancti. 1993. *Making Democracy Work: Civic Traditions in Modern Italy.* New Jersey: Princeton University Press.

Putnam, R.D. 1995. Bowling alone. America's declining social capital. *Journal of Democracy* 6: 65-78.

Rahman, O., J. Strauss, P. Gerter, D. Ashley, and K. Fox. 1994. Gender differences in adult health: an international comparison. Gerontologist. 34(4):463-469.

Raphael, D. and Bryant, T. 2000. Putting the Population in Population Health. *Can J. Public Health* 91(1): 9-10.

Raphael, D. and S. Farrell. 2002. Beyond medicine and lifestyle: addressing the societal determinants of cardiovascular disease in North America. *Leadership in Health Services* 15(4):i-v.

Raynor, D.A., Pogue-Geile, M.F., Kamarck, T.W., McCaffery, J.M., and S.B. Manuck. 2002. Covariation of psychosocial characteristics associated with cardiovascular disease: genetic and environmental influences. *Psychosomatic Medicine* 64(2): 191-203.

Rayo, L.I., and H.E. Marin 1998. Wine and heart. *Revista Espanola de Cardiologia.* 51(6):435-49.

Reddy, K.K., A.P. Rao, and T.P. Reddy. 2002. Socioeconomic status and the prevalence of coronary heart disease risk factors. *Asia Pacific Journal of Clinical Nutrition* 11(2):98-103.

Reeder, B. A., A. Angel, M. Ledoux, S. W. Rabkin, T. K. Young, and L. E. Sweet. 1992. Obesity and its relation to cardiovascular disease risk factors in Canadian adults. Canadian Heart Health Surveys Research Group. *CMAJ* 146: 2009-2019.

Reynes, J.E., T.M. Laster, H. Feldman, A.R. Assaf, and R.A. Carleton. 1993. Education and risk factors for coronary heart disease: Results from a New England community. *Am J Prev Med* 9:365-71.

Rith-Najarian, S.J., D.M. Gohdes, R. Shields, B. Skipper, K.R. Moore, B. Tolbert, T. Raymer, and K.J. Acton. 2002. Regional variation in cardiovascular disease risk factors among American Indians and Alaska Natives with diabetes. *Diabetes Care* 25(2):279-83.

Robinson, G.M. 1998. *Methods and Techniques in Human Geography.* New York: John Wiley & Sons.

Rose, G. 1992. *The strategy of preventive medicine.* Oxford: Oxford University Press.

Rosenberg, M.W. and K. Wilson. 2000. Gender, poverty and location: how much difference do they make in the geography of health inequalities? *SSM* 51(2):275-287.

Ross, N.A., M.W. Rosenberg, and D.C. Pross. 1994. Siting a women's health facility: a location-allocation study of breast cancer screening services in Eastern Ontario. *The Canadian Geographer* 38(2):150-61.

Roux, A., S. Merkin, D. Arnett, et al. 2001. Neighbourhood of residence and incidence of coronary heart disease. *New England Journal of Medicine* 345:99-106.

Rozanski, A., J.A. Blumenthal, and J. Kaplan. 1999. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation* 99(16):2192-217.

Runyan, D.K., W.M. Hunter, R.R. Socolar, L. Amaya-Jackson, D. English, J. Landsverk, H. Dubowitz, D.H. Browne, S.I. Bangdiwala, and R.M. Mathew. 1998. Children who prosper in unfavorable environments: the relationship to social capital. *Pediatrics* 101(1 Pt 1):12-18.

Rushton, G. 1998. Improving the geographic basis of health surveillance using GIS. In *GIS and Health,* eds, Gatrell, A. C. and M. Löytönen, 63-79. London ; Philadelphia, PA : Taylor & Francis.Sasson et al. 1993. Insulin resistance is an important determinant of left ventricular mass in the obese. *Circulation* 88 (part I):1431-6.

Sandelowski, M. (2000). Combining qualitative and quantitative sampling, data collection, and analysis techniques in mixed method studies. Research in Nursing in Health, 23, 246-255.

Saurel-Cubizolles, M.J., Romito, P., Ancel, P.Y., and Lelong, N. 2000. Unemployment and psychological distress one year after childbirth in France. *Journal of Epidemiology & Community Health* 54(3):185-91.

Saw, S.M. and U. Rajan. 1997 The epidemiology of obesity: a review. Annals of the Academy of Medicine, Singapore. 26(4):489-493.

Scarinci, I.C., B.M. Beech, W. Naumann, K.W. Kovach, L. Pugh, and B. Fapohunda. 2002. Depression, socioeconomic status, age, and marital status in black women: a national study. *Ethnicity & Disease* 12(3):421-8.

Scherer, M.J. and J.P. Lane. 1997. Assessing consumer profiles of 'ideal' assistive technologies in ten categories: an integration of quantitative and qualitative methods. Disability & Rehabilitation. 19(12):528-35.

Sellier, P. 1995. Physical activity in the cardiac patient. *Journal of Cardiovascular Pharmacology* 25 (Suppl. 1):S9-14.

Sesso, H.D., M.J. Stampfer, B. Rosner, C.H. Hennekens, J.E. Manson, and J.M. Gaziano. 2000. Seven-year changes in alcohol consumption and subsequent risk of cardiovascular disease in men. *Archives of Internal Medicine* 160(17):2605-12.

Shaper, A.G., and S.G. Wannamethee. 2000. Alcohol intake and mortality in middle-aged men with diagnosed coronary heart disease. *Heart* 83(4): 394-9.

Shapiro, S. 1997. Is Meta-Analysis a Valid Approach to the Evaluation of Small Effects in Observational Studies? *J. Clin Epidemiol* 50(3): 223-9.

SHEP Cooperative Research Group. 1991. Prevention of stroke by antihypertensive drug treatment in older persons with systolic hypertension. *Journal of the American Medical Association* 256: 3255-3264.

Shestov, D.B., Deev, A.D., Klimov, A.N., Davis, C.E., Tyroler, H.A. 1993. Increased risk of coronary heart disease death in men with low total and low-density lipoprotein cholesterol in the Russian Lipid Research Clinics Prevalence Follow-up Study *Circulation.* 88(3):846-53.

Sheth, T., et al. 1998. Ethnic differences in socioeconomic status and mortality. *Canadian Journal of Cardiology* 13(Suppl.B).

Shewry, M.C., Smith, W.C., Woodward, M, and Tunstall-Pedoe, H. 1992. Variation in coronary risk factors by social status: results from the Scottish Heart Health Study. *British Journal of General Practice* 42(363): 406-10.

Sjostrom, L.V. 1992. Mortality of severely obese subjects. *American Journal of Clinical Nutrition* 55(2 Suppl):516S-523S.

Smallman-Raynor, M., Muir, K.R., and S.J. Smith. 1998. The geographical assignment of cancer untis: patient accessibility as an optimal location problem. *Public Health* 112:379-83.

Solomon, C.G., F.B. Hu, M.J. Stampfer, G.A. Colditz, F.E. Speizer, E.B. Rimm, W.C. Willett, and J.E. Manson. 2000. Moderate alcohol consumption and risk of coronary heart disease among women with type 2 diabetes mellitus. *Circulation* 102(5):494-499.

Sorenson, P.S., et al, 1989. Long-term prognosis and quality of life after reversible cerebral ischemic attacks. *Acta Neurologica Scandinavica* 79:204-213.

Sorlie, P.D., E. Backlund, and J.B. Keller. 1995. US mortality by economic demographic, and social characteristics: The National Longitudinal Mortality Study. *American Journal of Public Health* 85:949-56.

Sparks, G., Craven, M.A., and C. Worth. 1994. Understanding differences between high and low childhood accident rate areas: The importance of qualitative data. *Journal of Public Health Medicine* 17:193-99.

Stein, J.H., and R.S. Rosenson. 1997. Lipoprotein Lp(a) excess and coronary heart disease. *Archives of Internal Medicine* 157(11):1170-6.

Steptoe, A. 1999. Psychosocial factors in the aetiology of coronary heart disease. *Heart* 82(3):258-259.

Sullivan, J.M. 1996. Practical aspects of preventing and managing atherosclerotic disease in post-menopausal women. *European Heart Journal* 17 Suppl D:32-7.

Sundquist, J. and S.E. Johansson. 1997. The influence of country of birth on mortality from all causes and cardiovascular disease in Sweden 1979-1993. *International Journal of Epidemiology* 26(2):279-87.

Sundquist, J. and S.E. Johansson. 1997. Self reported poor health and low educational level predictors for mortality: A population based follow up study or 39,156 people in Sweden. *Journal of Epidemiology & Community Health* 51:35-40.

Susic, D. 1997. Hypertension, aging, and atherosclerosis. The endothelial interface. *Medical Clinics of North America* 81(5):1231-40.

Susser, M. 1994. The Logic in Ecological: I. The Logic of Analysis. *American Journal of Public Health* 84(5): 825-829.

Syme, S.L. 1996. To prevent disease: the need for a new approach. D. Blane et al., Eds. *Health and social organization.* London: Routledge.

Tabachnick, B.G. and L. S. Fidell. 1989. *Using Multivariate Statistics.* New York, N.Y: HarperCollins Publishers.

Taft, C.T., A.S. Stern, L.A. King, and D.W. King. 1999. Modeling physical health and functional health status: the role of combat exposure, posttraumatic stress disorder, and personal resource attributes. *Journal of Traumatic Stress* 12(1):3-23.

Tennant, C. 1999. Life stress, social support and coronary heart disease. *Australian & New Zealand Journal of Psychiatry* 33(5):636-41.

Thomas, J.L., and P.A. Braus. 1998. Coronary artery disease in women. A historical perspective. *Archives of Internal Medicine* 158(4):333-7.

Thomas, R. 1992. *Geomedical systems. Intervention and Control.* New York: Routledge.

Thomson, H., Petticrew, M., Morrison, D. 2001. Health effects of housing improvement: systematic review of intervention studies. *BMJ* 323(7306):187-90.

Tiefelsdorf, M.D., Griffith, D., and B. Boots (1999). A variance-stabilizing coding scheme for spatial link matrices. *Environment and Planning A.* 31:165-180.

Toeller, M., A.E. Buyken, G. Heitkamp, W.A. Scherbaum, H.M. Krans, and J.H. Fuller. 1999. Associations of fat and cholesterol intake with serum lipid levels and cardiovascular disease: the EURODIAB IDDM Complications Study. *Experimental & Clinical Endocrinology & Diabetes* 107(8):512-521.

Tremblay, M.S., and J.D. Willms. 2000. Secular trends in the body mass index of Canadian children. *CMAJ* 163 (11): 1423-33.

Tucker, J.S., H.S. Friedman, D.L. Wingard, and J.E. Schwartz. 1996. Marital history at midlife as a predictor of longevity: Alternative explanations to the protective effect of marriage. *Health Psychology* 15:94-101.

Tunstall-Pedoe, H., Brown, C.A., Woodward, M., and Tavendale, R. 1995. Passive smoking by self report and serum cotinine and the prevalence of respiratory and coronary heart disease in the Scottish heart health study. *Journal of Epidemiology & Community Health* 49(2):139-43.

Tuomilehto, J., et al, 1996. Diabetes mellitus as a risk factor for death from stroke. Prospective study of a middle-aged Finnish population. *Stroke* 27(2): 202-5.

Turcotte, F. Book review: *Why are sone people healthy and others not? The determinants of health of populations. Evans, R.G., Barer, M.L., and Marmor, T.R. (Eds.), New York: Aldine de Gruyter,* 1994. *Health and Can Society* 1994;2(1):159-62.

van Doorslaer E, Wagstaff A, Bleichrodt H, *et al.* 1997. Income-related inequalities in health: Some international comparisons. *J Health Econ* 16:93–112

Veenstra, G. 2000. Social capital, SES and health: an individual-level analysis. *Social Science and Medicine* 50(2000) 619-629.

Vine, M., et al. 1997. Geographical information systems: their use in environmental epidemiologic research. *Environmental Health Perspectives* 105(6):598-605.

Vogt, T.M., J.P. Mullooly, D. Ernst, C.R. Pope, and J.F. Hollis. 1992. Social networks as predictors of ischemic heart disease. Cancer, stroke and hypertension: incidence, survival and mortality. *Journal of Clinical Epidiomology* 55:37-43.

Valle, M., F. Gascon, R. Martos, F.J. Ruz, F. Bermudo, R. Rios, and R. Canete. 2000. Infantile obesity: a situation of atherothrombotic risk? *Metabolism: Clinical & Experimental.* 49(5):672-675.

Veenstra, G. 2000. Social capital, SES and health: an individual-level analysis. *Soc Sci Med* 5001:619-629.

Wakefield, S.E., S.J. Elliott, D.C. Cole, and J.D. Eyles. 2001. Environmental risk and (re)action: air quality, health, and civic involvement in an urban industrial neighbourhood. *Health & Place* 7(3):163-77.

Walter, S. 1991a. The ecologic method in the study of environmental health: I. Overview of the method. *Env Hlth Persp* 94: 61-65

_____. 1991b. The ecologic method in the study of environmental health: II. Methodologic issues and feasibility. *Env Hlth Persp* 94: 67-73.

_____. 1992a. The Analysis of Regional Patterns in Health Data: I. Distributional Considerations. *American Journal of Epidemiology* 136(5): 730-741.

_____. 1992b. The Analysis of Regional Patterns in Health Data: I. The Power to Detect Environmental Effects. *American Journal of Epidemiology* 136(5): 730-741.

Walter, S.D., S.E. Birnie, L.D. Marrett, S.M. Taylor, D. Reynolds, J. Davies, J.J. Drake, and M. Hayes. 1994. The geographic variation of cancer incidence in Ontario. *American Journal of Public Health* 84(3):367-376

Walter, D.S., S.M. Taylor, and L.D. Marrett. 1999. An Analysis of Determinants of Regional Variation in Cancer Incidence: Ontario, Canada. In *Disease Mapping and Risk Assessment for Public Health*, A.B. Lawson et al., eds, 365-381, John Wiley & Sons Ltd.

Walters, V., R. Lenton, S. French, J. Eyles, J. Mayr, and B. Newbold. 1996. Paid work, unpaid work and social support: a study of the health of male and female nurses. *SSM* 43(11):1627-36.

Wannamethee, G., and A.B. Shaper. 1992. Physical activity and stroke in British middle-aged men. *British Medical Journal* 304:597-601.

Weber, A. and Lehnert, G. 1997. Unemployment and cardiovascular diseases: a causal relationship? *International Archives of Occupational & Environmental Health* 70(3):153-60.

Wells, A.J. 1994. Passive smoking as a cause of heart disease. J. Am Coll Cardiol 38:113-117.

Wenger, N.K. 1995. Hypertension and other cardiovascular risk factors in women. *American Journal of Hypertension*, 8(12 Pt 2):94s-99s.

Whaley, M.H., and S.N. Blair. 1995. Epidemiology of physical activity, physical fitness and coronary heart disease. *Journal of Cardiovascular Risk* 2(4):289-95.

WHO 1992. *The Victoria Declaration on heart health.* International Heart Health Conference, Victoria, Canada.

WHO.1994. *The Catalonia Declaration: Investing in Heart Health.* International Heart Health Conference, Barcelona, Spain, World Health Organization.

WHO. 1995. World Health Statistics Annual 1995, Geneva: WHO.

WHO. 1997. *The Jakarta Declaration on Leading Health Promotion into the 21st Century*. The Fourth International Conference on Health Promotion: New PLayers for a New Era -Leading Health Promotion into the 21st Century, Jakarta, Indonesia, World Health Organization.

WHO. 1998. *The Singapore Declaration: Forging the Will for Heart Health in the Next Millennium.* International Heart Health Conference, Singapore, Hong Kong, World Health Organization.

WHO. 1998. Population Health - Putting Concepts into Action.

WHO. 2001. *The Osaka Declaration: health, economics and political action: stemming the global tide of cardiovascular disease.* Fourth International Heart Health Conference, Osaka, Japan, World Health Organization.

Wigle, D. Book Review: *Why are sone people healthy and others not? The determinants of health of populations. Evans, R.G., Barer, M.L., and Marmor, T.R. (Eds.), New York: Aldine de Gruyter,* 1994. Canadian Journal of Public Health 1995;86(3):213.

Wilkinson, R. G. 1990. Income distribution and mortality: a natural experiment. *Social Health Illness.* 12:391-412.

Wilkinson, R. G. 1992. Income distribution and life expectancy. *BMJ* 304:165-168.

Wilkinson, R.G. 1994. The epidemiological transition: from material society to social disadvange? *Daedalus* 123:61-77.

Wilkinson, R.G. 1996. *Unhealthy Societies: The afflictions of Inequality.* Routledge: London.

Wilkinson, R.G. 1997. Socioeconomic determinants of health. Health inequalities: relative or absolute material standards? *BMJ* 314(7080):591-5.

Wilkinson, R.G. and M. Marmot. (Eds.) 1998. *Social Determinants of Health. The Solid Facts.* WHO Regional Office for Europe.

Wilkinson, R.G., IB. MacCallum, H. Rooijmans, DF. Murray, GD. Cockcroft, JR. McKnight, JA. Webb, D.J. 2000. Increased augmentation index and systolic stress in type 1 diabetes mellitus. *QJM.* 93(7):441-8.

Williams, J.I., and W. Young. 1996. *Inventory of Studies on the Accuracy of Canadian Health Administrative Databases.* Toronto: Institute for Clinical and Evaluative Sciences.

Wing S., M. Casper, W. Davis, C. Hayes, W. Riggan, and H.A. Tyroler. 1990. Trends in the geographic inequality of cardiovascular disease mortality in the United States, 1962-1982. *SSM* 30 (3): 261-266.

Wing, S. E. Barnett, M. Casper, and H.A. Tyroler. 1992. Geographic and socioeconomic variation in the onset of decline of coronary heart disease mortality in white women. *American Journal of Public Health* 82(2):204-9.

Winkleby, M.A., H.C. Kraemer, D.K. Ahn, and A.N. Varady. 1998. Ethnic and socioeconomic differences in cardiovascular disease risk factors: findings for women from the Third National Health and Nutrition Examination Survey, 1988-1994. *JAMA* 280(4):356-62.

Woodward, M., M.C. Shewry, W.C. Smith, and H. Tunstall-Pedoe. 1992. Social status and coronary heart disease: results from the Scottish Heart Health Study. *Preventive Medicine* 21(1):136-48.

Wolf, S., and J.G. Bruhn. 1993. *The Power of Clan: The Influence of Human Relationships on Heart Disease.* New Jersey: Transaction Publishers.

Wolf, P.A., et al. 1992. Epidemiology of stroke. In *Stroke, Pathophysiology, Diagnosis and Management,* eds. Barnett, J.J., 3-29. New York: Churchill Livingstone.

Wolfson, M., G. Kaplan, J. Lynch, N. Ross, and E. Backlund. 1999. Relation between income inequality and mortality: empirical demonstration. *BMJ* 319(7215):953-955.

York, E., R.E. Mitchell, and A. Graybiel. 1986. Cardiovascular epidemiology, exercise, and health: 40-year followup of the U.S. Navy's "1000 aviators". *Aviation Space & Environmental Medicine* 57(6):597-9.

Zock, P.L. and M.B. Katan. 1997. Trans fatty acids, lipoproteins, and coronary risk. *Canadian Journal of Physiology & Pharmacology* 75(3):211-216.

APPENDICES

Appendix 4.1 Significant PHUs with dissimilar neighbours (CVD mortality, both sexes combined, 1986-1989)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	2.30	-2.80	0.855	Halton	0.951
				Wellington-Dufferin-Guelph	1.036
				Simcoe	1.031
				York	0.952
				Toronto	0.852
York	1.29	-2.36	0.952	Peel	0.855
				Simcoe	1.031
				Durham	1.105
				Toronto	0.852
Wellington-Dufferin-Guelph	0.22	-2.20	1.036	Halton	0.951
				Hamilton-Wentworth	1.040
				Waterloo	1.017
				Perth	0.978
				Huron	1.051
				Bruce-Grey-Owen Sound	1.069
				Simcoe	1.031
				Peel	0.855
Durham	-0.36	-2.06	1.105	Toronto	0.852
				York	0.952
				Simcoe	1.031
				Haliburton-Karwatha	1.098
Toronto	2.18	-1.99	0.852	Peel	0.855
				York	0.952
				Durham	1.105

Appendix 4.2 Significant PHUs with dissimilar neighbours (CVD mortality, both sexes combined, 1990-1994)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	2.17	-2.89	0.847	Halton	0.868
				Wellington-Dufferin-Guelph	1.063
				Simcoe	1.082
				York	0.872
				Toronto	0.840
Toronto	2.79	-2.69	0.840	Peel	0.847
				York	0.872
				Durham	1.020
York	1.90	-2.46	0.872	Toronto	0.840
				Peel	0.847
				Simcoe	1.082
				Durham	1.020
Durham	0.47	-2.38	1.020	Toronto	0.840
				York	0.872
				Simcoe	0.082
				Haliburton-Karwatha	1.053

Appendix 4.3 Significant PHUs with dissimilar neighbours (Female CVD mortality, 1986-1989)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
York	0.74	-2.13	0.994	Durham	1.130
				Simcoe	1.025
				Peel	10.867
				Toronto	0.863
Peel	1.84	-2.26	0.867	Halton	1.001
				Wellington-Dufferin-Guelph	1.047
				Simcoe	1.025
				York	0.994
				Toronto	1.863
Wellington-Dufferin-Guelph	0.09	-2.15	1.047	Halton	1.001
				Hamilton-Wentworth	1.035
				Waterloo	0.998
				Perth	0.985
				Huron	1.037
				Bruce-Grey-Owen Sound	1.041
				Simcoe	1.025
				Peel	0.867

Appendix 4.4 Significant PHUs with dissimilar neighbours (Female CVD mortality, 1990-1994)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	1.71	-2.59	0.881	Halton	0.879
				Wellington-Dufferin-Guelph	1.050
				Simcoe	1.061
				York	0.941
				Toronto	0.844
Toronto	2.14	-2.08	0.844	Peel	0.881
				York	0.941
				Durham	1.029
York	1.17	-2.22	0.941	Toronto	0.844
				Peel	0.881
				Simcoe	1.061
				Durham	1.029
Durham	0.30	-2.09	1.029	Toronto	0.844
				York	0.941
				Simcoe	1.061
				Haluburton-Kawartha	1.067

Appendix 4.5 Significant PHUs with dissimilar neighbours (Male CVD mortality, 1986-1989)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Wellington-Dufferin-Guelph	0.32	-2.20	1.016	Peel	0.844
				Halton	0.899
				Hamilton-Wentworth	1.045
				Waterloo	1.046
				Perth	0.975
				Huron	1.038
				Bruce-Grey-Owen Sound	1.068
				Simcoe	1.021
Peel	2.47	-3.12	0.844	Halton	0.899
				Wellington-Dufferin-Guelph	1.016
				Simcoe	1.021
				York	0.905
				Toronto	0.852

Appendix 4.5 (continued)					
Toronto	2.35	-2.29	0.852	Peel	0.844
				York	0.905
				Durham	1.073
York	1.67	-2.45	0.905	Peel	0.844
				Simcoe	1.021
				Durham	1.073
				Toronto	0.852
Durham	-0.07	-2.43	1.073	Toronto	0.852
				York	0.905
				Simcoe	1.021
				Haliburton-Kawartha	1.055

Appendix 4.6 Significant PHUs with dissimilar neighbours Male CVD mortality, 1990-1994					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	2.39	-3.02	0.812	Halton	0.853
				Wellington-Dufferin-Guelph	1.067
				Simcoe	1.092
				York	0.797
				Toronto	0.843
Simcoe	-0.09	-2.26	1.092	York	0.797
				Peel	0.812
				Wellington-Dufferin-Guelph	1.067
				Bruce-Grey-Owen Sound	1.173
				Muskoka-Parry-Sound	0.960
				Haliburton-Kawartha	1.022
				Durham	1.006
Toronto	2.97	-3.07	0.843	Peel	0.812
				York	0.797
				Durham	1.006
York	2.40	-2.56	0.797	Toronto	0.843
				Peel	0.812
				Simcoe	1.092
				Durham	1.006
Durham	0.60	-2.56	1.006	Toronto	0.843
				York	0.797
				Simcoe	1.092
				Haliburton-Kawartha	1.022

Appendix 4.7 Significant PHUs with dissimilar neighbours CVD morbidity, both sexes combined, 1986-1989					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	1.04	-2.34	0.926	Halton	0.974
				Wellington-Dufferin-Guelph	1.000
				Simcoe	1.109
				York	0.845
				Toronto	0.834
Durham	0.52	-2.07	1.014	Toronto	0.834
				York	0.845
				Simcoe	1.109
				Haliburton-Karwatha	1.104
Toronto	1.75	-2.06	0.834	Peel	0.926
				York	0.845
				Durham	1.014

Appendix 4.8 Significant PHUs with dissimilar neighbours CVD morbidity, both sexes combined, 1990-1994					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Durham	0.55	-2.38	1.017	Toronto	0.847
				York	0.816
				Simcoe	1.088
				Haliburton-Karwatha	1.047
Peel	1.09	-2.29	0.899	Halton	0.973
				Wellington-Dufferin-Guelph	1.007
				Simcoe	1.088
				York	0.816
				Toronto	0.847
Toronto	1.62	-2.10	0.847	Peel	0.899
				York	0.816
				Durham	1.017

Appendix 4.9 Significant PHUs with dissimilar neighbours (Female CVD morbidity, 1986-1989)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	0.89	-2.16	0.932	Halton	0.986
				Wellington-Dufferin-Guelph	1.010
				Simcoe	1.125
				York	0.854
				Toronto	0.821
Durham	0.54	-2.25	1.022	Haliburton-Kawartha	1.088
				Simcoe	1.125
				York	0.854
				Toronto	0.821

Appendix 4.10 Significant PHUs with dissimilar neighbours (Female CVD morbidity, 1990-1994)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	0.91	-2.11	0.900	Halton	0.964
				Wellington-Dufferin-Guelph	1.026
				Simcoe	1.092
				York	0.814
				Toronto	0.839
Durham	0.48	-2.39	1.030	Toronto	0.839
				York	0.814
				Simcoe	1.092
				Haliburton-Kawartha	1.054

Appendix 4.11 Significant PHUs with dissimilar neighbours (Male CVD morbidity, 1986-1989)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	1.23	-2.53	0.916	Halton	0.958
				Wellington-Dufferin-Guelph	0.985
				Simcoe	1.087
				York	0.830
				Toronto	0.854
Toronto	1.93	2.28	0.854	Peel	0.816
				York	0.830
				Durham	0.999
York	1.56	2.02	0.830	Toronto	0.854
				Peel	0.916
				Simcoe	1.087
				Durham	0.999

Appendix 4.12 Significant PHUs with dissimilar neighbours (Male CVD morbidity, 1990-1994)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	1.32	-2.51	0.894	Halton	0.975
				Wellington-Dufferin-Guelph	0.985
				Simcoe	1.078
				York	0.809
				Toronto	0.861
York	1.66	-2.07	0.809	Toronto	0.861
				Peel	0.894
				Simcoe	1.078
				Durham	1.001
Toronto	1.91	-2.37	0.861	Peel	0.894
				York	0.809
				Durham	1.001
Durham	0.63	-2.37	1.001	Toronto	0.861
				York	0.809
				Simcoe	1.078
				Haliburton-Kawartha	1.031

Appendix 4.13 Significant PHUs with dissimilar neighbours (IHD mortality, both sexes combined, 1986-1989)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	2.22	-2.84	0.806	Halton	0.938
				Wellington-Dufferin-Guelph	0.994
				Simcoe	1.060
				York	0.879
				Toronto	0.813
York	1.53	-2.24	0.879	Peel	0.806
				Simcoe	1.060
				Durham	1.107
				Toronto	0.813
Toronto	2.26	-2.21	0.813	Peel	0.806
				York	0.879
				Durham	1.107
Durham	-0.16	-2.02	1.107	Toronto	0.813
				York	0.879
				Simcoe	1.060
				Haliburton-Kawartha	1.074

Appendix 4.14 Significant PHUs with dissimilar neighbours (IHD mortality, both sexes combined, 1990-1994)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	1.77	-2.61	0.787	Halton	0.880
				Wellington-Dufferin-Guelph	1.042
				Simcoe	1.087
				York	0.802
				Toronto	0.777
York	1.72	-2.32	0.802	Toronto	0.777
				Peel	0.787
				Simcoe	1.087
				Durham	1.004
Durham	0.52	-2.30	1.004	Toronto	0.777
				York	0.802
				Simcoe	1.087
				Haliburton-Kawartha	1.065

Appendix 4.15 Significant PHUs with dissimilar neighbours (Female IHD mortality, 1986-1989)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
York	0.95	-2.03	0.935	Toronto	0.818
				Peel	0.780
				Simcoe	1.056
				Durham	1.121
Peel	1.65	-2.11	0.780	Halton	1.021
				Wellington-Dufferin-Guelph	1.002
				Simcoe	1.056
				York	0.935
				Toronto	0.818

Appendix 4.16 Significant PHUs with dissimilar neighbours (Female IHD mortality, 1990-1994)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	1.29	-2.28	0.812	Halton	0.867
				Wellington-Dufferin-Guelph	1.007
				Simcoe	1.069
				York	0.861
				Toronto	0.776
York	1.07	-1.99	0.861	Toronto	0.776
				Peel	0.812
				Simcoe	1.069
				Durham	1.001
Durham	0.40	-1.99	1.001	Toronto	0.776
				York	0.861
				Simcoe	1.069
				Haliburton-Kawartha	1.061
Toronto	1.64	-0.07	0.776	Peel	0.812
				York	0.861
				Durham	1.001

Appendix 4.17 Significant PHUs with dissimilar neighbours (Male IHD mortality, 1986-1989)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	2.42	-3.32	0.830	Halton	0.864
				Wellington-Dufferin-Guelph	0.977
				Simcoe	1.043
				York	0.823
				Toronto	0.820
Toronto	2.41	-2.41	0.820	Peel	0.830
				York	0.823
				Durham	1.085
York	1.91	-2.29	0.823	Toronto	0.820
				Peel	0.830
				Simcoe	1.043
				Durham	1.085
Durham	-0.07	-2.40	1.085	Toronto	0.820
				York	0.823
				Simcoe	1.043
				Haliburton-Kawartha	1.042

Appendix 4.18 Significant PHUs with dissimilar neighbours (Male IHD mortality, 1990-1994)					
Hot spot	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	2.12	-2.82	0.765	Halton	0.885
				Wellington-Dufferin-Guelph	1.063
				Simcoe	1.088
				York	0.743
				Toronto	0.786
Toronto	2.81	-2.93	0.786	Peel	0.765
				York	0.743
				Durham	1.001
Simcoe	0.04	-2.24	1.088	York	0.743
				Peel	0.765
				Wellington-Dufferin-Guelph	1.063
				Bruce-Grey-Owen Sound	1.204
				Muskoka-Parry Sound	0.932
				Haliburton-Kawartha	1.044

Appendix 4.18 (continued)					
York	2.30	-2.55	0.743	Toronto	0.786
				Peel	0.765
				Simcoe	1.088
				Durham	1.001
Durham	0.60	-2.55	1.001	Toronto	0.786
				York	0.743
				Simcoe	1.088
				Haliburton-Kawartha	1.044

Appendix 4.19 Significant PHUs with dissimilar neighbours (IHD morbidity, both sexes combined, 1986-1989)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	1.06	-2.56	0.921	Halton	0.983
				Wellington-Dufferin-Guelph	0.903
				Simcoe	1.067
				York	0.799
				Toronto	0.815
Perth	0.61	-2.12	0.985	Wellington-Dufferin-Guelph	0.903
				Waterloo	0.888
				Oxford	1.081
				Middlesex-London	0.831
				Huron	1.067
Wellington-Dufferin-Guelph	0.72	-2.17	0.903	Peel	0.921
				Halton	0.983
				Hamilton-Wentworth	1.008
				Waterloo	0.858
				Perth	0.985
				Huron	1.067
				Bruce-Grey-Owen Sound	1.061
				Simcoe	1.067

Appendix 4.20 Significant PHUs with dissimilar neighbours (IHD morbidity, both sexes combined, 1990-1994)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Perth	0.87	-2.22	0.923	Huron	1.040
				Middlesex-London	0.798
				Oxford	1.096
				Waterloo	0.830
				Wellington-Dufferin-Guelph	0.908
Wellington-Dufferin-Guelph	0.70	-2.26	0.908	Bruce-Grey-Owen Sound	1.089
				Huron	1.040
				Perth	0.923
				Waterloo	0.830
				Hamilton-Wentworth	1.075
				Halton	0.972
				Peel	0.853
				Simcoe	1.090
Peel	1.25	-2.45	0.853	Halton	0.972
				Wellington-Dufferi-Guelph	0.908
				Simcoe	1.090
				York	0.804
				Toronto	0.808
Toronto	1.55	-1.99	0.808	Peel	0.853
				York	0.804
				Durham	1.047
Durham	0.35	-2.03	1.047	Toronto	0.808
				York	0.804
				Simcoe	1.090
				Haliburton-Kawartha	1.112

Appendix 4.21 Significant PHUs with dissimilar neighbours (Female IHD morbidity, 1986-1989)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Perth	0.62	-2.09	0.971	Huron	1.119
				Wellington-Dufferin-Guelph	0.887
				Waterloo	0.838
				Oxford	1.076
				Middlesex-London	0.747

Appendix 4.21 (continued)					
Peel	0.92	-2.23	0.896	Halton	1.020
				Wellington-Dufferin-Guelph	0.887
				Simcoe	1.110
				York	0.802
				Toronto	0.792

Appendix 4.22 Significant PHUs with dissimilar neighbours (Female IHD morbidity, 1990-1994)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Perth	0.65	-2.14	0.960	Middlesex-London	0.726
				Huron	1.088
				Wellington-Dufferin-Guelph	0.876
				Waterloo	0.811
				Oxford	1.077
Peel	1.19	-2.36	0.812	Halton	0.966
				Wellington-Dufferin-Guelph	0.876
				Simcoe	1.113
				York	0.744
				Toronto	0.784
Durham	0.28	-2.17	1.082	Toronto	0.784
				York	0.744
				Simcoe	1.113
				Haliburton-Kawartha	1.127

Appendix 4.23 Significant PHUs with dissimilar neighbours (Male IHD morbidity, 1986-1989)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Perth	0.52	-2.04	0.998	Oxford	1.083
				Middlesex-London	0.895
				Huron	1.020
				Wellington-Dufferin-Guelph	0.906
				Waterloo	0.845
Wellington-Dufferin-Guelph	0.82	-2.40	0.906	Halton	0.950
				Hamilton-Wentworth	1.014
				Waterloo	0.875
				Perth	0.998
				Huron	1.020
				Bruce-Grey-Owen Sound	1.012
				Simcoe	1.028
				Peel	0.927
Peel	1.17	-2.81	0.927	Halton	0.950
				Wellington-Dufferin-Guelph	0.906
				Simcoe	1.028
				York	0.784
				Toronto	0.842
York	1.56	-1.96	0.784	Toronto	0.842
				Peel	0.927
				Simcoe	1.028
				Durham	1.022
Toronto	1.65	-2.08	0.842	Peel	0.927
				York	0.784
				Durham	1.022

Appendix 4.24 Significant PHUs with dissimilar neighbours (Male IHD morbidity, 1990-1994)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Perth	1.02	-2.23	0.900	Waterloo	0.845
				Oxford	1.104
				Middlesex-London	0.852
				Huron	0.998
				Bruce-Grey-Owen Sound	1.021
				Wellington-Dufferin-Guelph	0.919
Peel	1.30	-2.52	0.871	Halton	0.967
				Wellington-Dufferin-Guelph	0.919
				Simcoe	1.068
				York	0.825
				Toronto	0.834
Simcoe	0.17	-2.01	1.068	Bruce-Grey-Owen Sound	1.021
				Wellington-Dufferin-Guelph	0.919
				Peel	0.871
				York	0.825
				Durham	1.018
				Haliburton-Kawartha	1.090
				Muskoka-Parry Sound	1.134
Toronto	1.64	-2.09	0.834	Peel	0.871
				York	0.825
				Durham	1.018
Wellington-Dufferin-Guelph	0.80	-2.53	0.919	Halton	0.967
				Hamilton-Wentworth	1.075
				Waterloo	0.845
				Perth	0.900
				Huron	0.998
				Bruce-Grey-Owen Sound	1.021
				Simcoe	1.068
				Peel	0.871
York	1.34	-1.97	0.825	Simcoe	1.068
				Peel	0.871
				Toronto	0.834
				Durham	1.018

Appendix 4.25 Significant PHUs with dissimilar neighbours (Female CBVD mortality, 1986-1989)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Leeds-Grenville-Lanark & Addington	-0.14	-2.19	1.062	Eastern Ontario Ottawa-Carlton Renfrew Kingston-Frontenac	0.918 0.801 1.093 0.845

Appendix 4.26 Significant PHUs with dissimilar neighbours (Female CBVD mortality, 1990-1994)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Sudbury District	-0.47	-2.01	1.076	Algoma Porcupine Timiskaming North Bay Muskoka-Parry Sound	1.074 0.876 0.858 0.852 1.034

Appendix 4.27 Significant PHUs with dissimilar neighbours (CBVD morbidity, both sexes combined, 1986-1989)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	1.08	-2.50	0.940	Halton Wellington-Dufferin-Guelph Simcoe York Toronto	0.914 1.065 1.079 0.839 0.833
Durham	0.22	-2.49	1.073	Toronto York Simcoe Haliburton-Kawartha	0.833 0.839 1.079 1.074

Appendix 4.28 Significant PHUs with dissimilar neighbours
(CBVD morbidity, both sexes combined, 1990-1994)

Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	0.90	-2.35	0.938	Halton	0.891
				Wellington-Dufferin-Guelph	1.053
				Simcoe	1.049
				York	0.810
				Toronto	0.865
Durham	0.17	-2.52	1.073	Toronto	0.865
				York	0.810
				Simcoe	1.049
				Haliburton-Kawartha	0.986

Appendix 4.29 Significant PHUs with dissimilar neighbours
(Female CBVD morbidity, 1986-1989)

Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Durham	0.20	-2.47	1.087	Haliburton-Kawartha	1.071
				Simcoe	1.076
				York	0.837
				Toronto	0.833
Peel	1.11	-2.46	0.935	Toronto	0.833
				York	0.837
				Simcoe	1.076
				Wellington-Dufferin-Guelph	1.075
				Halton	0.944

Appendix 4.30 Significant PHUs with dissimilar neighbours (Female CBVD morbidity, 1990-1994)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Durham	0.48	-2.39	1.096	Toronto	0.872
				York	0.854
				Simcoe	1.062
				Haliburton-Kawartha	0.981
Peel	0.91	-2.11	0.961	Halton	0.896
				Wellington-Dufferin-Guelph	1.091
				Simcoe	1.062
				York	0.854
				Toronto	0.872

Appendix 4.31 Significant PHUs with dissimilar neighbours (Male CBVD morbidity, 1986-1989)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	0.91	-2.38	0.944	Halton	0.880
				Wellington-Dufferin-Guelph	1.049
				Simcoe	1.074
				York	0.836
				Toronto	0.841
Durham	0.23	-2.37	1.053	Toronto	0.841
				York	0.836
				Simcoe	1.074
				Haliburton-Kawartha	1.066
				Peterborough	0.842

Appendix 4.32 Significant PHUs with dissimilar neighbours (Male CBVD morbidity, 1990-1994)					
Significant PHU	Moran's *I*	Z-score	CMF	First order neighbours	CMF
Peel	1.31	-2.84	0.914	Halton	0.882
				Wellington-Dufferin-Guelph	1.009
				Simcoe	1.028
				York	0.760
				Toronto	0.864
Halton	1.39	-1.96	0.882	Hamilton-Wentworth	0.856
				Wellington-Dufferin-Guelph	1.009
				Peel	0.914
Toronto	1.68	-2.20	0.864	Peel	0.914
				York	0.760
				Durham	1.046
Durham	0.36	-2.63	1.046	Toronto	0.864
				York	0.760
				Simcoe	1.028
				Haliburton-Kawartha	0.977
				Peterborough	0.955

Appendix 4.33 Bivariate Correlation Between CVD Mortality and Economic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Living in a rental unit	-0.192	0.256	-0.217	0.197	-0.118	0.488
Dwelling needs major repairs	0.449	0.003	0.313	0.030	0.469	0.002
Average dwelling value	-0.697	0.001	-0.559	0.001	-0.751	0.001
Average household income	-0.511	0.001	-0.398	0.007	-0.562	0.001
Income inequality	0.492	0.001	0.369	0.012	0.559	0.001
Unemployment rate	0.307	0.032	0.277	0.054	0.286	0.046
Incidence of low-income family	0.175	0.230	0.125	0.392	0.224	0.121
	1990-1994					
Living in a rental unit	-0.135	0.426	-0.146	0.389	-0.088	0.604
Dwelling needs major repairs	0.368	0.012	0.283	0.045	0.369	0.012
Average dwelling value	-0.743	0.001	-0.641	0.001	-0.781	0.001
Average household income	-0.604	0.001	-0.501	0.001	-0.654	0.001
Income inequality	0.544	0.001	0.439	0.003	0.605	0.001
Unemployment rate	0.203	0.161	0.190	0.191	0.199	0.171
Incidence of low-income family	0.134	0.360	0.117	0.424	0.163	0.263

Appendix 4.34 Bivariate Correlation Between CVD Morbidity and Economic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Living in a rental unit	-0.290	0.081	-0.328	0.047	-0.218	0.194
Dwelling needs major repairs	0.562	0.001	0.509	0.001	0.584	0.001
Average dwelling value	-0.757	0.001	-0.740	0.001	-0.756	0.001
Average household income	-0.591	0.001	-0.561	0.001	-0.615	0.001
Income inequality	0.534	0.001	0.486	0.001	0.581	0.001
Unemployment rate	0.406	0.004	0.371	0.009	0.424	0.002
Incidence of low-income family	0.166	0.254	0.114	0.437	0.239	0.098
	1990-1994					
Living in a rental unit	-0.255	0.128	-0.273	0.102	-0.206	0.221
Dwelling needs major repairs	0.543	0.001	0.510	0.001	0.540	0.001
Average dwelling value	-0.744	0.001	-0.723	0.001	-0.758	0.001
Average household income	-0.578	0.001	-0.545	0.001	-0.610	0.001
Income inequality	0.538	0.001	0.509	0.001	0.571	0.001
Unemployment rate	0.413	0.003	0.404	0.004	0.411	0.003
Incidence of low-income family	0.187	0.199	0.162	0.267	0.227	0.117

Appendix 4.35 Bivariate Correlation Between IHD Mortality and Economic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Living in a rental unit	-0.196	0.245	-0.262	0.117	-0.092	0.587
Dwelling needs major repairs	0.295	0.038	0.067	0.348	0.404	0.007
Average dwelling value	-0.662	0.001	-0.512	0.001	-0.728	0.001
Average household income	-0.551	0.001	-0.424	0.004	-0.611	0.001
Income inequality	0.495	0.001	0.349	0.017	0.582	0.001
Unemployment rate	0.275	0.056	0.202	0.164	0.289	0.044
Incidence of low-income family	0.180	0.215	0.119	0.415	0.239	0.098
	1990-1994					
Living in a rental unit	-0.140	0.407	-0.180	0.287	-0.080	0.638
Dwelling needs major repairs	0.293	0.039	0.239	0.077	0.284	0.044
Average dwelling value	-0.668	0.001	-0.571	0.001	-0.719	0.001
Average household income	-0.563	0.001	-0.489	0.001	-0.599	0.001
Income inequality	0.488	0.001	0.420	0.005	0.530	0.001
Unemployment rate	0.189	0.193	0.200	0.168	0.161	0.270
Incidence of low-income family	0.121	0.406	0.157	0.281	0.103	0.483

Appendix 4.36 Bivariate Correlation Between IHD Morbidity and Economic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Living in a rental unit	-0.214	0.202	-0.286	0.087	-0.122	0.471
Dwelling needs major repairs	0.489	0.001	0.424	0.004	0.522	0.001
Average dwelling value	-0.728	0.001	-0.693	0.001	-0.731	0.001
Average household income	-0.590	0.001	-0.532	0.001	-0.628	0.001
Income inequality	0.560	0.001	0.495	0.001	0.610	0.001
Unemployment rate	0.455	0.001	0.446	0.001	0.439	0.002
Incidence of low-income family	0.280	0.051	0.243	0.092	0.319	0.026
	1990-1994					
Living in a rental unit	-0.175	0.301	-0.197	0.243	-0.128	0.450
Dwelling needs major repairs	0.481	0.001	0.456	0.002	0.471	0.002
Average dwelling value	-0.704	0.001	-0.702	0.001	-0.688	0.001
Average household income	-0.575	0.001	-0.562	0.001	-0.579	0.001
Income inequality	0.553	0.001	0.551	0.001	0.551	0.001
Unemployment rate	0.468	0.001	0.474	0.001	0.446	0.001
Incidence of low-income family	0.279	0.052	0.273	0.058	0.291	0.042

Appendix 4.37 Bivariate Correlation Between CBVD Mortality and Economic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Living in a rental unit	-0.356	0.030	-0.281	0.092	-0.328	0.047
Dwelling needs major repairs	0.248	0.069	0.258	0.062	0.181	0.141
Average dwelling value	-0.143	0.199	0.021	0.452	-0.269	0.054
Average household income	-0.063	0.355	-0.022	0.448	-0.086	0.307
Income inequality	-0.003	0.492	-0.047	0.392	0.049	0.386
Unemployment rate	0.157	0.280	0.126	0.390	0.125	0.394
Incidence of low-income family	-0.158	0.277	-0.143	0.328	-0.097	0.506
	1990-1994					
Living in a rental unit	-0.275	0.099	-0.193	0.251	-0.216	0.199
Dwelling needs major repairs	0.043	0.401	-0.111	0.257	0.188	0.132
Average dwelling value	-0.140	0.204	0.066	0.349	-0.332	0.022
Average household income	-0.123	0.235	0.075	0.329	-0.322	0.026
Income inequality	0.021	0.451	-0.191	0.129	0.269	0.054
Unemployment rate	-0.137	0.349	-0.182	0.210	0.018	0.903
Incidence of low-income family	-0.209	0.150	-0.282	0.050	0.015	0.918

Appendix 4.38 Bivariate Correlation Between CBVD Morbidity and Economic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Living in a rental unit	-0.194	0.250	-0.232	0.167	-0.114	0.502
Dwelling needs major repairs	0.373	0.012	0.296	0.038	0.396	0.008
Average dwelling value	-0.743	0.001	-0.738	0.001	-0.690	0.001
Average household income	-0.568	0.001	-0.583	0.001	-0.511	0.001
Income inequality	0.462	0.002	0.433	0.004	0.458	0.002
Unemployment rate	0.201	0.166	0.198	0.172	0.175	0.228
Incidence of low-income family	0.050	0.732	-0.018	0.904	0.124	0.394
	1990-1994					
Living in a rental unit	-0.232	0.168	-0.226	0.178	-0.196	0.246
Dwelling needs major repairs	0.347	0.018	0.313	0.030	0.349	0.017
Average dwelling value	-0.727	0.001	-0.648	0.001	-0.778	0.001
Average household income	-0.535	0.001	-0.447	0.003	-0.608	0.001
Income inequality	0.443	0.003	0.361	0.014	0.515	0.001
Unemployment rate	0.251	0.081	0.260	0.072	0.221	0.128
Incidence of low-income family	0.076	0.602	0.032	0.827	0.128	0.382

Appendix 4.39 Bivariate Correlation Between CVD Mortality and Social Capital Variables							
Variables		1986-1989					
		Both sexes		Females		Males	
		Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Number of voluntary organizations		-0.235	0.161	-0.301	0.070	-0.210	0.212
Average charitable donations		-0.023	0.894	-0.456	0.005	-0.472	0.003
Licensed day care facilities		-0.529	0.001	-0.442	0.006	-0.526	0.001
Per capita municipal expenditure on:	environmental defense	-0.154	0.291	-0.112	0.442	-0.128	0.383
	social assistance	0.047	0.750	-0.062	0.672	0.136	0.353
	recreation	0.085	0.563	0.096	0.514	0.073	0.618
	culture	-0.399	0.004	-0.375	0.008	-0.330	0.021
		1990-1994					
Number of voluntary organizations		-0.354	0.031	-0.381	0.020	-0.333	0.044
Average charitable donations		0.106	0.531	-0.417	0.010	-0.448	0.005
Licensed day care facilities		-0.519	0.001	-0.424	0.009	-0.544	0.001
Per capita municipal expenditure on:	environmental defense	-0.237	0.102	-0.220	0.128	-0.204	0.159
	social assistance	-0.054	0.711	-0.094	0.520	0.003	0.984
	recreation	-0.108	0.460	-0.146	0.318	-0.062	0.674
	culture	-0.442	0.001	-0.438	0.002	-0.378	0.007

Appendix 4.40 Bivariate Correlation Between CVD Morbidity and Social Capital							
Variables		1986-1989					
		Both sexes		Females		Males	
		Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Number of voluntary organizations		-0.219	0.192	-0.132	0.438	-0.321	0.053
Average charitable donations		-0.500	0.002	-0.489	0.002	-0.488	0.002
Licensed day care facilities		-0.685	0.001	-0.683	0.001	-0.656	0.001
Per capita municipal expenditure on:	environmental defense	-0.271	0.059	-0.267	0.063	-0.243	0.092
	social assistance	-0.105	0.471	-0.109	0.455	-0.085	0.561
	recreation	0.052	0.725	0.079	0.591	0.017	0.907
	culture	-0.453	0.001	-0.412	0.003	-0.466	0.001
		1990-1994					
Number of voluntary organizations		-0.245	0.144	-0.197	0.243	-0.306	0.066
Average charitable donations		-0.463	0.004	-0.443	0.006	-0.467	0.004
Licensed day care facilities		-0.650	0.001	-0.632	0.001	-0.649	0.001
Per capita municipal expenditure on:	environmental defense	-0.251	0.082	-0.223	0.124	-0.262	0.069
	social assistance	-0.069	0.637	-0.077	0.597	-0.045	0.758
	recreation	0.061	0.679	0.108	0.462	0.008	0.958
	culture	-0.400	0.004	-0.371	0.009	-0.405	0.004

Appendix 4.41 Bivariate Correlation Between IHD Mortality and Social Capital Variables							
Variables		1986-1989					
		Both sexes		Females		Males	
		Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Number of voluntary organizations		-0.365	0.026	-0.378	0.021	-0.365	0.026
Average charitable donations		0.109	0.521	-0.390	0.017	-0.442	0.006
Licensed day care facilities		-0.531	0.001	-0.457	0.004	-0.513	0.001
Per capita municipal expenditure on:	environmental defense	-0.242	0.093	-0.246	0.089	-0.167	0.251
	social assistance	0.009	0.951	-0.106	0.468	0.120	0.410
	recreation	-0.091	0.533	-0.072	0.621	-0.092	0.530
	culture	-0.396	0.005	-0.318	0.026	-0.367	0.009
		1990-1994					
Number of voluntary organizations		-0.368	0.025	-0.397	0.015	-0.342	0.038
Average charitable donations		0.169	0.317	-0.342	0.039	-0.418	0.010
Licensed day care facilities		-0.492	0.002	-0.424	0.009	-0.503	0.002
Per capita municipal expenditure on:	environmental defense	-0.240	0.097	-0.202	0.165	-0.222	0.126
	social assistance	-0.092	0.528	-0.094	0.518	-0.061	0.677
	recreation	-0.166	0.254	-0.187	0.198	-0.132	0.367
	culture	-0.387	0.006	-0.339	0.017	-0.375	0.008

Appendix 4.42 Bivariate Correlation Between IHD Morbidity and Social Capital Variables							
Variables		1986-1989					
		Both sexes		Females		Males	
		Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Number of voluntary organizations		-0.324	0.051	-0.244	0.145	-0.399	0.014
Average charitable donations		-0.470	0.003	-0.444	0.006	-0.459	0.004
Licensed day care facilities		-0.615	0.001	-0.609	0.001	-0.581	0.001
Per capita municipal expenditure on:	environmental defense	-0.253	0.080	-0.262	0.069	-0.212	0.144
	social assistance	-0.036	0.804	-0.050	0.732	-0.006	0.966
	recreation	-0.024	0.872	0.058	0.693	-0.097	0.509
	culture	-0.488	0.001	-0.445	0.001	-0.481	0.001
		1990-1994					
Number of voluntary organizations		-0.311	0.061	-0.257	0.125	-0.364	0.027
Average charitable donations		-0.458	0.004	-0.439	0.007	-0.449	0.005
Licensed day care facilities		-0.604	0.001	-0.595	0.001	-0.584	0.001
Per capita municipal expenditure on:	environmental defense	-0.337	0.018	-0.282	0.050	-0.360	0.011
	social assistance	-0.043	0.769	-0.027	0.851	-0.037	0.800
	recreation	-0.041	0.782	0.040	0.783	-0.111	0.447
	culture	-0.452	0.001	-0.409	0.004	-0.456	0.001

Appendix 4.43 Bivariate Correlation Between CBVD Mortality and Social Capital Variables							
Variables		1986-1989					
		Both sexes		Females		Males	
		Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Number of voluntary organizations		0.351	0.033	0.192	0.254	0.389	0.017
Average charitable donations		-0.195	0.248	-0.198	0.240	-0.268	0.109
Licensed day care facilities		-0.376	0.022	-0.256	0.126	-0.384	0.019
Per capita municipal expenditure on:	environmental defense	-0.071	0.627	-0.030	0.837	-0.067	0.648
	social assistance	-0.094	0.523	-0.152	0.297	-0.003	0.985
	recreation	0.248	0.086	0.089	0.541	0.311	0.030
	culture	-0.065	0.657	-0.079	0.589	-0.023	0.875
		1990-1994					
Number of voluntary organizations		0.065	0.702	0.125	0.460	-0.019	0.912
Average charitable donations		0.009	0.959	-0.156	0.355	-0.208	0.216
Licensed day care facilities		-0.304	0.067	-0.154	0.363	-0.376	0.022
Per capita municipal expenditure on:	environmental defense	-0.129	0.377	-0.270	0.060	0.097	0.509
	social assistance	-0.032	0.830	-0.210	0.148	0.201	0.165
	recreation	-0.108	0.459	-0.218	0.132	0.103	0.480
	culture	-0.120	0.412	-0.192	0.187	0.038	0.797

Appendix 4.44 Bivariate Correlation Between CBVD Morbidity and Social Capital Variables							
Variables		1986-1989					
		Both sexes		Females		Males	
		Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Number of voluntary organizations		-0.220	0.191	-0.132	0.436	-0.298	0.073
Average charitable donations		-0.504	0.001	-0.514	0.001	-0.444	0.006
Licensed day care facilities		-0.650	0.001	-0.685	0.001	-0.553	0.001
Per capita municipal expenditure on:	environmental defense	-0.286	0.046	-0.263	0.067	-0.272	0.058
	social assistance	-0.047	0.747	-0.044	0.762	-0.038	0.796
	recreation	0.041	0.781	0.003	0.983	0.078	0.594
	culture	-0.465	0.001	-0.435	0.002	-0.447	0.001
		1990-1994					
Number of voluntary organizations		-0.180	0.288	-0.166	0.325	-0.196	0.246
Average charitable donations		-0.428	0.008	-0.380	0.020	-0.450	0.005
Licensed day care facilities		-0.588	0.001	-0.521	0.001	-0.624	0.001
Per capita municipal expenditure on:	environmental defense	-0.174	0.232	-0.170	0.242	-0.153	0.294
	social assistance	-0.027	0.853	-0.050	0.731	0.010	0.948
	recreation	0.101	0.491	0.078	0.594	0.125	0.393
	culture	-0.378	0.007	-0.362	0.011	-0.354	0.013

Appendix 4.45 Bivariate Correlation Between CVD Mortality and Demographic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Less than high school education	0.420	0.005	0.323	0.025	0.405	0.006
Being unmarried	-0.222	0.093	-0.207	0.109	-0.136	0.212
Doctors' location quotient	-0.322	0.024	-0.285	0.047	-0.293	0.041
	1990-1994					
Less than high school education	0.456	0.002	0.346	0.018	0.489	0.001
Being unmarried	-0.227	0.089	-0.128	0.225	-0.206	0.111
Doctors' location quotient	-0.419	0.003	-0.440	0.002	-0.360	0.011

Appendix 4.46 Bivariate Correlation Between CVD Morbidity and Demographic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Less than high school education	0.630	0.001	0.624	0.001	0.589	0.001
Being unmarried	-0.394	0.008	-0.469	0.002	-0.274	0.050
Doctors' location quotient	-0.192	0.187	-0.137	0.349	-0.243	0.092
	1990-1994					
Less than high school education	0.600	0.001	0.612	0.001	0.546	0.001
Being unmarried	-0.334	0.022	-0.386	0.009	-0.222	0.094
Doctors' location quotient	-0.118	0.421	-0.080	0.586	-0.149	0.308

Appendix 4.47 Bivariate Correlation Between IHD Mortality and Demographic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Less than high school education	0.298	0.036	0.159	0.173	0.317	0.028
Being unmarried	-0.231	0.084	-0.214	0.101	-0.171	0.156
Doctors' location quotient	-0.360	0.011	-0.291	0.043	-0.354	0.013
	1990-1994					
Less than high school education	0.368	0.012	0.263	0.058	0.406	0.006
Being unmarried	-0.200	0.118	-0.106	0.266	-0.203	0.114
Doctors' location quotient	-0.390	0.006	-0.401	0.004	-0.351	0.013

Appendix 4.48 Bivariate Correlation Between IHD Morbidity and Demographic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Less than high school education	0.475	0.001	0.467	0.002	0.444	0.003
Being unmarried	-0.252	0.066	-0.283	0.045	-0.216	0.099
Doctors' location quotient	-0.222	0.125	-0.177	0.223	-0.243	0.092
	1990-1994					
Less than high school education	0.506	0.001	0.526	0.000	0.443	0.003
Being unmarried	-0.220	0.096	-0.232	0.084	-0.172	0.154
Doctors' location quotient	-0.091	0.536	-0.052	0.721	-0.112	0.444

Appendix 4.49 Bivariate Correlation Between CBVD Mortality and Demographic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Less than high school education	0.386	0.009	0.305	0.033	0.341	0.020
Being unmarried	-0.262	0.059	-0.187	0.134	-0.107	0.265
Doctors' location quotient	0.035	0.811	0.042	0.773	0.019	0.896
	1990-1994					
Less than high school education	0.171	0.155	0.069	0.343	0.216	0.099
Being unmarried	-0.249	0.068	-0.214	0.102	-0.035	0.418
Doctors' location quotient	-0.230	0.113	-0.094	0.522	-0.271	0.060

Appendix 4.50 Bivariate Correlation Between CBVD Morbidity and Demographic Variables						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Less than high school education	0.561	0.001	0.565	0.001	0.476	0.001
Being unmarried	-0.481	0.001	-0.556	0.001	-0.330	0.023
Doctors' location quotient	-0.191	0.188	-0.157	0.282	-0.205	0.158
	1990-1994					
Less than high school education	0.544	0.001	0.521	0.001	0.519	0.001
Being unmarried	-0.417	0.005	-0.434	0.004	-0.306	0.033
Doctors' location quotient	-0.192	0.187	-0.149	0.306	-0.218	0.133

Appendix 4.51 Bivariate Correlation Between CVD Mortality and Psychosocial Health and Wellbeing						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Experiencing stressful life	-0.277	0.048	-0.116	0.247	-0.370	0.012
Perceived dissatisfaction with social life	-0.322	0.026	-0.076	0.327	-0.355	0.016
Unhappiness in life	-0.029	0.432	0.057	0.369	-0.064	0.354
Perceived dissatisfaction with health	0.274	0.051	0.058	0.367	0.368	0.012
Perceived low well-being	0.095	0.287	0.004	0.492	0.155	0.179
Self-reported poor health status	0.537	0.001	0.233	0.083	0.656	0.001
Physical activity limitation	0.423	0.005	0.126	0.228	0.551	0.001
	1990-1994					
Experiencing stressful life	-0.177	0.148	0.015	0.465	-0.322	0.026
Perceived dissatisfaction with social life	-0.227	0.089	-0.034	0.422	-0.200	0.118
Unhappiness in life	0.016	0.464	0.076	0.327	-0.008	0.481
Perceived dissatisfaction with health	0.100	0.278	-0.138	0.208	0.289	0.041
Perceived low well-being	0.219	0.096	0.114	0.250	0.317	0.028
Self-reported poor health status	0.365	0.013	0.099	0.279	0.509	0.001
Physical activity limitation	0.394	0.008	0.177	0.148	0.490	0.001

Appendix 4.52 Bivariate Correlation Between CVD Morbidity and Psychosocial Health and Wellbeing						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Experiencing stressful life	-0.514	0.001	-0.400	0.007	-0.545	0.001
Perceived dissatisfaction with social life	-0.415	0.005	-0.278	0.048	-0.356	0.015
Unhappiness in life	-0.136	0.212	0.084	0.311	-0.184	0.138
Perceived dissatisfaction with health	0.138	0.208	0.089	0.300	0.180	0.143
Perceived low well-being	0.034	0.421	0.060	0.363	0.063	0.356
Self-reported poor health status	0.499	0.001	0.311	0.031	0.579	0.001
Physical activity limitation	0.505	0.001	0.363	0.014	0.583	0.001
	1990-1994					
Experiencing stressful life	-0.429	0.004	-0.338	0.020	-0.432	0.004
Perceived dissatisfaction with social life	-0.395	0.008	-0.249	0.068	-0.382	0.010
Unhappiness in life	-0.068	0.345	0.119	0.242	-0.163	0.167
Perceived dissatisfaction with health	0.175	0.150	0.114	0.251	0.200	0.117
Perceived low well-being	0.087	0.305	0.122	0.236	0.055	0.374
Self-reported poor health status	0.527	0.001	0.367	0.013	0.578	0.001
Physical activity limitation	0.513	0.001	0.355	0.016	0.596	0.001

Appendix 4.53 Bivariate Correlation Between IHD Mortality and Psychosocial Health and Wellbeing						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Experiencing stressful life	-0.256	0.063	-0.102	0.274	-0.339	0.020
Perceived dissatisfaction with social life	-0.276	0.049	-0.021	0.451	-0.318	0.028
Unhappiness in life	-0.082	0.315	0.002	0.494	-0.083	0.312
Perceived dissatisfaction with health	0.091	0.297	-0.224	0.092	0.299	0.036
Perceived low wellbeing	0.064	0.354	-0.106	0.265	0.193	0.127
Self-reported poor health status	0.365	0.013	-0.011	0.474	0.536	0.001
Physical activity limitation	0.314	0.029	-0.022	0.449	0.513	0.001
	1990-1994					
Experiencing stressful life	-0.118	0.243	-0.017	0.459	-0.206	0.110
Perceived dissatisfaction with social life	-0.157	0.176	-0.008	0.480	-0.139	0.206
Unhappiness in life	-0.008	0.481	0.035	0.419	-0.035	0.418
Perceived dissatisfaction with health	0.043	0.401	-0.205	0.112	0.237	0.078
Perceived low wellbeing	0.235	0.080	0.101	0.276	0.322	0.026
Self-reported poor health status	0.304	0.034	0.065	0.351	0.459	0.002
Physical activity limitation	0.326	0.024	0.110	0.259	0.434	0.004

Appendix 4.54 Bivariate Correlation Between IHD Morbidity and Psychosocial Health and Wellbeing						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Experiencing stressful life	-0.433	0.004	-0.286	0.043	-0.471	0.002
Perceived dissatisfaction with social life	-0.300	0.035	-0.104	0.269	-0.306	0.033
Unhappiness in life	-0.113	0.253	0.101	0.276	-0.191	0.129
Perceived dissatisfaction with health	0.155	0.180	0.052	0.379	0.176	0.149
Perceived low wellbeing	0.109	0.260	0.135	0.214	0.101	0.277
Self-reported poor health status	0.538	0.001	0.293	0.039	0.615	0.001
Physical activity limitation	0.472	0.002	0.279	0.047	0.566	0.001
	1990-1994					
Experiencing stressful life	-0.356	0.015	-0.261	0.059	-0.350	0.017
Perceived dissatisfaction with social life	-0.279	0.047	-0.125	0.230	-0.296	0.038
Unhappiness in life	-0.043	0.400	0.093	0.292	-0.094	0.291
Perceived dissatisfaction with health	0.249	0.069	0.102	0.275	0.272	0.051
Perceived low wellbeing	0.205	0.112	0.226	0.089	0.137	0.210
Self-reported poor health status	0.585	0.001	0.350	0.017	0.660	0.001
Physical activity limitation	0.590	0.001	0.418	0.005	0.656	0.001

Appendix 4.55 Bivariate Correlation Between CBVD Mortality and Psychosocial Health and Wellbeing						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Experiencing stressful life	-0.211	0.106	-0.289	0.042	-0.171	0.156
Perceived dissatisfaction with social life	-0.285	0.044	-0.197	0.121	-0.269	0.054
Unhappiness in life	0.057	0.369	-0.010	0.477	-0.039	0.409
Perceived dissatisfaction with health	0.191	0.129	0.098	0.282	0.176	0.149
Perceived low wellbeing	-0.160	0.173	-0.100	0.277	-0.174	0.152
Self-reported poor health status	0.213	0.103	0.137	0.209	0.302	0.035
Physical activity limitation	0.141	0.203	0.057	0.369	0.130	0.221
	1990-1994					
Experiencing stressful life	-0.113	0.253	0.002	0.495	-0.276	0.049
Perceived dissatisfaction with social life	-0.186	0.135	0.204	0.225	-0.294	0.039
Unhappiness in life	0.140	0.205	0.232	0.083	0.092	0.294
Perceived dissatisfaction with health	0.117	0.245	0.088	0.303	0.269	0.054
Perceived low wellbeing	-0.129	0.223	-0.066	0.349	0.026	0.440
Self-reported poor health status	0.024	0.443	-0.035	0.418	0.157	0.177
Physical activity limitation	0.060	0.361	-0.015	0.465	0.172	0.155

Appendix 4.56 Bivariate Correlation Between CBVD Morbidity and Psychosocial Health and Wellbeing						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Experiencing stressful life	-0.383	0.010	-0.355	0.015	-0.369	0.012
Perceived dissatisfaction with social life	-0.322	0.026	-0.227	0.088	-0.228	0.087
Unhappiness in life	-0.051	0.383	0.153	0.183	-0.064	0.353
Perceived dissatisfaction with health	0.136	0.211	0.020	0.453	0.262	0.059
Perceived low wellbeing	0.020	0.454	-0.050	0.385	0.091	0.297
Self-reported poor health status	0.513	0.001	0.278	0.048	0.629	0.000
Physical activity limitation	0.429	0.004	0.316	0.029	0.459	0.002
	1990-1994					
Experiencing stressful life	-0.353	0.016	-0.225	0.090	-0.393	0.008
Perceived dissatisfaction with social life	-0.432	0.004	-0.182	0.141	-0.427	0.004
Unhappiness in life	-0.087	0.305	0.165	0.165	-0.266	0.056
Perceived dissatisfaction with health	0.005	0.489	0.058	0.367	-0.005	0.487
Perceived low wellbeing	-0.055	0.374	0.016	0.463	-0.044	0.398
Self-reported poor health status	0.368	0.012	0.268	0.055	0.424	0.004
Physical activity limitation	0.324	0.025	0.189	0.131	0.372	0.012

Appendix 4.57 Bivariate Correlation Between CVD Mortality and Risk Factor Behaviours						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Current daily smoking	0.595	0.001	0.452	0.002	0.539	0.001
Physical inactivity	0.070	0.340	0.185	0.136	0.020	0.452
More than 30 per cent fat in diet	0.247	0.070	0.256	0.063	0.186	0.135
Excessive drinking	na	na	-0.267	0.110	-0.076	0.656
	1990-1994					
Current daily smoking	0.504	0.001	0.360	0.014	0.534	0.001
Physical inactivity	0.130	0.221	0.183	0.139	0.109	0.260
More than 30 per cent fat in diet	0.313	0.030	0.275	0.050	0.272	0.052
Excessive drinking	na	na	-0.267	0.110	-0.124	0.465

Appendix 4.58 Bivariate Correlation Between CVD Morbidity and Risk Factor Behaviours						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Current daily smoking	0.621	0.001	0.534	0.001	0.572	0.001
Physical inactivity	0.148	0.190	0.222	0.093	0.113	0.253
More than 30 per cent fat in diet	0.248	0.069	0.285	0.044	0.155	0.180
Excessive drinking	na	na	-0.314	0.058	-0.175	0.299
	1990-1994					
Current daily smoking	0.616	0.001	0.545	0.001	0.558	0.001
Physical inactivity	0.167	0.161	0.221	0.094	0.118	0.244
More than 30 per cent fat in diet	0.229	0.086	0.261	0.059	0.142	0.201
Excessive drinking	na	na	-0.254	0.129	-0.189	0.261

Appendix 4.59 Bivariate Correlation Between IHD Mortality and Risk Factor Behaviours						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Current daily smoking	0.429	0.004	0.214	0.101	0.466	0.002
Physical inactivity	0.080	0.319	0.157	0.177	0.067	0.347
More than 30 per cent fat in diet	0.266	0.056	0.268	0.055	0.217	0.099
Excessive drinking	na	na	-0.228	0.174	-0.166	0.327
	1990-1994					
Current daily smoking	0.400	0.007	0.250	0.068	0.467	0.002
Physical inactivity	0.189	0.132	0.215	0.101	0.151	0.186
More than 30 per cent fat in diet	0.323	0.026	0.269	0.054	0.301	0.035
Excessive drinking	na	na	-0.161	0.342	-0.173	0.305

Appendix 4.60 Bivariate Correlation Between IHD Morbidity and Risk Factor Behaviours						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Current daily smoking	0.618	0.001	0.556	0.001	0.524	0.001
Physical inactivity	0.084	0.311	0.180	0.143	0.071	0.338
More than 30 per cent fat in diet	0.197	0.121	0.203	0.114	0.148	0.191
Excessive drinking	na	na	-0.307	0.064	-0.193	0.252
	1990-1994					
Current daily smoking	0.657	0.001	0.621	0.001	0.554	0.001
Physical inactivity	0.186	0.135	0.213	0.103	0.170	0.158
More than 30 per cent fat in diet	0.247	0.071	0.267	0.055	0.169	0.159
Excessive drinking	na	na	-0.205	0.223	-0.143	0.397

Appendix 4.61 Bivariate Correlation Between CBVD Mortality and Risk Factor Behaviours						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Current daily smoking	0.299	0.036	0.164	0.332	0.243	0.074
Physical inactivity	0.120	0.239	0.229	0.086	-0.078	0.324
More than 30 per cent fat in diet	0.114	0.252	0.076	0.327	0.089	0.300
Excessive drinking	na	na	-0.033	0.845	0.213	0.207
	1990-1994					
Current daily smoking	0.216	0.100	0.086	0.613	0.235	0.080
Physical inactivity	-0.034	0.422	0.116	0.248	-0.175	0.150
More than 30 per cent fat in diet	0.026	0.439	0.082	0.314	-0.069	0.342
Excessive drinking	na	na	-0.190	0.260	0.060	0.725

Appendix 4.62 Bivariate Correlation Between CBVD Morbidity and Risk Factor Behaviours						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Current daily smoking	0.599	0.001	0.527	0.001	0.539	0.001
Physical inactivity	0.060	0.361	0.123	0.233	0.020	0.454
More than 30 per cent fat in diet	0.285	0.044	0.333	0.022	0.173	0.153
Excessive drinking	na	na	-0.302	0.069	-0.109	0.519
	1990-1994					
Current daily smoking	0.498	0.001	0.413	0.006	0.461	0.002
Physical inactivity	0.027	0.436	0.135	0.214	-0.082	0.315
More than 30 per cent fat in diet	0.152	0.185	0.172	0.155	0.069	0.343
Excessive drinking	na	na	-0.351	0.033	-0.164	0.332

Appendix 4.63 Bivariate Correlation Between CVD Mortality and Social Support						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Low social participation (ages 16-59)	0.325	0.050	0.166	0.327	0.363	0.027
Low social participation (ages 60 and over)	0.106	0.532	0.110	0.517	0.071	0.678
Dysfunctional family	0.380	0.010	0.365	0.013	0.332	0.022
Membership in voluntary organizations	0.301	0.035	0.234	0.081	0.308	0.032
No help from family and friends in times of need	-0.004	0.491	-0.041	0.406	0.132	0.218
No friend or family member to confide in	0.322	0.026	0.170	0.157	0.356	0.015
Living alone	0.556	0.001	0.439	0.003	0.622	0.001
	1990-1994					
Low social participation (ages 16-59)	0.348	0.035	0.285	0.088	0.283	0.089
Low social participation (ages 60 and over)	0.007	0.966	0.030	0.858	0.067	0.695
Dysfunctional family	0.446	0.003	0.459	0.002	0.355	0.016
Membership in voluntary organizations	0.444	0.003	0.379	0.010	0.439	0.003
No help from family and friends in times of need	0.230	0.086	-0.027	0.437	0.338	0.020
No friend or family member to confide in	0.262	0.059	0.130	0.221	0.278	0.048
Living alone	0.643	0.001	0.539	0.001	0.704	0.001

Appendix 4.64 Bivariate Correlation Between CVD Morbidity and Social Support						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Low social participation (ages 16-59)	0.283	0.090	0.307	0.064	0.173	0.305
Low social participation (ages 60 and over)	0.020	0.907	0.044	0.794	0.141	0.404
Dysfunctional family	0.306	0.033	0.413	0.006	0.141	0.203
Membership in voluntary organizations	0.389	0.009	0.407	0.006	0.325	0.025
No help from family and friends in times of need	0.186	0.135	-0.029	0.432	0.284	0.044
No friend or family member to confide in	0.251	0.067	0.134	0.215	0.181	0.141
Living alone	0.631	0.001	0.610	0.001	0.645	0.001
	1990-1994					
Low social participation (ages 16-59)	0.316	0.056	0.351	0.033	0.192	0.254
Low social participation (ages 60 and over)	-0.025	0.883	-0.007	0.966	0.109	0.520
Dysfunctional family	0.359	0.015	0.469	0.002	0.183	0.140
Membership in voluntary organizations	0.374	0.011	0.362	0.014	0.329	0.023
No help from family and friends in times of need	0.230	0.085	-0.037	0.414	0.318	0.028
No friend or family member to confide in	0.279	0.047	0.132	0.219	0.246	0.071
Living alone	0.652	0.001	0.650	0.001	0.652	0.001

Appendix 4.65 Bivariate Correlation Between IHD Mortality and Social Support						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Low social participation (ages 16-59)	0.262	0.118	0.155	0.361	0.280	0.093
Low social participation (ages 60 and over)	0.143	0.397	0.167	0.323	0.117	0.490
Dysfunctional family	0.296	0.037	0.294	0.039	0.231	0.084
Membership in voluntary organizations	0.358	0.015	0.350	0.017	0.321	0.026
No help from family and friends in times of need	0.120	0.240	0.003	0.492	0.227	0.088
No friend or family member to confide in	0.299	0.036	0.216	0.100	0.318	0.027
Living alone	0.513	0.001	0.382	0.010	0.589	0.001
	1990-1994					
Low social participation (ages 16-59)	0.302	0.069	0.251	0.134	0.278	0.095
Low social participation (ages 60 and over)	0.065	0.704	0.110	0.515	0.064	0.705
Dysfunctional family	0.395	0.008	0.388	0.009	0.333	0.022
Membership in voluntary organizations	0.432	0.004	0.363	0.014	0.441	0.003
No help from family and friends in times of need	0.248	0.070	0.026	0.439	0.310	0.031
No friend or family member to confide in	0.266	0.056	0.181	0.142	0.247	0.071
Living alone	0.555	0.001	0.477	0.001	0.611	0.001

Appendix 4.66 Bivariate Correlation Between IHD Morbidity and Social Support						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Low social participation (ages 16-59)	0.435	0.007	0.425	0.009	0.331	0.046
Low social participation (ages 60 and over)	0.189	0.262	0.186	0.271	0.175	0.299
Dysfunctional family	0.257	0.063	0.381	0.010	0.085	0.309
Membership in voluntary organizations	0.222	0.093	0.240	0.076	0.183	0.140
No help from family and friends in times of need	0.254	0.065	0.129	0.223	0.248	0.069
No friend or family member to confide in	0.293	0.039	0.222	0.093	0.204	0.113
Living alone	0.573	0.001	0.557	0.001	0.573	0.001
	1990-1994					
Low social participation (ages 16-59)	0.461	0.004	0.461	0.004	0.331	0.046
Low social participation (ages 60 and over)	0.006	0.971	0.061	0.721	0.123	0.468
Dysfunctional family	0.378	0.011	0.441	0.003	0.214	0.102
Membership in voluntary organizations	0.210	0.106	0.235	0.080	0.144	0.198
No help from family and friends in times of need	0.305	0.033	0.090	0.299	0.359	0.014
No friend or family member to confide in	0.354	0.016	0.244	0.073	0.292	0.040
Living alone	0.565	0.001	0.594	0.001	0.530	0.001

Appendix 4.67 Bivariate Correlation Between CBVD Mortality and Social Support						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Low social participation (ages 16-59)	-0.143	0.399	-0.260	0.120	0.086	0.612
Low social participation (ages 60 and over)	-0.106	0.534	-0.066	0.699	-0.157	0.355
Dysfunctional family	-0.031	0.427	-0.204	0.112	0.168	0.161
Membership in voluntary organizations	0.164	0.167	0.030	0.431	0.249	0.068
No help from family and friends in times of need	-0.343	0.019	-0.162	0.170	-0.165	0.165
No friend or family member to confide in	0.094	0.291	-0.182	0.140	0.254	0.065
Living alone	0.085	0.308	-0.028	0.435	0.190	0.130
	1990-1994					
Low social participation (ages 16-59)	-0.131	0.440	-0.107	0.527	-0.109	0.519
Low social participation (ages 60 and over)	-0.164	0.331	-0.129	0.446	0.007	0.965
Dysfunctional family	0.066	0.350	0.019	0.455	0.116	0.247
Membership in voluntary organizations	0.208	0.108	0.068	0.344	0.277	0.049
No help from family and friends in times of need	-0.163	0.167	-0.255	0.064	0.099	0.279
No friend or family member to confide in	0.102	0.275	-0.052	0.380	0.218	0.098
Living alone	0.076	0.328	-0.140	0.205	0.303	0.034

Appendix 4.68 Bivariate Correlation Between CBVD Morbidity and Social Support						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Low social participation (ages 16-59)	0.296	0.075	0.241	0.150	0.258	0.122
Low social participation (ages 60 and over)	-0.006	0.971	0.020	0.905	0.104	0.541
Dysfunctional family	0.416	0.005	0.470	0.002	0.305	0.033
Membership in voluntary organizations	0.418	0.005	0.478	0.001	0.309	0.031
No help from family and friends in times of need	0.148	0.192	-0.059	0.365	0.184	0.138
No friend or family member to confide in	0.344	0.019	0.126	0.228	0.243	0.074
Living alone	0.554	0.001	0.521	0.001	0.547	0.001
	1990-1994					
Low social participation (ages 16-59)	0.240	0.153	0.316	0.057	0.080	0.639
Low social participation (ages 60 and over)	-0.021	0.901	-0.072	0.672	0.026	0.879
Dysfunctional family	0.341	0.019	0.513	0.001	0.128	0.225
Membership in voluntary organizations	0.472	0.002	0.371	0.012	0.520	0.001
No help from family and friends in times of need	0.158	0.176	-0.097	0.284	0.176	0.149
No friend or family member to confide in	0.249	0.069	0.077	0.325	0.184	0.138
Living alone	0.618	0.001	0.559	0.001	0.659	0.001

Appendix 4.69 Bivariate Correlation Between CVD Mortality and Physiological Characteristics						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Hypertension	0.161	0.170	0.133	0.216	0.023	0.445
Diabetes	0.318	0.028	0.390	0.009	0.160	0.172
Obesity	0.440	0.003	0.377	0.011	0.252	0.066
	1990-1994					
Hypertension	0.260	0.060	0.311	0.030	0.011	0.473
Diabetes	0.361	0.014	0.367	0.013	0.185	0.136
Obesity	0.446	0.003	0.400	0.007	0.248	0.070

Appendix 4.70 Bivariate Correlation Between CVD Morbidity and Physiological Characteristics						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Hypertension	0.329	0.023	0.315	0.029	0.133	0.217
Diabetes	0.328	0.024	0.409	0.006	0.120	0.239
Obesity	0.641	0.001	0.704	0.001	0.346	0.018
	1990-1994					
Hypertension	0.376	0.011	0.381	0.010	0.144	0.198
Diabetes	0.341	0.019	0.393	0.008	0.144	0.198
Obesity	0.598	0.001	0.684	0.001	0.313	0.030

Appendix 4.71 Bivariate Correlation Between IHD Mortality and Physiological Characteristics						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Hypertension	0.134	0.214	0.123	0.234	0.053	0.379
Diabetes	0.338	0.020	0.387	0.009	0.182	0.140
Obesity	0.324	0.025	0.286	0.043	0.129	0.224
	1990-1994					
Hypertension	0.224	0.091	0.244	0.073	-0.020	0.452
Diabetes	0.390	0.009	0.391	0.008	0.232	0.084
Obesity	0.350	0.017	0.322	0.026	0.194	0.125

Appendix 4.72 Bivariate Correlation Between IHD Morbidity and Physiological Characteristics						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Hypertension	0.253	0.066	0.212	0.104	0.141	0.203
Diabetes	0.352	0.016	0.426	0.004	0.195	0.124
Obesity	0.456	0.002	0.547	0.000	0.206	0.111
	1990-1994					
Hypertension	0.372	0.012	0.346	0.018	0.122	0.236
Diabetes	0.362	0.014	0.387	0.009	0.219	0.096
Obesity	0.507	0.001	0.597	0.000	0.265	0.056

Appendix 4.73 Bivariate Correlation Between CBVD Mortality and Physiological Characteristics						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Hypertension	0.038	0.411	-0.116	0.246	-0.090	0.298
Diabetes	0.075	0.329	0.102	0.275	-0.074	0.333
Obesity	0.248	0.070	-0.040	0.407	0.386	0.009
	1990-1994					
Hypertension	0.072	0.337	0.128	0.225	0.014	0.467
Diabetes	0.086	0.306	0.064	0.353	-0.125	0.230
Obesity	0.176	0.149	0.049	0.386	0.148	0.191

Appendix 4.74 Bivariate Correlation Between CBVD Morbidity and Physiological Characteristics						
Variables	1986-1989					
	Both sexes		Females		Males	
	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value	Pearson's Corr. Coeff.	2-tailed p-value
Hypertension	0.246	0.071	0.295	0.038	0.058	0.367
Diabetes	0.273	0.051	0.378	0.010	0.023	0.445
Obesity	0.586	0.001	0.646	0.001	0.294	0.039
	1990-1994					
Hypertension	0.288	0.042	0.391	0.008	0.041	0.404
Diabetes	0.338	0.020	0.347	0.018	0.073	0.334
Obesity	0.557	0.001	0.649	0.001	0.241	0.075