The Epidemiology of Diabetes in Manitoba: An Exploration Through Time and Space

by

Chris Green, B.A., M.H.Sc.

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A Thesis/Practicum submitted to the Faculty of Graduate Studies of The University of Manitoba in partial fulfillment of the requirement of the degree

Of

DOCTOR OF PHILOSOPHY

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Abstract

Diabetes mellitus (DM) is becoming epidemic world wide, with the number of cases projected to increase from 171 million cases in the year 2000 to 366 million cases in the year 2030. The number of cases of DM in Manitoba is also rapidly increasing. In 1998, it was estimated that there were 54,926 adults living with diagnosed DM in Manitoba, a significant increase from 30,104 cases in 1986. The purpose of this study was to describe the temporal and geographic variability of DM in the province of Manitoba and to assess the degree to which this variability is associated with underlying population characteristics. This was accomplished in four separate studies. The first study focused on describing and modeling the spatial distribution of DM prevalence in the City of Winnipeg, Manitoba in 1998. The second study compared the demographic, temporal and geographic patterns of DM incidence and prevalence in the Manitoba First Nation population to the non-First Nation population from 1989 to 1998. The third study used a number of diverse spatial techniques to visualize, explore and model the incidence of DM in the province of Manitoba between 1989 and 1998. The fourth study used a component cohort projection model to back-cast and forecast the prevalence of DM in Manitoba from 1950 to 2050. All analyses were based on DM incidence and prevalence data derived from the Manitoba diabetes database. The results of these four studies suggest that the number of DM cases will continue to rapidly increase into the foreseeable future in Manitoba. They also suggest that despite an observed gradient in DM risk by age, socio-economic and First Nation status, all population groups are at significant risk for developing the disease. It is concluded that prevention programs focusing only on groups

at highest risk for developing DM will have little chance of success and that population based prevention approaches which tackle the fabric of everyday life will be required to stem the epidemic. It is also concluded that population based prevention efforts will need to be informed by further research utilizing diverse historical, anthropological and geographical research methods which can identify the range of forces affecting DM risk, and the types and scales of interventions which may be required simultaneously at individual, local, regional, national, and even international levels to deal effectively with the DM epidemic.

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Chapter 1

Introduction

Statement of the Problem:

Diabetes Mellitus (DM) is one of the most common non-communicable diseases in the world today (Amos, McCarty & Zimmet, 1997). Ninety five percent of all cases of DM are Type 2 Diabetes Mellitus (T2DM) which is characterized by insulin resistance and adult onset. Type 1 Diabetes Mellitus (T1DM) which makes up less than five percent of all DM cases results from pancreatic beta-cell destruction and has an initial onset primarily in adolescents and young adults (Harris, 1995; Meltzer, Leiter, Daneman, Gerstein, Lau, Ludwig et al. 1998).

It is projected that the number of cases of DM around the world will increase rapidly over the next 25 years from 171 million cases in 2000 to 366 million cases in 2030 (Wild, Roglic, Green, Sicree & King, 2004). In Canada in the fiscal year 1999/2000, it was estimated that there were 1.2 million adults living with diagnosed DM (Center for Chronic Disease Prevention and Control, 2003). However, the actual number of persons with DM in Canada is likely to be much higher since 30 to 50 percent of all DM cases remain undiagnosed (Harris, Flegal, Cowie, Eberhardt, Goldestein & Little, 1998; Young & Mustard, 2001). In Manitoba, in 1998, there were over 54,926 adults living with diagnosed DM and this number is expected to increase significantly over the next 25 years (Blanchard, Green & Wajda, 1998). Of urgent concern are the extremely high rates of DM observed in the Manitoba First Nation population. In 1998, rates of DM in First Nation adult males and females were 2.8 and 4.6 times higher than in the non-First Nation

population (Green, Blanchard, Young & Griffith, 2003). Also of significant concern is the recent emergence of T2DM in Manitoba First Nation children less than 15 years of age (Dean, 1998; Dean, Mundy & Moffatt, 1992; Dean, Young, Flett & Wood-Steiman, 1998; Sellers, Eisenbarth, Young & Dean, 2000; Young, Dean, Flett & Wood-Steiman, ; Young, Martens, Taback, Sellers, Dean, Cheang et al. 2002).

The complications and costs associated with DM are staggering. Compared to individuals without DM, individuals with DM are 17 times more likely to develop kidney disease, have a two to eight fold increase in cardiovascular mortality, have twice the prevalence of hypertension, a 15 fold risk of lower limb amputation, and is one of the leading causes of blindness in adults (Amos et al., 1997). In Canada in 1998 it was estimated that the total economic cost of DM and its chronic complications was likely between 7 and 7.8 billion dollars (Dawson, Gomes, Gerstein, Blanchard & Kahler, 2002). Extrapolated to the Manitoba population, the estimated cost of DM to the Manitoba economy in 1998 was 262 million dollars. The cost of DM to the Manitoba economy is expected to increase dramatically over the next 25 years as the number of persons with DM continues to rise (Blanchard et al., 1998).

A better understanding of the basic epidemiology of DM and its relationship to population characteristics is critical in order to develop program and policy responses to effectively deal with this increasingly expensive and devastating disease. Over the long-term, population level information on the incidence and prevalence of DM is necessary to effectively design and target primary prevention programs which can prevent the disease from occurring in the first place. In the short term, population level information is

required to target secondary prevention programs which minimize the impact of complications in persons who have already developed DM. Effective program and policy development also requires knowledge of the degree to which DM is concentrated in geographically defined sub-populations and whether this concentration is increasing or decreasing over time.

Until recently, geographically specific DM databases having complete population coverage were not available anywhere in the world. With the development of the Manitoba DM database (MDD) geographically specific DM data for the whole population is now available (Blanchard, Ludwig, Wajda, Dean, Anderson, Kendal et al. 1996). This study which uses the MDD to better understand the geographic and socio-demographic factors associated with the disease fills an important void in the literature. With the exception of the recently published Diabetes Practice Atlas for Ontario (Hux & Tang, 2002) and several recent Ontario studies (Booth & Hux, 2003; Shah & Hux, 2003), population based studies have been restricted to analysis of data collected through population surveys or through DM registries of small sub-populations.

Purpose of the Study:

The overall aim of this study is to describe the spatial and temporal trends in the epidemiology of DM in Manitoba and their relationship to individual and population level characteristics.

The four main objectives of the study are:

a. To describe the epidemiology of DM in Manitoba by year, age, gender, First

Nation status, and region of residence.

b. To identify the socio-demographic factors associated at the population level with variability in DM rates across Manitoba.

c. To identify the long term historical and future trends in DM prevalence in Manitoba, and the population level explanatory factors/dynamics that are consistent with these trends.

e. To describe the program, policy and future research implications of the spatial, temporal and demographic trends in DM identified in the study.

Organization of the Thesis:

The body of the thesis document consists of four papers written in publishable format. Two of these papers have been extracted from manuscripts which have already been published. The four papers are:

1. Geographic analysis of diabetes prevalence in an urban area, published in Social Science and Medicine, 2003 (Green, Hoppa, Young & Blanchard, 2003). The study models the spatial distribution of DM prevalence in 1998 in the City of Winnipeg, Manitoba, Canada using two techniques - spatial regression, and the spatial scan statistic paired with analysis of variance.

2. The epidemiology of diabetes in the Manitoba Registered First Nation population: current patterns and comparative trends, published in Diabetes Care, 2003 (Green, Blanchard, Young & Griffith, 2003). This second study compares the demographic, temporal and broad geographic patterns of DM incidence and

prevalence in the Manitoba First Nation population to the non-First Nation population from 1989 to 1998.

3. Visualization, exploration and modeling of diabetes incidence in Manitoba, 1989-1998. This third study uses several spatial and epidemiological techniques including the production of continuous surface smoothed maps, the Gini coefficient, the global and local Moran's I, the spatial scan statistic, the population attributable risk, and Poisson regression to visualize, explore and model the incidence of DM in the province of Manitoba between 1989 and 1998.

4. 100 years of diabetes: re-tracing and predicting and epidemic. The fourth study uses a component cohort projection model to back-cast and forecast the prevalence of DM in Manitoba from 1950 to 2050, and calculates the percentage of growth in DM cases which can be attributed to changes in population growth, population aging, and increased risk.

As a body of work, these four papers build a comprehensive picture of the dynamics of the emerging DM epidemic in Manitoba. The document also contains an overall theoretical framework, literature review, discussion and a common bibliography.

It is important to note that the two papers included as part of this thesis which were previously published in 2003 (see Chapters 4 and 5) are referenced throughout the rest of the thesis document This is because the remaining sections of the thesis document were written and assembled in 2004, and would be have been incomplete without reference to these contributions to the published literature on the epidemiology of DM in Manitoba.

Chapter 2

Theoretical Approach

This study adopts a population health/ecological paradigm to guide the research process and the analysis and interpretation of its results. In contrast to the traditional biomedical paradigm which tends to focus on the capacity of the health care system to repair the individual body when it dysfunctions, this approach argues that there are a multiplicity of factors outside of the health care system which have significant impacts on the health of the population (Evans & Barer, 1994; VanLeeuwen, Waltner-Toews, Abernathy & Smitt, 1999; Waltner-Toews, 2004). These include factors in the social and physical environments such as income, education, social support networks, quality of community life, housing and working conditions, and at the personal level, genetic predisposition, health practices, individual capacity and coping skills. The population health perspective suggests that the health of the population can be best maintained through strategies that address the entire range of individual and collective factors affecting the health of the population (Canadian Population Health Inititiative, 2004).

More fundamentally, the population health/ecological approach proposed here challenges the view of the body as being static and machine like (which can be easily repaired when it is broken) and suggests that the body is an emergent process open to many larger scales of influence as it continuously exchanges the materials of life with the larger world around it. It assumes that the human body is embedded within a multi-layered hierarchically organized biological and socio-cultural landscape which is in a continuous process of reconstituting itself. This suggests that the physical body (as part of this continually

reconstituting landscape) does not have physical permanence and integrity until death, but rather is in a continual process of decomposition and renewal, and is never the same from moment to moment. This dynamic and flexible view of the body and the ecology in which it is embedded, moves beyond the biomedical view of the body as a machine, and can facilitate a productive exploration of how and why biological variability (i.e. health and disease) occurs systematically across space and time. [1]

The Butterfly Model of Health (Figure 2.1) embodies the population health/ecological paradigm (VanLeeuwen et al., 1999). This model overcomes the limitations of many previous population health models which tended to focus on simple one-way linear relationships between health determinants and outcomes and which did not address the complex ways in which socio-cultural and biological dynamics interact at multiple scales of influence (Hayes, Foster & Foster, 1994; VanLeeuwen et al., 1999). In the Butterfly model, the human body, which is located at the local intersection of the biophysical and socio-cultural environments, is open to and constituted (co-created) out of the complex interaction of biology and culture. As illustrated in Figure 2.1, the local biological and socio-cultural environments (large circles), are themselves embedded within more global (external) biological and socio-cultural environments. At the human level, biological and behavioural filters in the model refer to the innate biological (genetic and otherwise) and behavioural abilities of individuals and populations to maintain health and fight disease. The bi-directional

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[1] It appears that the human body is continually and very dynamically renewing itself. Radio isotopic techniques which allow the tracing of chemicals that enter and leave the body have suggested that 98% of the 10 (28thpower) atoms of the body are replaced annually, with each body structure having its own rate of reformation. Though the rates of bodily tissue replacement differ, it is estimated that within five years the entire body is completely renewed (Davis, 1980; Dossey, 1982).

arrows between all elements emphasize that no components of the system are fixed; rather that all components are in a continuous process of change and regeneration, with all components affecting all other components to a lesser or greater degree.

The population health framework outlined here needs to be informed by a number of perspectives which acknowledge that the models or frameworks we develop to guide research necessarily simplify the world and therefore cannot be used without considerable reflection. In other words, the population health framework presented here cannot be used mechanically to inform the research process since there are many complexities and interconnections between things which cannot possibly be communicated through a simple diagram or framework. The model needs to be seen then, only as an initial starting point for thinking about the dynamics of human health, whose implications can be most usefully explored when paired with a number of critical social science perspectives. These perspectives can help one use the framework heuristically as a broad guide and for interpreting research into the connections between factors affecting population health. The implications of the Butterfly model in light of a number of critical social science perspectives are described below.

First, the Butterfly model makes explicit the importance of scale. The model assumes that the world is organized hierarchically on the human side, from genes to individuals to families to communities to regions to nations, and ultimately to the human race, and that each level in the hierarchy has its own set of dynamics which are embedded within the dynamics of the levels above and below it. This means that studies which restrict themselves to an analysis at only one level and do not explore cross-scale dynamics, may generate an incomplete

understanding of disease ecology (McMichael, 1995,1999, 2001; Sobal, 2001; Waltner-Toews, 2004). Biomedical epidemiology has tended to operate in this way, with the result that many epidemiologists use individuals as the unit of analysis, usually in one population and one place, and interpret their findings using physiological explanations. The Butterfly model challenges this approach and suggests that a comprehensive explanation of disease causation will need to explore how patterns of disease observed in individuals and local populations are connected by forces and dynamics operating at a variety of scales, both larger and smaller than the individual or the local population. This approach is consistent with the critical realist approach which rejects the atomistic idea that events and objects are independent - i.e. that society is made up of a structureless aggregate of individuals, and argues for the existence of deep structures, processes and mechanisms which tie reality (biological and social) together at different levels in very complex ways (Cloke, Philo & Sadler, 1991; Sayer, 1992).

A large body of research has emerged over the past two decades which has empirically demonstrated that this sort of cross scale analysis is valid and useful. This research has consistently shown a strong relationship between socioeconomic factors and environmental conditions operating at broad scales (community, region, nation) and health status (Berkman & Kawachi, 2000; Hertzman, Frank & Evans, 1994; Marmot, 1999; Williams, 2001), and has proposed and tested a number of specific pathways and mechanisms at the individual and physiological levels which may mediate these relationships. These include the generalized stress response, the direct and cumulative effects of material deprivation on biological functioning, early child-hood intrauterine experiences, restricted

access to the resources required to adopt and maintain healthy lifestyle habits, the direct mental and physical risks of living in socially and materially hazardous environments, the disempowering effects of racism and poverty, and the local perpetuation of cultural traditions and practices which promote and reinforce health damaging ways of life (Baum, Garofalo & Yali, 1999; Elreedy, Krieger, Ryan, Sparrow, Weiss & Hu, 1999; Hales, Desai & Ozanne, 1997; Krieger, 2000, 2001b, 2001c; Raphael, 2001; Williams, 2001).

Failure to appreciate the importance of cross scale dynamics in disease studies can result in a reification of socially constructed categories such as race and gender. Often these categories come to be portrayed as important causes of chronic diseases such as diabetes, heart disease and hypertension (King, 1997). Expanding the scale of analysis provides an opportunity to explore the ways in which racial and ethnic categories may have more to do with deeper social processes and connections (that need to be explained) than with the essential or genetic characteristics of sub-populations. In order to address this issue of scale, Krieger (2001a) and Krieger & Davey (2004) have proposed the concept of "embodiment", the way in which social forces and influences become embedded in the physical characteristics of populations, resulting in what is referred to as "racialized biology". Racialized biology suggests that the biology of racially defined groups varies primarily because of the life situation in which these groups find themselves, the dynamics of which can only be fully appreciated by examining forces acting at spatial and temporal scales greater than the individual. These include the social history of vulnerable groups, their insertion into the social and economic systems, their cultural responses and practices, and their material conditions of life. Hertzman (1999) uses the term "biological embedding" to express a

similar concept, arguing that the characteristics of a person's biology can only be comprehensively understood through an examination of the broad material and psychosocial conditions they have encountered on an everyday basis through the course of their life.

Secondly, the Butterfly model with its emphasis on the importance of scale, stresses the need to consider the impact of globalization on the health of local populations. The globalization perspective suggests that global forces of the late 20th century / early 21st century are leading to a homogenization of ways of life around the world (urbanization, fast food, sedentary life style etc.), and this is leading to a rapid proliferation of chronic diseases (WHO, 2000). Although difficult to define, Sobal, (2001) and Ritzer (2003) suggest that globalization is the process by which previously local, national and regional phenomena are now becoming integrated and unified into larger global systems. The new units of analysis, these authors suggest, are global governments, global corporations, global media, and global food systems which are leading to global patterns of disease. The implication of the globalization perspective is that in order to comprehensively understand the determinants of ill-health and disease of local populations, epidemiological research will need to move beyond biology and individual behaviours to an examination of global forces and trends operating at large temporal and spatial scales. Goodman and Leatherman (2001) , Farmer (1999), and McMichael (2001) argue, for example, that it is vital that biocultural anthropology concern itself with how global systems and history interact with local systems and history in creating the contexts for understanding the actions of people, and the biological outcomes of those actions.

Thirdly, the Butterfly model challenges researchers to speculate about the deeper

social and biological forces that create and activate its components. This raises questions about how the contemporary configurations of the socio-economic and biophysical environments came into being in the first place and what sustains them over time and space (Hayes et al., 1994; Mitchell, 1993,1994,2000). A political economy or Marxist perspective, for example, might focus on the relationship of global capitalism to local economies and material environments and the impact that these have on the well-being of individuals and communities.

Fourthly, the Butterfly model makes explicit the need to move beyond the concept of space as a geometric container to place as a human construction. As illustrated in Figure 2.1 by the bi-directional arrows between the biophysical and socio-cultural environments, with humans at the intersection of these two domains, the model reinforces the idea that local places and their populations gain their characteristics through the complex and dialectical interaction of biological and cultural forces. Massey (1993) suggests that critical perspectives on place need to be informed by an appreciation of the ways in which local and global forces come together to shape the unique characteristics of places. Gesler & Kearns (2002) and Kearns & Gesler (1998) go even further and stress the importance of people's perceptions and experiences in shaping the health related characteristics of local places.

Fifthly, two insights from Rose on the epidemiology of population health need to be integrated into the use of the population health framework (Rose 1985, 1992) First, Rose argues that since the determinants of health (or ill-health) are distributed on a continuous gradient throughout society as a whole, the majority of disease cases often occur in low-risk groups. Although disease rates are significantly higher in high risk groups, the small

population size of the high risk groups means that these groups contribute very few cases to the overall population incidence of the disease. The implication for prevention is that programs focusing on high risk groups, even if they were 100% successful, would do little to diminish the overall population rate of the disease.

Rose also argues that population based studies which focus on homogenous populations often cannot demonstrate population level influences on health and tend to privilege individual level explanations for disease variability. This is because the homogeneity of exposure within the study population provides an insufficient range of predictor values to account for biological outcomes. It is only when one compares the same population across time, or very different populations across space do relationships between predictors and outcomes become visible at the population level.

Taken together, Rose's perspectives suggest that in order to ensure the availability of sufficient variability in exposure and outcome variables necessary to identify important relationships at the population level, population health research designs should attempt to compare the same population across long time frames, or should include identifiable and diverse geographically and culturally based groups. Rose's perspective also bring into the question the practice of using population based research results to focus policy and program responses on identified high risk groups since these groups may contribute little to the overall prevalence of the health issue.

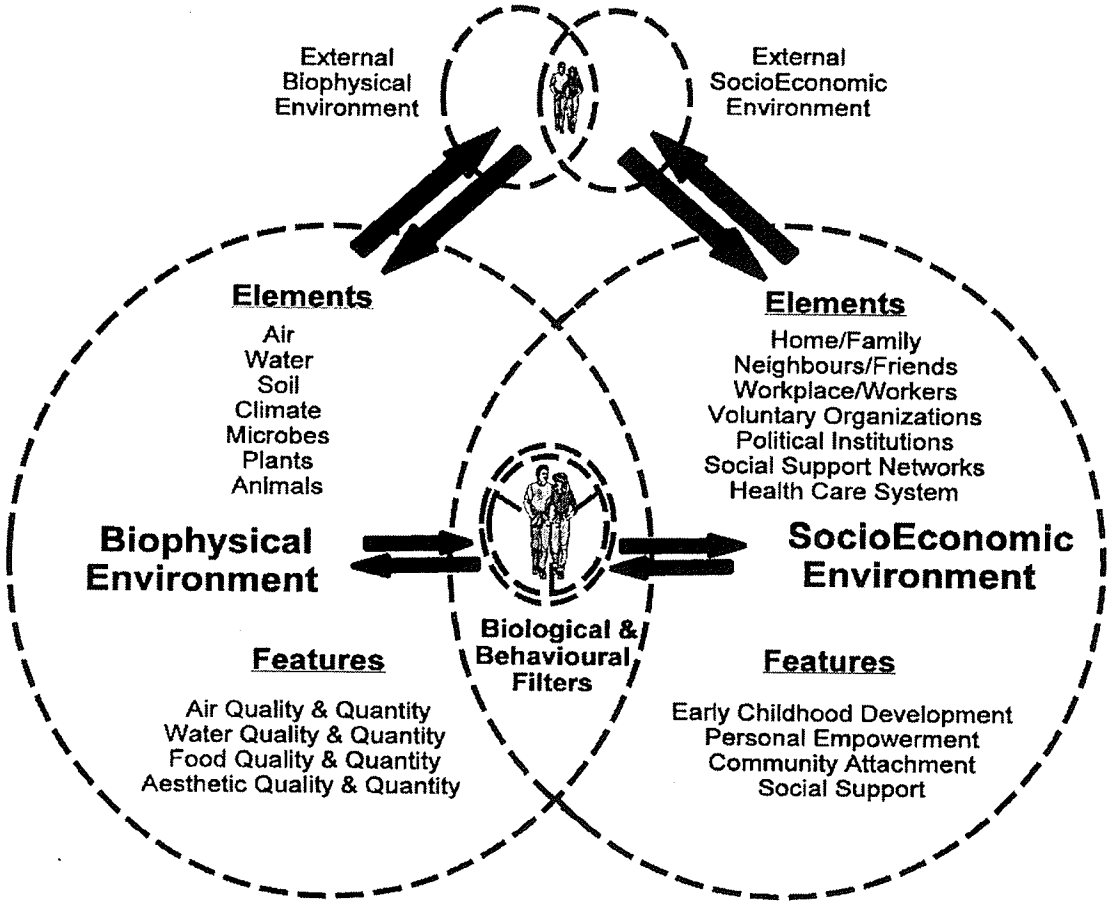
Sixthly, it is important to acknowledge that the construction of scientific knowledge guided by the population health framework proposed here is a social process and hence its production and deployment is connected to larger social processes, the reproduction of

culture, and the maintenance of social power relationships (Lupton, 1994, 1996, 1997; Rawlinson, 1987). This means that the scientific "representations" created through this research process (maps, statistical tables and outputs), and their interpretations cannot be considered objective and true in the traditional scientific sense (Harley, 2001). Rather, it should be expected that the results of this research study may be legitimately contested or challenged by others in society who are situated differently and as a result have different knowledge bases, perspectives and interests. This is not to suggest that the scientific knowledge produced using the population health framework is completely socially constructed and arbitrary; rather that the results of studies undertaken using the framework, and the conclusions drawn, are a function of both the underlying "reality" one is studying, and of the methods and theoretical conceptions used to investigate that reality (Sayer, 1992).

Finally, the Butterfly framework suggests that health research needs to be predicated upon an understanding of the complexity of the social and biological landscape in which human populations are embedded. In complex systems, there is a dense web of causal connections among the components (Gunderson & Holling, 2002; Holling, 1994, 2001). Components have so many links to each other that they affect each other in many ways; a change in one component affects others in a way that eventually loops back to affect the original component. This suggests that one should not expect to find easily modeled linear, one-way and universal relationships between framework components. Rather, non-linear relationships, often contingent upon local conditions, and often exhibiting significant threshold effects should be expected. A variety of approaches including ethnographic, historical, experimental modeling, and scenario building methods may need to be employed to

come to a more complete appreciation of the complex dynamics at play between framework components.

Figure 2.1:
Butterfly Model



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Chapter 3

Literature Review

Definition of DM:

Diabetes Mellitus (DM) refers to a group of heterogeneous metabolic disorders with the common elements of hyperglycaemia and glucose intolerance, due to insulin deficiency, impaired effectiveness of insulin action, or both (Harris, 1995). The most common characteristic feature of DM is elevated levels of blood glucose. It is important to note that prior to 1979 there were no universally accepted criteria for DM which could be used for diagnosis and surveillance of the disease and which could differentiate between the two major categories of DM. By 1979 it had become recognized that DM actually consisted to two very different diseases with distinct etiologies and treatments. The standard criteria adopted in 1979 differentiated the two major types of DM on the basis of the distinct treatments each disease required. The two classifications were insulin-dependent diabetes mellitus (IDDM) and non-insulin dependent diabetes (NIDDM) (National Diabetes Data Group, 1979).

In 1995, an international expert committee working under the auspices of the American Diabetes Association reviewed the 1979 classification and diagnosis criteria for DM. This review culminated in a new classification and set of diagnostic criteria for DM which were adopted by the Canadian Diabetes Association in 1998 and subsequently by Manitoba Health (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 1997). This new classification, which eliminated the terms IDDM and NIDDM,

defined the two major types of DM based upon pathogenesis rather than treatment since persons with any form of diabetes may require insulin treatment as some stage of their disease. These new diagnostic criteria also lowered the level of fasting venous plasma glucose at which DM is defined. The contemporary definitions of the two major forms of DM are described below.

Type 1 Diabetes Mellitus (T1DM) encompasses diabetes that is primarily a result of pancreatic beta-cell destruction and that is prone to ketoacidosis (Meltzer, Leiter, Daneman, Gerstein, Lau, Ludwig et al. 1998). This type DM results in the loss of insulin production and the inability of the body to metabolize carbohydrates properly. Initial onset of T1DM is primarily in youth less than 35 years of age, but can occur at any age. Individuals with T1DDM have low or absent levels of circulating endogenous insulin and are therefore dependent on injected insulin to prevent ketosis and sustain life. T1DM is hypothesized to be caused by a cellular mediated autoimmune response which destroys the pancreatic islet beta cells. T1DM constitutes approximately 5% of all cases of DM.

Type 2 Diabetes Mellitus (T2DM) is characterized by insulin resistance and relative insulin deficiency rather than complete lack of insulin production as is the case with T1DM (Meltzer et al., 1998). The diagnosis of T2DM usually occurs after the age of 40 years of age, although it is becoming increasing common in young adults and even adolescents. T2DM can be asymptomatic for many years and diagnosis is often made from associated complications or through an abnormal blood or urine glucose test. T2DM constitutes around 95% of all DM cases. The diagnostic criteria for TD2M is a fasting venous plasma glucose level (FPG) equal to or greater than 7 mmol/L or a 2 hour 75 gram

oral glucose level (2 hr OGTT) greater than or equal to 11.1 mmol/L.

There are also three types of pre-T2DM. Impaired fasting glucose (IFG) is defined as an FPG between 6.1 and 6.9; impaired glucose tolerance (IGT) is defined as an FPG with a 2 hour OGTT between 7.8 and 11.0; gestational diabetes in pregnant women is defined as a 2 hour OGTT > 8.9 mmol/L. Although these types of pre-T2DM do not have the diabetes associated risk for microvascular disease that persons with T2DM have, these individuals have a higher risk for the development of T2DM and cardiovascular disease than the general population.

In 2003, the 1998 Canadian Practice Guidelines for diabetes were updated by the Canadian Diabetes Association but this did not result in a change in the diagnostic criteria for any of the forms of diabetes (Clinical Practice Guidelines Expert Committee, 2003).

Most administrative databases cannot differentiate between T1DM and T2DM. The ICD9 Code 250 for DM does not have any sub-classifications for the different types of DM. This means that research and surveillance databases for DM developed using administrative databases cannot easily differentiate between T1DM and T2DM. Previous conventions which used the age 30 as a cut-point between T2DM and T1DM have come into question with the increasing prevalence of early onset T2DM (Fagot-Compagna, Narayan & Imperatore, 2001).

Historical and Projected Trends in DM:

DM in the General American and Canadian Populations:

Long-term historical rates of DM in Canada are not available since this data was

never collected systematically in disease registries or in health surveys. Data from the American National Health Interview Surveys, however, does provide information on self-reported levels of DM between 1935 and 1993 in the general U.S. population (Kenny, Aubert & Geiss, 1995). This data shows that there has been a steady increase in both the number of people with DM in the U.S. and the prevalence rate over a 60 year period The 1993 average DM prevalence rate of 2.97% was more than three times the rate in 1960 (.91%) and eight times the rate in 1935 (.37%). These comparative rates need to be treated with some caution since the increase in prevalence may have been due in part to the aging of the U.S. population, a reduction in the mortality of persons with diabetes since the early 1970's, and changes in the sensitivity of the criteria used to diagnosis DM. However, the magnitude of the prevalence rate changes revealed through an examination of age specific rates powerfully show that these increases in DM prevalence are likely real. For example, in the 45 years and younger age group, the prevalence of DM increased almost nine-fold, from 1/1000 to 8.6/1000 between 1935 and 1993. In the 65 - 74 year age group the prevalence of DM increased seven fold, from 14.6/1000 in 1935 to 106/1000 in 1993.

In Canada, population level DM estimates derived by applying a nationally agreed upon algorithm to routinely collected hospital and physician utilization data suggest that over 5.1% of Canadian adults (1.2 million) were living with diagnosed DM in the fiscal year 1999/2000 (Center for Chronic Disease Prevention and Control, 2003). However, the actual number of persons with DM in Canada in that year was likely much higher since 30 to 50 percent of all DM cases remain undiagnosed (Harris, Flegal, Cowie, Eberhardt,

Goldestein & Little, 1998; Young & Mustard, 2001)

In Manitoba in 1998, DM prevalence estimates derived from hospital and physician utilization data suggest that the prevalence of DM in the general non-First Nation Manitoba population has been increasing steadily since the late 1980's (Green, Blanchard, Young & Griffith, 2003a). Between 1989 and 1998 the age standardized prevalence of DM in adult non-First Nation men increased 1.4 fold from 41.9/1000 to 59.63/1000; in non-First Nation women prevalence rates also increased 1.4 fold, from 37.1/1000 to 53.5/1000. In 1998, it was estimated that there were 49,513 DM cases in the non-First Nation Manitoba population.

DM in First Nation Populations:

DM was virtually unheard of in North American First Nation populations prior to the 1940's (West, 1974, 1978). Medical surveys prior to the 1950s revealed that First Nation populations were suffering primarily from the combined effects of hunger, famine and epidemic infectious diseases such as tuberculosis (Moore, Kruse & Tisdall, 1946; Vivian, McMillan, Moore, Robertson, Sebrell, Tisdall et al. 1948; Young, 1993). However, by the 1960's and 70's it became apparent that DM was becoming a serious problem in First Nation populations. The most intensively studied First Nation population in the U.S. has been the Pima. Knowler, Bennett, Hamman & Miller (1978) observed age and sex adjusted incidence and prevalence rates of DM 13 and 19 times higher than the predominantly white population of Rochester Minnesota

In 1990 Young, Szathmary, Evers & Wheatley (1990) demonstrated not only that DM was becoming epidemic in Canadian First Nation populations, but that there was a

high level of variability in DM between geographically and culturally defined First Nation groups. This study, based on case registry data maintained by Health Canada's First Nation and Inuit Health Branch, showed that the age-sex adjusted rate of DM varied among Canadian First Nations from a low of .8 percent in the N.W. Territories to a high of 8.7 percent in the Atlantic region. Among the Inuit, the prevalence was 0.4 percent. In comparison to the general Canadian population, only in British Columbia, the Yukon, and the Northwest Territories were DM prevalence rates lower in First Nations. In other regions of Canada, the DM prevalence rates in First Nations were 2 to 5 times higher than in all other Canadians. This same study, which studied ecological correlates of DM prevalence, demonstrated that the best predictors of NIDDM in First Nations were latitude and the six composite language phylum areas. The authors concluded that both environmental and genetic mechanisms were involved in DM. Increasing latitude was used as proxy for the influence of acculturation while the language phylum culture area served as an index of genetic relationship.

More recent studies have demonstrated that the prevalence of DM in Canadian First Nations is high and continuing to increase rapidly. Harris, Gittelsohn, Hanley, Barnie, Wolever, Gao et al. (1997) reported age standardized T2DM prevalence rates of 28% and 24.2% in 10 years and older females and males respectively in the First Nation community of Sandy Lake, Ontario. These rates are among the highest reported rates in the world. In a Manitoba study, age standardized DM prevalence rates in adult First Nations were observed to be 2.8 times (males) and 4.6 times (females) higher than the general Manitoba population (Green et al., 2003a). Between 1989 and 1998 estimated

DM prevalence in First Nations males increased from 104.2/1000 in 1989 to 170/1000 in 1998; among First Nation females, DM prevalence rates were observed to increase from 181.6/1000 to 248.7/1000 in 1998.

Increasingly T2DM is being observed in First Nation children and adolescents (Dean, 1998; Dean, Mundy & Moffatt, 1992; Harris, Perkins & Whalen-Brough, 1996). In the Island Lake region of northeastern Manitoba, new cases in children as young as 8 years of age have been identified through screening of fasting plasma glucose levels (Dean, Young, Flett & Wood-Steiman, 1998). The prevalence of T2DM among female adolescents aged 10- 19 years of age was reported to be 3.6%.

DM in Black, Mexican American and Southeast Asian Populations:

DM has also been observed to be elevated in Black and Mexican American populations in the U.S. and in the South East Asian population in Canada. The prevalence of DM in Blacks has been observed to be 1.4 times higher than in whites and 2 to 3 times higher in Mexican Americans than in non-Hispanic whites (Carter, Pugh & Monterrosa, 1996; Stern & Mitchell, 1995; Tull & Roseman, 1995). In Ontario, DM prevalence was observed to be 2.7 times higher in the South East Asian population than in the general population (Manuel & Schultz, 2002).

Projected Trends in DM:

Using a simple age standardization approach which modeled the impact of the aging population on DM prevalence, King, Aubert & Herman (1998) estimated that the number of DM cases around the world would increase rapidly over the next 25 years, from 154 million estimated cases in 2000 to 300 million cases in 2025. In the same study,

King and colleagues also estimated that the number of cases of DM in Canada would increase from 1,7 million cases, in 2000 to 2.6 million cases in 2025. World diabetes estimates and projections were recently updated by Wild, Roglic, Green, Sicree & King (2004) who estimated that the total number of persons with diabetes would rise from 171 million in 2000 to 366 million in 2030.

Green, Blanchard, Wajda, Depew, Cooke, Brazeau et al. (1997) used a component cohort projection methodology to forecast DM prevalence in the 25 years and older Manitoba Registered First Nation population from 1996 to 2016. The model assumed that the age specific DM incidence and diabetes related mortality rates would remain constant at 1995 levels throughout the projection period. This study predicted that the number of cases of DM in the 25 years and older Registered First Nation population would triple, from 6722 cases in 1996 to 19,740 cases by the year 2016.

Blanchard, Green & Wajda (1998), also using a component cohort projection methodology projected DM prevalence from 1995 to 2025 for the total Manitoba population. The model assumed that the age specific DM incidence and diabetes related mortality rates would remain constant at 1995 levels throughout the projection period. This study predicted that the number of cases of DM in the total Manitoba population would increase from 45,948 cases in 1995 to 104,239 cases in 2025, a 2.2 fold increase.

b. Complications and Costs of DM:

Complications of DM:

Complications associated with DM are very debilitating and life-threatening.

Compared to individuals without DM, individuals with DM are 17 times more likely to develop kidney disease, have a two to eight fold increased risk in cardiovascular mortality, have twice the prevalence of hypertension, and have a 15 fold risk of lower limb amputation. DM is one of the leading causes of blindness in adults (Amos, McCarty & Zimmet, 1997).

According to the recently released ICES Practice Atlas: Diabetes in Ontario, people with DM make up only 6% of Ontario's population while accounting for 32% of heart attacks, 43% of heart failure cases, 30% of strokes, 51% of new dialysis patients and 70% of amputations (Hux, Booth & Lepaucis, 2002). Also in Ontario, Shah & Hux (2003) demonstrated that the risk of infectious disease related hospitalization was 2.17 times higher in persons with DM as compared to persons without DM, while the risk ratio for death attributable to infection was 1.92.

In the overall Manitoba population, it was estimated that in 1991 approximately 25% of hospitalizations due to heart disease and stroke were among persons with DM and that by 1993 over 40% of persons initiating dialysis had DM. It was also estimated that in 1991 lower limb amputations were 10 times higher in persons with DM than in those without DM. In the Manitoba First Nation population, persons with DM were estimated to account for 91% of lower limb amputations, 60% of heart disease hospitalizations, 50% of hospitalizations due to stroke, 41% of all hospital days, and 30% of hospitalizations (Diabetes and Chronic Diseases Unit, 1998).

Costs of DM:

In Canada in 1998 it was estimated that the total economic cost of DM and its

chronic complications was likely to be between 7 and 7.8 billion dollars (Dawson, Gomes, Gerstein, Blanchard & Kahler, 2002). These cost estimates included direct medical costs such hospital services, physician services, and medicines consumed by people with diabetes, as well as mortality related productivity losses. Of the costs associated with the complications of DM, cardiovascular disease was the major contributor to the direct costs of DM. Extrapolated to the Manitoba population, the estimated cost of DM to the Manitoba economy in 1998 was 262 million dollars. The cost of DM to the Manitoba economy is expected to increase dramatically over the next 25 years as the number of persons with DM continues to rise (Blanchard et al., 1998).

Per capita health expenditures associated with DM were also calculated for Manitoba for the years 1995/1996. After standardizing for age, the annual per capita cost for hospital, personal care home, physician and dialysis services were estimated to be twice as high for adults with DM as compared to the non-DM general population ($2169 vs. $1011). In the First Nation population, per capita costs among adults with DM were estimated to be three times higher than in adults without DM (Diabetes and Chronic Diseases Unit, 1998).

D. Etiology of DM:

Genes vs. Environment Debate:

There is great debate about the cause of the DM epidemic (Swinburn, 1996). Although there is general consensus that DM has both genetic and social roots, there is little consensus on the relative contributions of these factors (Carter et al., 1996; Fujimoto,

1996; Haffner, 1998; Hales & Barker, 1992; Hales, Desai & Ozanne, 1997; McDermott, 1998; Ozanne & Hales, 1998). Up until recently, genetic explanations have dominated the discussion as to what causes DM. This was likely due to the observation in the 1960's and 1970's of extremely high rates of DM in minority First Nation, Hispanic and Black populations in North America and in South Pacific Polynesian populations. However, with the recent explosion of DM in non-minority populations over the past 10 years, explanations for the causes of DM are now being expanded beyond the genetic paradigm. Aging of the population, obesity, nutrition and physical activity, and the effects of the intrauterine environment are now being explored as factors which may be involved in the etiology of DM.

Genetic Explanation for T2DM:

In 1962, James Neel postulated the existence of the "thrifty gene" in order to explain the paradox of high rates of DM in Native North American Aboriginal people (Neel, 1962). This evolutionary model suggested that in the early years of life the diabetic genotype was exceptionally thrifty in the utilization of carbohydrates, rapidly converting excess glucose to stored fat. It would therefore confer a survival advantage in times of food shortages, as would commonly occur in the case of hunter-gatherer and pre-industrial agricultural societies. However, in the modern environment in which high fat energy dense foods are abundantly available, the thrifty gene no longer confers a survival advantage, but renders it owners more susceptible to obesity and diabetes. Twenty years after Neel originally proposed the existence of the thrifty gene he confined his genetic explanation for DM to T2DM as a result of the new classification scheme which now

delineated between T1DM and T2DM (McDermott, 1998). The thrifty gene has been used to explain the high prevalence of T2DM in the non-European Aboriginal, Hispanic and Black populations of North America who are assumed to all have recent hunter and gatherer roots.

Since the publication of Neel's thrifty gene hypothesis there has been a concerted research effort to identify the specific genes responsible for T2DM (Swinburn, 1996). Most of this effort has been focused on finding evidence for the "thrifty" gene in Hispanic and Aboriginal populations in North America. A significant number of studies have successfully demonstrated a relationship between genetic variability in these populations and the prevalence of T2DM and its immediate risk factors, impaired glucose tolerance, obesity and hyperinsulinemia (Bogardus, Lillioja, Nyomba, Freymond, Zurlo, Swinburn et al. 1988; Bogardus, Lillioja, Nyomba, Freymond, Zurlo, Swinburn et al. 1989; Brosseau, 1993; Comuzzie, Hixson, Almasy, Mitchell, Mahaney, Dyer et al. 1997; Hanis, Chakraborty, Ferrell & Schull, 1986; Hegele, Cao, Harris, Hanley & Zinman, 1999c; Stern, Duggirala, Mitchell, Reinhart, Shivakumar, Shipman et al. 1996; Stern, Mitchell, Blangero, Reinhart, Krammerer, Harrison et al. 1996; Williams, Long, Hanson, Sievers & Knowler, 2000). These studies have used a variety of study methodologies including segregation and pedigree analysis, linkage analysis, admixture analysis, analysis of population biological variability, twin concordance studies, and population epidemiology.

Moving Beyond the Genetic Paradigm for T2DM:

However, the significance and strength of the genetic evidence has been challenged on several fronts. These challenges, which are summarized below, open up consideration

to a variety of non-genetic pathways and influences which may be involved in the etiology of DM.

First, a number of assumptions underlying the thrifty gene hypothesis, as it specifically applies to First Nation populations in North America, have been brought into question. The thrifty gene hypothesis assumes that the linguistically and culturally distinct indigenous populations across North and South America today are all descendants of a single population that existed in a uniform high carbohydrate nutritional environment in the pre-European contact era. The concept of a single ancestral population is built upon an anthropological hypothesis that the peopling of America happened during one single migration. There is, however, increasing evidence for multiple points of entry into the Americas by genetically diverse populations who remained isolated from each other for periods of up to 3,000 years, ample time for genetic differences to accumulate between them (Dewar, 2001; Greenberg, Turner & Zegura, 1986; Gruhn, 1988; Meltzer, 2004; Szathmary, 1984). The assumption of a high carbohydrate nutritional environment has also been brought into question. Szathmary (1990) suggests that periodic carbohydrate overload could not have happened in the arctic environment of the proposed route of entry to the Americas. The pre-contact diet of Eskimos and sub-arctic Indians suggests that the diet would been chronically low in carbohydrates and high in fat and protein.

Secondly, unlike the situation of well-established balanced population polymorphisms such as sickle cell anemia and single gene defects such as cystic fibrosis, no single gene has been found to consistently account for T2DM or its precursors, insulin resistance and obesity (McDermott, 1998; Neel, 1999; Swinburn, 1996). The genetic

basis for mature onset diabetes in youth (MODY) has been well established, but this type of T2DM represents only a small proportion (less than 5%) of all T2DM cases (Neel, 1999). In a study of Aboriginal populations in Canada, Hegele, Cao, Harris, Hanley & Zinman (1999a) and Hegele, Cao, Harris, Hanley & Zinman (1999) speculate that the genes conferring susceptibility to some forms of T2DM in these populations may be private to those populations. The failure to find one specific gene which is strongly and consistently associated with T2DM after 40 years of intensive research suggests the genetic basis of T2DM is likely very weak and very hetereogenous - that there are diverse biological pathways to the disease which operate differentially and unpredictability in different contexts.

Thirdly, uncontrolled sociodemographic and lifestyle confounding bring into question the results of many of the studies which have examined the relationship between genetic variability and T2DM and its precursors. The majority of these studies have confined their investigations into the more individually proximate predictors of T2DM such as genetic variability, body mass index, and insulin response and have not controlled for the effects of socio-economic status or lifestyle in any rigorous way. There is however, increasing evidence that the prevalence of T2DM is powerfully graded by socio-demographic and lifestyle factors at both the population and individual levels. Green, Hoppa, Young & Blanchard (2003b) observed that at the population level the prevalence of DM in the City of Winnipeg was inversely and powerfully correlated with a number of sociodemographic factors including income and education levels. In Ontario, the prevalence of T2DM was observed to be 40% higher in individuals in the lowest income

quartile as compared to individuals in the highest quartile, and 30% higher in individuals with less than a high school education as compared to individuals with a college or university education (Manuel & Schultz, 2002). In two recent British studies, T2DM was observed to be inversely correlated with measures of socio-economic status, while T1DM was not observed to be correlated with socio-economic status (Connolly, Unwin, Sherriff, Bilous & Kelly, 2000; Evans, Newton, Ruta, MacDonald & Morris, 2000). Hegele (1999a, 1999b), in comparing the prevalence of T2DM in the Sandy Lake Oji-Cree and the Keewatin Inuit concluded that candidate genes had only small effects upon the complex traits associated with T2DM. The effect of genetic variability, he concluded, were overshadowed by the much larger contributions to variation from non-genetic factors such as age, gender, obesity and most importantly, adherence to traditional diet and lifestyle. The Sandy Lake Oji-Cree have a T2DM prevalence rate 5 times that of the general Canadian population, while the Keewatin Inuit, who have a more traditional and active lifestyle, have rates of T2DM one third that of the Canadian population. A number of other studies have also demonstrated an inverse correlation between socioeconomic status and T2DM (Auslander W F, Haire-Joshu, Houston & Fisher E B, 1992; Gardner, Jr., Stern, Haffner, Gaskill, Hazuda, Relethford et al. 1984; Hazuda & Monterrosa, 1992; Hendricks & Haas, 1991; Leonetti, Tsunehara, Wahl & Fujimoto, 1992; Marshall, Hamman, Baxter, Mayer-Davis, Fulton & Orleans, 1993).

Fourthly, most genetic studies have focused on detecting the impact of genetic variability on T2DM between individuals within small homogenous and high risk Aboriginal and Hispanic populations. As pointed out by Rose (1985, 1992), this type of

study design which focuses on individual variation within one homogenous sub-population has a low probability of detecting broader lifestyle and population level influences on health outcomes and tends to privilege individual level explanations (i.e. genetic variability) for disease variabilty. It is only when one compares diverse populations possessing an expanded range of lifestyle and socio-demographic characteristics does the impact of broader lifestyle and population characteristics on health become visible.

Focusing exclusively on high risk populations who may have already exceeded their adaptive capacity to maintain healthy energy metabolism may also amplify the observed relationship between genetic variability and T2DM. Genetic variability may be more tightly coupled to T2DM in these populations than in low prevalence / low risk populations because the genes which would normally be phenotypically silent in low risk populations have exceeded their adaptive capacity and are now conferring variability in T2DM prevalence (Weiss, 1993).

Fifthly, an emerging literature is now suggesting the perturbations in the intrauterine environment may produce familial and sibling similarities in glucose intolerance which may mimic a genetic predisposition to T2DM (Hales & Barker, 1992; Hales et al., 1997). This idea, sometimes referred to as the thrifty phenotype hypothesis, is loosely based on Norbert Freinkel's hypothesis of fuel-mediated teratogensis and suggests that a fetus exposed to hyperglycemia or other types of inadequate nutrition *in utero* has long term anthropometric and metabolic effects, including increased susceptibility to obesity and T2DM (Freinkel, 1980). It is also proposed that undernutrition during critical periods of organ development may cause impairment of

insulin action and/or insulin secretion which if challenged later in life by factors such as obesity will result in T2DM (Ravelli, van der Meulen, Michels, Osmond, Barker, Hales et al. 1998).

The large cohort studies in the Pima Indians tend to support this hypothesis. Pettit and colleagues (Pettitt, Nelson, Saad, Bennett & Knowler, 1993) reported that the 19-24 year old offspring of Pima Indian women with T2DM were more obese and had a much higher prevalence of diabetes (50%) than offspring of non-diabetic women (1.4%) or of women who developed diabetes after pregnancy (8.6%). These effects persisted after controlling for age, father's diabetic status and age of onset of the mothers' T2DM. These observations not only support Freinkel's theory, but further suggest that the very early age of onset of T2DM in the female offspring of these diabetic mothers sets up a vicious cycle where this generation was exposing the next to a diabetogenic intrauterine environment, thus amplifying the risk through the generations. Other studies within the Pima have demonstrated that both high and low birthweight are strongly predictive of T2DM in this population (McCance, Pettit, Hanson, Jacobsson, Knowler & Bennett, 1994; McCance, Pettitt, Hanson, Jacobsson, Knowler & Bennett, 1993)

A number of other studies also support the idea that early life events can affect lifelong susceptibility to T2DM. A recent study by Lawlor, Ebrahim & Davey (2002) which used a cross-sectional study design, to assess the association between childhood and adulthood social class and insulin resistance, found that adverse social circumstances in childhood, as well as adulthood, were strongly and independently associated with increased risk of insulin resistance and other metabolic risk factors. Young, Martens,

Taback, Sellers, Dean, Cheang et al. (2002) used a case control design to examine the relationship between prenatal and early infancy risk factors and the development of T2DM among native Canadian children. Multiple logistic regression modeling identified preexisting maternal DM, gestational diabetes, and failing to breastfeed for longer than 12 months as significant predictors of diabetic status. Other factors such as low and high birthweight were also associated with T2DM but were not statistically significant. Finally, studies by Minuk, Meyers, Legare, Sadri & Lautt (1998) and Lautt (1999) have demonstrated a relationship in rat models between fetal exposure to alcohol and adult insulin resistance.

Finally, the emerging observation that the prevalence of T2DM and its precursors (obesity) are rapidly increasingly around the world, and that most of the new cases of T2DM in western countries such as Canada and U.S. are occurring (and are projected to occur) in non-Aboriginal populations brings into question the strength and significance of genetic explanations for the disease (Amos et al., 1997; Blanchard et al., 1998; Gray, Robbins, Wang, Yeh, Fabsitz, Cowan et al. 1997; King et al., 1998; Mokdad, Ford, Bowman, Nelson, Engelgau, Vinicor et al. 2000) Clearly, the dramatic increase in the prevalence of DM and its strongest predictor, obesity over the past 20 years cannot be attributed to a change in the genetic make up of the population (Cruickshank, Mbanya, Wilks, Balkau, McFarlane-Anderson & Forrester, 2001; Mokdad et al., 2000; Smith & Ebrahim, 2001) The rapid increase in the disease in such a short time frame suggests that the primary driving influences on the disease do not lie within the genome, but at scales of influence much higher than the genome (Chopra, Galbraith & Darnton-Hill, 2002; Sobal,

2001; Zimmet, 2000; Zimmet, Alberti & Shaw, 2001; Zimmet, Shaw & Alberti, 2003). The two major factors driving the rapid increase in T2DM appear to be increasing population obesity levels and the aging of population.

Obesity is the single strongest predictive risk factor for the development of T2DM. In the Nurse's Health Study (Carey, Walters, Colditz, Solomon, Willett, Rosner et al. 1997) and the Health Professionals Follow-up Study (Chan, Rimm, Colditz, Stampfer & Willett, 1994) it was found that compared with the lowest BMI category, risks for developing T2DM were increased more than tenfold among women with BMIs higher than 29 and among men with BMIs greater than 31. These studies also demonstrated that being moderately overweight was closely related to the onset of T2DM. In Canada, an analysis of the 1996-97 National Population Health Survey found that Canadians with a BMI greater than 30 were four times as likely to have DM than Canadians with a BMI less than or equal to 30 (Gilmore, 1999). In Ontario, overweight individuals (BMI 27-29.9) and obese individuals (BMI 30 or greater) had T2DM prevalence ratios of 1.7 and 2.8 respectively (Manuel & Schultz, 2002).

The rates of obesity in Canada and around the world have increased dramatically over the past 20 years and are now reaching epidemic proportions (Andersen, 2000; Bray & Macdiarmid, 2000; Dausch, 2002; Friedrich, 2002; Harmon & Dachman, 2002; Herpertz & Saller, 2000; Katzmarzyk, 2002; WHO, 2000). In Canada, for example, the percentage of the 20-64 year old adult population considered overweight (a BMI >= 30) increased from 5.6% in 1985 to 14.9% in 2000 (Raine, 2004). In Manitoba, a similar trend has been observed, with rates of overweight in adults increasing from 16% in

1985 to 35% in 2000. Although some of this increase is due to the aging of the population, much of the increase is due to increases in age specific rates of overweight in younger age cohorts. In Manitoba for example, the rate of overweight in young adults 20-34 years of age increased from 26% in 1995 to 30% in 2000; in the 35-44 year age group, the rate of overweight increased from 29% in 1995 to 35% in 2000 (Statistics Canada, 2004).

Rates of overweight in Canadian children are also increasing. Tremblay & Willms (2000), using data from a number of national representative surveys, estimated that the prevalence of overweight in 7 - 13 year old boys increased from 15% in 1981 to 28.8% in 1996, and among girls from 15% to 23.6% during this same time period. Of even greater concern is the increase in rates of obesity in Canadian children. Between 1981 and 2001 the rate of obesity in Canadian children 7 - 13 years of age increased 5 fold from 2% in 1981 to 10% in 2001 (Canadian Population Health Inititiative, 2004) .

It is important to note that like T2DM, rates of obesity have been observed to be correlated with minority and low socio-economic status in both children and adults. Strauss & Pollack (2001), using data from the National Longitudinal Survey Youth reported that between 1986 and 1998, overweight increased significantly and steadily among American children, with the greatest levels of obesity and greatest increases in obesity occurring in African American and Hispanic children and children living in poor economic circumstances. Mokdad, Bowman, Ford, Vinicor, Marks & Koplan (2001) , reported similar results for American adults using data derived a cross sectional survey undertaken by the Centers for Disease Control and Prevention. Using a BMI of >= 30 as

a definition of obese, this study demonstrated that rates of obesity were significantly higher in non-white adults with low levels of education. In Canada, higher rates of obesity and overweight have been observed among First Nations (Young, 2001), and among adults with lower levels of education (ACPH, 1999).

The close relationship between obesity and T2DM suggests that an understanding of the epidemiology of T2DM requires an understanding of the epidemiology of obesity. At its simplest level, overweight and obesity are the result of an energy imbalance, caused by excess energy intake, insufficient energy expenditure, or by a combination of both. The rapid increase in obesity over the past 20 years suggests that although genetics may be involved in obesity, the causes of the recent obesity epidemic likely have more to do with forces and influences less proximate to the individual. These include the increasingly environmentally pervasive factors which promote the over-consumption and under-expenditure of calories - the structure of the (fast) food system and associated advertising, reliance on automobiles, the lack of free time for home food preparation, recreation and exercise, excessive television viewing, and the loss of physical activity curricula in schools (Chopra et al., 2002; Nestle & Jacobson, 2000; Sobal, 2001).

Although rates of overweight and obesity have historically been higher in groups of low socioeconomic status, the rapid increase in obesity in all groups over the past 20 years (and associated levels of T2DM) suggests that these rate differentials may be on the verge of diminishing. In other words, the historical concentration of obesity and T2DM in spatially concentrated minority groups of low SES may begin to diminish over the next 20 years as the environmental forces promoting energy imbalance gather force and affect

more homogenously all groups in society.

Aging of the population is also responsible for much of the observed increase in T2DM in Canada and North America since age specific rates of T2DM increase rapidly with advancing age. The 1995 prevalence of DM in the general Manitoba population, for example, increased from .4% in the 20 - 24 year age group to 14% in the 70-74 year age group (Blanchard et al., 1998). As referred to earlier in this paper, it is estimated that the impact of the aging of the population in Manitoba will be considerable, leading to a 2 - 3 fold increase in the number of persons with T2DM in the province between 1998 and 2025.

E. Spatial and Population Methods for Studying DM:

There are few geographically specific population based studies of DM reported in the literature. This is because until recently population based data on DM simply have not been available. Unlike cancer or communicable diseases, there are no centralized registries with complete coverage for DM in either Canada or the U.S (Blanchard, Ludwig, Wajda, Dean, Anderson, Kendal et al. 1996; Hux & Tang, 2002). As a result, most epidemiological studies on DM have either focused on small discrete populations or have analyzed broad population health surveys. These approaches have provided valuable information on the epidemiology of DM but have been limited in their ability to generate insights into the small area geographic variability of DM. Research based on surveys have also suffered from biases due to low response rates and under-reporting of DM by survey respondents and usually have not provided DM incidence estimates (Mackenbach,

Looman & van der Meer, 1996).

However, with the recent development in Manitoba of the Manitoba Diabetes Database (MDD), which uses routinely collected administrative data (hospital and physican utilization data) to generate DM estimates (Blanchard et al., 1996), and the adoption of this methodology by other jurisdictions across Canada, a number of population based studies of DM have been undertaken. In Ontario for example, this methodology has been used to develop the Diabetes Practice Atlas which comprehensively describes the incidence and prevalence of DM and its complications (Hux & Tang, 2002). Although the Ontario study is the most comprehensive study undertaken to date using population based estimates for DM, its use of geographic methods was limited primarily to map illustration and basic descriptive epidemiology. The study used either the 50 Ontario counties with an average population size of 200,000 individuals or 16 District Health Council areas with an average population size of 625,000 in its descriptive analyses and did not undertake any formal modeling of DM data using more advanced spatial methods.

Green et al. (2003b), using DM estimates derived from the Manitoba Diabetes database, applied both ecological regression and the spatial scan statistic to model the small area spatial variability of DM in the city of Winnipeg, Manitoba. This study observed a high level of spatial variability in DM prevalence strongly correlated to measures of socioeconomic status. Green et al. (2003a) also used the Manitoba Diabetes database to describe the spatial and temporal variability in DM incidence and prevalence in Manitoba from 1989 to 1998. This study demonstrated distinct spatial patterns of DM incidence and prevalence for First Nation and non-First Nation populations.

Although geographic and spatial methods have not been applied extensively to the study of DM, spatial methods have been used to study a wide range of health issues of public health importance (Cromley & McLafferty, 2002; Elliott, Wakefield, Best & Briggs, 2000; Melnick, 2002; Waller & Gotway, 2004). These methods include summary measures of spatial clustering and spatial auto-correlation, spatial smoothing methods to stabilize the small area rate estimates, and data modeling techniques to formally explore the relationships between predictor and outcome variables across space and time, often in a multi-level manner.

It is important to note that the application of spatial methods to epidemiology and public health is still in its infancy and there is currently no consensus on the appropriate methods to use. As a result a number of authors suggest that multiple spatial methods and approaches should be triangulated where possible to ensure that study results and conclusions are valid (Gatrell, 2002; Gatrell & Bailey, 1996; Hanson & Wieczorek, 2002; Melnick, 2002). It has been observed, for example, that analyses undertaken at different geographic scales or using different configurations of areal units can generate quite different and often contradictory results (Meade & Earickson, 2000). This issue, known as the modifiable areal unit problem (MAUP) is well recognized in geography, but is only now becoming appreciated in epidemiology. As well, the issue of the ecological fallacy, which consists of incorrectly inferring that relationships observed at the population level also hold at the individual level can seriously confound the conclusions drawn from spatial analyses (Macintyre & Ellaway, 2000). Again, the use of multiple methods employed at different scales of analysis, and being clear on issues related to construct validity (i.e.

variables measured at the individual level and the population level may be measuring very different things) can help guard against drawing erroneous conclusions.

Chapter 4

Geographic Analysis of Diabetes Prevalence in an Urban Area[1]

Introduction:

Type 2 Diabetes Mellitus (T2DM) is one of the most common non-communicable diseases in the world today (Amos, McCarty & Zimmet, 1997). It is projected that the number of cases of T2DM around the world will increase rapidly over the next 25 years, from 154 million estimated cases in 2000 to 300 million cases in 2025 (King, Aubert & Herman, 1998). There is great debate about the cause of the T2DM epidemic (Swinburn, 1996). Although there is general consensus that T2DM has both genetic and social roots, there is little consensus on the relative contribution of these factors (Carter, Pugh & Monterrosa, 1996; Fujimoto, 1996; Haffner, 1998; Hales & Barker, 1992; Hales, Desai & Ozanne, 1997; McDermott, 1998; Ozanne & Hales, 1998).

This study used two spatial techniques to explore the geographic variability of Diabetes Mellitus (DM) prevalence in the City of Winnipeg, Manitoba. Since 95% of all cases of DM are estimated to be T2DM (Harris, 1995), DM prevalence was used in this study as a proxy for T2DM. A common problem in geographic epidemiology is that observed rates, especially in low incidence or prevalence situations, can often be artifacts of the areal geographic units to which individual events are aggregated for analysis. This can have the effect of rendering invisible small geographic areas that have significantly elevated rates of disease (Meade & Earickson, 2000) The spatial scan statistic, the first

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geographic method used in this study, avoids this problem by iteratively creating a number of statistically significant high and low rate cluster areas from small geographic regions. The spatial scan has been used in a number of recent studies to identify spatial clusters of cancer (Hjalmars & Gustafsson, 1999; Hjalmars, Kulldorff, Gustafsson & Nagarwalla, 1996; Hjalmars, Kulldorff, Wahlqvist & Lannering, 1999; Jemal, Devesa, Kulldorff, Hayes & Fraumeni, 2000; Kulldorff, Athas, Feurer, Miller & Key, 1998; Kulldorff, Feuer, Miller & Freedman, 1997; Kulldorff & Nagarwalla, 1995), child-hood mortality (Sankoh, Ye, Sauerborn, Muller & Becher, 2001), aviation crashes (Grabowski & Li, 2001) and acute respiratory disease in cattle (Norstrom, Pfeiffer & Jarp, 1999). This spatial scan method is compared to the more traditional approach of aggregating event data to pre-existing and large geographic administrative areas.

Materials and Methods:

Study Setting:

The study was conducted in the City of Winnipeg, Manitoba, Canada. Winnipeg has a population of 632,000 and is the only large metropolitan city in the province. Over the past 20 years, Winnipeg has experienced significant social and physical deterioration of its central core and downtown area, paired with rapid growth of its peripheral suburbs. The majority of the population historically have been of European descent. However, because of in-migration from rural communities and natural population increases, an increasing percentage of the population is of Aboriginal descent. Manitoba has a universal health insurance plan and all residents of the province are eligible to receive health care

services without cost.

Data Sources:

Sociodemographic data including self-reported Aboriginal status were obtained from the 1996 Census Canada microdata files (Statistics Canada, 1996). Data on the quality of the physical and social environment in 1999 were obtained from the City of Winnipeg (City of Winnipeg, 2000). Data on smoking rates and DM prevalence data for 1998 were obtained from the Manitoba Health Epidemiology Unit. Definitions for the sociodemographic, lifestyle and environmental predictor variables are included in Table 4.2. The methodology used to generate population based DM prevalence estimates has been described previously (Blanchard, Ludwig, Wajda, Dean, Anderson, Kendal et al. 1996). This method used the standardized case definition of either one hospital visit or two physician visits with a DM diagnosis (ICD 250) within a two-year period in order generate DM incidence and prevalence estimates from hospital and physician claims data. This methodology was unable, however, to distinguish between T2DM and Type 1 insulin dependent diabetes mellitus. Hospital and physician claims data were available for all residents of Manitoba since the entire population is covered by a universal health care program. Population denominator data were obtained from the Manitoba Health population registry of all citizens insured for health services in the province. All data were aggregated initially to the neighborhood level (n=230) using the geocoding functionality within the GIS software Arc-View 3.2 (Environmental Systems Research Institute, 1999).

Spatial Methods:

Two methods were used to explore the geographic variability and clustering of

DM within the City of Winnipeg and to identify, using ecological methods, the social and environmental factors associated with variability in DM. The first is the spatial scan method and the second is the pre-existing regions method. Linear regression and analysis of variance were used with both these methods to identify the socioeconomic, environmental and lifestyle factors ecologically associated with this variability. These factors included self-reported Aboriginal status, education, income, family structure, unemployment, housing conditions, crime and smoking rates.

Spatial Scan Method

The spatial scan statistic was used to test for the presence of clusters of DM and to identify their approximate location. The open domain software Statscan distributed by the National Cancer Institute was employed for this purpose (Kulldorff, Rand, Gherman, Williams & DeFrancesco, 1998). The spatial scan statistic, which works by aggregating together the unique combinations of small area geographies which have a high probability of being clusters is an especially powerful tool to use in low prevalence and low incidence situations. Traditional epidemiological approaches which require that rare events be aggregated to pre-existing higher level geographies can often mask the existence of real clusters. The statistic assumes the number of cases in each geographic region to be Poisson distributed. The method tests the null hypothesis that within any age and gender group, the risk of having DM is the same as in all regions combined. This means that the expected age and gender adjusted prevalence rate is constant over the whole area.

The spatial scan statistic places a circular window of varying size on the map surface and allows its center to move so that at any given position and size, the window

includes different sets of adjacent neighborhoods. If the window contains the centroid of a neighborhood, then the whole neighborhood is included in the window. As the window is placed at each neighborhood centroid, its radius is varied continuously from zero up to a maximum radius which never includes more than 50% of the total population. The method creates a large number of distinct circular windows, each containing a distinct set of adjacent neighborhoods, and each a possible candidate for containing a cluster of prevalent diabetes cases. For each window, the method uses a Monte Carlo simulation to test the null hypothesis that there is an elevated risk of DM prevalence. The Statscan software allows any number of covariates to be implemented into the model, and calculates indirectly standardized rates. Details of the how the likelihood function is maximized over all windows under the Poisson assumption have been described elsewhere (Kulldorff et al., 1997).

In this study, the Statscan software was applied to the 230 Winnipeg neighborhoods in order to generate possible clusters of DM prevalence. Age and gender were applied as covariates. Two iterations were undertaken. The first iteration used the default setting within the Statscan software which maximizes the cluster size at 50% of the total study population. The second iteration set the maximum generated cluster size at 10% of the total study population. Smaller maximum cluster sizes result in a larger number of smaller clusters with more extreme values. The Monte Carlo simulation used to test significance was set at 999 iterations. The software was set to generate both high and low clusters. Only statistically significant clusters were retained for analysis. Non-cluster areas were aggregated together into one cluster area and assumed to have a

relative risk of 1.0. Clusters were initially mapped using Arc-View 3.2 (Environmental Systems Research Institute, 1999) in order to identify their physical location. Social and environmental predictor variables were then aggregated to the cluster areas in order to identify their possible relationship to DM prevalence. Where appropriate, analysis of variance and non-spatial and spatial linear regression techniques were used to formally explore the relationship between predictor variables and DM prevalence.

Pre-Existing Regions Method

In the second method, DM prevalence and predictor data were further aggregated to the 23 Health regions used to organize the delivery of services within the City of Winnipeg. DM prevalence was directly standardized by age and gender to the 1998 Winnipeg population. Chloropleth maps of all variables were generated to visually examine their spatial distribution. Spatial clustering of all variables were assessed using the Moran's I statistic. Non-spatial and spatial regression techniques were used to explore the relationship between predictor variables and the standardized DM prevalence.

Analysis of Variance and Regression Analysis:

In both the spatial scan and pre-existing regions methods, variables were log transformed when necessary in order to ensure that the regression assumptions of normality and heteroskedascity were not violated. Regression analyses were undertaken using the S-PLUS Spatial Statistics extension for Arc-View 3.2 (Mathsoft, 1999). A simultaneous autoregressive model was used for spatial regressions. Analysis of variance was undertaken using NCSS(Hintz, 2001).

Results:

In Winnipeg in 1998 there were 29,885 prevalent cases of DM, resulting in an overall DM prevalence rate of 47.3 cases/1000 population. Prevalence rates of DM were observed to be higher in men than in women and to increase rapidly in the age 65 and over age group.

Spatial Scan Results:

Table 4.1 shows the results applying the spatial scan statistic. With the maximum cluster size set at 50% of the total study population, two significant ($p<0.001$) clusters were generated with relative risks of 1.3 and .84. Figure 4.1 shows that the high relative risk cluster is located in the central and northern core of the City of Winnipeg, while the low relative risk cluster is located in the southern suburbs of the City. The high relative risk cluster is the most likely one, with a log likelihood ratio of 291.56.

With the maximum cluster size set at 10% of the total study population, the spatial scan statistic generated 10 significant ($p<0.009$) high and low clusters. Relative risks ranged from 0.69 to 1.45. Figure 4.2 shows that the high relative risk clusters are again all located in the central and northern core of the City of Winnipeg, while the low relative risk clusters are located in the southern suburbs of the city. The most likely cluster, with a relative risk of 1.45, and a log likelihood ratio of 282.79 is located in the central core of the city.

Table 4.2 shows the predictor variables aggregated to cluster areas generated by the spatial scan method when the maximum cluster size was set to 50% of the total population. This table illustrates that high diabetes prevalence is clustering in those areas

of the City of Winnipeg which have a high percentage of Aboriginal population, low educational levels, low family income, a high percentage of lone parent families, high levels of unemployment, high numbers of vacant and placarded houses, high levels of crime, and high rates of smoking. Analysis of variance was undertaken for all predictor variables. In all cases, the between cluster variance was significant at the $p < 0.005$ level.

Regression analysis of predictor variables against the relative risk of the cluster areas generated when the maximum cluster size was set to 10% produced similar results (Table 4.3). Non-spatial regression resulted in very high Pearson R values ranging from 0.69 to 0.97, with all regressions significant at the $p < 0.001$ level. Education had the greatest predictive value. Spatial regression, which accounts for the spatial clustering of variables in a regression equation, did not appreciably change either the non-spatial regression coefficients or significance levels. With one exception, regression equations generated using spatial regression techniques did not result in any residual spatial autocorrelation, indicating that the regression model was successful in fully accounting for any spatial correlation in the DM prevalence rates.

Pre-Existing Regions Method:

When aggregated to the 23 health regions, all variables used in the model were highly spatially clustered. Visual inspection of chloropleth maps showed clustering of DM prevalence in the central core of the City of Winnipeg (Figure 4.3), associated with a larger Aboriginal population, low education, low family income, lone parent families, high unemployment, poor housing stock, high crime rates, and high rates of smoking. This visual impression was confirmed by significant Morans I values ($p < 0.001$) for all values.

Standardized DM prevalence rates ranged from 37.7/1000 to 78.8/1000 and were significantly different from the mean in all but one region.

Regression of predictor variables against the standardized diabetes prevalence rates for the 23 health regions within the City of Winnipeg generated results similar in strength and direction to the spatial scan analysis. Non-spatial regression again resulted in very high Pearson R values (Table 4.4). All regression models were significant at the $p < 0.001$ level. Unemployment had the greatest predictive value. Spatial regression did not appreciably change either the non-spatial correlation coefficients or significance levels and did not result in any residual spatial autocorrelation.

Multiple regression of predictor variables against diabetes prevalence resulted in a model incorporating family income and unemployment, with a Pearsons R value of 0.944. Spatial regression analysis of these predictor variables did not result in any appreciable change in either the regression coefficients or significance levels and did not result in any residual spatial autocorrelation. In this model, diabetes prevalence was positively associated with unemployment and negatively associated with family income. Additional variables could not be incorporated into the model because of the high level of multi-collinearity of predictor variables (Table 4.5).

Discussion:

This study has demonstrated substantial clustering and small area variations in the prevalence of DM in the City of Winnipeg, and that these variations are associated with variations in socioeconomic, environmental and lifestyle characteristics of the population.

This study has also demonstrated that two distinct approaches to spatial analysis, the spatial scan statistic and the pre-existing regions method generate very similar results in terms of identifying the geographic location of DM clusters and of the population characteristic ecologically correlated to those clusters. Finally, our results have shown that when high levels of non-spatial correlation exist between predictor and dependent variables, spatial regression approaches do not appreciably change the strength or direction of the regression coefficients.

The study has a number of methodological limitations. First, it has relied exclusively on data derived from administrative databases in order to estimate the DM prevalence rates. Since cases have not been individually verified, this approach could result in either an overestimate or underestimate of prevalence rates. However, we have previously studied the accuracy of this approach and found that the specificity is high when compared to local registries of DM (Blanchard et al., 1996). It is possible that some of the small area variations that we observed are due to variability in health care access and diagnostic practices. This is unlikely, however, since Manitoba provides universal health care to its residents so restricted access to physician and hospital services is not likely.

Secondly, the administrative databases from which the diabetes prevalence rates were derived cannot distinguish between T2DM and Type 1 Insulin Dependent Diabetes. However, given that it is estimated that approximately 95% of all DM cases are T2DM it is likely that the variability in DM prevalence observed in this study reflects primarily the impact of T2DM (Harris, 1995)

Thirdly, the small number of observations used in regression analysis within both

the spatial scan and pre-existing regions approaches means that regression results must be used with some caution. Tests for normality and heteroscadiscity may not have been sensitive to violations of regression assumptions because of the small numbers of observations. However, given the strength of the direction and significance of the generated correlation coefficients, and their consistency between the two spatial methods, the observed correlations are likely real and significant.

Fourthly, the ecological approach used in this study has been frequently criticized as being a weak design and commits what is known as the ecological fallacy. The ecological fallacy suggests that it is a mistake to apply characteristics measured at the scale of the population or geographic level to individuals living within those geographies or populations (Morgenstein, 1982, 1995). The ecological design used in this study therefore restricts us to making statements about the characteristics of the populations living in specific geographies. Statements made about individuals living within those geographies can only be made with caution. However, given the arguments by Rose and others (Rose, 1985, 1992; Wilkinson, 1996, 1999) on the primary importance of population and geographic level factors on population health, this study design legitimately provides important clues to the etiology of DM at the population level. It suggests that DM prevalence at the population level is powerfully graded by socioeconomic status, environmental quality, and lifestyle.

Finally, the study used only one lifestyle variable, smoking in mothers of newborns on discharge from hospital, as a proxy for overall lifestyle quality. Given that this variable may be a relatively weak proxy for lifestyle attributes relevant to DM, caution must be

taken in concluding that lifestyle is associated with diabetes prevalence at the ecological level. Lifestyle measures more directly related to DM prevalence such as diet, exercise and obesity were not available at the geographic levels required for this study.

The high level of consistency between the results of the spatial scan statistic and the pre-existing regions method in identifying etiological factors associated with DM is encouraging. Previous studies that have used the spatial scan statistic to identify cancer clusters have not attempted to systematically explore possible etiological factors associated with clusters using analysis of variance and linear regression (Hjalmars et al., 1996; Hjalmars et al., 1999; Jemal et al., 2000; Kulldorff et al., 1998; Kulldorff et al., 1997; Kulldorff & Nagarwalla, 1995; Sankoh et al., 2001; Walsh & Fenster, 1997). This study suggests that the spatial scan statistic in conjunction with analysis of variance and linear regression may be a useful tool in exploring the etiology of cancer and other chronic diseases.

The relationship observed between DM prevalence and low levels of socioeconmic status, environmental quality and lifestyle at the geographic level is consistent with previous studies (Auslander W F, Haire-Joshu, Houston & Fisher E B, 1992; Hanis, Chakraborty, Ferrell & Schull, 1986; Hazuda & Monterrosa, 1992; Hendricks & Haas, 1991; Leonetti, Tsunehara, Wahl & Fujimoto, 1992; Marshall, Hamman, Baxter, Mayer-Davis, Fulton & Orleans, 1993) . This study provides some of the strongest evidence to date of this relationship, with DM prevalence estimates based upon diabetes prevalence estimates covering the whole population of Winnipeg. Previous studies have not been population based and were often restricted in scope to limited surveys of specific sub-populations.

This study demonstrates that the highest rates of DM are occurring in geographic areas that have the highest concentration of Aboriginal people. It has been hypothesized that populations of Aboriginal, Black, and Mexican American origin are genetically predisposed to develop T2DM supposedly due to the high frequency of the "thrifty gene" in their respective population gene pools. The thrifty gene, it is proposed, conferred an adaptive advantage in historical times of feast and famine. However, in modern conditions of relative plenty, the thrifty gene predisposes individuals to the development of obesity and increased frequency of DM (McDermott, 1998; Neel, 1962, 1982, 1999). In this study, it was observed that the geographic areas with the highest prevalence of DM also had the lowest socioeconomic status, the poorest lifestyles, and the lowest levels of environmental quality. Regression analyses demonstrated that broad neighborhood characteristics such as education and income were more predictive of DM prevalence than Aboriginal status. Once family income and unemployment were used in the regression analysis as predictors, Aboriginal status lost all of its significance as a predictor of DM. This suggests that it may be more the impact of low socioeconomic status that is putting populations at risk of DM in Winnipeg rather than genetic background. This also suggests that population based studies using race as a covariate need to critically question their use of racial constructs by examining the social and physical circumstances in which particular racially defined groups find themselves. These studies may need to examine whether it is these circumstances which are predisposing these groups to disease and ill-health rather than something inherent in their "race" (King, 1997). There may indeed be a genetic component that confers some variability in DM between individuals, but at the level of the

population it appears that larger socioeconomic and environmental factors are more important. Further studies which stratify the analysis by Aboriginal status in order to explore whether the socioeconomic and environmental gradients in DM prevalence observed in this study apply to the non-Aboriginal population alone would add strength to these conclusions.

The high level of multi-collinearity observed between predictor variables also suggests that attempts at disentangling the independent relationships between these variables and DM prevalence may be counter-productive since all predictor variables may in fact be measuring aspects of the same phenomena (Evans & Barer, 1994; Hertzman, Frank & Evans, 1994; Marmot, 1999; Wilkinson, 1996; Wilkinson, 1999). This phenomenon is likely related to social position, access to real life choices, and a sense of personal empowerment. This suggests that ecological studies utilizing socioeconomic predictors should start to locate their analyses within well developed perspectives on how the social position of particular groups become established, spatially concentrated, reproduced over time, and results in poor health outcomes. The specific pathways by which low social position becomes translated into poor health outcomes through the generalized stress response, poor lifestyle practices, and reduced opportunities are becoming increasingly recognized (Baum, Garofalo & Yali, 1999; Cohen, 1999; Kawachi, 1999; Lundberg, 1999; McEwen & Seeman, 2001; Pickering, 1999; Williams, 2001).

This study raises questions about how we need to better understand the powerful and predictable impact that place has on the health of populations. This study has documented that geographies in the central core of Winnipeg are associated with high

levels of DM. These core area neighborhoods are places that seemed to have emerged as gathering places for individuals low on the social hierarchy with few social choices. This has likely occurred as a result of historical, political, and economic forces. The result has been the transformation of the physical and social fabric of these geographies into places of risk with ecological characteristics having strong association with health status. In order to more fully understand how this has happened over time, the unique history of how these high-risk geographies have evolved over time have to be explored more carefully through the use of diverse historical, political, economic and ethnographic methods.

Finally, the results of this study suggest that high rates of DM are tightly embedded within a context of poverty and disempowerment. Population based prevention programs which focus only on lifestyle modification would likely not be successful. As illustrated by this study, lifestyle quality indicators like smoking are highly highly correlated with income and education. This suggests that DM prevention programs, to be successful, would require comprehensive policy interventions above and beyond lifestyle modification. These interventions have to address the socioeconomic resources and opportunities available to individuals.

Table 4.1:
DM prevalence analysis, City of Winnipeg, Manitoba, 1998, using the spatial scan statistic

Max. Cluster Size	Cluster Type !	Cases	Expected	RR*	LLR#	Pvalue
A. 50%	High	7335	5644	1.3	291.56	p < 0.001
	Non	12782	n/a	1.0	n/a	n/a
	Low	9768	11578	0.84	236.17	p < 0.001
B. 10%	High	4101	2825	1.45	282.79	p < 0.001
	High	912	778	1.171	11.07	p < 0.009
	High	2056	1820	1.13	15.64	p < 0.001
	Non	17527	n/a	1.0	n/a	n/a
	Low	2457	2916	0.84	42.16	p < 0.001
	Low	677	840	0.81	17.47	p < 0.001
	Low	1799	236	0.76	78.66	p < 0.001
	Low	199	272	0.73	11.09	p < 0.009
	Low	362	509	0.71	23.96	p < 0.001
	Low	464	659	0.70	33.07	p < 0.001
	Low	243	353	0.69	19.74	p <0 001

! **Cluster Type:** High - cluster with relative risk > 1, Non - aggregation of non clustered population, Low - cluster with relative risk < 1;
* **RR:** Relative Risk - Observed DM Prevalence / Expected DM Prevalence;
 # LLR: Log Likelihood Ratio

Table 4.2:
Analysis of variance, predictor variables aggregated to spatial scan generated cluster areas for DM prevalence, maximum cluster size set to 50%

Predictor	Clusters			Analysis of Variance
	Low Cluster (#RR=0.84)	Non-Clustered (#RR=1.0)	High Cluster (#RR=1.30)	
Aboriginal Status *	3.8	5.5	16.9	$p < 0.005$
Less than Grade 9 *	5.3	8.9	17.3	$p < 0.005$
Average Family Income *	62994	50810	37392	$p < 0.005$
Lone Parent *	13.7	16.1	23.6	$p < 0.005$
Unemployment *	6.1	7.7	14.5	$p < 0.005$
Vacant House *	0.6	0.8	15.1	$p < 0.005$
Crime *	56.6	88.5	157.6	$p < 0.005$
Smoking *	18.1	26.7	35	$p < 0.005$
DM Cases by Cluster	n = 9768	n = 12,782	n = 7335	
Study Population by Cluster	n = 245528	n = 258849	n = 127623	

* **Aboriginal Status** - % of the population reporting Aboriginal Status; **Less than Grade 9** - % of the population 15 yrs + reporting less than grade 9 education; **Average Family Income** - average family income; **Lone Parent** - % of families reporting being headed by a lone-parent; **Unemployment** - % of the population 15+ in the labour force that is unemployed; **Vacant House** - no. of houses/1000 residential properties that are vacant or placarded; **Crime** - no. of crimes against property and persons/1000 population; **Smoking** - % of mothers of newborns smoking on discharge from hospital

 # RR: Relative Risk - Observed DM Prevalence / Expected DM Prevalence;

Table 4.3:
Regression analysis of DM prevalence relative risk vs. predictor variables, using spatial scan generated cluster areas, maximum cluster size set to 10%

	Non-SpatialRegression			Spatial Regression		
Predictor	**R #**	**Regression Coefficient**	**P Value**	**Regression Coefficient**	**P Value**	**Residual Spatial Autocorrelation !**
Aboriginal Status *	0.90	0.034	$p < 0.001$	0.0398	$p < 0.001$	N.S
Less Than Grade 9 *	0.97	0.0452	$p < 0.001$	0.0456	$p < 0.001$	N.S.
Average Family Income*	-0.93	- 0.0000139	$p < 0.001$	-0.000013	$p < 0.001$	N.S.
Lone Parent *	0.89	0.0342	$p < 0.001$	0.0240	$p < 0.001$	N.S.
Unemployment *	0.93	0.0504	$p < 0.001$	0.0558	$p < 0.001$	N.S.
Vacant House *	0.69	0.0202	$p < 0.001$	0.0079	$p < 0.098$	$p < 0.05$
Crime *	0.90	0.00433	$p < 0.001$	0.0049	$p < 0.001$	N.S.
Smoking *	0.88	0.218	$p < 0.001$	0.023	$p < 0.001$	N.S.

*** Definitions in Table 2;**
 # R Pearsons R
! Spatial autocorrelation of regression residuals. Significance is based upon the Moran's I statistic.

Table 4.4:
Regression analysis, age standardized DM prevalence rates vs. predictor variables, using existing health boundaries for the City of Winnipeg, Manitoba

	Non-Spatial Regression			Spatial Regression		
Predictor	R #	Regression Coefficient	P Value	Regression Coefficient	P Value	Residual Spatial Autocorrelation !
Aboriginal Status *	0.90	1.009	p < 0.001	0.969	p <.0 001	N.S.
Less Than Grade 9 *	0.90	1.584	p < 0.001	1.586	p < 0.001	N.S.
Avg. Family Income*	-0.89	-0.0006	p < 0.001	-0.0006	p < 0.001	N.S.
Lone Parent *	0.86	1.156	p < 0.001	1.153	p < 0.001	N.S.
Unemployment *	0.92	1.682	p < 0.001	1.76	p < 0.001	N.S.
Vacant House *	0.74	0.480	p < 0.001	0.4008	p < 0.001	N.S.
Crime *	0.80	0.110	p < 0.001	0.1026	p < 0.001	N.S.
Smoking*	0.84	0.729	p < 0.001	0.766	p < 0.001	N.S.

*** Definitions in Table 2**
 # Pearsons R
! Spatial autocorrelation of regression residuals. Significance is based upon the Moran's I statistic

Table 4.5:
Covariance of predictor variables, Pearsons R, using existing health boundaries for the City of Winnipeg

	Aboriginal Status	Less Than Grade 9	Average Family Income	Lone Parent	Unemployment	Vacant House	Crime	Smoking
Aboriginal Status *	1.0	0.90	-0.82	0.94	0.98	0.83	0.86	0.89
Less than Grade 9*	0.90	1.0	-0.87	0.87	0.90	0.74	0.77	0.86
Average Family Income*	-0.82	-0.87	1.0	-0.89	-0.86	-0.78	-0.82	-0.87
Lone Parent*	0.94	0.87	-0.89	1.0	0.96	0.85	0.90	0.93
Unemployment*	0.98	0.90	-0.86	0.96	1.0	0.83	0.90	0.88
Vacant House*	0.83	0.74	-0.78	0.85	0.84	1.0	0.89	0.79
Crime *	0.86	0.77	-0.82	0.90	0.90	0.89	1.0	0.78
Smoking *	0.89	0.86	-0.87	0.93	0.87	0.79	0.79	1.0

*** Definitions in Table 2**

Figure 4.1:
DM prevalence analysis, City of Winnipeg, 1998, using the spatial scan statistic, maximum cluster size set at 50% of the study population

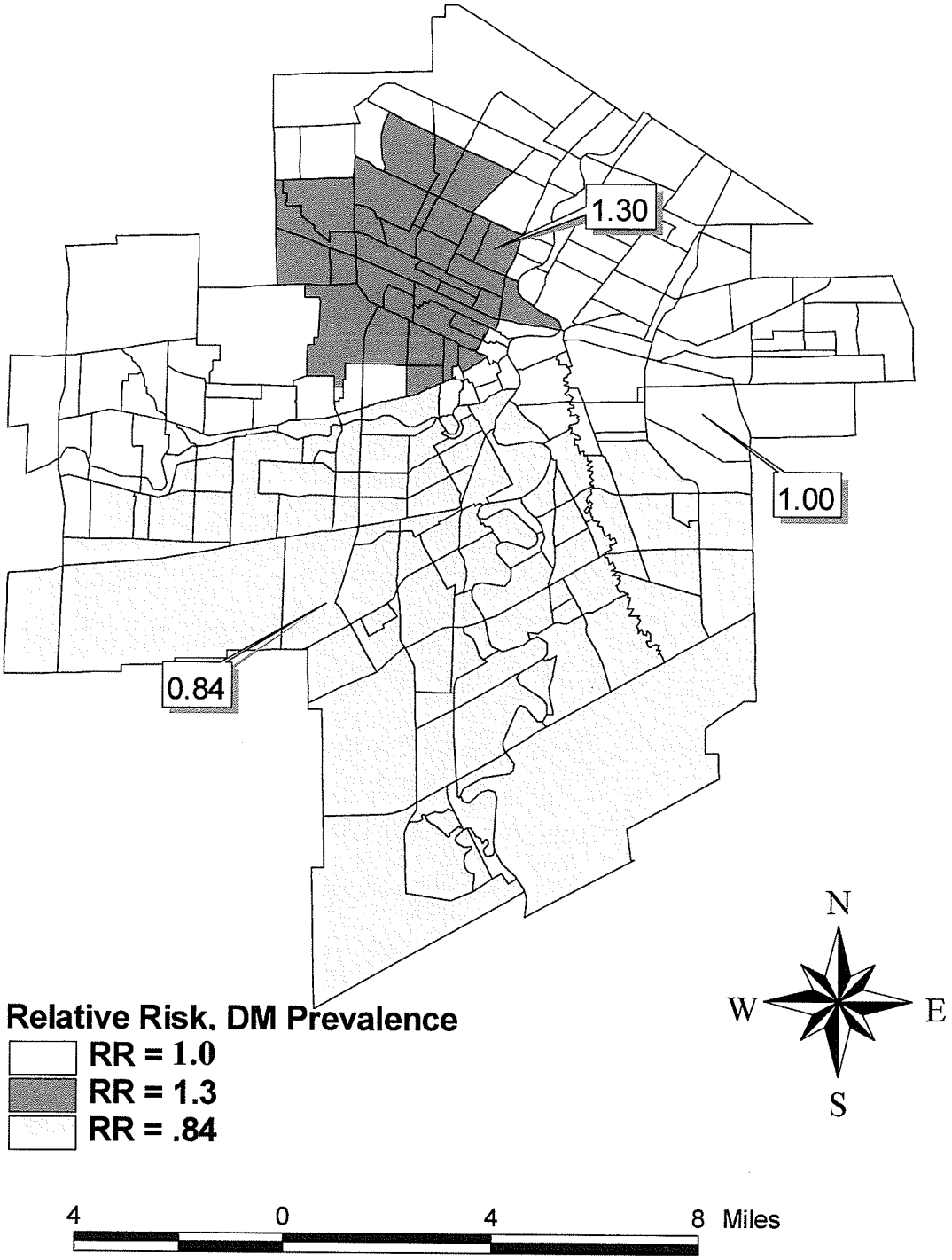


Figure 4.2:
DM prevalence analysis, City of Winnipeg, 1998, using the spatial scan statistic, maximum cluster size set at 10% of the study population

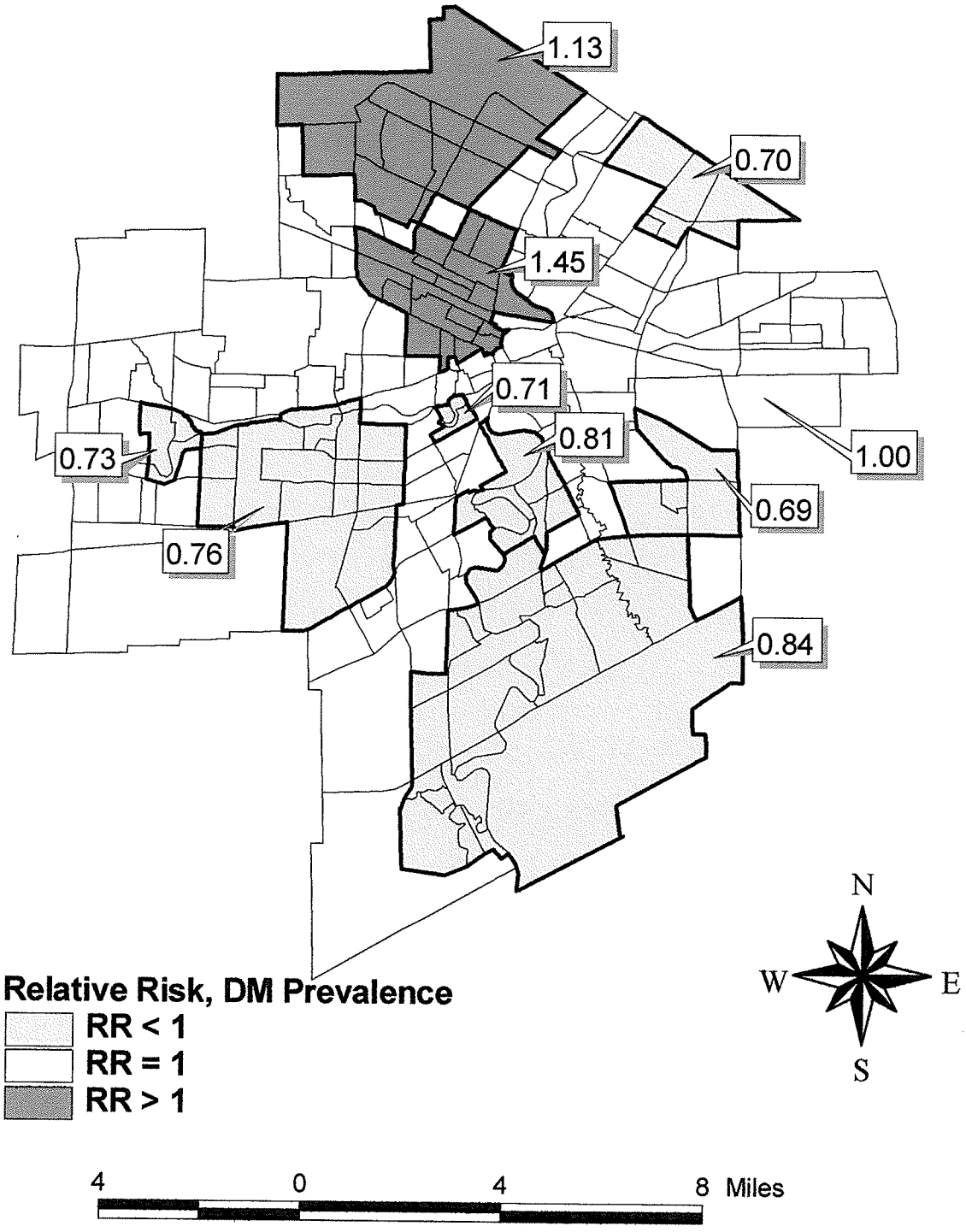
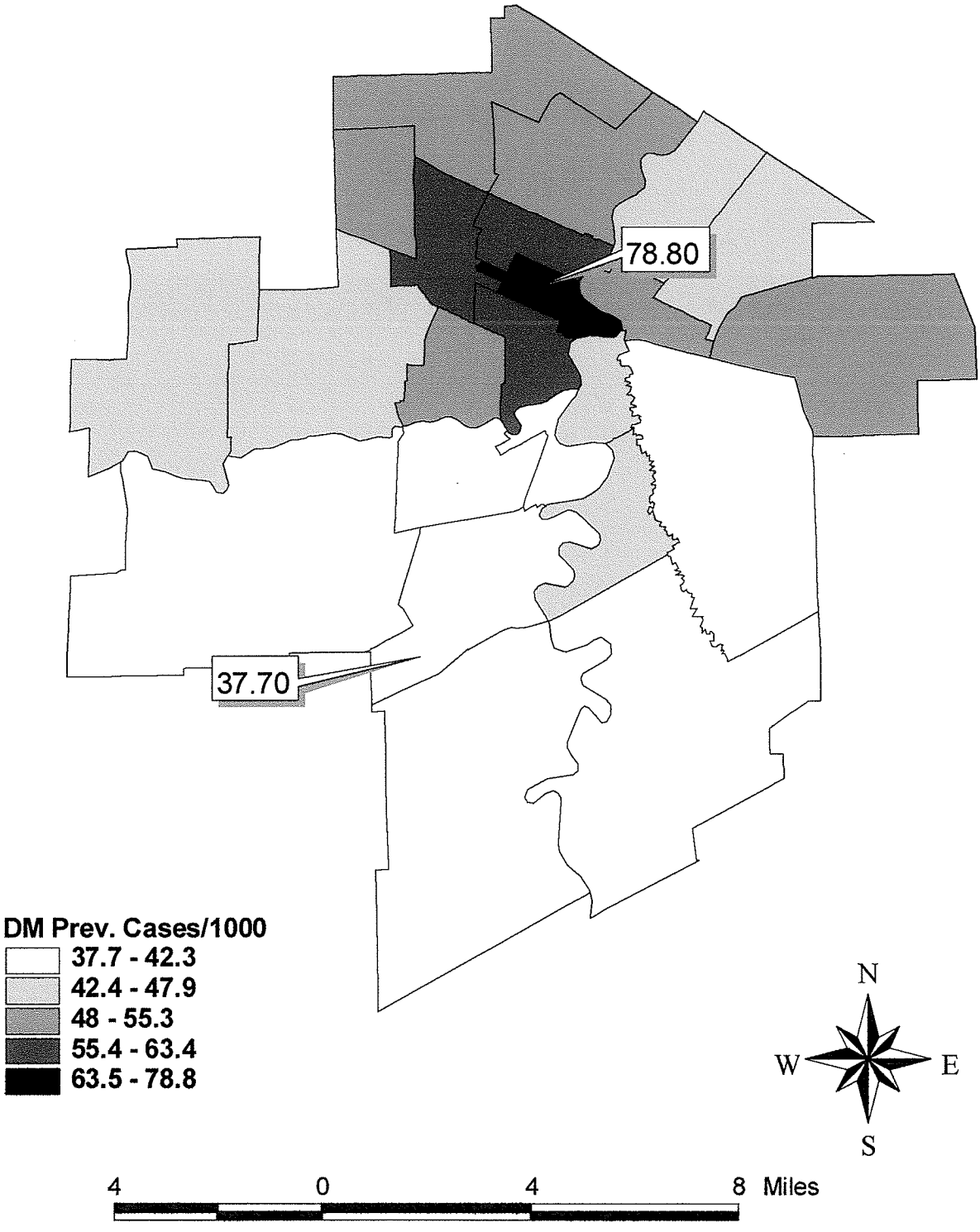


Figure 4.3:
Standardized DM prevalence rate, City of Winnipeg, 1998 by health region



Chapter 5

The Epidemiology of Diabetes in the Manitoba Registered First Nation Population: Current Patterns, Comparative Trends[1]

Introduction:

Diabetes Mellitus (DM) is increasingly responsible for substantial morbidity and mortality in Canada's First Nation populations. Planning and implementing effective primary and secondary intervention programs to deal with this disease and its devastating effects in First Nations people requires accurate population-based data on the temporal trends and geographic distribution of DM (Young, Reading, Elias & O'Neil, 2000).

This study uses Manitoba Health administrative databases to examine trends in the incidence and prevalence of DM among Registered First Nation adults in Manitoba from 1989 to 1998. Comparisons are made to the non-First Nation adult population in order to highlight the magnitude of the DM epidemic in First Nations people. The geographic variation in DM rates across Manitoba are also examined.

The study was conducted in the Canadian province of Manitoba. Manitoba has a population of 1.14 million people of which more than half (645,000) reside in Winnipeg, the provincial capital. The majority of Manitobans are of European descent, while approximately 10% of the population are self-identified as having Aboriginal ancestry (Statistics Canada, 1996). Manitoba has a universal health insurance plan and all residents of the province are eligible to receive health care services with no payments required at the

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time of service.

Research Design and Methods:

Data Sources:

The data used for this study were derived from the Manitoba Diabetes Database (MDD) which has been described previously (Blanchard, Ludwig, Wajda, Dean, Anderson, Kendal et al. 1996). This database contains a longitudinal record for Manitoba residents of all physician contacts and hospital separation records which cited a diagnosis of DM (ICD-9-CM code 250) between April 1, 1984 and March 31, 1999. Individuals are categorized as having DM if they have had at least 2 separate physician contacts for DM within 2 years of each other, or at least one hospital separation for DM. Cases of gestational diabetes are excluded from the MDD. The sensitivity of this database for detecting clinically diagnosed cases of DM has been demonstrated and the validity of the methodology has been discussed previously (Blanchard et al., 1996). This methodology is unable to distinguish between Type 2 and Type 1 DM. Individuals who move from the province subsequent to being identified as having DM are included in the MDD only for the years they are resident in the province.

Manitoba's First Nations are comprised of persons who are registered under the Indian Act of Canada and living in Manitoba. To identify persons belonging to Manitoba's First Nations, the Manitoba Health population registry was used. As part of the process for registration within Manitoba Health's insurance system, an attempt is made to verify whether new registrants are Registered Indian. If so, their First Nation

affiliation is recorded. It is important to note that for a variety of complex historical and political reasons many individuals of Aboriginal ancestry are not eligible for registration under the Indian Act of Canada. These individuals are therefore not identified as First Nations in this study. As well, approximately only 70% of Registered First Nations living in the province of Manitoba are identified as being First Nation in the Manitoba Health population registry.

Estimating Incidence and Prevalence:

The diagnosis date of incident cases was defined based on the first physician contact for a diagnosis of DM which was followed within 2 years by a subsequent physician contact, or by the hospitalization for DM, whichever came first. The annual incidence rates were calculated using the mid-year population at risk based on the Manitoba population registry. The population at risk is the population at mid-year without DM. The average annual incidence rates for the years 1994-98 were computed by cumulating all incident cases and summing the mid-year population at risk for those years. The point prevalence at December of each year was estimated by determining the number of cases which had previously been diagnosed who had neither died nor left the province at that time. This was done using the Manitoba Health population registry which is routinely updated and closely matches census population estimates. The average annual prevalence rates for 1994- 98 were computed by cumulating the point prevalent cases for each of the 5-year periods and summing the mid-year populations for those years. Average annual incidence and prevalence rates for 1994-1998 were calculated in order to maximize rate stability when making geographic comparisons. In order to facilitate

comparisons across time and with other published reports and studies, age- adjusted rates were computed for both incidence and prevalence by the direct method using the 1991 Canadian population as the standard.

To examine the geographic variations in cases of DM, the province was divided into 22 regions. The City of Winnipeg was divided into 12 Community Characterization Areas (CCAs). CCAs are administrative areas used by the Winnipeg Regional Health Authority (WHRA) to deliver health services. CCAs were used in this study since over 60% of the province's population resides within the City of Winnipeg. Rural Manitoba was divided into 10 Regional Health Authority (RHAs) areas. The two northern RHAs of Burntwood and Churchill were combined for this study in order to maintain the population size required to ensure stable rate calculations. The average population size of each region was approximately 50,000. Age adjusted incidence and prevalence rates for each region were calculated using the 1991Canadian population as a standard.

Results:

Time Trends:

The age-adjusted prevalence of DM among persons 20 years and older increased steadily between 1989 and 1998 for First Nation and non-First Nation men and women (Figure 5.1). Among First Nation women, the age adjusted prevalence increased by 37% from 181.6/1000 in 1989 to 248.7/1000 in 1998. Among First Nation men, there was a 1.6 fold increase from 104.2/1000 in 1989 to 170/1000 in 1998. There were also increases in prevalence among non-First Nation men (1.4 fold, from 41.9 to 59.63/1000) and women

(1.4 fold, from 37.1 to 53.5/1000). Throughout the study period, First Nation males had an average DM prevalence rate 2.5 times higher than non-First Nation males, while First Nation females had an average DM prevalence rate 4.5 times higher than non-First Nation females.

The age adjusted incidence of DM among persons 20 years and older increased slightly for First Nation males, and Non-First Nation males and females between 1989 and 1998. The incidence in First Nation females did not change during this period. Among First Nation men, there was a 1.4 fold increase from 15.3/1000 in 1989 to 21.1/1000 in 1998. There were also increases in incidence among non-First Nation men (1.25 fold, from 5.44/1000 to 6.8/1000) and women (1.2 fold, from 4.7/1000 to 5.7/1000). Throughout the study period, First Nation males had an average DM incidence rate 3 times higher than non-First Nation males, while First Nation females had an average DM incidence rate 3.7 times higher than non-First Nation females.

Age and Gender Specific Incidence and Prevalence:

Age specific incidence and prevalence of DM were substantially higher among First Nations as compared non-First Nations persons in all age groups. Whereas the incidence and prevalence among non-First Nations persons increased steadily with age, among First Nations both incidence and prevalence peaked in the 60-69 year age group and began to fall in the 70 plus age group. The highest prevalence difference between First Nation and non-First Nation women occurred in the 45-49 year age group, with a prevalence ratio of 7.2 (Table 5.1). In men, the highest prevalence difference occurred in 40-44 year old age group (prevalence ratio of 5.2).

In First Nations both incidence and prevalence of DM were higher among women than in men. The opposite trend was observed in the non-First Nation population.

Distribution by Geographic Region:

Although the incidence and prevalence of DM was substantially higher among First Nations than non-First Nations in all geographic areas, the geographic patterns differed (Figure 5.2). In the First Nations population, the highest prevalence of DM was among those who lived in the southwestern rural area of the province (290/1000) and in the eastern and western sections of the City of Winnipeg. The lowest prevalence was among those living primarily in the more remote northern rural areas of the province and in the southern section of the City of Winnipeg. In contrast, among non-First Nation populations, the highest rates were found in the more remote northern rural areas of the province and in the central downtown area of the City of Winnipeg. Similar patterns were observed for DM incidence.

Relationship between Incidence, Mortality and Increasing Prevalence:

Figure 5.3 illustrates that the annual incidence of DM exceeded annual mortality in First Nations with DM between 1989 and 1998, leading to net annual increases in DM cases over the entire study period.

Discussion:

The prevalence of DM in First Nations observed in this study are comparable to those observed in other studies. Harris, Gittelsohn, Hanley, Barnie, Wolever, Gao et al. (1997), using data collected through an intensive community-wide prevalence survey,

reported age standardized prevalence rates (standardized to the 1991 Canadian population) of DM in the Sandy Lake First Nation population of 280/1000 and 242/1000 for women and men respectively. This compares to rates of 248/1000 and 209/1000 for First Nation women and men observed in this study. Schraer, Adler, Mayer, Halderson & Trimble (1997) and Burrows, Geiss, Engelgau & Acton (2000), drawing upon data derived from a variety of patient registries, observed somewhat lower age specific prevalence rates in American Indians and Alaska Natives between 1990 and 1997. Consistent with this study, however, both Schraer and Burrows observed increasing prevalence of DM over time. It is important to note that prevalence comparisons between these studies are difficult because of differences in data capture methodologies, age ranges and time frames employed, and age standardization techniques used.

This study has a number of methodological limitations which must be kept in mind when interpreting its results. First, it has relied exclusively on data derived from administrative databases in order to estimate DM prevalence and incidence rates. Since this approach depends upon DM cases being recognized, diagnosed and recorded through routine interaction with the health care system, it most certainly underestimates the actual incidence and prevalence of DM. As described by Young & Mustard (2001) and Harris, Flegal, Cowie, Eberhardt, Goldestein & Little (1998), up to one third of DM cases are undiagnosed. Some of the increasing prevalence of DM observed in this study may actually have been due to movement of individuals from the undiagnosed DM pool as a result of increased screening by health care providers. As well, as a result of the recent lowering of the cutoff point for diagnosing DM in Canada from a fasting plasma glucose

level of 7.8 to 7 mmol/L, the pool of undiagnosed individuals with DM has increased. Movement of individuals from the undiagnosed to the diagnosed DM pool may be an important factor in driving future increases in observed DM prevalence. Despite likely underestimation of DM incidence and prevalence, the specificity of our approach is high when compared to existing registries of Type 2 DM (Blanchard et al., 1996) and to abstracted patient charts of randomly selected physicians (Hux et al., 2002).

Secondly, the restriction of First Nation designation in this study to those individuals registered under the Indian Act of Canada, and the under-recording of First Nation status in the Manitoba Health Registry, likely diminishes the observed differences between the First Nation and the non-First Nation population. This is probably not a major issue since those individuals identified as First Nation are highly likely to be of First Nation ancestry whereas those missed and thus classified as non-First Nation are unlikely to affect the rate in the much larger non-First Nation population.

Thirdly, this study does not differentiate between Type 1 DM and Type 2 DM since the case definition for DM used in the generation of MDD from administrative databases is based upon ICD-CM- 250. No subclassifications exist in these administrative databases which would allow differentiation between Type 1 and Type 2 DM. However, given that Type 2 DM makes up approximately 90 - 95%of all DM cases, the use of DM in this study is likely a valid proxy for Type 2 DM (Harris, 1995).

The results of this study have two important implications for First Nation DM prevention and management programs. First, it appears that DM prevalence rates will almost certainly continue to rise in Manitoba First Nations over the next two decades. As

illustrated in figure 5.3, as long as the number of incident cases of DM exceeds the number of deaths in persons with DM, DM prevalence will continue to rise. The rapid aging of the currently very young First Nation population into high risk older age groups, paired with emerging life prolonging DM treatments, will maintain the spread between incidence and mortality into the forseeable future. Even if incidence rates were flat or declining due to a breakthrough in DM prevention, prevalence rates would continue to rise as incidence outpaces mortality. This observation is consistent with others who are predicting world wide increases in DM prevalence over the next several decades in all population groups (Boyle, Honeycutt, Narayan, Hoerger, Geiss, Chen et al. 2001; King, Aubert & Herman, 1998).

As a result, the health burden due to all types of diabetic complications will likely continue to rise in Manitoba First Nations. This means that the health care and social service systems should start preparing now to provide the secondary prevention and support services and systems a large number of First Nation adults with DM are going to require to maintain a quality of life. These include DM screening programs, foot-care programs, accessible dialysis services, dietary counseling services, and enhanced infrastructure at the community level to facilitate independent living by adults with limited mobility and eyesight.

Secondly, "upstream" population based primary prevention programs need to be aggressively implemented to ensure that DM incidence among First Nations begins to decrease in the future. The dramatically higher rates of DM in Manitoba First Nations as compared to the non-First Nations population highlights the urgency of this activity.

Since DM appears to be closely related to the adoption by First Nations people of many aspects of the modern lifestyle including diet and low levels of physical activity, prevention programs which draw upon Aboriginal traditions and ways of life and which focus on the lifestyle habits of Aboriginal youth need to be implemented (Dyck, 1995; Szathmary, Ritenbaugh & Goodby, 1987). Currently, almost 50% of the First Nation population is under the age of 20 years and are still in the process of forming life-long lifestyle habits which will affect their future susceptibility to developing DM and its complications. A number of very promising primary prevention programs which draw upon Aboriginal traditions and ways of life have been implemented across Canada (Daniel, Gamble, Henderson & Burgess, 1995; Daniel, Green, Marion, Gamble, Herbert, Hertzman et al. 1999; Gittelsohn, 1995; Gittelsohn, Harris, Burris, Kakegamic, Landman, Sharma et al. 1996; Macaulay, 1988; Macaulay, Paradis, Potvin, Cross, Saad-Haddad, McComber et al. 1997; McComber, Macaulay, Kirby, Desrosiers, Cross & Saad-Haddad, 1998).

The results of this study are also suggestive of a number of future research priorities. First, the observation in this study of lower DM prevalence in more northern and remote areas of the province suggests that living in these areas has a protective effect on DM. Further research is required to determine if this is due to a greater adherence to traditional lifestyle practices such as hunting, fishing, and consumption of wild game, to broader community level factors, or to genetic factors.

Secondly, the reason for the higher prevalence of DM in First Nation women observed in this study also needs to be better understood. The relationship to earlier episodes of gestational DM should be investigated as one possible pathway that increases

the susceptibility of First Nation women to DM.

In conclusion, this paper has demonstrated the value of having accurate population based information on the epidemiology of DM in First Nations. By providing information on the trajectory and the geography of the DM epidemic in First Nations, and the intensity of the epidemic in comparison to non-First Nation populations, it provides important clues as to the magnitude and structure of the primary and secondary intervention programs which will be required to effectively manage this disease in First Nations people. It also highlights the important need to undertake further research into the community and individual level factors which appear to place some First Nation population groups at lower risk for developing DM.

Table 5.1:
Age specific and age adjusted prevalence (per 1000 adults) of DM in Manitoba, 1998, First nations compared to non-First nations

	First Nation		Non-First Nation		Comparison
Sex and age group (years)	Cases	Prevalence per 1,000	Cases	Prevalence per 1,000	Prevalence Ratio*
Females					
20-24	78	26.79	261	7.47	3.58
25-29	173	57.99	433	12.23	4.74
30-34	262	94.14	743	19.468	4.83
35-39	343	143.81	1129	25.14	5.72
40-44	368	203.20	1335	31.09	6.53
45-49	393	313.39	1666	43.30	7.22
50-54	410	415.82	2135	64.77	6.41
55-59	333	471.67	2145	84.27	5.59
60-64	316	539.25	2406	110.06	4.89
65-69	216	535.9	2836	133.47	4.01
70+	359	509.21	10249	150.95	3.37
Total (Crude)	3251	185.63	25338	62.68	2.96
Age-adjusted#	---	248.70	---	53.54	4.64
Males					
20-24	28	9.93	210	5.90	1.68
25-29	73	26.85	305	8.55	3.14
30-34	133	51.01	481	12.56	4.06
35-39	184	80.98	788	17.35	4.66
40-44	232	139.33	1157	26.77	5.20
45-49	302	236.86	1778	46.57	5.08
50-54	351	336.20	2454	74.30	4.52
55-59	262	340.25	2617	104.95	3.24
60-64	218	371.37	2864	135.63	2.73
65-69	162	390.36	3214	163.72	2.38
70+	217	317.25	8307	184.73	1.71
Total (crude)	2162	128.27	24175	63.62	2.01
Age-adjusted#	---	170.01	---	59.38	2.86
Total (crude)	5413	157.50	49513	63.13	2.34
Age Adjusted#	---	209.70	---	56.06	3.74

*** Ratio of First Nation Prevalence to Non-First Nation Prevalence**
 # Adjusted to the 1991 Canadian Population

Figure 5.1:
Point prevalence of DM in the Manitoba population, aged 20+, standardized to the 1991 Canadian population

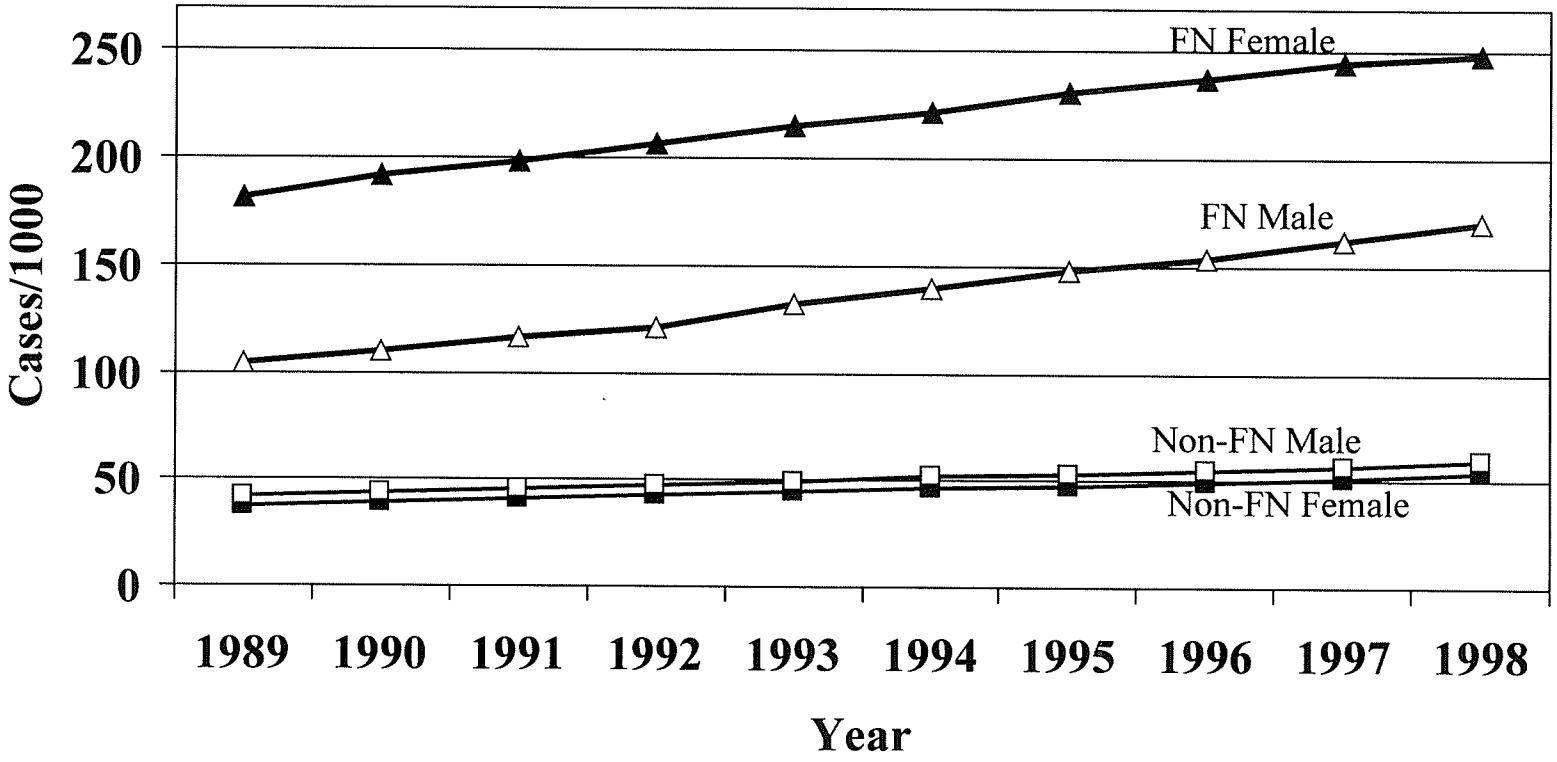
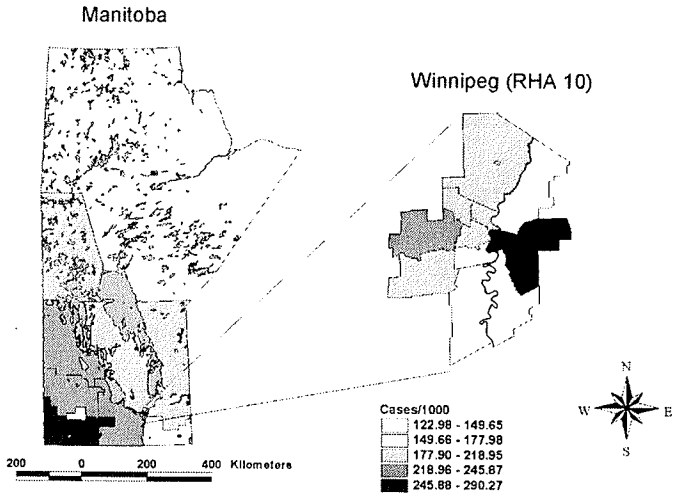


Figure 5.2:
Geographic variation in DM prevalence, First Nation population compared with the non-First Nation population

First Nation DM Average Prevalence Rate, 1994-1998 Standardized to the 1991 Canadian Population



Non-First Nation DM Average Prevalence Rate, 1994-1998 Standardized to the 1991 Canadian Population

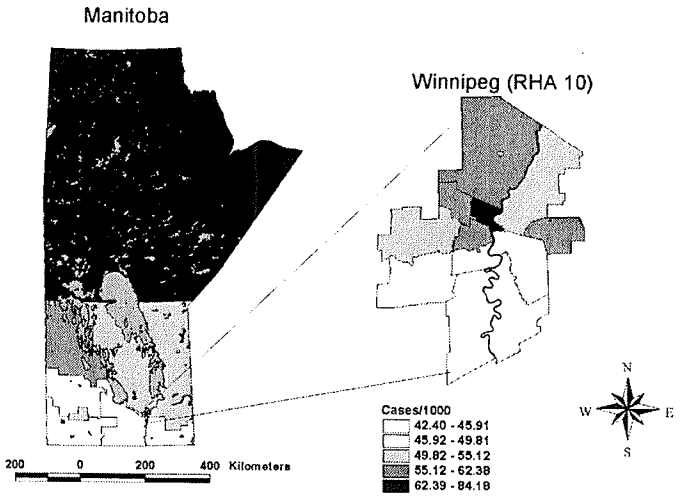
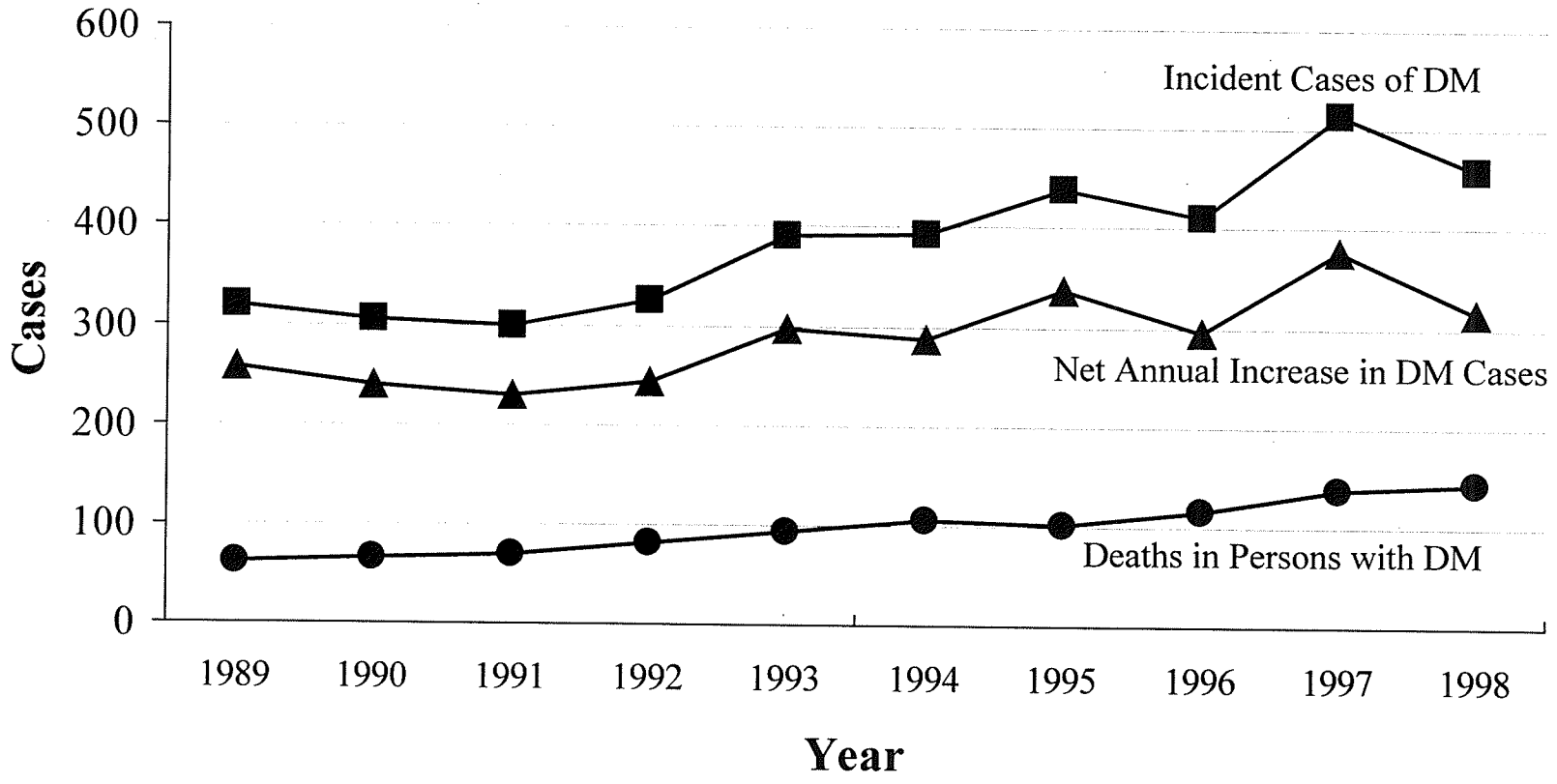


Figure 5.3:
Incident cases of DM, DM related deaths, and net annual increase in DM cases, Registered First Nations, Manitoba, 1989 - 1998



Chapter 6

Visualization, Exploration and Modeling of Diabetes Incidence in Manitoba, 1989-1998

Introduction:

Diabetes Mellitus (DM) is one of the most common non-communicable diseases in the world today. It is projected that the number of cases of DM around the world will increase rapidly over the next 25 years, from 171 million estimated cases in 2000 to 366 million cases by the year 2025 (Wild, Roglic, Green, Sicree & King, 2004). Planning and implementing effective primary and secondary prevention programs to deal with the emerging DM epidemic at the local level requires accurate population based data in order to assess the geographic distribution and concentration of the disease, and to model its individual and population level correlates. This study demonstrates the utility of employing a number of diverse spatial and epidemiological techniques to visualize, explore and model DM incidence, using the province of Manitoba for the years 1989 to 1998 as a case study. The study also illustrates the utility of applying population based health perspectives to the interpretation of study results and to the identification of their program, policy and future research implications. This study builds upon previous work exploring the spatial distribution of DM in Manitoba (Green, Blanchard, Young & Griffith, 2003a; Green, Hoppa, Young & Blanchard, 2003b)

Methods:

Study Setting:

The study was conducted in the western Canadian province of Manitoba. Manitoba has a population of 1.1 million people, more than one half (645,000) reside in the City of Winnipeg, the provincial capital. The majority of Manitobans are of European descent whereas 10% of the population is self-identified as having Aboriginal ancestry. Manitoba has a universal health insurance plan, and all residents of the province are eligible to receive health care services with no payments required at the time of service.

Data Sources:

DM incidence data was obtained for the adult population 20 years and older from the Manitoba Diabetes Database (MDD), which has been previously described (Blanchard, Ludwig, Wajda, Dean, Anderson, Kendal et al. 1996). This database contains a longitudinal record for Manitoba residents of all physician contacts and hospital separation records that cited a diagnosis of diabetes (ICD-9CM code 250) between April 1 1984 and March 31, 1999. Individuals are categorized as having DM if they have had at least two separate physician contacts of diabetes within 2 years of each other or at least one hospital separation for diabetes. Cases of gestational diabetes are excluded from the MDD. The sensitivity of this database for detecting clinically diagnosed cases of diabetes has been demonstrated, and the validity of the methodology has been discussed previously (Blanchard et al., 1996). The methodology is unable to distinguish between Type 2 and Type 1 DM. DM incidence was calculated using the mid-year population at risk based on the Manitoba population registry.

Sociodemographic predictor data obtained from the 1996 Census Canada microdata files were used to estimate small area average family income, educational status, unemployment rates, percentage of parents headed by a single parent, the percentage of the population reporting recent immigrant status, and the percentage of the population reporting Aboriginal ancestry (Statistics Canada, 1996) Adult smoking rate estimates were obtained from the Public Health Branch, Manitoba Health. Registered First Nation (RFN) status and Dene cultural affiliation were obtained from the Manitoba Health population registry. It is important to note that for a variety of complex historical and political reasons many individuals of Aboriginal ancestry are not eligible for registration under the Indian Act of Canada. These individuals are therefore not identified as RFN in this study. Also, only 70% of the RFN population living in the province of Manitoba are identified as being RFN in the Manitoba Health population registry.

Spatial Methods:

Records in the MDD were geocoded to neighborhoods in the City of Winnipeg (n=230) using postal code and to health municipalities in rural areas (n=268) using the Manitoba health municipal code. The resulting set of 498 geographic areas had an average population size of 2000. Data analysis was undertaken in three steps according to the framework for spatial analysis proposed by Bailey & Gatrell (1995).

In the first step, data visualization, directly standardized DM incidence rates for the years 1989 - 1998 combined were estimated for each of the 498 geographic areas used in the study. In order to control for unstable rate estimates resulting from small case counts, rate estimates for DM incidence were generated using an adaptive mean nearest

neighbor smoothing algorithm that was implemented using a program written in Epi Info 6.04d (Centers for Disease Control, 2001). This program utilized a spatial weights table calibrated to the population size of each geographic area and its first, second and third order neighbors. Geographic areas with a population size of 5,000 or more were not smoothed; geographic areas with a population size of less than 5,000 were smoothed by adaptively borrowing both numerator and denominator data from neighboring geographic areas to the degree required to generate a denominator of approximately constant size (5000 persons). Directly standardized rates using the 1991 general Canadian population as the standard were then calculated using the data borrowed in the smoothing process. The Spatial Analyst extension in Arc-View (Environmental Systems Research Institute, 1999) was used to generate a continuous surface isopleth map of DM incidence for Manitoba.

In the second step, data exploration, the local and global Moran's I, the spatial scan statistic, the Gini coefficient, and the population attributable risk (PAR) were calculated in order to summarize and describe the degree of spatial clustering and geographic inequality in DM incidence rates across the province. This step is important to confirm that disease patterns identified through the data visualization stage are real and not due to random spatial variation. By describing the inequality in disease rates across geographic regions, this step can also provide important clues to disease etiology and the potential impact of prevention measures. The Gini coefficient was calculated for two time periods, 1989 - 1993 and 1994 - 1998 to assess whether there was an increase in geographic inequality in DM incidence over the study period.

The local and global Moran's I were calculated with the GeoDa software (Anselin, 2004) using a weights file containing 10 nearest neighbors, with significance testing based upon 999 Monte Carlo simulations. The local Moran's I identifies the location and statistical significance of high and low rate disease clusters and was calculated with the significance filter set at $p<0.01$. The global Moran's I is an overall summary measure of spatial clustering which compares observed rates of disease with rates estimated using the average of disease rates in spatial neighbors. A high Moran's I value indicates strong spatial clustering; a Moran's I value near zero indicates lack of clustering (Anselin, 2000; Anselin, 1995).

The spatial scan statistic, which identifies high and low rate cluster areas through the aggregation of contiguous geographic regions was calculated using the Satscan 2.1 software (Kulldorff, Rand, Gherman, Williams & DeFrancesco, 1998). The software was set to find age adjusted clusters with a maximum size of 50% of the study population, and detected clusters were tested using 999 Monte Carlo simulations. The program assumes a Poisson distribution and calculates indirectly standardized rates (expressed as relative risk - observed divided by expected) for each identified geographic cluster. The specifics of the spatial scan statistic (Kulldorff, Athas, Feurer, Miller & Key, 1998) and its application to the study of spatial variation in DM have been described previously (Green et al., 2003b).

The Gini coefficient, a measure of inequality ranging from 0 (absolute equality) to 1 (absolute inequality), was calculated using the formula proposed by Castillo-Salgado, Schneider, Loyola, Mujica, Roca & Yerg (2001) and was implemented using a program

written for this purpose in Epi Info 6.04d (Center for Disease Control, 2001). The Gini coefficient was calculated from the generated cumulative proportions of DM cases and population at risk. Gini coefficient calculations were double checked using the EPIDAT 3.0 software package (Pan American Health Organization, 2003).

The population attributable risk (PAR) was then calculated using two methods employing the technique described by Alleyne, Castillo-Salgado, Schneider, Loyola & Vidaurre (2002). In the first method, geographic areas in the province were ranked by DM rate and the combined incidence rate in the lowest rate geographic areas containing 10% of the provincial population was calculated. This rate was then applied to the total Manitoba population on an age and gender specific basis. In the second method, the DM incidence rate for the total Manitoba population was calculated and applied to the 10% of the population living in the highest rate geographic areas of the province on an age and gender specific basis.

In the third step, data modeling, the socio-demographic characteristics associated with each of the 498 geographic areas studied were classified into quintiles using the Jenks natural breaks algorithm. These were then assigned to individual records in the database. Using the Poisson regression functionality in the NCSS software (Hintz, 2001), rate ratios adjusted for age and gender were then calculated for each predictor variable. Rate ratios were also calculated for several individual and geographic level predictors including First Nation Status, Dene/non-Dene, on/off reserve, and urban/rural designation. Dene/non-Dene comparisons were undertaken since previous studies have suggested that this population has a significantly lower risk of DM as compared to other First Nation groups

(Young, Szathmary, Evers & Wheatley, 1990). Confidence intervals were corrected for over-dispersion of the variance using the dispersion phi multiplier function in the NCSS software. Poisson regression is considered to be an appropriate statistical approach to use for analyzing public health data when disease incidence is less than 10% (Frome & Checkoway, 1985).

Results:

The age adjusted incidence of DM increased from 53.9 cases/10,000 in 1989 to 69.1 cases/10,000 in 1998. The average incidence rate during the study period was 56.1 cases/10,000. In 1989 there were 4,129 incident cases of DM in Manitoba which increased to 5,299 cases by 1998. There were 43,955 incident DM cases during the study period.

Data Visualization:

The continuous surface isopleth map of DM incidence (Figure 6.1) shows that there is marked geographic variability in the rate of DM incidence across the province of Manitoba, with DM incidence rates ranging from 29 to 239 cases per 10,000 population at risk. The highest rates of DM can be observed in the northern regions of the province. High rates can also be observed in individual First Nation communities scattered throughout both the northern and southern regions of Manitoba. Slightly elevated rates are also visible in the central core of the City of Winnipeg.

Data Exploration:

The local Moran's I, often referred to as the LISA statistic (local indicator of

spatial association), identified a large high rate cluster of DM incidence in the northern region of Manitoba and several low rate clusters of DM incidence in southwestern Manitoba and in geographic regions surrounding and extending into the City of Winnipeg (Figure 6.2). High rate cluster areas are red in color, low rate cluster areas are blue in color, and non-clustered areas are non-colored. Geographic areas are considered to be clustered if they are surrounded by geographic neighbors having similar values, with the congregation of similar values assessed through a Monte Carlo simulation as being unlikely to have occurred by chance alone. The Global Moran's I of 0.4031 for DM incidence generated in this study was significant at the $p<0.001$ level, indicating a high level of overall clustering of DM incidence rates. The Moran's I scatterplot (Figure 6.2) illustrates the regression slope of observed DM incidence rates vs. estimated DM incidence rates which were calculated using information from the 10 nearest geographic neighbors (values are plotted in standard deviations). The upwardly rising regression line shows that the observed DM incidence rates (X axis) are positively correlated with rates of DM estimated using information from geographic neighbors alone (Y axis).

The spatial scan statistic identified a high rate cluster of DM incidence in northern Manitoba (relative risk of 2.07, $p< .01$) and in the central core of the City of Winnipeg (relative risk of 1.348, $p<0.05$), as well as in several First Nation communities in southeastern and southwestern Manitoba (Figure 6.3). Low rate clusters were identified in southern Manitoba in geographic areas extending into the City of Winnipeg, with relative risks of 0.846 ($p< 0.05$) and 0.815 ($p<0.05$).

The identification of statistically significant high and low rate clusters of DM

incidence using the local Moran's I and the spatial scan statistic confirms that the spatial patterns of DM incidence observed in the data visualization stage of analysis are real and unlikely to have occurred by chance alone.

The Gini coefficients for 1989-1993 and 1994-1998 time periods were 0.13 and 0.14 respectively, indicating a relatively low and stable level of geographic inequality in DM incidence rates over the study period. The associated Lorenz curves used to calculate the Gini coefficients (Figure 6.4) plot the cumulative proportion of DM incident cases against the cumulative proportion of the population at risk. These charts show that as the cumulative proportion of the population increases (X axis), the cumulative proportion of DM cases (Y axis) fails to keeps step, resulting in a deflection of the Lorenz curve downward from the 45 degree axis. If the DM cases were equally distributed among geographic areas in Manitoba in proportion to the population at risk, the Lorenz curve would follow the 45 degree line exactly. The relatively minor deflection of the Lorenz curve downwards from the 45 degree diagonal line confirms that DM incident cases in both time periods are not highly spatially concentrated in a small number of geographic areas in Manitoba; rather DM cases are spread out quite equally relative to the underlying population at risk. Reading directly from the Lorenz curves, it can be calculated that the highest rate geographic areas for DM incidence which contain 10% of the population include only 15.6% and 16.4% of the incident DM cases respectively in the 1989-1993 and 1994-1998 time periods.

The population attributable risk calculated by applying the DM incident rates observed in the 10% of the population living in the lowest rate geographic areas of the

'province to the total Manitoba population was 33.8 %. This indicates that if a hypothetical prevention program had been put in place which resulted in all geographic areas having the same DM incidence rate as the rate observed in the lowest 10% of the population, there would be 33.8% fewer DM cases (n=14,871) occurring in Manitoba during the 1989 - 1998 study period.

The population attributed risk calculated by applying the DM incidence rate for Manitoba as a whole to the 10% of the population living in the highest rate geographic areas for DM incidence was 7.9%. This indicates that if a hypothetical prevention program was put in place which resulted in the highest risk areas for DM containing 10% of the population at risk having the same DM incidence rate as that observed in the province as a whole, there would be 7.9% fewer DM cases (n=3505) occurring in Manitoba during the 1989 - 1998 study period.

Data Modeling:

Poisson regression analysis shows that DM incidence varies significantly by a range of individual, geographic and socio-demographic factors. Table 6.1 express the outputs of the poisson regression analysis in terms of rate ratios. A rate ratio is the ratio of the incidence rate in the category of interest compared to the rate in the reference or comparison category. As indicated, rate ratios have been adjusted for both age and gender where appropriate.

At the geographic level, the DM incidence rate was observed to be 8% higher in rural Manitoba than in Winnipeg, and 4.12 times higher in First Nation reserve communities than in non-reserve communities across the province.

At the individual level, DM incidence was observed to rise steadily with age with no significant difference observed between males and females. DM incidence was observed to be 4.25 times higher in Registered First Nation (RFN) individuals than in the non-RFN population. When the regression model was further adjusted for average family income, less than grade 9 education and unemployment, the rate ratio for RFN status decreased to 3.45, suggesting that the relationship between RFN status and DM incidence is mediated by socio-economic status. Individuals living in Dene communities in the far north of Manitoba were observed to have a DM incidence rate only 28% that of the general non-Dene Manitoba population and only 8% that of the non-Dene RFN population.

DM prevalence was also observed to be strongly and consistently graded by measures of socio-economic status. Adjusting only for age and sex, DM incidence was observed to be the highest in geographic areas with the greatest percentage of the population self-reporting Aboriginal status(rate ratio=4.35, $p<0.05$), the lowest average family income (rate ratio=2.97, $p<0.05$), the lowest levels of educational achievement (rate ratio=2.64 $p<0.05$), the highest percentage of lone parent families(rate ratio=1.78, $p<0.05$),the highest rate of unemployment (rate ratio=3.24, $p<0.05$), and the highest rates of adult smoking (rate ratio=2.42, $p<0.05$). The percentage of the population reporting landed immigrant status was not consistently related to DM incidence, although the highest quintile for this predictor had a significant rate ratio of 1.18 ($p<0.05$). As with the RFN population, when the regression model was further adjusted for socioeconomic status, the rate ratio for self-reported Aboriginal status decreased to 3.11 from 4.35.

Discussion:

Spatial Structure:

This study has analyzed the spatial variability of DM incidence in Manitoba using the three step process of data visualization, data exploration and data modeling proposed by Bailey and Gatrell (1995). It has shown through the production of a continuous surface isopleth map of DM incidence that there is a pronounced geographic concentration of DM incidence in Northern Manitoba, in First Nation communities throughout the province, and to some degree in the core area of the City of Winnipeg. The highly significant Moran's I test for spatial clustering and the spatial scan statistic both suggest that these observed spatial patterns are real and unlikely to have occurred by chance alone. The existence of true spatial patterns in DM incidence is further confirmed by the observation of a strong and significant grading of DM incidence by a number of geographic, individual and socio-demographic predictor variables.

Public Health Significance of Spatial Equality of DM Incidence:

Paradoxically, the pronounced variation in DM rates across geographic areas observed in this study may have less direct public health significance than first appearances would suggest The Gini coefficient for DM incidence was observed to be very low, suggesting that although there is broad variation in DM rates across geographic areas, there is overall, a relatively equal distribution of DM cases across Manitoba geographies in relationship to the population at risk. In other words, the low Gini coefficient suggests that it is not the case that the majority of DM cases are highly concentrated in a small number of geographic areas of the province. It is insightful to note in comparison that

studies of sexually transmitted diseases and West Nile Virus in Manitoba have reported Gini coefficients of 0.4 (Elliott, Blanchard, Beaudoin, Green, Nowicki, Matusko et al. 2002) and 0.83 (Green, 2004) respectively, indicating that in contrast to DM, these diseases are quite highly concentrated in a small number of geographic areas.

The low Gini coefficient has two major implications. First, from an etiological perspective, it suggests that the major "causes" or "driving forces" behind the DM epidemic are themselves not spatially concentrated in particular micro-environments which affect only a small sub-set of the population (as would for example be the case in localized water contamination causing an outbreak of disease); rather the low Gini coefficient suggests that the major forces driving the DM epidemic are insidious, distributed widely, and permeate all levels of Manitoba society. Although the variability in DM rates that does exist within Manitoba can be successfully modeled against a number of individual and ecological level predictors, the low Gini coefficient suggests that there are probably larger and more important driving forces affecting DM rates in the Manitoba population.[1]

As suggested by Rose (1985,1992), the broad causes of population health are often very difficult to demonstrate through traditional epidemiological analyses when these causes are distributed homogeneously throughout the population. He argues these forces

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This is not to suggest that previous studies such as the recent paper by Green et. al. (2003b) which have focused on modeling the relationship between socio-economic status and DM incidence/prevalence are mis-directed or wrong; rather that these studies may be blind to the effects of long-term forces acting on the population as a whole. If viewed over a long time period, the impact of these long-term forces may dwarf the effects of socio-economic status on rates of DM. See further discussion by Rose in the following paragraph.

can only be rendered visible through comparison of the study population to earlier historical epochs (when things were very different) or through comparison to populations having very different social, economic, cultural and ecological characteristics. This would suggest that in order to comprehensively understand the large forces which are driving the DM epidemic in Manitoba, it would be critical to examine long-term historical trends in DM, and how these long-term historical trends are connected in time and space with macro level changes in factors such as the structure of the food system, the urban and rural landscapes, medical practice, and personal lifestyles. These factors have been suggested as being critically important in affecting population rates of both obesity and DM (2002; American Dietetic Association, 1998; CIHR, 2003; Frank, Engelke & Schmid, 2003; Nestle & Jacobson, 2000; World Health Organization, 2000; Young & Nestle, 2002).

Secondly, the low Gini coefficient observed in this study brings into question the implementation of prevention programs for DM which focus exclusively on geographically defined high risk groups. If the objective of public health prevention efforts is to reduce the overall disease burden in a population, there may be little to be gained from implementing a high risk approach to preventing disease when high risk groups for the disease are small in size. Although the disease rates may be dramatically higher in groups considered at high risk, the small population size of the high risk groups means that these groups contribute very few cases to the overall population incidence of the disease. Programs focusing on high risk groups, even if they were 100% successful, would do little to diminish the overall population rate of the disease. The corollary is that prevention

programs focusing on the general population might achieve greater impact on diminishing disease rates. In this study, the low Gini coefficient suggests that high rate geographical areas for DM in Manitoba do not contain a large number of persons with DM and that high risk prevention programs would have a marginal impact on the overall population rate of DM. This is confirmed by population attributable risk calculations reported in this study which suggest that hypothetical prevention efforts focusing only on the 10% of the population living in the highest risk geographic areas of the province would have the potential to decrease the incidence of DM by only 7.9%, while prevention efforts focusing on the population as a whole would perform much better and would have the potential to decrease DM rates by a maximum of 33.8%.

Thirdly, the observation that the Gini coefficient has not increased significantly over the study period suggests that the causes of DM incidence are well established and are not becoming more concentrated in high risk populations over time.

Grading of DM by Sociodemographic Factors:

Despite the low levels of spatial inequality observed in this study, incidence rates of DM were observed to model tightly against a number of socio-demographic predictor variables. The observation that DM incidence rates were significantly higher in the RFN population and in populations living in geographic areas with lower socio-economic status is consistent with a number of previous studies (Auslander W F, Haire-Joshu, Houston & Fisher E B, 1992; Blanchard, Green & Wajda, 1998; Connolly, Unwin, Sherriff, Bilous & Kelly, 2000; Evans, Newton, Ruta, MacDonald & Morris, 2000; Gardner, Jr., Stern, Haffner, Gaskill, Hazuda, Relethford et al. 1984; Green et al., 2003a;

Green et al., 2003b; Hazuda & Monterrosa, 1992; Hendricks & Haas, 1991; Leonetti, Tsunehara, Wahl & Fujimoto, 1992; Manuel & Schultz, 2002; Young et al., 1990). In this study, rates of DM were observed to be significantly higher in populations with low levels of education, low family incomes, high rates of unemployment, high rates of lone parent families and high rates of smoking. The relationship of these factors to elevated rates of DM is consistent with the explanatory paradigm for the development of DM which suggests that persons lower on the social hierarchy have a restricted set of life choices and as result have fewer opportunities for self-care, adoption of healthy lifestyles, suffer higher rates of stress, and thereby experience higher rates of chronic disease such as DM (Brown, Ettner, Piette, Weinberger, Gregg, Shapiro et al. 2004; Krieger & Davey, 2004).

The higher rates of DM in the First Nation and Aboriginal populations observed in this study are consistent with the conclusions of previous studies which have suggested that in general (with some exceptions) Aboriginal people have a higher a rate of DM because of a genetic predisposition to the disease (Bogardus, Lillioja, Nyomba, Freymond, Zurlo, Swinburn et al. 1988; Haffner, 1998; Neel, 1999; Neel, 1962; Neel, 1982; Stern, Mitchell, Blangero, Reinhart, Krammerer, Harrison et al. 1996; Williams, Long, Hanson, Sievers & Knowler, 2000). However, the mediation of the relationship between DM and Aboriginality by socio-economic status observed in this study suggests that in part the higher rate of DM in Aboriginal populations is due to the effects of low socio-economic status as noted above.

Strikingly, this study has demonstrated that persons living in Dene communities in the northern parts of Manitoba have dramatically lower incidence rates of DM as

compared to the general population of Manitoba and the non-Dene First Nations population. This observation is consistent with previous observations made by Young et al. (1990), who suggested that lower DM rates in the Dene may be the result of both the unique genetic resiliency of this Aboriginal group and the retention of a more traditional lifestyle protective against the development of DM.

Study Limitations:

This study has a number of methodological limitations that must be kept in mind when interpreting its results. First, the data modeling component of the study is based upon an ecological design. This means that the measures of socio-demographic status that were used were measured at the geographical level and cannot be considered direct properties of individuals. This design was adopted since the socio-demographic data required for the study (other than age, sex, and First Nation status) were simply not available on the individual health record. The ecological design has been frequently criticized as a weak design and commits what is referred to as the ecological fallacy (Macintyre & Ellaway, 2000; Morgenstein, 1982; Morgenstein, 1995). The ecological fallacy suggests that it is a mistake to apply characteristics measured at the scale of the population or small geographic area to individuals living within those areas. The ecological design used in this study therefore restricts us somewhat to making statements about the characteristics of the populations rather than individuals. However, given the recent work by Rose (1985, 1992), Kawchi, Kennedy & Wilkinson (1999), Krieger, Chen, Waterman, Soobader, Subramanian & Carson (2002), Krieger, Chen, Waterman, Soobader, Subramanian & Carson (2003), Krieger, Waterman, Chen, Soobader &

Subramanian, (2003), and Mustard, Derksen, Berthelot & Wolfson (1999), which highlight the important and independent role that population level factors have on the health of individuals, and of the frequent convergence between socio-demographic factors measured at a small area level and the same factors measured at the individual level, the powerful grading of DM by socio-economic status observed in this study should be taken seriously. It is important to note that in this study the estimates of socio-demographic status that were applied to individual cases of DM were generated from a set of spatial units with an average population size of only 2000 persons, minimizing the possibility of significant heterogeneity within those geographic areas biasing individual level sociodemographic estimates. The strength and validity of the ecological approach used in this study is confirmed by the observation that the individual and ecological measures of Aboriginal status generated similar rate ratios for DM incidence (4.25 vs. 4.35).

Secondly, DM incidence rate estimates were generated from data derived from administrative databases. Since cases of DM were not individually verified, this approach could result in either an over-estimate or under-estimate of DM incidence rates. However, previous studies have studied the accuracy of this approach and found the specificity is high when compared to local registries of DM (Blanchard et al., 1996). It is possible that some of the small area variations that were observed in this study could be due to variability in health care access and diagnostic variability. This is quite possible in the more northern and remote areas of the province of Manitoba where health services are more difficult to deliver comprehensively. In these areas the problem could be exacerbated by the increasing use of "dummy billing" where physicians are being paid on

a per diem basis. In this situation, physicians are not financially penalized for failing to record and submit patient level diagnostic information for inclusion into provincial administrative databases.

Thirdly, the administrative databases from which the DM incidence rates were derived cannot distinguish between Type 1 insulin dependent and Type 2 adult onset DM. However, given that it is estimated that approximately 95% of all DM cases are Type 2 it is likely that the variability in DM incidence reported in this study reflect primarily the impact of Type 2 DM (Harris, 1995).

Fourthly, the statistical properties of the Gini Coefficient statistic that was used in this study as a measure of spatial inequality have not been well established (Lee, 1997). This makes its use in formally comparing trends in spatial inequality over time and space difficult. However, in its current implementation, the Gini coefficient does appear to be a powerful tool to initially explore and communicate the implications of spatial inequality for disease etiology and public health interventions (Alleyne et al., 2002; Castillo-Salgado et al., 2001).

Fifthly, the data modeling that was undertaken in this study generated only global measures of association. It is unlikely that the dynamics of the DM epidemic in Manitoba are the same across all regions and global measures may tend to "gloss over" and render invisible local heterogeneities of significant importance. Techniques such as the multi-variate LISA statistic (Anselin, 2004; Anselin, 1995) and geographically weighted regression (Fotheringham, Brundson & Charlton, 2003), with applicable software for their calculation have only recently become available. The use of local measures of association

warrant application to the study of DM in Manitoba, but are beyond the scope of the current study.

Sixthly, this study has not visualized, explored and modeled the incidence of DM separately for the RFN and non-RFN populations. This is because the very small size and often sparse distribution of the RFN population in Manitoba makes it difficult to develop the stable small area rate estimates required for data visualization and exploration. As well, using the ecological characteristics pertaining to the total population to model DM incidence in a population subset such as the RFN population is problematic.

Finally, the Moran's I statistic (global and local) generated in this study as a measure of spatial clustering may be biased since smoothed rate estimates for DM incidence were used as inputs into its calculation. The smoothing process itself can generate significant spatial auto-correlation in the data which can inflate the value of the Moran's I. However the inflation of the Moran's I statistic generated in this study was likely quite minimal since only a very moderate amount of spatial smoothing was used to stabilize small area incidence rates. As well, the pattern of clusters identified by the local Moran's I (LISA) was very similar to the pattern of clusters identified using the spatial scan statistic, suggesting that the Moran's I statistics were not greatly biased by smoothed rate inputs.

Policy and Program Implications:

This study has three important implications for the development of policies, programs, and future research activities that are required to effectively respond to the emerging DM epidemic in Manitoba.

First, this study suggests that program and policy directions should never be based upon an analysis of a simple map depicting high and low rate disease areas. These maps seductively suggest that there may be high rate areas for disease, and these geographic areas are where prevention programs should be focused. However, as illustrated by this study, it is important to formally assess the implications of the degree to which disease cases are equally distributed. If it is the case, as was observed in this study, that the disease in question has very few cases located in high risk geographic areas (despite the visual dominance of these high risk areas on isopleth maps), then prevention programs focusing their efforts only on high risk areas may be based upon an overly optimistic estimate of the potential of those programs to reduce the incidence of the disease in the population. In this circumstance, it may be justifiable to focus prevention efforts on geographically defined populations at highest risk for the disease. However, the objectives of the prevention program would have to be aimed at issues of social equity (e.g. reducing the burden of illness in the most vulnerable populations), but not on significantly reducing the overall population incidence of the disease.

Secondly, this study suggests that despite a clear grading of DM incidence and prevalence by socio-economic status, the major "causes" of the DM epidemic appear to be much larger than this. The very low level of spatial inequality in DM incidence in Manitoba suggests that the causes of DM are everywhere and located in the fundamental structure of how we feed, transport, and medically treat ourselves. This is consistent with the globalization paradigm proposed by Ritzer (2003) and Sobal (2001) which suggests that global forces of the late 20th century/early 21st century are leading to a

homogenization of ways of life around the world (urbanization, fast food, sedentary life style) and that this is resulting in a rapid proliferation of chronic diseases such as DM. Over time, these forces of globalization are putting all members of society at risk for DM, but more so individuals of low socio-economic since they have fewer capacities and resources to deal with the deleterious effects of global forces. This perspective suggests that programs and policies attempting to deal with the emerging DM epidemic must move beyond genetic, lifestyle, and socio-demographic explanations for the disease and embrace an explanatory paradigm which critically acknowledges the ways in which the determinants of health in local geographic areas are becoming increasingly structured by global forces. This would suggest that preventative interventions which focus on either high risk individuals or populations will have a low chance of success unless they are supported by national and international level initiatives which address the ways in which global forces are increasingly able to structure local landscapes in ways that lead to negative population health outcomes.

Thirdly, this study challenges the use of space and place as simple spatial containers in epidemiological research. The results of this study suggest that while the root causes of the DM epidemic are globally derived, their effects vary locally as a function of socio-economic status and ethnicity. This indicates that uncritically aggregating numerator and denominator data to geographic areas for the purposes of epidemiological analysis may ignore how the characteristics of local places and the health of the people who live there are shaped in complex and dialectical ways by the impacts, interactions, and historical trajectories of local and global forces (Crutchfield, Farmer &

Packard, 1986; Gesler & Kearns, 2002; Goodman & Leatherman, 2001; Hayes, Foster & Foster, 1994; Kearns, 1993; Kearns, 1994; Kearns & Joseph, 1993; Kearns & Gesler, 1998; Massey, 1993). In order to understand what is causing variability in DM rates across space, diverse historical, political, economic and ethnographic methods need to be employed (Waltner-Toews, 2004). Robust policy and program development for DM prevention urgently requires information from this type of diverse research approach in order to develop effective interventions. This research strategy would allow the identification of the local forces protective against DM, the ways in which global forces become embedded at the local level, and the types and scales of interventions which may be required simultaneously at individual, local, regional, national and even international levels to deal effectively with the emerging DM epidemic.

Table 6.1:
Poisson regression analysis, DM incidence Manitoba, 1989-1998 combined

Predictors	Rate Ratio	95% CI		Predictors	Rate Ratio	95% CI	
		Upper	Lower			Upper	Lower
Urban				*** Avg. Family Income**			
Urban	1.0	n/a	n/a	1. 21,137 - 31.543	**2.97**	**2.83**	**3.11**
Rural	**1.08**	**1.06**	**1.10**	2. 31,544 - 42,568	1.46	1.4	1.53
Reserve				3. 42,569 - 53,790	1.27	1.22	1.33
Off Reserve	1.0	n/a	n/a	4. 53,790 - 70,818	1.19	1.13	1.24
On-Reserve	**4.12**	**3.96**	**4.28**	5. 70,818 - 108,715	1.0	n/a	n/a
Gender				*** Less than Grade 9**			
Male	1.0	n/a	n/a	1. 1.34 - 7.2	1.0	n/a	n/a
Female	**0.97**	**0.95**	**0.98**	2. 7.3 - 12.32	1.2	1.16	1.23
Agegroup				3. 12.33 - 18.87	1.19	1.16	1.22
20-39	1.0	n/a	n/a	4. 18.88 - 27.58	1.36	1.32	1.4
40-59	3.71	3.6	3.82	5. 27.59 - 38.45	**2.64**	**2.53**	**2.75**
60-69	7.07	6.84	7.3	***Single Parent Families**			
70plus	**7.49**	**7.26**	**7.72**	1. 3.52 - 9.48	1.0	n/a	n/a
*** RFN Status**				2. 9.49 - 14.82	1.07	1.04	1.1
Non-Status	1.0	n/a	n/a	3. 14.83 - 20.60	1.3	1.26	1.33
Status	**4.25**	**4.10**	**4.39**	4. 20.61 - 30.21	1.53	1.49	1.57
***# RFN Status (Adj. by SES)**				5. 30.21 - 42.85	**1.78**	**1.69**	**1.86**
Non-Status	1.0	n/a	n/a	*** Unemployment Rate**			
Status	**3.45**	**3.23**	**3.70**	1. 1.09 - 5.46	1.0	n/a	n/a
*** Aboriginal Status**				2. 5.47 - 8.84	1.15	1.12	1.18
1. 0.22 - 6.3	1.0	n/a	n/a	3. 8.85 - 15.0	1.28	1.24	1.31
2. 6.4 - 18.16	1.15	1.13	1.18	4. 15.0 - 23.66	1.93	1.86	2
3. 18.17 - 37.70	1.48	1.43	1.52	5. 23.66 - 38.4	**3.24**	**3.12**	**3.37**
4. 37.71 - 73.53	1.83	1.73	1.93	*** Smoking Rate**			
5. 73.54 - 99.14	**4.35**	**4.18**	**4.52**	1. 3.45 - 14.45	1.0	n/a	n/a
***# Aboriginal Status(Adj. by SES)**				2. 14.46 - 21.56	1.14	1.07	1.22
1. 0.22 - 6.3	1.0	n/a	n/a	3. 21.57 - 30.48	1.25	1.18	1.34
2. 6.4 - 18.16	1.03	0.98	1.08	4. 30.49 - 45.66	1.4	1.31	1.5
3. 18.17 - 37.70	1.13	1.05	1.23	5. 45.67 - 73.61	**2.42**	**2.25**	**2.59**
4. 37.71 - 73.53	1.43	1.28	1.61	*** Immigrants**			
5. 73.54 - 99.14	**3.11**	**2.72**	**3.55**	1. 0 - 15.97	1.0	n/a	n/a
*** Dene**				2. 15.98 - 40.64	0.81	0.77	0.85
Non-Dene Manitoba Populatio	1.0	n/a	n/a	3. 40.65 - 77.42	0.8	0.75	0.84
Dene	**0.28**	**0.12**	**0.67**	4. 77.43 - 133.82	1.02	0.95	1.09
***Dene**				5. 133.83 - 252.49	**1.18**	**1.09**	**1.28**
Non-Dene RFN Population	1.0	n/a	n/a				
Dene	**0.08**	**0.03**	**0.19**				

* Adjusted by Age and Gender

Adjusted by Average Family Income, Less than Grade 9, Unemployment Rate

RFN Status - Registered First Nation Status (individual level); Aboriginal Status - % of the population reporting Aboriginal status; Immigrants - persons/1000 receiving landed immigrant status between 1980 and 1996; Average Family Income - average family income; Single Parent Families - % of famlies reporting being headed by a lone-parent; Unemployment Rate - % of the population 15+ in the labour force that is unemployed; Smoking Rate - estimated percentage of the adult population who smoke; Less than Grade 9 - % of the 15+ population with less than grade 9;

Figure 6.1:
DM incidence, Manitoba, 1989 - 1998, smoothed, age standardized to the 1991 Canadian population

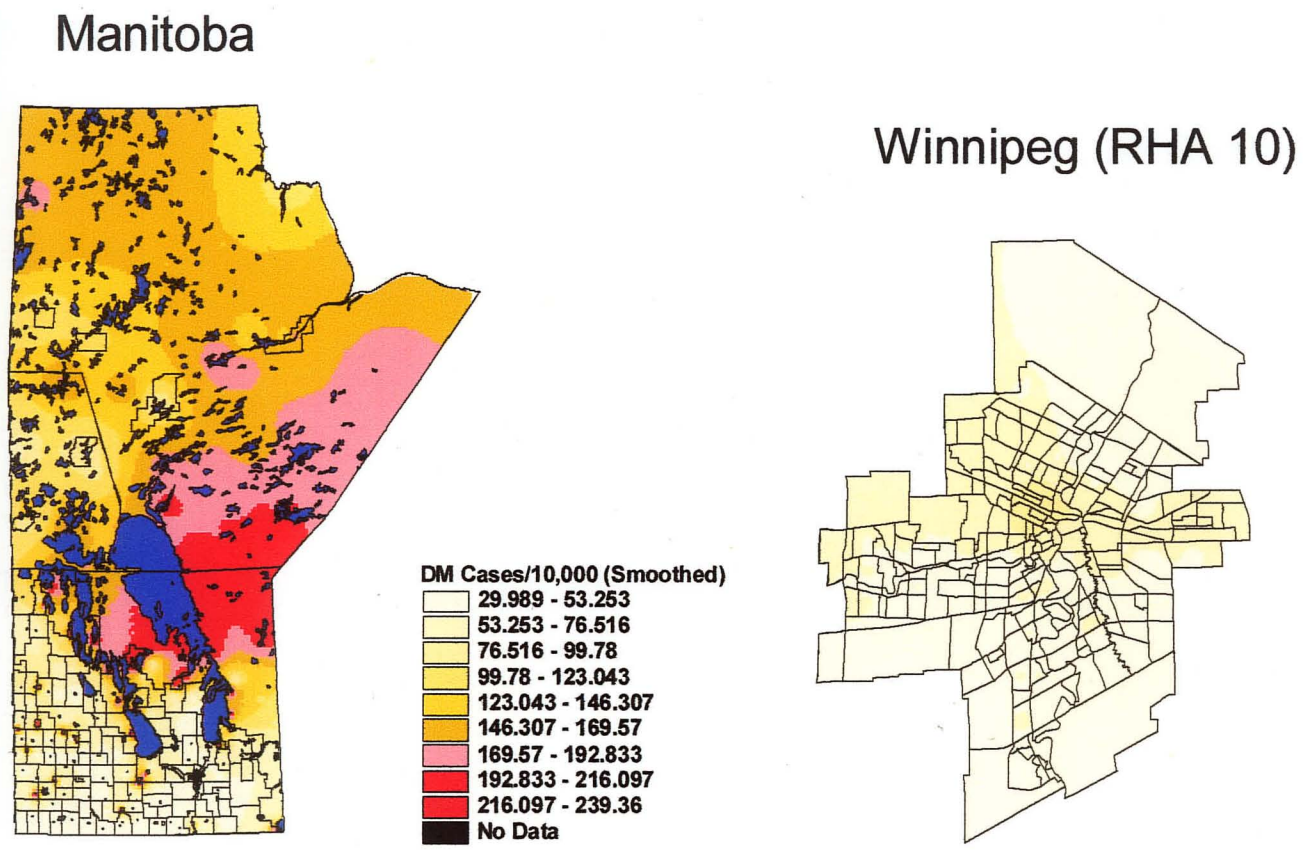
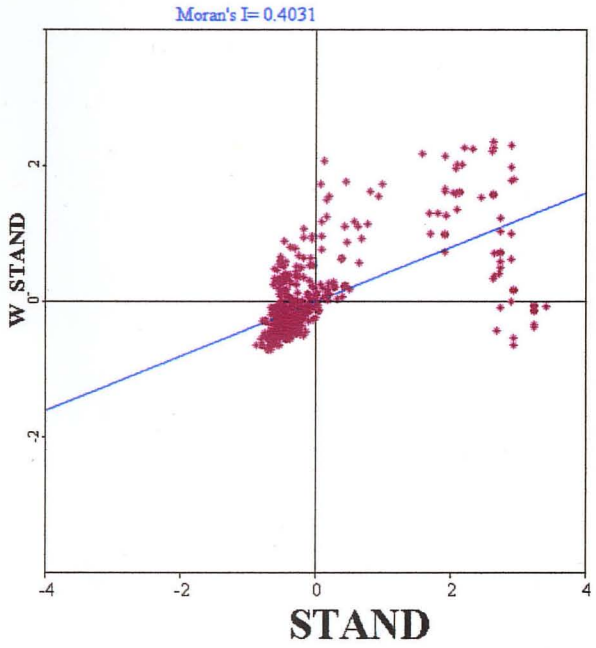


Figure 6.2:
Global and local Moran's I analysis, DM incidence, Manitoba, 1989-1998

Moran's I scatterplot



Local indicator of spatial association (LISA) map significance filter (p<.01)

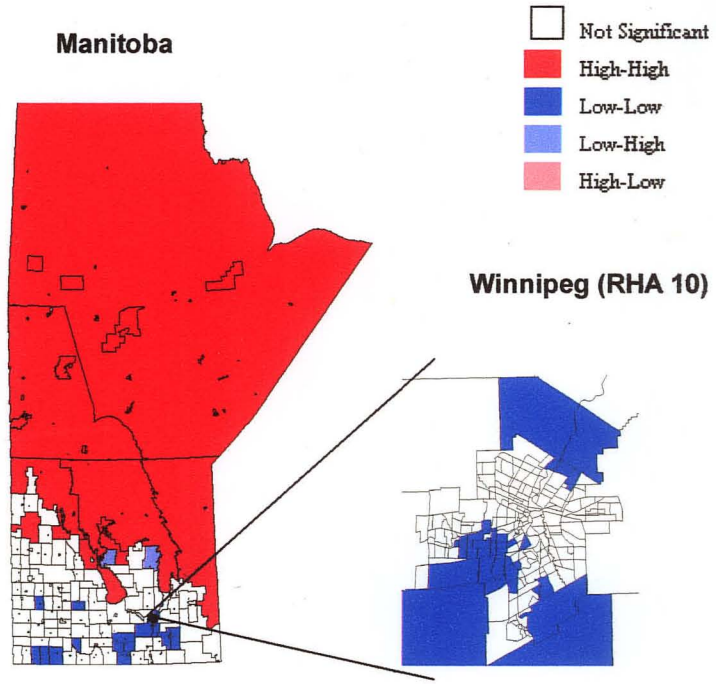


Figure 6.3:
DM incidence analysis, Manitoba, 1989-1998 using the spatial scan statistic, maximum cluster set at 50% of the study population

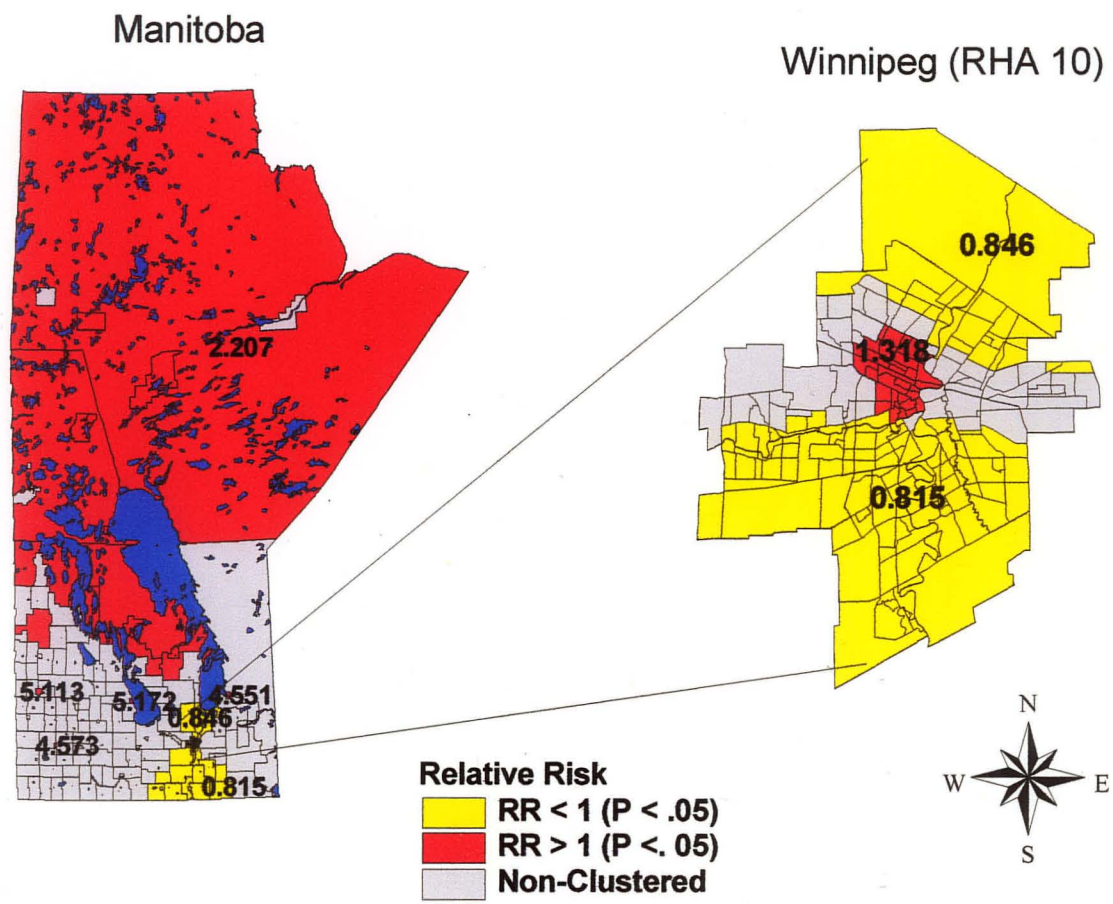
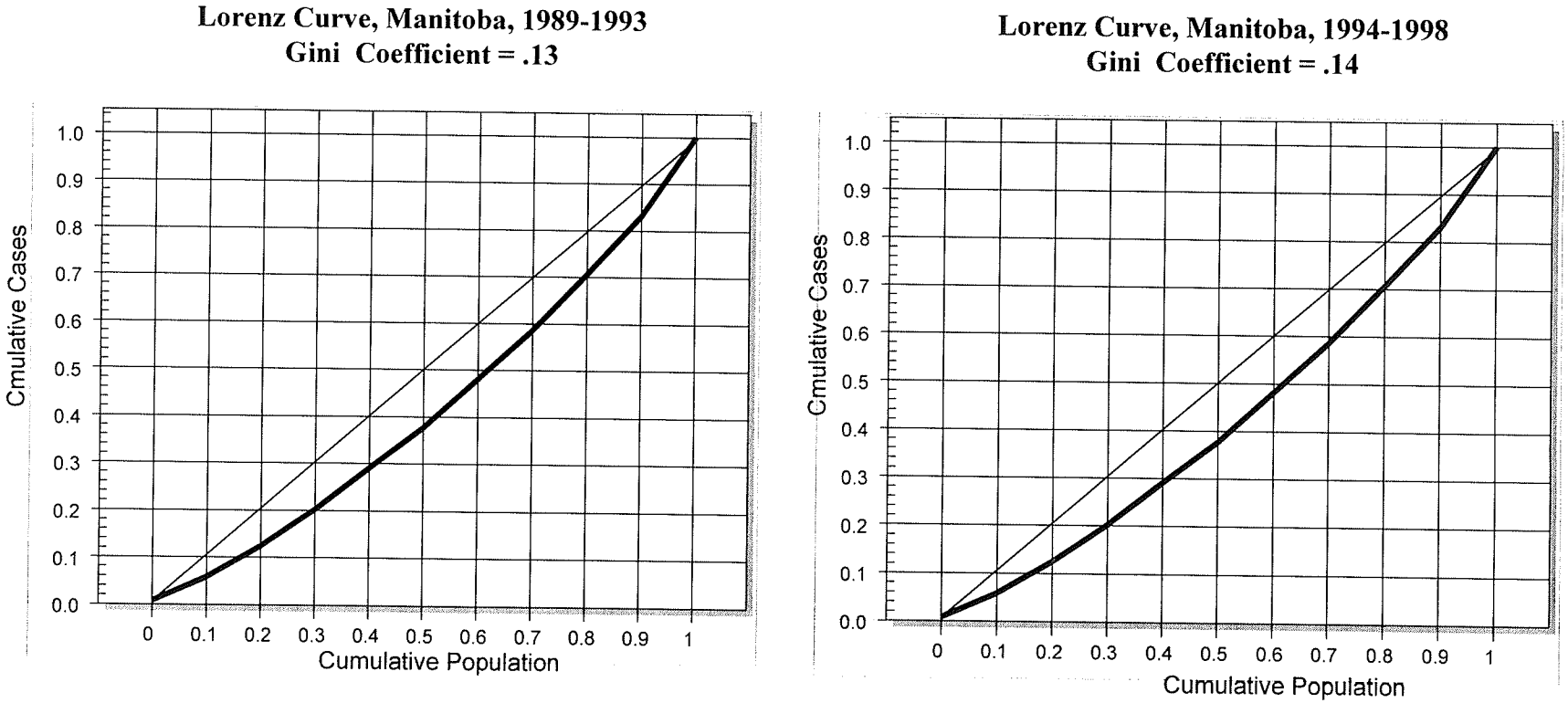


Figure 6.4:
Gini coefficient, DM incidence, Manitoba, 1989-1993 compared to 1994-1998



Chapter 7

100 Years of Diabetes: Re-Tracing and Predicting an Epidemic

1. Introduction:

Background:

Diabetes Mellitus (DM) is one of the most common non-communicable diseases in the world today and appears to be increasing rapidly in both the developed and developing countries (Amos, McCarty & Zimmet, 1997). A number of studies have been published over the past decade which have projected the future prevalence of DM (Amos et al., 1997; Bagust, Hopkinson, Maslove & Currie, 2002; Blanchard, Green & Wajda, 1998; Boyle, Honeycutt, Narayan, Hoerger, Geiss, Chen et al. 2001; Green, Blanchard, Wajda, Depew, Cooke, Brazeau et al. 1997; Helms, 1992; King, Aubert & Herman, 1998; Ruwaard, Hoogenveen, Verkleij, Kromhout, Casparie & van der Veen, 1993; Wild, Roglic, Green, Sicree & King, 2004). All of these studies suggest that DM prevalence will continue to rapidly increase in the foreseeable future. Wild et al. (2004) for example, suggests that between the years 2000 and 2025 the number of DM cases world-wide will more than double from 171 million to 366 million cases. In Canada, there are currently estimated to be over 1.2 million people living with DM (Center for Chronic Disease Prevention and Control, 2003).

Most studies to date which have undertaken DM projections have used the relatively simple technique of applying the age and sex specific rates of DM in the base year of the projection to a future population structure in order to generate estimates of

the number of future DM cases. This type of simple projection approach has two primary short-comings. First, it assumes a constant age and gender specific DM prevalence rate over the projection period. This could lead to a significant underestimation of future DM cases, especially if DM prevalence rates are increasing rapidly over time. Secondly, the simple age standardization approach does not allow base inputs to be modeled independently and dynamically. This makes it difficult to create a variety of future projection scenarios based upon varying assumptions about trends in the base inputs used in the model.

Ruwaard et al. (1993) for example, compared the use of the simple age standardization technique for DM projections to a more complex component cohort modeling approach in which individual demographic components driving future DM prevalence were modeled dynamically from 1980 to 2005. These components included baseline DM prevalence, trends in DM incidence, and trends in DM related mortality. Baseline DM prevalence, incidence, and mortality data were obtained from a Dutch sentinel network of general practitioners. Ruwaard concluded that the traditional age and sex standardization approach significantly underestimated future DM prevalence as compared to the more dynamic component cohort modeling approach by 30% - 40% depending upon the configuration of model inputs.

In order to ensure that policies and programs targeting the emerging DM epidemic are informed by a realistic appreciation of what the future may hold, it is critical that projections of future DM prevalence be undertaken using a dynamic forecasting methodology which allows base inputs to be modeled independently. This will help ensure

that policy and program decisions are critically informed by a number of "what if" scenarios which play out varying assumptions about both the impacts of prevention programs and the effects of wider social forces and factors on future DM prevalence.

Current Study:

This study uses a dynamic component cohort projection model to back-cast and forecast the incidence and prevalence of DM in Manitoba from 1950 to 2050. In order to model the trajectory of DM over the past 50 years, the model was iteratively seeded with base inputs and run forward from 1950 to 1998. These inputs included population estimates from 1950 to 1998, estimated DM incidence, prevalence and DM related mortality in 1950 and their trends to 1998. This resulted in a historical model of DM that was consistent with both the published historical record of DM rates in 1950 and with DM rates actually observed in 1998. The model was then run forward from 1998 to 2050 using several different sets of inputs in order to create four future scenarios of DM prevalence in Manitoba.

The model used in this study to forecast and back-cast the DM epidemic in Manitoba is based upon emerging work in the area of complex adaptive systems (Berkes, Colding & Folke, 2003; Byrne, 1998; Chu, Strand & Fjelland, 2003; Elliot & Kiel, 1999; Gunderson & Holling, 2002; Holling, 2001; Phillippe & Mansi, 1998; Waltner-Toews, 2004). This perspective questions the traditional linear methods often employed in epidemiological research. It suggests that systems have emergent properties which can often only be explored through the use of dynamic simulation models. These models, which require only a small number of inputs, can generate quite complex results, and allow

the development of future scenarios. By running these models forward in time (either from an historical point in time to the present, or from the present into the future), one can model the emergent and complex properties of a system of relationships. By modifying initial inputs, one can explore what the impact of changes in initial starting conditions would be over time. Outputs from the model can be checked against real world observations when possible in order to validate the legitimacy of model inputs and assumptions. A major assumption of the complex adaptive systems perspective is that the future is inherently unknowable (and unpredictable) and that it is important to generate and prepare for a number of divergent futures.

The approach used in this study of using simulations to model both the historic and future trajectories of chronic disease is important and unique because it helps overcome the limitation of having only a small number of years of reliable disease data. When only a small number of years of data is available, identification of the long-term disease trends and their relationship to large scale historical and ecological forces is difficult to ascertain (Brand, 1999). As a result, many analyses of trends in DM have focused on modeling the individual (lifestyle and genetics) and geographic variability in disease rates within the restricted time frames of the research database (Connolly, Unwin, Sherriff, Bilous & Kelly, 2000; Evans, Newton, Ruta, MacDonald & Morris, 2000; Green, Blanchard, Young & Griffith, 2003; Green, Hoppa, Young & Blanchard, 2003; Manuel & Schultz, 2002; Young, Szathmary, Evers & Wheatley, 1990). However, as Rose (1985, 1992) suggests, when exploring the causes of a disease in a population, it is critical to identify the large scale forces which may be causing the disease at the population level. These causes affect

everyone in the population and their lack of variability across the population renders their relationship to disease invisible to traditional epidemiological methods. He argues that these macro level forces can only be rendered visible by examining long-term trends within a study population, or through comparison to other populations having very different social, economic, cultural and ecological characteristics. Failure to identify the impact of these large scale forces, Rose suggests, will result in a focusing on the more proximate causes of disease in individuals such as genetics and lifestyle.

This study, by generating an empirically based and historically plausible 100 year composite picture of the emerging DM epidemic, sets the stage for examination of the large scale factors in the social and ecological environments which may be driving and shaping the course of the DM epidemic in Manitoba. These factors, which affect everyone in the population, include the structure of the food system, the urban and rural landscapes, medical practice, and personal lifestyles. These factors have been suggested as being critically important in affecting population rates of both obesity and DM (CIHR, 2003; Nestle & Jacobson, 2000a; Poston & Foreyt, 1999; WHO, 2000). In this study, these larger factors are referred to as LLM (landscape, lifestyle and medical practice) and an attempt is made to model their combined and cumulative contribution to the growth in DM cases from 1950 to 2050.

This study also generates four future scenarios of the DM epidemic in Manitoba. In doing so, it provides important insights into the range of disease futures for which Manitoba will have to prepare. Depending upon which future scenario prevails, there may be very different societal impacts of the disease, and very different sets of policy options

and prevention programs that become necessary in order to effectively manage the epidemic. It is important that policy makers and planners consider that the worst case scenario could actually occur, and how society needs to respond now in order to either prevent this epidemic from occurring or to prepare for its impacts.

Separate analyses were undertaken in this study for the Registered First Nation (RFN) and non-RFN populations in Manitoba. The RFN population refers to a sub-set of individuals of Aboriginal ancestry eligible for registration under the Indian Act of Canada. For a variety of a complex historical and political reasons, not all individuals of Aboriginal ancestry are eligible for RFN status (Nault, Chen, George & Norris, 1993).

It is important to note that modeling DM backwards and forwards in time is complicated by the fact that both the case definition for DM and the rate at which it is diagnosed in the population has changed significantly over the past 50 years (Harris, 2003; Harris, Meltzer & Zinman, 1998; Meltzer, Leiter, Daneman, Gerstein, Lau, Ludwig et al. 1998; National Diabetes Data Group, 1979). It is anticipated that both the case definition and the case detection rates for the disease will continue to change in the future. This makes it difficult to make comparisons between different time periods. For the purposes of this paper, however, DM will be considered to be diagnosed DM that has been brought to the attention of the health care system. As a result, it is likely that a portion of the increase in DM rates modeled in this paper since 1950 may be accounted for by the more sensitive case definitions for the disease that have appeared over the past 50 years and by improved case detection of the disease by physicians. In this paper, there has been no attempt to disentangle actual disease from diagnosed DM as there is not an empirical set

of data that would facilitate this. It is also important to note that there has been no attempt made in this paper to separately model Type 1 and Type 2 DM. This is because Type 1 and Type 2 DM cannot be easily distinguished within the administrative databases used for this study. However, given that approximately 95% of all DM cases are Type 2 DM (Harris, 1995), this paper is primarily about the trajectory of the Type 2 DM in Manitoba.

Materials and Methods:

Study Setting:

The study was conducted in the Canadian province of Manitoba. Manitoba has a population of 1.14 million people, of whom more than one-half (645,000) reside in the City of Winnipeg, the provincial capital. The majority of Manitobans are of European descent, whereas 10% of the population is self-identified as having Aboriginal ancestry (Statistics Canada, 2001a). Manitoba has a universal health insurance plan, and all residents of the province are eligible to receive health care services with no payments required at the time of service.

Diabetes Projection Model (DPM):

A component cohort Diabetes Projection Model (DPM) was developed using the Epi-Info 6.04d software package(Centers for Disease Control, 2001). This program, which starts with a base-year age and gender specific DM prevalence estimate, applies DM incidence and mortality rates to each gender and age covariate in order to estimate for each year the number of new cases of DM and the number of deaths among those with DM. At each anniversary, the DM and non-DM cohorts are aged by one year. This

process is repeated for each successive year of the projection. Inputs required by the DPM include age and gender specific estimates of the total population for each year of the projection, and DM prevalence, incidence, and mortality rates for the first year of the projection. The model allows increasing or decreasing trends in incidence and mortality rates to be modeled throughout the projection period. Outputs of the program include year, age and gender specific estimates of DM prevalence.

DPM Model Inputs:

Overall Population Estimates, 1950 - 2050:

Separate population estimates for the Registered First Nations (RFN) and the non-Registered First Nations (non-RFN) population were initially generated for the years 1950 to 2050. This task was complicated by the fact that the detailed year, gender and age specific population estimates required for this study were frequently not available in published form. For example, population estimates for Registered First Nations were not available for all years between 1950 and 1997, and when they were available they contained only very broad age and gender breakdowns. In addition, published population projections for both the RFN population and the Manitoba population as a whole were available only to the year 2025. As a result, detailed and continuous population estimates from 1950 to 2050 had to be generated using a variety of interpolation, component cohort projection and subtraction techniques which are described in detail below.

RFN Population Estimates, 1950 - 2050:

a. Future Projections: Population estimates for Registered First Nations (RFN) in Manitoba for the years 1998 to 2002 were obtained from the First Nation and Inuit Health

Branch, Winnipeg office (First Nation and Inuit Health Branch, 2003). Corrections were made for late reporting of births using the approach described by (Nault et al., 1993).

Using the corrected 2002 base year population and published trends in life expectancy and fertility (Indian and Northern Affairs, 2003; Nault et al., 1993), the population was projected to the year 2050 using the DemProj population projection software (Futures Group, 2003). Total fertility and age specific fertility were assumed to converge to non-First Nation patterns by the year 2050 (George, Loh, Verma & Shin, 2001). The Coale Demeny Model West life table [1], modified to reflect the mortality experience of the 0 - 19 year age group was used to model patterns of mortality. In comparison to other available life tables in the DemProj software package, patterns of age specific mortality generated using the Coale Demeny Model West life table most closely matched the actual patterns of mortality observed in the RFN population for Manitoba for the year 2000 (First Nation and Inuuit Health Branch, 2003). Net migration was assumed to be zero since the available information on RFN migration suggests that interregional migration historically has had a negligible effect on the growth of regional RFN populations in Canada (Loh, 1990). Projected population estimates were validated against published projection numbers to the year 2021 (Indian and Northern Affairs, 2003).

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[1]Life tables are used to estimate current and anticipated patterns of mortality in a population when the exact patterns of mortality are unknown (as is the case when undertaking population projections). The Coale and Demeny regional model life tables used in the DemProj software were first published in 1966 and updated in 1983 (Coale, A., Demeny & Vaughn, 1983). In their original form, the Coale and Demeny regional model life tables consisted of 25 separate versions or levels, each corresponding to an anticipated population life expectancy. The DemoProj software combines the 25 levels of each family of life tables into one master life table which is organized by life expectancy. When a projected population life expectancy in a particular year falls between discrete categories of life expectancy in the life table, interpolation is used to estimate age and gender specific survival rates.

b. Historical Estimates: The RFN population for Manitoba in 1950 was obtained from Indian and Northern Affairs Canada (2004). Since this population estimate did not contain the detailed age and gender break down of the population required for this study, the detailed age structure of the population was estimated by applying the age structure of the population reporting an Aboriginal language as their first language from the 1951 Canadian census (Statistics Canada, 1953) to the 1950 Indian and Northern Affairs Canada (INAC) population estimate. The resulting age structure was further decomposed from 10 year age categories into five year age categories using the Carrier-Farrag formula (Arriaga, 1994). The Carrier-Farrag formula is a demographic technique which uses a number of validated interpolation and subtraction techniques to decompose broad age categories into the finer age categories which may be required for further demographic analyses.

In order to generate year specific population estimates from 1950 to 1998 with the fine age and gender breakdowns required for this study, the 1950 estimated population was projected to 1998 using the DemProj projection software (Futures Group, 2003) using estimated trends in life expectancy and fertility (Young, 1994). The Coale Demeny West life table was again used for the projection. Corrections for the in-migration of Bill-C31[2] registrants were made, based upon published estimates (Nault et al., 1993). Net migration was assumed to be zero. Projected population estimates were validated against

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[2] Amendments to the Indian Act of Canada, passed as Bill C-31 in June 1985, provided for the restoration of Indian status to individuals (and their children) who had lost them under the previous Indian act (Nault et al., 1993). From 1985 to 1991, approximately 80,000 individuals were reinstated as result of the Bill C31 amendments.

published population estimates published by INAC in 1965, 1985 (Indian and Northern Affairs, 2004) and against the 1998 population data provided by FNIHB.

Non-RFN Population Estimates, 1950 - 2050:

a. Future Projections: Population estimates for the total Manitoba population were obtained from the provincial Public Health Branch, Manitoba Health for the years 1985 to 2002. Since the Manitoba Health population database under-reports the number of Registered First Nations by approximately 30%, the non-RFN population of Manitoba was generated by subtracting the corrected RFN population estimates (described above) from the total Manitoba population for the years 1998 to 2002.

Using the 2002 base year estimate of the non-RFN Nation population and published trends in life expectancy and fertility (George et al., 2001), the population was projected to the year 2050 using the DemProj projection software (Futures Group, 2003). The Coale Demeny North life table was used for the projection and net migration was assumed to be zero. In comparison to other available life tables in the DemProj software package, patterns of age specific mortality generated using the Coale Demeny North life table most closely matched the actual patterns of mortality observed in the general Manitoba population in the year 2001 (Statistics Canada, 2003) Projected population estimates were validated by summing the separate population projections undertaken for the RFN and non-RFN populations and comparing year specific totals against published projections for Manitoba to the year 2025 (George et al., 2001).

b. Historical Estimates: Year and gender specific population estimates for the total Manitoba population were obtained from Statistics Canada for the years 1951 to 1984

(Statistics Canada, 2001b). Since these population estimates did not contain all of the detailed five year age categories required for this study, the reported age structure was decomposed into the required five year age categories using the Carrier-Farrag formula (Arriaga, 1994). Prior to 1961, population data were available only for the years 1941, 1951 and 1956. Intercensal population estimates were generated using the linear interpolation function of the PAS software (Arriaga, 1994). Population estimates for the total Manitoba population, 1950 to 1984 were combined with the population estimates obtained for the Manitoba Population from Manitoba Health for the years 1985 - 1997. The non-RFN population was generated by subtracting the estimated RFN population from the estimated total Manitoba population for the years 1950 to 1997.

1998 DM Prevalence Estimates

DM prevalence data was obtained from the Manitoba diabetes database (MDD), which has been previously described (Blanchard, Ludwig, Wajda, Dean, Anderson, Kendal et al. 1996). This database contains a longitudinal record for Manitoba residents of all physician contacts and hospital separation records that cited a diagnosis of DM (ICD-9CM code 250) between 1 April 1984 and March 31, 1999. Individuals are categorized as having DM if they have had a least two separate physician contacts for DM within 2 years of each other or at least one hospital separation for DM. Cases of gestational diabetes are excluded from the MDD. The sensitivity of this database for detecting clinically diagnosed cases of DM has been demonstrated, and the validity of the methodology has been discussed previously (Blanchard et al., 1996). As previously mentioned, the methodology is unable to distinguish between type 2 and type 1 DM.

Since the Manitoba population registry underestimates the number of persons who are RFN by approximately 30%, DM prevalence estimates for 1998 were generated for the RFN population by applying the age and gender specific 1998 DM prevalence rates for individuals identified as RFN in the Manitoba Health population registry database to the corrected population estimates for RFNs provided by FNIHB. The DM prevalence estimates for the non-RFN Population was generated by subtracting the estimated number of DM cases in the RFN population (above) from the total number of DM cases in Manitoba in 1998.

Historical Projection: 1950 - 1998:

In order to generate a plausible scenario for the development of DM from 1950 to 1998, the DPM was "seeded" with estimates of 1950 DM base prevalence that were consistent with the published literature. For the non-RFN population, the 1958 DM prevalence rate of 9.5 cases/1000 obtained from the U.S. National Health Interview Survey was used (Kenny, Aubert & Geiss, 1995).

For the RFN population, the DM prevalence for 1950 was obtained from three published DM prevalence studies undertaken in nine Aboriginal communities in the early 1950's in the American southwest (Cohen, 1954; Drevets, 1965; Shochet, 1958). The prevalence estimate obtained by averaging the DM prevalence estimates in the nine study communities, 20.3 cases/1000, was used to seed the DPM model.

Mortality rates in persons with DM in 1950 could not be estimated using published literature on historical trends since this data has not been recorded consistently over time (Geiss, Herman & Smith, 1995). As a result, mortality rates in persons with DM were

assumed to be 50% higher in 1950 than they were in the years 1994 to 1998. This assumption was based upon the belief that improvements in medical care and technology since 1950 have significantly improved the survival of persons with DM through earlier detection and improved secondary prevention (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus 1997). In the DPM model, mortality rates in 1950 were incremented in linear fashion to 1994 - 1998 rates by the year 1998.

Base incidence rates and their trends to 1998 were then iteratively entered into the DPM Model. The model was run forward from 1950 to 1998, the results inspected, base incidence rates modified, and then re-run until the DPM model generated DM case counts for 1998 similar to those actually observed in 1998.

Separate scenarios were developed for the RFN and the non-RFN populations. The RFN DPM also modeled the in-flow of Bill-C31 registrants. Bill C-31 registrants were assumed to have the same year, age and gender specific prevalence rates of DM as RFN individuals already identified in the MDD.

Future Scenarios: 1998 - 2050:

Based upon long-term trends in DM incidence and mortality identified during the development of the historical scenarios (above), four scenarios were developed for the future projection of DM in Manitoba. These four scenarios were developed in order to illustrate the impact of different assumptions on future DM prevalence and to generate the range of possible "futures". Separate sets of scenarios were developed for the RFN and the non-RFN populations and run from 1998 to 2050. In all four scenarios, the assumption of decreasing mortality associated with DM was used since it was felt that

medical management of DM and its complications would continue to improve over the next half century (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 2003). Assumptions regarding future trends in DM incidence were chosen to illustrate the effects of deviations from historical trends on future DM prevalence. The four scenarios were:

a. Successful Prevention: Assumes that the average DM incidence observed between 1994 and 1998 will decrease by 50% by the year 2050 and the average mortality in persons with DM between 1994 and 1998 will decrease by 25% by the year 2050.

b. Status Quo: Assumes that the average DM incidence observed between 1994 and 1998 will stay constant between 1998 and 2050 and the average mortality in persons with DM between 1994 and 1998 will decrease by 25% by the year 2050.

c. Moderate Growth (Most Likely): Assumes that the average DM incidence observed between 1994 and 1998 will double between 1998 and 2050 and the average mortality in persons with DM between 1994 and 1998 will decrease by 25% by the year 2050.

d. Rapid Growth: Assumes that the average DM incidence observed between 1994 and 1998 will increase at the same rate between 1998 and 2050 as it did between 1950 and 1998, and average mortality in persons with DM between 1994 and 1998 will decrease by 25% by the year 2050. In this scenario, RFN incidence rates of DM were assumed to increase 5 fold by 2050 while non-RFN incidence rates of DM were assumed to increase 4 fold by 2050.

Analysis of Growth Drivers: 1950 - 2050:

The increase in DM cases attributable to three different growth drivers were then calculated for 1950 to 2050 using the method proposed by Bashir & Esteve (2000). The moderate growth (most likely) scenario for 1998 - 2050 was used for these calculations. This method partitions the increase in the total number of cases of DM between time periods into three components: (1) differences due to population size; (2) differences due to population structure (aging); (3) differences due to increased risk. This is accomplished by standardizing for the effects of population size and aging on the increase in total DM cases between time periods, and then inferring the proportion of increase in DM cases due to increased risk through subtraction. In this paper, increased risk is attributed to changes in landscape, lifestyle and medical practice (LLM).

Results:

Population Estimates, 1950 - 2050:

Total Population

As illustrated in Figure 7.1A, between 1950 and 1998, there was a 49% increase in the size of the total Manitoba population, from 765,415 in 1950 to 1,142,465 in 1998. It is projected that the total Manitoba population will continue to increase in size to the year 2025 (1,215,671). Between 2025 and 2050 the total Manitoba population is projected to decrease slightly to 1,116,748.

The projected total population generated in this study compares favorably to published projections for Manitoba in 2025. In 2025, Statistics Canada (George et al.,

2001) estimated that the total Manitoba population would range between 1,131,100 and 1,332,000, while the population projection estimate generated in this study for the year 2025 was 1,215,671.

Registered First Nations

Figure 7.1B illustrates that between 1950 and 1998 there was a five fold increase in the size of the RFN population, from 17,514 in 1950 to 101,234 in 1998. It is projected that the RFN population will continue to increase in size after 1998, to 176,084 in 2025 and 236,228 in 2050. Figure 7.1B also illustrates that prior to 1998 the RFN population was primarily a very young population. After 1998 it is anticipated that there will be a rapid movement of the RFN population into older age categories.

The historical RFN population figures generated in this study by projecting the 1950 RFN population forward to1998 using the Dem Proj software compares favorably to published population totals for a number of intervening years. For the years 1965 and 1985, Indian and Northern Affairs (2004) reported RFN population totals for Manitoba of 29,957 and 51,376, while the projection estimates generated in this study were 29,155 and 54,974 for the same years. In 1998, the corrected FNIHB population estimates for RFN in Manitoba was 101,234 while the projected population estimate was 100,878.

The projected future RFN population generated in this study also compares favorably to published projections for the Manitoba RFN population. Indian and Northern Affairs (2003) estimated that the Manitoba RFN population would be 160,758 in 2021 while this study projected a 2021 population of 164,969.

Non-Registered First Nations

Figure 7.1C illustrates that the non-RFN population grew from 747,901 in 1950 to 1,041,231 in 1998. In contrast to the RFN population, it is projected that the non-RFN population will decrease in size to 1,039,587 by 2025 and 880,520 by 2050. During this same time period, it is projected that the non-RFN population will experience significant aging with an increasing proportion of the population moving into older age groups.

Historical DM Projection, 1950 to 1998:

Tables 7.1A, 7.1B, and 7.1C depict the number of DM cases by year for the total Manitoba population, the RFN population and the non-RFN population from 1950 to 1997 which were generated using the diabetes projection model (DPM). DM case numbers reported in these tables for 1998 were those that were actually observed (and not generated by the DPM). As illustrated, the model estimates that there was a continuous increase in the number of DM cases from 1950 to 1998 in the total, the RFN and non-RFN populations.

Inputs into the DPM for the RFN population were as follows: 1950 DM prevalence - 356 cases; 1950 DM incidence - 1/5 of the observed 1994-1998 average DM incidence, incrementing in linear fashion to the 1994-1998 average DM incidence by 1998; 1950 mortality - 1.5 times that of the 1994-1998 average DM mortality, incrementing in linear fashion to the 1994-1998 average level by 1998. This model suggests that the number of cases of DM in the RFN population may have increased 23 fold between 1950 and 1998, from 356 cases in 1950 to 8336 cases in 1998.

Inputs into the DPM for the non-RFN population were as follows: 1950 DM

prevalence - 6941 cases; 1950 DM incidence - 1/4 of the observed 1994-1998 average DM incidence, incrementing in linear fashion to the 1994-1998 average DM incidence by 1998; 1950 mortality - 1.5 times that of the 1994-1998 average DM mortality, incrementing in linear fashion to the 1994-1998 average levels by 1998. This model suggests that the number of cases of DM in the non-RFN population may have increased 6.8 fold, from 6941 cases in 1950 to 47,406 cases in 1998.

Table 7.2 compares the observed DM Cases in 1998 to the number of DM cases estimated using the diabetes projection model (DPM) run from 1950 to 1998 by Registered First Nation status, age, and gender. As illustrated, the total number of DM cases and the age structure of the DM cases estimated using the DPM closely approximates the pattern of DM cases actually observed in 1998 for both the RFN and non-RFN population in the majority of age and gender categories. In the RFN population the DPM model estimated that there would be 8188 persons with DM in 1998 while 8336 cases of DM were observed in this population. In the non-RFN population, the DPM model estimated that there would be 47,737 cases of DM while 47,406 cases of DM were observed.

Future Scenarios, 1998 - 2050:

Figure 7.2A depicts the results of the four DM projection scenarios from 1998 to 2050 for the total Manitoba population, superimposed on the estimated growth in DM from 1950 to 1998. This chart suggests that there has been continuous growth in the number of cases in DM since 1950, which will continue to the year 2050, regardless of the projection scenario adopted. As illustrated, the prevention scenario projects a doubling of

the number of DM cases from 1998 to 2025 (55,724 to 99,682), decreasing slightly to 97,236 DM cases by 2050. The status quo scenario projects 117,382 DM cases in 2025 and 144,060 cases by 2050; the moderate /most likely scenario projects 139,866 DM cases by 2025 and 214,382 cases by 2050; and the rapid growth scenario projects a tripling of DM cases by 2025 (176,839) with a further increase to 343,215 cases by 2050.

Figure 7.2B depicts the results of the four DM projections scenarios from 1998 to 2050 for the RFN population, superimposed on the estimated growth in DM from 1950 to 1998. As illustrated, the prevention scenario projects 22045 cases of DM by 2025 and 31087 cases by 2050; the status quo scenario projects 25,727 cases of DM by 2025 and 45072 cases by 2050; the moderate growth scenario projects 30,225 cases by 2025 and 64031 cases by 2050; and the rapid growth scenario projects 35,635 cases by 2025 and 86829 cases by 2050. In all scenarios, it is anticipated that there will be continued growth in DM cases in the RFN population beyond the year 2050.

Figure 7.2C depicts the results of the four DM projections scenarios from 1998 to 2050 for the non-RFN population, superimposed on the estimated growth in DM from 1950 to 1998. As illustrated, the prevention scenario projects 77,637 cases of DM by 2025 and 66,149 cases by 2050, with the total number of DM cases peaking in 2029; the status quo scenario projects 91655 cases of DM by 2025 and 98,988 cases by 2050, with the total number of DM cases peaking in 2042; the moderate growth scenario projects 109,643 cases by 2025 and 150,351 cases by 2050, with growth in DM cases continuing past 2050; and the rapid growth scenario projects 141,204 cases by 2025 and 256,386 cases by 2050, again with growth in DM cases continuing past 2050.

Figures 7.2A, 7.2B, and 7.2C all suggest that the number of DM cases began to rapidly climb in the mid-1980's in the total, RFN and non-RFN populations, with continued growth in DM cases expected into the first quarter of the current century regardless of the projection scenario one chooses as the most likely to occur.

Year specific DM case count and crude rate prevalence estimates from 1998 to 2050 by scenario and by the total Manitoba population (Tables 7.3A, 7.3B, 7.3C, 7.3D), the RFN population (Tables 7.4A, 7.4B, 7.4C, 7.4D) and the non-RFN population (Tables 7.5A, 7.5B, 7.5C, 7.5D) are appended.

The moderate growth scenario was chosen as the most likely projection scenario for further consideration since it continues trends in DM incidence and mortality observed between 1950 - 1998, but dampens the trajectory of these trends downward significantly to approximately one half of their estimated 1950 - 1998 levels. The other three projection scenarios were considered less likely since it was not felt that the societal response to the DM epidemic over the next 25 - 50 years would be sufficiently strong to reverse the current increasing trend in DM incidence (prevention and status quo scenarios), nor was it felt that the rate of increase in DM incidence rates estimated between 1950 and 1998 would be sustained to the year 2050. The assumptions used to designate the moderate growth scenario as the most likely projection scenario are supported by the observation that rates of obesity in Canadian children and adults are continuing to rapidly grow (Canadian Population Health Inititiative, 2004; Raine, 2004; Tremblay & Willms, 2000) and by the general observation that "run away" trends in social and biological systems rarely go unchecked (Schwartz, 1991; Waltner-Toews,

2004).

Table 7.6 depicts DM prevalence from 1998 to 2050 by Registered First Nation Status, age, gender and year, using the moderate growth projection scenario for 2025 and 2050. This table suggests that the crude rate of DM in the total population will increase from 4.88% in 1998 to 11.5% in 2025 and 19.1% in 2050, while the crude rate in the RFN population will increase from 8.23% in 1998 to 17.1% in 2025 and 27.1% in 2050. The crude rate of DM in the non-RFN population is projected to increase from 4.5% in 1998 to 10.5% in 2025 and 17.0% in 2050.

These results suggest that over the next 50 years, an increasing percentage of the DM cases in Manitoba will occur in the RFN population. In 1998, 15% of all DM cases were in the RFN population; by 2025 it is anticipated that 21.6% of all DM cases will be in the RFN population, and by 2050 this percentage is expected to increase to 29.8%.

These projection results also indicate that DM cases will be increasingly concentrated in older age groups in all population groups. In 1998, 33.2%, 10.2% and 37% of the DM cases in the total, RFN and non-RFN populations respectively were found in the 70 plus age groups; by 2050 the percentage of DM cases in the 70 plus age group is expected to increase to 39.8 %, 21.6% and 47.6% respectively. This is confirmed by examination of Figures 7.3A, 7.3B, and 7.3C which illustrate the projected changes in population size and age structure of persons with DM in the total, RFN and non-RFN populations in Manitoba from 1950 to 2050. These charts illustrate not only the rapid growth in DM cases from 1950 to 2050, but also the increasing concentration of DM cases in older age categories in all population groups.

Increase in DM Cases Attributable to Growth Drivers:

Figure 7.4 summarize the increase in DM cases from 1950 to 2050 which can be attributed to changes in population size, changes in population structure, and changes in risk - landscape, lifestyle, and medical practice (LLM). As alluded to earlier in this paper, LLM is a "catch-all" category which reflects the cumulative impact on DM prevalence of changes in the structure of the material landscape, lifestyle habits of the population, and medical practice (changes in the case definition of DM, its level of diagnosis, and its medical management). LLM is what is left over after the impact of demographic changes on DM prevalence have been accounted for.

Overall, between 1950 and 1998, 86.3% of the increase in DM cases (41,807) in the total Manitoba population was attributable to changes in LLM, while 9.1% and 4.6% of the increase was attributable to population growth and changes in age structure respectively.

In the RFN population between 1950 and 1998, 78.3% of the increase in DM cases (6252) population was attributable to changes in LLM, while only 21.3% and .3% of the increase was attributable to population growth and changes in age structure respectively. This compares to the non-RFN population during the same time period in which 87.8% of the increase in DM cases (35,555) was attributable to changes in LLM and 6.7% and 5.4% of the increase was attributable to population growth and changes in age structure. What this suggests is that while changes in the demographic structure (size and age composition) were responsible for some of the growth in DM cases in both the RFN and non-RFN populations between 1950 and 1998, the contribution of demographic

factors to the growth DM were very small compared to the impact of non-demographic factors (LLM) in the larger social environment. During this time period, the RFN population grew in size, but not did not age significantly, while the non-RFN population both grew and aged significantly.

Between 1998 and 2050 it is anticipated that demographic change will be a much stronger contributor to the growth in DM cases in both the RFN and non-RFN populations. Between 1998 and 2025, when the RFN population is anticipated to both increase in size and experience significant aging, it is predicted that 28.1% and 32.3% of the increase in DM cases (21,889) in the RFN population will be attributable to population growth and population aging respectively, while LLM will contribute 39.4%. During the same time period, the non-RFN population is anticipated to decrease in size but age significantly. As a result, it is projected that a decrease in the size of the non-RFN population will lead to a 0.2% decrease in the number of DM cases between 1998 and 2025, while continued population aging and LLM will drive the number of DM cases up by 28.1% and 72% respectively.

Between 2025 and 2050, it is anticipated that population growth, population aging, and LLM will contribute almost equally to the growth in DM cases in the RFN population. During this time period population growth, population aging and LLM are anticipated to contribute 30.39%, 37.28% and 32.32% respectively to the increase in DM cases. In the non-RFN population, where there will be an overall decrease in population size, it is anticipated that there will still be an increase in overall DM cases (40,708), driven primarily by a continued aging of the population and continued changes in LLM.

During this time period, decreasing population size will drive the number of DM cases downwards by 16,775 cases while continued aging of the population and LLM will drive the number of DM cases upwards by 14,537 cases and 42,946 cases respectively.

Discussion:

Overview of Results:

Through the application of a dynamic cohort-component estimation approach, this study has back-casted and forecasted the prevalence of diagnosed DM in Manitoba from 1950 to 2050. In creating an empirically based model of the trajectory of DM prevalence which is consistent with the published literature, the study provides important insights into the historical and future dynamics of the DM epidemic. The historical simulation model used in this study, which allowed model inputs from 1950 to dynamically play themselves out over a 48 year period was able to generate accurate DM prevalence estimates to the year 1998. The consistency of model predictions with observed DM prevalence rates indicates that model inputs can be taken seriously. These suggest that the prevalence of diagnosed DM was very low in 1950 and has increased significantly over the past five decades as a result of decreasing diabetes related mortality, a 4 to 5 fold increase in diagnosed DM incidence, and population growth and aging. The positive performance of the historical simulation model used in this study provides a solid justification for its use in projecting future DM prevalence.

This study indicates that the number of diagnosed DM cases in Manitoba may have increased from 7279 cases in 1950 to 55,742 cases in 1998, an eight fold increase. The

study also suggests that regardless of the future scenario adopted, the number of DM cases will likely continue to increase in Manitoba to the year 2050. Depending upon the assumptions one makes about the trajectory of future DM incidence, it is projected that the number of DM cases will increase to between 99,682 and 176,839 cases in 2025 and 97,236 and 343,215 cases by 2050. This represents a 2 to 3 fold increase in DM cases by the year 2025 and a 2 to 6 fold increase by the year 2050. Sobering is the observation that DM cases will still almost double in number by the year 2025 even if prevention efforts are successfully put into place which are able to reduce the incidence rate of DM to 75% of 1994-98 average levels by the year 2025. Growth of DM cases in the RFN population is anticipated to be much faster than in either the total Manitoba population or in the non-RFN population, with 3 to 4 fold increase in DM cases projected by the year 2025 and a 4 to 10 fold increase projected by the year 2050. In all population groups, as the number of cases of DM increase, an increasing percentage of the DM cases are projected to become concentrated in older age groups. The results of this study suggest that as a society, Manitoba is going to have to consider and prepare for a future in which a high burden of illness from DM is will likely be a constant part of the social and medical landscape.

Comparison of Projected Results to Published Studies:

Historical Estimates

DM prevalence estimates generated in this study for the early 1950's compare favorably to the published DM estimates which do exist since these published estimates were used as a basis for initially seeding the DPM with its inputs in 1950. In 1958, the NHIS reported a crude prevalence rate of DM in the U.S. population of 9.3 cases/1000

(Kenny et al., 1995). This compares to the crude rate of 10.9 cases/1000 in 1958 estimated in this study for the total Manitoba population (see Table 7.1A). Similarly, in 1975, the NHIS estimated a prevalence of 22.9/1000, while this study projected a slightly lower crude prevalence rate of 18.9 cases/1000. It is difficult to compare these rates directly since they are not age standardized and therefore do not reflect the impact of differential age structure on crude DM prevalence rate estimates.

The historical projection results generated in this study also compare well with DM case estimates from the Manitoba Diabetes Database (MDD) published previously (Blanchard et. al, 1996). This current study estimated that there were 31,945 persons with diagnosed DM in the total Manitoba population in 1986 while previously published estimates from the MDD estimated that there were 30,104 persons with diagnosed DM in 1986. As summarized in Table 7.2, 1998 total and age specific estimates for DM generated by the DPM are also very closely aligned to the number of DM cases actually observed (in the MDD) for the total Manitoba population (55,925 cases vs. 55,742 cases).

The results of this study which suggest that there were very few cases of diagnosed DM in the Manitoba RFN population in the early 1950's is generally consistent with the observation by West (1974, 1978) that DM was virtually unheard of in the Aboriginal population prior to the 1940's . Although there are numerous published historical estimates of DM prevalence for Aboriginal people in North America, these estimates are difficult to interpret and compare because they were collected using a variety of methods including self reported surveys, chart reviews and clinical studies (Bruce,

1993). DM was diagnosed in these studies using a variety of different diagnostic tests including fasting blood glucose, glucose tolerance test, and the random blood glucose test. As well, estimated rates of DM seem to vary considerably by geographic area of North America and linguistic group, making validation of the current study results for Manitoba using these data difficult. Most relevant is the study by Young et al. (1990) who reported a crude prevalence of DM in the Manitoba Registered First Nation population in 1987 of 28 cases per 1000, a rate that is approximately one half of the rate estimated in this study (57.5 cases/1,000). However, in a review of DM in Native American Communities in the U.S., Valway, Freeman, Kaufman, Welty, Helgerson & Gohdes (1993) reported a crude 1987 DM prevalence rate of 45 cases/1000 for all communities covered by the Indian Health Service, a rate that is only about 25% lower than the rate for 1987 estimated in this study. Valway reported that the crude DM prevalence rate ranged from a low of 9 cases/1000 to a high of 76 cases/1,000 in 1987.

Future Estimates

The "most likely" scenario used in this study generates future DM prevalence estimates which are often significantly higher than those reported in previous studies. This is because the most likely scenario used in this study makes the assumption that age specific DM incidence will continue to rise over the next 50 years, resulting in a constant increase in the age specific prevalence of diagnosed DM in most age groups to the year 2050. As indicated earlier, most previous projection studies either assumed a constant age specific prevalence and/or constant incidence rate throughout the projection period, resulting in generally lower future DM estimates.

The most likely scenario of the current study projects that the number of cases of DM in the total Manitoba population will increase 2.5 fold between 1998 and 2025 and by 3.8 fold between 1998 and 2050. This compares to much lower estimates by Helms (1992) for the U.S. population that DM cases would rise by only 40 % between 2000 and 2025, and by 50% between 2000 and 2050. Helms applied a constant age specific prevalence rate of DM to the projected U.S. population in order to generate future estimates of DM. Using a similar methodology in projecting the global burden of DM from 1995 to 2025, King et al. (1998) estimated that the number of DM cases in Canada would increase by only 50% between 2000 and 2025. Wild et al. (2004), also assuming constant age and gender specific prevalence rates of DM, projected a 2.1 fold increase in the number of DM cases world-wide, from 171 million cases in 2000 to 366 million cases in 2025. The author indicates that these projections are likely significant underestimates of future DM prevalence because rapidly rising obesity rates world-wide and increased survival in persons with DM are anticipated to lead to much higher future age and gender specific DM prevalence rates. More closely aligned to the results generated in this study, Boyle et al. (2001) applied rising age specific DM prevalence rates to the projected U.S. population in order to generate future DM prevalence estimates. His study predicted that DM cases in the U.S. would increase by between 2.4 and 3.6 fold between 2000 and 2050 depending upon the assumptions one made about population growth during this period.

Green et al. (1997) used a component cohort projection methodology to forecast DM prevalence in the 25 years and older Manitoba Registered First Nation population from 1995 to 2016. The model assumed that the age specific DM incidence and diabetes

related mortality rates would remain constant at 1995 levels throughout the projection period. This study predicted that by the year 2016 there would be 19,740 cases of DM in the 25+ RFN population in Manitoba. This compares favorably to both the most likely and the status quo scenarios of the current study which projected 21,137 and 19,289 DM cases in the total Manitoba RFN population by 2016.

Blanchard et al. (1998), also using a component cohort projection methodology projected DM prevalence from 1995 to 2025 for the total Manitoba population. The model assumed that the age specific DM incidence and diabetes related mortality rates would remain constant at 1995 levels throughout the projection period. This study predicted that by the year 2025 there would be 104,239 individuals cases of diagnosed DM in Manitoba. This projected number of DM cases is considerably lower than the 139,868 cases (more likely scenario) projected in the current study. It is, however, quite comparable to the "status quo" scenario of the current study which projects that by 2025 there will be 117,382 cases of DM in the total Manitoba population. The status quo scenario assumes a constant age specific incidence rate (1994-1998 average) and a gradually decreasing mortality rate throughout the projection period. Blanchard also projected that by 2025 there would be 23,161 cases of DM in the Manitoba RFN population. Again, this projected number of DM cases is considerably lower than the 30,225 cases projected in the current study using the most likely scenario, but is quite comparable to the status quo scenario which projected 25,727 DM cases by 2025 in the RFN population.

100 Year Perspective on DM in Manitoba:

This study has generated an empirically based and historically plausible 100 year composite picture of the emerging DM epidemic in Manitoba. In doing so, it has set the stage for serious consideration of the large scale factors in the social and ecological environments which are driving and shaping the DM epidemic. The projection models developed in this study suggest that between 1950 and 1998, only a small percentage (13.7%) of the increase in DM prevalence during this time period could be attributed to changes in population size and structure and that 86% of the increase could be attributed to changes in lifestyle, landscape and medical practice. The study has also highlighted that in both the past 50 years and the next 50 years, most of the cases of DM occurred, and will likely occur, in the non-RFN population, populations traditionally considered at low risk of developing DM. These two observations together suggest that explanations for the emerging DM epidemic need to move beyond both genetic and demographic explanations for increasing DM prevalence rates and begin to examine changes in the larger environment which are now affecting rates of DM in the population as a whole. As suggested earlier, some of the reported increase in DM prevalence over the past 50 years are undoubtedly due to increases in detection of the disease. However, given that the single major risk factor for the development of DM is obesity (Carey, Walters, Colditz, Solomon, Willett, Rosner et al. 1997; Chan, Rimm, Colditz, Stampfer & Willett, 1994) , the examination of the wider determinants of DM must evaluate the long-term obesity trend, its causes, and its relationship to rising rates of DM. Adults in Canada who are obese have rates of DM which are 3 to 10 times higher than in individuals who are not

obese (Canadian Fitness and Lifestyle Research Institute, 2004).

In both Canada and the U.S. the rates of obesity in both children and adults have increased dramatically over the past two decades, paralleling the increase in DM. In 2001 in the U.S., the rate of obesity among adults was 20.9%, an increase of 74% since 1991 (Centers for Disease Control, 2004). In Canada, obesity rates in adults increased from 5.6% in 1985 to 14.9% in 2001, a 2.6 fold increase (Raine, 2004). Rates of obesity in children have also increased significantly over the past several decades. Between 1981 and 2001 the rate of obesity in Canadian children 7 to 13 years of age increased 5 fold from 2% in 1981 to 10% in 2001 (Canadian Population Health Inititiative, 2004). An emerging literature is now suggesting that the rising obesity epidemic in North America is due to changes in the physical and socio-cultural landscapes in which people live and that this has resulted in an "engineering out" of opportunities to expend energy through physical activity, and the "engineering in" of opportunities to consume excess calories (Canadian Population Health Inititiative, 2004; CIHR, 2003; Frank, Engelke & Schmid, 2003; Nestle & Jacobson, 2000b; Raine, 2004). These changes, which appear to have been rapidly accelerating since the early 1950's, include the mass production and distribution of the car, the invention of the suburb and the resulting culture of driving (at the expense of walking and bicycling), changes in the structure of the food system which has resulting in increased availability and consumption of energy dense snack foods, the increased popularity of sedentary activities such as television watching and the internet, the loss of school based physical activity curricula with concomitant emphasis on intellectual development, and concerns about childhood safety which has resulted in

lowered rates of walking to school and outside spontaneous play. Although individuals of lower socio-economic status appear to be more susceptible to the deleterious effects of these broad social changes, as evidenced by higher rates of obesity (ACPH, 1999; Mokdad, Bowman, Ford, Vinicor, Marks & Koplan, 2001; Strauss & Pollack, 2001), everyone, rich or poor appears to be affected as evidenced by the dramatic increased in population obesity rates over the past two decades. Current upward trends in obesity suggest that these forces are continuing to increase in intensity and will continue to push DM prevalence rates to ever-higher levels in the immediate future (Zimmet, Shaw & Alberti, 2003). Especially concerning is the impact that the currently high rates of child-hood obesity will have on the adult prevalence of DM several decades in the future (Tremblay & Willms, 2000).

Study Limitations:

This study has a number of methodological limitations which need to be taken into account when interpreting its results. First, this study has relied exclusively on data derived from administrative databases in order to estimate the DM incidence and prevalence rates used as a basis for DM forecasts and back-casts. Since cases have not been individually verified, this approach could result in either an over-estimate or an underestimate of base DM rates in 1998. However, the accuracy of this approach has been studied and found that the specificity is high when compared to local registries of DM (Blanchard et al., 1996).

Secondly, the administrative databases from which the base DM incidence and prevalence rates were derived cannot distinguish between Type 2 adult onset DM and

Type 1 insulin-dependent DM. However, given that it is estimated that approximately 95% of all DM cases are Type 2, it is likely that most of the cases of DM used in this study are Type 2 (Harris, 1995).

Thirdly, the results of this study, which focused on modeling the number of diagnosed cases of DM, may be significantly confounded by the fact that one third of actual DM cases are undiagnosed and that the number of undiagnosed cases of DM may be changing over time (Young & Mustard, 2001). As a result, increases in diagnosed DM cases between 1950 and 1998 which were attributed primarily to changes in the social and physical environments may be due in part to increased case detection of DM between 1950 and 1998.

Fourthly, it is impossible to directly validate the estimated historical and future DM case counts generated in this study. However, their consistency with both the historical record, and other projection studies suggest that they are reasonable.

Fifthly, the linear growth assumptions for DM incidence and DM related mortality used within the projection model for both the back-casting and forecasting of DM cases may be overly simplistic. For example, it is very possible that the trends in DM incidence and DM related mortality used to model DM rates between 1950 and 1998 did not increment smoothly (as assumed in the model), but may have changed more rapidly in certain decades than in others. Although the unique combination of input variables used in the historical projection model generated DM prevalence patterns very consistent with those actually observed in 1998, it is conceivable that very different trends in DM incidence and DM related mortality could generate similar results.

Sixthly, the DM prevalence counts for the RFN and the non-RFN population in 1998 are estimates. The 1998 DM case count for the RFN population was derived by applying the age and gender specific rates for the Registered First Nation population that were identified in the Manitoba diabetes database to the estimated RFN population in Manitoba in 1998 obtained from FNIHB. The non-RFN DM case count was derived by subtracting the RFN DM case count (calculated above) from the total number of DM cases observed in Manitoba in 1998. This was done because the RFN population is under-counted in the Manitoba Health population registry by approximately 30%. Using unadjusted DM case counts for the RFN population would unfairly discount estimates of current and projected DM cases in the RFN population.

Seventh, the population projections used as a basis for DM projections did not assume any population migration into Manitoba. There may be significant migration into countries such as Canada (with a very low below replacement fertility rate) over the next 50 years and this may cause the population projections used in this study for Manitoba to be increasingly in error (Conference Board of Canada, 2004). This could lead to an increasingly conservative estimate over time of the projected number of persons living with diagnosed DM in Manitoba. However, given that the population projections generated in this study were close to the published estimates from Statistics Canada and Indian and Northern Affairs, they can be used with confidence in the short-term (10 - 20 years).

Finally, both the population projections and the DM projections generated by this study need to be treated not as facts, but rather as a range of possibilities. This is

because they are based upon a number of assumptions about base inputs and time trends which can often not be easily justified or supported empirically. Population projections more than 20 - 25 years into the future are rarely taken seriously since they often turn out to be quite inaccurate (Cohen, 1995; Shyrock, Siegel & Stockwell, 1976). In the same vein, the DM projections generated by this study should be treated with extreme caution after 2025. However, the 4 different scenarios generated by the study do provide a low and high range of possible futures for DM, and can provide insight into the different ways in which current trends may play themselves out. From this perspective then, the results of this study provide an important tool for policy makers to think about the implications of current trends, and to ask new "what if" questions which could then be modeled through using the DPM in order to test out the implications of proposed policy alternatives.

Policy, Program and Future Research Implications:

The results of this study have a number of important implications for research, program planning, and policy development. First, from a future research perspective, more definitively linking the rapid increases in DM over the past 50 years to concomitant changes in the social and physical environments during the same time period will require moving beyond the traditional linear epidemiological paradigm of simple cause and effect. For example, linking increasing rates of DM to obesity, and obesity to specific changes in the social and physical landscapes (as was alluded to earlier in this paper) will require embracing diverse historical and geographical research methods which can be used to defensively and credibly establish the broad connections between such factors at different temporal and geographic scales (Brand, 1999; Neustadt & May, 1986; Waltner-Toews,

2004).

Secondly, this study has produced 4 separate future scenarios for growth of DM in Manitoba. All of these scenarios suggest that the number of DM cases will continue to grow over the next 25 - 50 years. Even the most optimistic prevention scenarios suggest that there will be a higher number of cases of DM in the future than in 1998. What this suggests is that as program and policy makers grapple with how to deal with the emerging DM epidemic, they must not be naive about the impact of their intervention programs on the overall population rates of DM. As illustrated by this study, the continued aging of the population over the next five decades can easily over-ride the impact of successful prevention efforts and result in a continued increase in DM cases despite declining incidence. This means that despite the efforts that are put into DM prevention, Manitoba is going to have to prepare for a future in which there will be high burden of illness from DM and its complications. Plans will have to be put into place for extensive and comprehensive programs focusing on secondary prevention which can help manage and ameliorate the impacts of diabetes related complications. Both the public and policy makers are going to have to come to terms with how to organize and pay for the supports and medical interventions that are going to be required to deal with the serious complications associated with increasing rates of DM. These include kidney failure, limb amputation, loss of vision and cardiovascular disease. This will be especially challenging in the emerging context of an aging population in which there will be fewer persons of working age generating the wealth required to pay for medical and community support programs.

Thirdly, the rapidly increasing rates of DM in the population indicates that interventions to prevent and manage the DM epidemic are going to have to address factors affecting the population as a whole. As suggested by the results of this study, the "causes" of the diabetes epidemic appear to be located in those aspects of the larger physical and social environments which are placing all members of the population at risk of becoming obese and developing DM. Programs and policies which focus only on the risky behaviors and susceptibilities of high risk groups and which do not address the larger societal causes of the disease will be unlikely to slow down the growth in the population rates of DM. Unless prevention efforts are willing to tackle the fabric of everyday life - the structure of the urban and rural environments, the structure of the food system, the structure of family life, lifestyles, and the forces that reinforce poor lifestyles, neither the prevention nor even the moderate growth scenarios played out in this study are likely to occur. What is more plausible is the emergence of the moderate or rapid growth scenarios (Zimmet, 2000; Zimmet, Shaw & Alberti, 2003). Both of these scenarios lead to future rates of DM which may at first seem improbable; however, it is important to note that both the moderate and rapid growth scenarios are based upon an extrapolation of historical trends in DM incidence from 1950 to 1998. As a result, these scenarios need to be taken seriously as possible "worst case" scenarios which may actually come to pass. Playing out the "what if's" and the implications of these scenarios for both the public and for policy and decision makers should be used to make the case now for the importance of prevention efforts to address the root causes of the DM epidemic.

Finally, specific policies and programs will need to be put into place which address

the impacts of DM and its complications in the RFN population. As described in this study, the burden of illness from DM experienced by the RFN population is significantly higher than in the general Manitoba population. Although most of the current and future cases of DM in Manitoba will occur in the non-RFN population, an increasing percentage of DM cases is anticipated to occur in the RFN population. This study has estimated that in 1998 approximately 15% of all DM cases were found in the RFN population, and that this proportion is projected to increase to 30% by the year 2050. Providing supports to the RFN population living in First Nation communities will be especially challenging because of issues related to remote geography and poorly developed community and health service infrastructure(Young, Reading, Elias & O'Neil, 2000). This means that a concerted effort is going to have to be made to ensure that the RFN population receives at minimum the same primary and secondary prevention programs available to the non-RFN population. However, significantly enhanced primary and secondary prevention programs focusing on this population will need to be designed in such a way that acknowledge the unique cultural background and challenges facing the RFN population (Abonyi, 2001; Young et al., 2000).

Table 7.1A:
Historical projection, DM cases, 1950 - 1998, total Manitoba population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1950	4031	387313.00	1.04	3248	378102.00	0.86	7279	765415.00	0.95
1951	4129	394812.00	1.05	3381	381723.00	0.89	7510	776535.00	0.97
1952	4235	402375.00	1.05	3520	389770.00	0.90	7755	792145.00	0.98
1953	4347	409894.00	1.06	3667	396279.00	0.93	8014	806173.00	0.99
1954	4468	417417.00	1.07	3820	402790.00	0.95	8288	820207.00	1.01
1955	4596	424936.00	1.08	3981	409302.00	0.97	8577	834238.00	1.03
1956	4734	432478.00	1.09	4151	417561.00	0.99	8885	850039.00	1.05
1957	4880	439694.00	1.11	4332	425545.00	1.02	9212	865239.00	1.06
1958	5036	446892.00	1.13	4522	432021.00	1.05	9558	878913.00	1.09
1959	5202	454090.00	1.15	4722	438494.00	1.08	9924	892584.00	1.11
1960	5378	461308.00	1.17	4932	444987.00	1.11	10310	906295.00	1.14
1961	5566	468503.00	1.19	5155	453183.00	1.14	10721	921686.00	1.16
1962	5765	474878.00	1.21	5391	461006.00	1.17	11156	935884.00	1.19
1963	5977	480877.00	1.24	5641	468121.00	1.21	11618	948998.00	1.22
1964	6200	484958.00	1.28	5905	474293.00	1.24	12105	959251.00	1.26
1965	6434	486877.00	1.32	6182	478131.00	1.29	12616	965008.00	1.31
1966	6677	484266.00	1.38	6471	478800.00	1.35	13149	963066.00	1.37
1967	6932	483585.00	1.43	6774	479415.00	1.41	13706	963000.00	1.42
1968	7201	487390.00	1.48	7094	483604.00	1.47	14294	970994.00	1.47
1969	7486	490904.00	1.52	7430	488094.00	1.52	14916	978998.00	1.52
1970	7783	492105.00	1.58	7782	490910.00	1.59	15565	983015.00	1.58
1971	8101	502117.00	1.61	8155	498689.00	1.64	16256	1000806.00	1.62
1972	8436	502908.00	1.68	8546	500684.00	1.71	16981	1003592.00	1.69
1973	8787	505156.00	1.74	8957	504148.00	1.78	17744	1009304.00	1.76
1974	9158	509969.00	1.80	9391	510189.00	1.84	18549	1020158.00	1.82
1975	9547	512732.00	1.86	9849	514201.00	1.92	19396	1026933.00	1.89
1976	9955	515500.00	1.93	10332	518236.00	1.99	20287	1033736.00	1.96
1977	10384	517830.00	2.01	10839	522172.00	2.08	21224	1040002.00	2.04
1978	10832	518654.00	2.09	11372	524605.00	2.17	22205	1043259.00	2.13
1979	11297	515954.00	2.19	11928	523473.00	2.28	23225	1039427.00	2.23
1980	11778	513803.00	2.29	12506	522886.00	2.39	24285	1036689.00	2.34
1981	12280	513899.00	2.39	13111	524587.00	2.50	25391	1038486.00	2.45
1982	12806	519249.00	2.47	13746	529644.00	2.60	26551	1048893.00	2.53
1983	13358	526555.00	2.54	14413	536689.00	2.69	27771	1063244.00	2.61
1984	13935	532135.00	2.62	15112	541964.00	2.79	29047	1074099.00	2.70
1985	14561	548821.00	2.65	15864	559939.00	2.83	30425	1108760.00	2.74
1986	15239	552158.00	2.76	16707	563426.00	2.97	31945	1115584.00	2.86
1987	15975	554947.00	2.88	17649	566179.00	3.12	33623	1121126.00	3.00
1988	16737	556735.00	3.01	18620	568679.00	3.27	35357	1125414.00	3.14
1989	17542	558836.00	3.14	19654	570974.00	3.44	37196	1129810.00	3.29
1990	18331	559136.00	3.28	20629	571709.00	3.61	38960	1130845.00	3.45
1991	19147	559782.00	3.42	21625	573335.00	3.77	40772	1133117.00	3.60
1992	20003	559510.00	3.58	22667	573610.00	3.95	42671	1133120.00	3.77
1993	20901	560918.00	3.73	23754	575939.00	4.12	44654	1136857.00	3.93
1994	21847	565499.00	3.86	24888	580268.00	4.29	46735	1145767.00	4.08
1995	22835	565395.00	4.04	26062	581600.00	4.48	48896	1146995.00	4.26
1996	23864	564195.00	4.23	27275	580446.00	4.70	51139	1144641.00	4.47
1997	24946	565069.00	4.41	28535	581262.00	4.91	53481	1146331.00	4.67
1998*	26740	563057.00	4.75	29002	579408.00	5.01	55742	1142465.00	4.88

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.1B:
Historical projection, DM cases, 1950-1998, Registered First Nation population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1950	154	9027	1.70	202	8487.00	2.38	356	17514	2.03
1951	161	9339	1.72	213	8789.00	2.43	374	18128	2.06
1952	168	9663	1.74	225	9104.00	2.47	393	18767	2.10
1953	176	10004	1.76	238	9429.00	2.52	414	19433	2.13
1954	185	10353	1.78	251	9772.00	2.57	436	20125	2.17
1955	194	10713	1.81	265	10121.00	2.62	459	20834	2.20
1956	203	11085	1.83	281	10485.00	2.68	484	21570	2.24
1957	214	11466	1.86	297	10862.00	2.73	511	22328	2.29
1958	225	11860	1.89	314	11248.00	2.79	539	23108	2.33
1959	236	12261	1.93	333	11648.00	2.86	569	23909	2.38
1960	249	12675	1.96	353	12059.00	2.93	601	24734	2.43
1961	262	13092	2.00	374	12480.00	3.00	636	25572	2.49
1962	275	13524	2.04	397	12912.00	3.07	672	26436	2.54
1963	290	13965	2.08	421	13353.00	3.16	711	27318	2.60
1964	305	14418	2.12	447	13808.00	3.24	753	28226	2.67
1965	322	14877	2.16	475	14278.00	3.33	797	29155	2.73
1966	339	15355	2.21	505	14758.00	3.42	844	30113	2.80
1967	358	15846	2.26	536	15253.00	3.52	894	31099	2.88
1968	378	16357	2.31	570	15771.00	3.61	948	32128	2.95
1969	399	16885	2.36	606	16300.00	3.72	1005	33185	3.03
1970	422	17430	2.42	644	16848.00	3.82	1066	34278	3.11
1971	446	17992	2.48	685	17416.00	3.93	1131	35408	3.19
1972	471	18577	2.54	729	17998.00	4.05	1200	36575	3.28
1973	498	19185	2.60	775	18602.00	4.17	1274	37787	3.37
1974	527	19809	2.66	825	19227.00	4.29	1353	39036	3.46
1975	558	20454	2.73	878	19872.00	4.42	1436	40326	3.56
1976	591	21119	2.80	935	20533.00	4.55	1526	41652	3.66
1977	626	21799	2.87	996	21209.00	4.70	1622	43008	3.77
1978	664	22498	2.95	1061	21902.00	4.84	1725	44400	3.89
1979	705	23215	3.04	1130	22613.00	5.00	1835	45828	4.00
1980	748	23942	3.12	1205	23337.00	5.16	1953	47279	4.13
1981	794	24687	3.22	1284	24072.00	5.33	2078	48759	4.26
1982	844	25446	3.32	1369	24825.00	5.51	2213	50271	4.40
1983	897	26221	3.42	1459	25594.00	5.70	2356	51815	4.55
1984	954	27006	3.53	1556	26373.00	5.90	2510	53379	4.70
1985	1020	27805	3.67	1669	27169.00	6.14	2689	54974	4.89
1986	1119	28622	3.91	1851	28110.00	6.59	2970	56732	5.23
1987	1255	30343	4.13	2111	29822.00	7.08	3366	60165	5.59
1988	1396	32923	4.24	2379	32388.00	7.34	3774	65311	5.78
1989	1556	35440	4.39	2685	34896.00	7.69	4241	70336	6.03
1990	1682	38270	4.39	2914	37714.00	7.73	4596	75984	6.05
1991	1808	39927	4.53	3141	39369.00	7.98	4949	79296	6.24
1992	1946	41387	4.70	3390	40830.00	8.30	5336	82217	6.49
1993	2095	42959	4.88	3654	42404.00	8.62	5749	85363	6.73
1994	2252	44541	5.06	3933	43998.00	8.94	6185	88539	6.99
1995	2420	46112	5.25	4226	45579.00	9.27	6646	91691	7.25
1996	2598	47657	5.45	4533	47140.00	9.62	7132	94797	7.52
1997	2788	49173	5.67	4858	48679.00	9.98	7646	97852	7.81
1998	3085	50376	6.12	5251	50858.00	10.32	8336	101234	8.23

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.1C:
Historical projection, DM cases, 1950 - 1998, Non-Registered First Nation population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1950	3877	378286.00	1.02	3046	369615.00	0.82	6923	747901.00	0.93
1951	3969	385473.00	1.03	3167	372934.00	0.85	7136	758407.00	0.94
1952	4066	392712.00	1.04	3295	380666.00	0.87	7362	773378.00	0.95
1953	4171	399890.00	1.04	3429	386850.00	0.89	7600	786740.00	0.97
1954	4283	407064.00	1.05	3569	393018.00	0.91	7852	800082.00	0.98
1955	4403	414223.00	1.06	3716	399181.00	0.93	8118	813404.00	1.00
1956	4530	421393.00	1.08	3871	407076.00	0.95	8401	828469.00	1.01
1957	4666	428228.00	1.09	4035	414683.00	0.97	8702	842911.00	1.03
1958	4811	435032.00	1.11	4208	420773.00	1.00	9019	855805.00	1.05
1959	4966	441829.00	1.12	4389	426846.00	1.03	9354	868675.00	1.08
1960	5130	448633.00	1.14	4579	432928.00	1.06	9709	881561.00	1.10
1961	5305	455411.00	1.16	4780	440703.00	1.08	10084	896114.00	1.13
1962	5490	461354.00	1.19	4994	448094.00	1.11	10484	909448.00	1.15
1963	5687	466912.00	1.22	5220	454768.00	1.15	10906	921680.00	1.18
1964	5894	470540.00	1.25	5457	460485.00	1.19	11351	931025.00	1.22
1965	6112	472000.00	1.29	5706	463853.00	1.23	11818	935853.00	1.26
1966	6338	468911.00	1.35	5967	464042.00	1.29	12304	932953.00	1.32
1967	6574	467739.00	1.41	6238	464162.00	1.34	12811	931901.00	1.37
1968	6823	471033.00	1.45	6523	467833.00	1.39	13346	938866.00	1.42
1969	7086	474019.00	1.49	6824	471794.00	1.45	13910	945813.00	1.47
1970	7361	474675.00	1.55	7138	474062.00	1.51	14499	948737.00	1.53
1971	7655	484125.00	1.58	7469	481273.00	1.55	15124	965398.00	1.57
1972	7965	484331.00	1.64	7817	482686.00	1.62	15781	967017.00	1.63
1973	8289	485971.00	1.71	8182	485546.00	1.69	16470	971517.00	1.70
1974	8630	490160.00	1.76	8566	490962.00	1.74	17196	981122.00	1.75
1975	8988	492278.00	1.83	8971	494329.00	1.81	17959	986607.00	1.82
1976	9364	494381.00	1.89	9396	497703.00	1.89	18760	992084.00	1.89
1977	9758	496031.00	1.97	9844	500963.00	1.96	19601	996994.00	1.97
1978	10168	496156.00	2.05	10312	502703.00	2.05	20479	998859.00	2.05
1979	10593	492739.00	2.15	10797	500860.00	2.16	21390	993599.00	2.15
1980	11030	489861.00	2.25	11302	499549.00	2.26	22332	989410.00	2.26
1981	11486	489212.00	2.35	11827	500515.00	2.36	23312	989727.00	2.36
1982	11962	493803.00	2.42	12377	504819.00	2.45	24338	998622.00	2.44
1983	12461	500334.00	2.49	12954	511095.00	2.53	25414	1011429.00	2.51
1984	12981	505129.00	2.57	13556	515591.00	2.63	26537	1020720.00	2.60
1985	13541	521016.00	2.60	14195	532770.00	2.66	27735	1053786.00	2.63
1986	14120	523536.00	2.70	14856	535316.00	2.78	28975	1058852.00	2.74
1987	14720	524604.00	2.81	15538	536357.00	2.90	30257	1060961.00	2.85
1988	15341	523812.00	2.93	16242	536291.00	3.03	31582	1060103.00	2.98
1989	15986	523396.00	3.05	16969	536078.00	3.17	32954	1059474.00	3.11
1990	16650	520866.00	3.20	17714	533995.00	3.32	34364	1054861.00	3.26
1991	17339	519855.00	3.34	18484	533966.00	3.46	35823	1053821.00	3.40
1992	18057	518123.00	3.49	19278	532780.00	3.62	37334	1050903.00	3.55
1993	18806	517959.00	3.63	20100	533535.00	3.77	38905	1051494.00	3.70
1994	19595	520958.00	3.76	20955	536270.00	3.91	40550	1057228.00	3.84
1995	20415	519283.00	3.93	21836	536021.00	4.07	42250	1055304.00	4.00
1996	21265	516538.00	4.12	22742	533306.00	4.26	44006	1049844.00	4.19
1997	22157	515896.00	4.29	23678	532583.00	4.45	45835	1048479.00	4.37
1998	23655	512681.00	4.61	23751	528550.00	4.49	47406	1041231.00	4.55

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.2:

Comparison of 1998 observed DM cases to estimated 1998 DM cases generated using the diabetes projection model with inputs from 1950

	Registered First Nations						Non-First Nations					
	Male		Female		Total		Male		Female		Total	
Age	Obs.	Est.	Obs	Est.	Obs..	Est.	Obs.	Est.	Obs.	Est.	Obs.	Est.
0-4	2	2	0	0	2	2	15	16	15	19	30	34
5-9	3	3	3	0	6	3	84	42	54	40	138	83
10-14	4	5	22	22	26	27	119	75	133	75	252	151
15-19	15	23	40	48	55	71	184	113	185	147	369	260
20-24	45	47	117	114	162	161	205	183	224	251	429	435
25-29	109	106	255	217	364	323	276	319	395	484	671	803
30-34	211	204	383	351	594	555	459	542	657	788	1116	1330
35-39	279	208	535	504	814	712	707	822	982	1133	1689	1955
40-44	349	329	560	632	909	960	1107	1200	1191	1445	2298	2645
45-49	434	404	660	658	1094	1062	1714	1721	1394	1764	3108	3485
50-54	483	408	641	644	1124	1052	2360	2193	1942	2035	4302	4227
55-59	333	369	605	581	938	950	2555	2518	1899	2258	4454	4776
60-64	313	314	512	488	825	801	2836	2830	2247	2500	5083	5330
65-69	208	230	358	366	566	597	3112	2970	2676	2741	5788	5710
70-74	131	155	234	254	365	409	2093	2816	3054	2796	5147	5611
75-79	86	88	159	156	245	245	2452	2196	2950	2517	5402	4713
80plus	80	96	167	162	247	259	2377	2534	3753	3655	6130	6189
Total	3085	2990	5251	5198	8336	8188	23655	23091	23751	24647	47406	47737

Obs. - Number. of Diagnosed DM Cases observed in 1998; Est. - Number of Diagnosed DM Cases estimated using the Diabetes Projection Model, using inputs from 1950.

Table 7.3A:
Prevention scenario, DM cases, 1998-2050, total Manitoba population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998	26740	563057.00	4.75	29002	579408.00	5.01	55742	1142465.00	4.88
1999	27908	563744.00	4.95	30324	580026.00	5.23	58232	1143770.00	5.09
2000	29024	566781.00	5.12	31599	583121.00	5.42	60623	1149902.00	5.27
2001	30090	568162.00	5.30	32823	584817.00	5.61	62913	1152979.00	5.46
2002	31115	569655.00	5.46	34005	586560.00	5.80	65120	1156215.00	5.63
2003	32104	571913.00	5.61	35142	587950.00	5.98	67247	1159863.00	5.80
2004	33065	574053.00	5.76	36240	589273.00	6.15	69304	1163326.00	5.96
2005	33996	576139.00	5.90	37304	590607.00	6.32	71300	1166746.00	6.11
2006	34901	578191.00	6.04	38339	591984.00	6.48	73239	1170175.00	6.26
2007	35781	580179.00	6.17	39346	593377.00	6.63	75127	1173556.00	6.40
2008	36638	582137.00	6.29	40330	594826.00	6.78	76968	1176963.00	6.54
2009	37472	584041.00	6.42	41291	596295.00	6.92	78763	1180336.00	6.67
2010	38281	585915.00	6.53	42230	597805.00	7.06	80510	1183720.00	6.80
2011	39066	587751.00	6.65	43147	599343.00	7.20	82213	1187094.00	6.93
2012	39826	589507.00	6.76	44041	600874.00	7.33	83868	1190381.00	7.05
2013	40559	591220.00	6.86	44915	602416.00	7.46	85473	1193636.00	7.16
2014	41263	592835.00	6.96	45764	603931.00	7.58	87026	1196766.00	7.27
2015	41937	594372.00	7.06	46590	605426.00	7.70	88526	1199798.00	7.38
2016	42577	595816.00	7.15	47389	606881.00	7.81	89966	1202697.00	7.48
2017	43184	597120.00	7.23	48162	608249.00	7.92	91345	1205369.00	7.58
2018	43753	598294.00	7.31	48905	609531.00	8.02	92657	1207825.00	7.67
2019	44284	599332.00	7.39	49616	610714.00	8.12	93900	1210046.00	7.76
2020	44774	600186.00	7.46	50295	611745.00	8.22	95070	1211931.00	7.84
2021	45222	600859.00	7.53	50938	612619.00	8.31	96161	1213478.00	7.92
2022	45628	601311.00	7.59	51543	613296.00	8.40	97171	1214607.00	8.00
2023	45987	601574.00	7.64	52108	613797.00	8.49	98096	1215371.00	8.07
2024	46302	601642.00	7.70	52633	614111.00	8.57	98934	1215753.00	8.14
2025	46569	601475.00	7.74	53113	614196.00	8.65	99682	1215671.00	8.20
2026	46791	601117.00	7.78	53551	614081.00	8.72	100342	1215198.00	8.26
2027	46969	600563.00	7.82	53944	613757.00	8.79	100912	1214320.00	8.31
2028	47102	599793.00	7.85	54291	613202.00	8.85	101393	1212995.00	8.36
2029	47194	598849.00	7.88	54594	612444.00	8.91	101788	1211293.00	8.40
2030	47246	597709.00	7.90	54854	611463.00	8.97	102099	1209172.00	8.44
2031	47260	596420.00	7.92	55070	610298.00	9.02	102330	1206718.00	8.48
2032	47239	594984.00	7.94	55244	608948.00	9.07	102482	1203932.00	8.51
2033	47183	593388.00	7.95	55377	607404.00	9.12	102561	1200792.00	8.54
2034	47097	591675.00	7.96	55472	605694.00	9.16	102570	1197369.00	8.57
2035	46983	589840.00	7.97	55529	603831.00	9.20	102512	1193671.00	8.59
2036	46844	587874.00	7.97	55552	601800.00	9.23	102396	1189674.00	8.61
2037	46683	585824.00	7.97	55543	599640.00	9.26	102225	1185464.00	8.62
2038	46503	583661.00	7.97	55504	597328.00	9.29	102007	1180989.00	8.64
2039	46309	581419.00	7.96	55436	594903.00	9.32	101745	1176322.00	8.65
2040	46102	579109.00	7.96	55345	592368.00	9.34	101446	1171477.00	8.66
2041	45883	576713.00	7.96	55231	589722.00	9.37	101114	1166435.00	8.67
2042	45658	574269.00	7.95	55095	586988.00	9.39	100753	1161257.00	8.68
2043	45425	571786.00	7.94	54940	584186.00	9.40	100366	1155972.00	8.68
2044	45188	569251.00	7.94	54770	581310.00	9.42	99959	1150561.00	8.69
2045	44949	566689.00	7.93	54585	578387.00	9.44	99533	1145076.00	8.69
2046	44707	564084.00	7.93	54388	575400.00	9.45	99094	1139484.00	8.70
2047	44463	561478.00	7.92	54179	572392.00	9.47	98643	1133870.00	8.70
2048	44221	558858.00	7.91	53961	569366.00	9.48	98181	1128224.00	8.70
2049	43977	556195.00	7.91	53735	566296.00	9.49	97712	1122491.00	8.70
2050	43734	553524.00	7.90	53502	563224.00	9.50	97236	1116748.00	8.71

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.3B:
Status Quo scenario, DM cases, 1998-2050, total Manitoba population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998.00	26740	563057.00	4.75	29002	579408.00	5.01	55742	1142465.00	4.88
1999.00	27908	563744.00	4.95	30324	580026.00	5.23	58232	1143770.00	5.09
2000.00	29054	566781.00	5.13	31631	583121.00	5.42	60686	1149902.00	5.28
2001.00	30181	568162.00	5.31	32918	584817.00	5.63	63101	1152979.00	5.47
2002.00	31296	569655.00	5.49	34195	586560.00	5.83	65490	1156215.00	5.66
2003.00	32404	571913.00	5.67	35454	587950.00	6.03	67858	1159863.00	5.85
2004.00	33509	574053.00	5.84	36703	589273.00	6.23	70212	1163326.00	6.04
2005.00	34614	576139.00	6.01	37946	590607.00	6.42	72560	1166746.00	6.22
2006.00	35719	578191.00	6.18	39186	591984.00	6.62	74906	1170175.00	6.40
2007.00	36826	580179.00	6.35	40427	593377.00	6.81	77253	1173556.00	6.58
2008.00	37934	582137.00	6.52	41669	594826.00	7.01	79603	1176963.00	6.76
2009.00	39045	584041.00	6.69	42914	596295.00	7.20	81958	1180336.00	6.94
2010.00	40155	585915.00	6.85	44161	597805.00	7.39	84317	1183720.00	7.12
2011.00	41264	587751.00	7.02	45412	599343.00	7.58	86677	1187094.00	7.30
2012.00	42372	589507.00	7.19	46665	600874.00	7.77	89037	1190381.00	7.48
2013.00	43473	591220.00	7.35	47919	602416.00	7.95	91392	1193636.00	7.66
2014.00	44568	592835.00	7.52	49173	603931.00	8.14	93740	1196766.00	7.83
2015.00	45650	594372.00	7.68	50424	605426.00	8.33	96075	1199798.00	8.01
2016.00	46720	595816.00	7.84	51671	606881.00	8.51	98391	1202697.00	8.18
2017.00	47772	597120.00	8.00	52912	608249.00	8.70	100683	1205369.00	8.35
2018.00	48804	598294.00	8.16	54141	609531.00	8.88	102944	1207825.00	8.52
2019.00	49811	599332.00	8.31	55359	610714.00	9.06	105169	1210046.00	8.69
2020.00	50792	600186.00	8.46	56560	611745.00	9.25	107351	1211931.00	8.86
2021.00	51742	600859.00	8.61	57741	612619.00	9.43	109482	1213478.00	9.02
2022.00	52658	601311.00	8.76	58899	613296.00	9.60	111558	1214607.00	9.18
2023.00	53538	601574.00	8.90	60030	613797.00	9.78	113569	1215371.00	9.34
2024.00	54379	601642.00	9.04	61133	614111.00	9.95	115512	1215753.00	9.50
2025.00	55180	601475.00	9.17	62202	614196.00	10.13	117382	1215671.00	9.66
2026.00	55939	601117.00	9.31	63237	614081.00	10.30	119175	1215198.00	9.81
2027.00	56656	600563.00	9.43	64233	613757.00	10.47	120889	1214320.00	9.96
2028.00	57332	599793.00	9.56	65192	613202.00	10.63	122523	1212995.00	10.10
2029.00	57968	598849.00	9.68	66110	612444.00	10.79	124079	1211293.00	10.24
2030.00	58566	597709.00	9.80	66990	611463.00	10.96	125555	1209172.00	10.38
2031.00	59125	596420.00	9.91	67828	610298.00	11.11	126954	1206718.00	10.52
2032.00	59649	594984.00	10.03	68628	608948.00	11.27	128276	1203932.00	10.65
2033.00	60138	593388.00	10.13	69387	607404.00	11.42	129526	1200792.00	10.79
2034.00	60595	591675.00	10.24	70108	605694.00	11.57	130704	1197369.00	10.92
2035.00	61024	589840.00	10.35	70794	603831.00	11.72	131817	1193671.00	11.04
2036.00	61426	587874.00	10.45	71444	601800.00	11.87	132869	1189674.00	11.17
2037.00	61805	585824.00	10.55	72060	599640.00	12.02	133865	1185464.00	11.29
2038.00	62166	583661.00	10.65	72647	597328.00	12.16	134813	1180989.00	11.42
2039.00	62511	581419.00	10.75	73206	594903.00	12.31	135718	1176322.00	11.54
2040.00	62846	579109.00	10.85	73738	592368.00	12.45	136584	1171477.00	11.66
2041.00	63170	576713.00	10.95	74248	589722.00	12.59	137417	1166435.00	11.78
2042.00	63487	574269.00	11.06	74735	586988.00	12.73	138222	1161257.00	11.90
2043.00	63798	571786.00	11.16	75203	584186.00	12.87	139001	1155972.00	12.02
2044.00	64107	569251.00	11.26	75653	581310.00	13.01	139760	1150561.00	12.15
2045.00	64415	566689.00	11.37	76087	578387.00	13.16	140502	1145076.00	12.27
2046.00	64722	564084.00	11.47	76509	575400.00	13.30	141230	1139484.00	12.39
2047.00	65030	561478.00	11.58	76918	572392.00	13.44	141948	1133870.00	12.52
2048.00	65339	558858.00	11.69	77318	569366.00	13.58	142658	1128224.00	12.64
2049.00	65650	556195.00	11.80	77711	566296.00	13.72	143362	1122491.00	12.77
2050.00	65962	553524.00	11.92	78097	563224.00	13.87	144060	1116748.00	12.90

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.3C:
Most-Likely scenario, DM cases, 1998-2050, total Manitoba population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998.00	26740	563057.00	4.75	29002	579408.00	5.01	55742	1142465.00	4.88
1999.00	27908	563744.00	4.95	30324	580026.00	5.23	58231	1143770.00	5.09
2000.00	29086	566781.00	5.13	31664	583121.00	5.43	60751	1149902.00	5.28
2001.00	30277	568162.00	5.33	33018	584817.00	5.65	63296	1152979.00	5.49
2002.00	31488	569655.00	5.53	34394	586560.00	5.86	65882	1156215.00	5.70
2003.00	32724	571913.00	5.72	35786	587950.00	6.09	68510	1159863.00	5.91
2004.00	33989	574053.00	5.92	37201	589273.00	6.31	71190	1163326.00	6.12
2005.00	35285	576139.00	6.12	38642	590607.00	6.54	73927	1166746.00	6.34
2006.00	36614	578191.00	6.33	40113	591984.00	6.78	76727	1170175.00	6.56
2007.00	37978	580179.00	6.55	41617	593377.00	7.01	79596	1173556.00	6.78
2008.00	39376	582137.00	6.76	43157	594826.00	7.26	82534	1176963.00	7.01
2009.00	40809	584041.00	6.99	44733	596295.00	7.50	85543	1180336.00	7.25
2010.00	42275	585915.00	7.22	46345	597805.00	7.75	88620	1183720.00	7.49
2011.00	43773	587751.00	7.45	47995	599343.00	8.01	91768	1187094.00	7.73
2012.00	45300	589507.00	7.68	49679	600874.00	8.27	94979	1190381.00	7.98
2013.00	46854	591220.00	7.93	51399	602416.00	8.53	98253	1193636.00	8.23
2014.00	48432	592835.00	8.17	53152	603931.00	8.80	101583	1196766.00	8.49
2015.00	50029	594372.00	8.42	54935	605426.00	9.07	104965	1199798.00	8.75
2016.00	51643	595816.00	8.67	56748	606881.00	9.35	108391	1202697.00	9.01
2017.00	53268	597120.00	8.92	58586	608249.00	9.63	111854	1205369.00	9.28
2018.00	54901	598294.00	9.18	60446	609531.00	9.92	115348	1207825.00	9.55
2019.00	56536	599332.00	9.43	62324	610714.00	10.21	118860	1210046.00	9.82
2020.00	58170	600186.00	9.69	64217	611745.00	10.50	122387	1211931.00	10.10
2021.00	59797	600859.00	9.95	66118	612619.00	10.79	125915	1213478.00	10.38
2022.00	61411	601311.00	10.21	68025	613296.00	11.09	129436	1214607.00	10.66
2023.00	63010	601574.00	10.47	69931	613797.00	11.39	132941	1215371.00	10.94
2024.00	64589	601642.00	10.74	71832	614111.00	11.70	136421	1215753.00	11.22
2025.00	66144	601475.00	11.00	73723	614196.00	12.00	139867	1215671.00	11.51
2026.00	67675	601117.00	11.26	75601	614081.00	12.31	143276	1215198.00	11.79
2027.00	69177	600563.00	11.52	77462	613757.00	12.62	146639	1214320.00	12.08
2028.00	70652	599793.00	11.78	79303	613202.00	12.93	149955	1212995.00	12.36
2029.00	72100	598849.00	12.04	81122	612444.00	13.25	153222	1211293.00	12.65
2030.00	73522	597709.00	12.30	82918	611463.00	13.56	156440	1209172.00	12.94
2031.00	74917	596420.00	12.56	84688	610298.00	13.88	159605	1206718.00	13.23
2032.00	76288	594984.00	12.82	86432	608948.00	14.19	162719	1203932.00	13.52
2033.00	77633	593388.00	13.08	88149	607404.00	14.51	165782	1200792.00	13.81
2034.00	78958	591675.00	13.34	89840	605694.00	14.83	168798	1197369.00	14.10
2035.00	80262	589840.00	13.61	91505	603831.00	15.15	171767	1193671.00	14.39
2036.00	81551	587874.00	13.87	93146	601800.00	15.48	174697	1189674.00	14.68
2037.00	82828	585824.00	14.14	94764	599640.00	15.80	177592	1185464.00	14.98
2038.00	84097	583661.00	14.41	96362	597328.00	16.13	180459	1180989.00	15.28
2039.00	85364	581419.00	14.68	97940	594903.00	16.46	183304	1176322.00	15.58
2040.00	86631	579109.00	14.96	99503	592368.00	16.80	186134	1171477.00	15.89
2041.00	87901	576713.00	15.24	101051	589722.00	17.14	188951	1166435.00	16.20
2042.00	89176	574269.00	15.53	102584	586988.00	17.48	191760	1161257.00	16.51
2043.00	90458	571786.00	15.82	104107	584186.00	17.82	194565	1155972.00	16.83
2044.00	91750	569251.00	16.12	105620	581310.00	18.17	197370	1150561.00	17.15
2045.00	93054	566689.00	16.42	107125	578387.00	18.52	200179	1145076.00	17.48
2046.00	94372	564084.00	16.73	108624	575400.00	18.88	202996	1139484.00	17.81
2047.00	95702	561478.00	17.04	110120	572392.00	19.24	205822	1133870.00	18.15
2048.00	97047	558858.00	17.37	111614	569366.00	19.60	208661	1128224.00	18.49
2049.00	98405	556195.00	17.69	113109	566296.00	19.97	211514	1122491.00	18.84
2050.00	99776	553524.00	18.03	114606	563224.00	20.35	214382	1116748.00	19.20

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.3D:

Rapid growth scenario, DM cases, 1998-2050, total Manitoba population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998	23659	563057.00	4.20	29002	579408.00	5.01	55742	1142465.00	4.88
1999	24641	563744.00	4.37	30324	580026.00	5.23	58232	1143770.00	5.09
2000	25661	566781.00	4.53	31708	583121.00	5.44	60837	1149902.00	5.29
2001	26722	568162.00	4.70	33151	584817.00	5.67	63557	1152979.00	5.51
2002	27833	569655.00	4.89	34660	586560.00	5.91	66408	1156215.00	5.74
2003	29004	571913.00	5.07	36234	587950.00	6.16	69395	1159863.00	5.98
2004	30239	574053.00	5.27	37877	589273.00	6.43	72528	1163326.00	6.23
2005	31540	576139.00	5.47	39596	590607.00	6.70	75819	1166746.00	6.50
2006	32910	578191.00	5.69	41397	591984.00	6.99	79275	1170175.00	6.77
2007	34351	580179.00	5.92	43282	593377.00	7.29	82902	1173556.00	7.06
2008	35863	582137.00	6.16	45258	594826.00	7.61	86708	1176963.00	7.37
2009	37447	584041.00	6.41	47325	596295.00	7.94	90695	1180336.00	7.68
2010	39102	585915.00	6.67	49486	597805.00	8.28	94867	1183720.00	8.01
2011	40829	587751.00	6.95	51742	599343.00	8.63	99223	1187094.00	8.36
2012	42624	589507.00	7.23	54094	600874.00	9.00	103763	1190381.00	8.72
2013	44487	591220.00	7.52	56541	602416.00	9.39	108486	1193636.00	9.09
2014	46414	592835.00	7.83	59084	603931.00	9.78	113387	1196766.00	9.47
2015	48402	594372.00	8.14	61723	605426.00	10.19	118463	1199798.00	9.87
2016	50447	595816.00	8.47	64453	606881.00	10.62	123706	1202697.00	10.29
2017	52545	597120.00	8.80	67272	608249.00	11.06	129111	1205369.00	10.71
2018	54689	598294.00	9.14	70179	609531.00	11.51	134667	1207825.00	11.15
2019	56874	599332.00	9.49	73168	610714.00	11.98	140365	1210046.00	11.60
2020	59094	600186.00	9.85	76234	611745.00	12.46	146193	1211931.00	12.06
2021	61343	600859.00	10.21	79372	612619.00	12.96	152139	1213478.00	12.54
2022	63615	601311.00	10.58	82575	613296.00	13.46	158191	1214607.00	13.02
2023	65903	601574.00	10.96	85836	613797.00	13.98	164333	1215371.00	13.52
2024	68200	601642.00	11.34	89150	614111.00	14.52	170553	1215753.00	14.03
2025	70502	601475.00	11.72	92509	614196.00	15.06	176839	1215671.00	14.55
2026	72803	601117.00	12.11	95907	614081.00	15.62	183179	1215198.00	15.07
2027	75098	600563.00	12.50	99337	613757.00	16.19	189564	1214320.00	15.61
2028	77385	599793.00	12.90	102795	613202.00	16.76	195987	1212995.00	16.16
2029	79659	598849.00	13.30	106275	612444.00	17.35	202441	1211293.00	16.71
2030	81920	597709.00	13.71	109773	611463.00	17.95	208921	1209172.00	17.28
2031	84164	596420.00	14.11	113286	610298.00	18.56	215422	1206718.00	17.85
2032	86393	594984.00	14.52	116809	608948.00	19.18	221940	1203932.00	18.43
2033	88605	593388.00	14.93	120340	607404.00	19.81	228473	1200792.00	19.03
2034	90801	591675.00	15.35	123877	605694.00	20.45	235017	1197369.00	19.63
2035	92984	589840.00	15.76	127420	603831.00	21.10	241576	1193671.00	20.24
2036	95153	587874.00	16.19	130966	601800.00	21.76	248149	1189674.00	20.86
2037	97313	585824.00	16.61	44096	599640.00	7.35	254742	1185464.00	21.49
2038	99466	583661.00	17.04	45485	597328.00	7.61	261360	1180989.00	22.13
2039	10165	581419.00	1.75	46892	594903.00	7.88	268006	1176322.00	22.78
2040	10379	579109.00	1.79	48318	592368.00	8.16	274686	1171477.00	23.45
2041	10594	576713.00	1.84	49761	589722.00	8.44	281397	1166435.00	24.12
2042	10810	574269.00	1.88	51218	586988.00	8.73	288138	1161257.00	24.81
2043	11026	571786.00	1.93	52686	584186.00	9.02	294912	1155972.00	25.51
2044	11243	569251.00	1.98	54165	581310.00	9.32	301719	1150561.00	26.22
2045	11460	566689.00	2.02	55654	578387.00	9.62	308560	1145076.00	26.95
2046	11679	564084.00	2.07	57151	575400.00	9.93	315435	1139484.00	27.68
2047	11898	561478.00	2.12	58657	572392.00	10.25	322337	1133870.00	28.43
2048	12117	558858.00	2.17	60172	569366.00	10.57	329271	1128224.00	29.18
2049	12337	556195.00	2.22	61697	566296.00	10.89	336232	1122491.00	29.95
2050	12556	553524.00	2.27	63230	563224.00	11.23	343215	1116748.00	30.73

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.4A:
Prevention scenario, DM cases, 1998-2050, Registered First Nations population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998	3085	50376	6.12	5251	50858.00	10.32	8336	101234	8.23
1999	3271	51996.00	6.29	5573	52490.00	10.62	8843	104486.00	8.46
2000	3461	53658.00	6.45	5902	54130.00	10.90	9363	107788.00	8.69
2001	3652	55317.00	6.60	6233	55791.00	11.17	9885	111108.00	8.90
2002	3845	56806.00	6.77	6568	57242.00	11.47	10413	114048.00	9.13
2003	4036	57980.00	6.96	6902	58448.00	11.81	10938	116428.00	9.39
2004	4228	59167.00	7.15	7235	59662.00	12.13	11462	118829.00	9.65
2005	4419	60375.00	7.32	7567	60898.00	12.43	11985	121273.00	9.88
2006	4610	61603.00	7.48	7899	62153.00	12.71	12508	123756.00	10.11
2007	4802	62853.00	7.64	8229	63433.00	12.97	13030	126286.00	10.32
2008	4994	64122.00	7.79	8559	64730.00	13.22	13553	128852.00	10.52
2009	5188	65421.00	7.93	8888	66050.00	13.46	14076	131471.00	10.71
2010	5382	66739.00	8.06	9217	67391.00	13.68	14598	134130.00	10.88
2011	5577	68080.00	8.19	9544	68752.00	13.88	15121	136832.00	11.05
2012	5773	69439.00	8.31	9869	70132.00	14.07	15642	139571.00	11.21
2013	5969	70822.00	8.43	10193	71526.00	14.25	16161	142348.00	11.35
2014	6165	72216.00	8.54	10514	72937.00	14.42	16678	145153.00	11.49
2015	6360	73618.00	8.64	10834	74352.00	14.57	17193	147970.00	11.62
2016	6554	75028.00	8.73	11150	75772.00	14.72	17703	150800.00	11.74
2017	6747	76444.00	8.83	11464	77198.00	14.85	18210	153642.00	11.85
2018	6938	77857.00	8.91	11775	78618.00	14.98	18712	156475.00	11.96
2019	7128	79270.00	8.99	12082	80041.00	15.10	19209	159311.00	12.06
2020	7315	80688.00	9.07	12386	81465.00	15.20	19701	162153.00	12.15
2021	7500	82093.00	9.14	12686	82876.00	15.31	20186	164969.00	12.24
2022	7683	83492.00	9.20	12981	84282.00	15.40	20664	167774.00	12.32
2023	7862	84886.00	9.26	13271	85677.00	15.49	21133	170563.00	12.39
2024	8038	86274.00	9.32	13557	87065.00	15.57	21594	173339.00	12.46
2025	8210	87648.00	9.37	13836	88436.00	15.65	22045	176084.00	12.52
2026	8378	89016.00	9.41	14110	89799.00	15.71	22488	178815.00	12.58
2027	8544	90375.00	9.45	14379	91147.00	15.78	22922	181522.00	12.63
2028	8706	91723.00	9.49	14641	92481.00	15.83	23347	184204.00	12.67
2029	8866	93064.00	9.53	14898	93801.00	15.88	23764	186865.00	12.72
2030	9024	94399.00	9.56	15150	95111.00	15.93	24173	189510.00	12.76
2031	9179	95726.00	9.59	15396	96410.00	15.97	24575	192136.00	12.79
2032	9333	97044.00	9.62	15637	97689.00	16.01	24969	194733.00	12.82
2033	9484	98355.00	9.64	15872	98962.00	16.04	25356	197317.00	12.85
2034	9633	99664.00	9.67	16102	100216.00	16.07	25735	199880.00	12.88
2035	9780	100956.00	9.69	16327	101455.00	16.09	26106	202411.00	12.90
2036	9925	102242.00	9.71	16547	102679.00	16.12	26472	204921.00	12.92
2037	10069	103521.00	9.73	16763	103889.00	16.14	26831	207410.00	12.94
2038	10212	104790.00	9.75	16975	105080.00	16.15	27186	209870.00	12.95
2039	10355	106042.00	9.76	17182	106250.00	16.17	27537	212292.00	12.97
2040	10498	107281.00	9.79	17387	107401.00	16.19	27884	214682.00	12.99
2041	10639	108506.00	9.81	17588	108531.00	16.21	28227	217037.00	13.01
2042	10781	109711.00	9.83	17785	109633.00	16.22	28565	219344.00	13.02
2043	10921	110900.00	9.85	17977	110716.00	16.24	28898	221616.00	13.04
2044	11060	112071.00	9.87	18166	111776.00	16.25	29226	223847.00	13.06
2045	11198	113217.00	9.89	18351	112810.00	16.27	29548	226027.00	13.07
2046	11335	114345.00	9.91	18532	113818.00	16.28	29866	228163.00	13.09
2047	11470	115455.00	9.93	18709	114800.00	16.30	30179	230255.00	13.11
2048	11605	116543.00	9.96	18882	115757.00	16.31	30486	232300.00	13.12
2049	11738	117601.00	9.98	19052	116683.00	16.33	30789	234284.00	13.14
2050	11870	118640.00	10.01	19218	117588.00	16.34	31087	236228.00	13.16

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.4B:

Status-quo scenario, DM cases, 1998-2050, Registered First Nations population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998	3085	50376	6.12	5251	50858.00	10.32	8336	101234	8.23
1999	3271	51996.00	6.29	5573	52490.00	10.62	8843	104486.00	8.46
2000	3464	53658.00	6.46	5908	54130.00	10.91	9373	107788.00	8.70
2001	3663	55317.00	6.62	6251	55791.00	11.21	9915	111108.00	8.92
2002	3868	56806.00	6.81	6605	57242.00	11.54	10472	114048.00	9.18
2003	4075	57980.00	7.03	6963	58448.00	11.91	11038	116428.00	9.48
2004	4286	59167.00	7.24	7327	59662.00	12.28	11612	118829.00	9.77
2005	4501	60375.00	7.45	7696	60898.00	12.64	12196	121273.00	10.06
2006	4720	61603.00	7.66	8070	62153.00	12.98	12790	123756.00	10.33
2007	4944	62853.00	7.87	8450	63433.00	13.32	13394	126286.00	10.61
2008	5173	64122.00	8.07	8836	64730.00	13.65	14008	128852.00	10.87
2009	5408	65421.00	8.27	9227	66050.00	13.97	14634	131471.00	11.13
2010	5648	66739.00	8.46	9623	67391.00	14.28	15271	134130.00	11.39
2011	5893	68080.00	8.66	10025	68752.00	14.58	15918	136832.00	11.63
2012	6144	69439.00	8.85	10432	70132.00	14.87	16575	139571.00	11.88
2013	6399	70822.00	9.04	10843	71526.00	15.16	17241	142348.00	12.11
2014	6659	72216.00	9.22	11258	72937.00	15.43	17916	145153.00	12.34
2015	6922	73618.00	9.40	11677	74352.00	15.71	18599	147970.00	12.57
2016	7190	75028.00	9.58	12100	75772.00	15.97	19289	150800.00	12.79
2017	7461	76444.00	9.76	12527	77198.00	16.23	19987	153642.00	13.01
2018	7735	77857.00	9.93	12956	78618.00	16.48	20690	156475.00	13.22
2019	8011	79270.00	10.11	13389	80041.00	16.73	21399	159311.00	13.43
2020	8291	80688.00	10.27	13824	81465.00	16.97	22114	162153.00	13.64
2021	8572	82093.00	10.44	14261	82876.00	17.21	22832	164969.00	13.84
2022	8854	83492.00	10.61	14700	84282.00	17.44	23554	167774.00	14.04
2023	9138	84886.00	10.77	15139	85677.00	17.67	24277	170563.00	14.23
2024	9422	86274.00	10.92	15580	87065.00	17.89	25002	173339.00	14.42
2025	9707	87648.00	11.07	16021	88436.00	18.12	25727	176084.00	14.61
2026	9992	89016.00	11.23	16462	89799.00	18.33	26453	178815.00	14.79
2027	10278	90375.00	11.37	16903	91147.00	18.54	27180	181522.00	14.97
2028	10566	91723.00	11.52	17344	92481.00	18.75	27909	184204.00	15.15
2029	10856	93064.00	11.67	17785	93801.00	18.96	28641	186865.00	15.33
2030	11149	94399.00	11.81	18227	95111.00	19.16	29375	189510.00	15.50
2031	11444	95726.00	11.96	18669	96410.00	19.36	30113	192136.00	15.67
2032	11742	97044.00	12.10	19112	97689.00	19.56	30853	194733.00	15.84
2033	12042	98355.00	12.24	19555	98962.00	19.76	31597	197317.00	16.01
2034	12345	99664.00	12.39	19998	100216.00	19.96	32343	199880.00	16.18
2035	12651	100956.00	12.53	20443	101455.00	20.15	33093	202411.00	16.35
2036	12960	102242.00	12.68	20888	102679.00	20.34	33847	204921.00	16.52
2037	13273	103521.00	12.82	21335	103889.00	20.54	34607	207410.00	16.69
2038	13590	104790.00	12.97	21784	105080.00	20.73	35374	209870.00	16.86
2039	13913	106042.00	13.12	22236	106250.00	20.93	36149	212292.00	17.03
2040	14242	107281.00	13.28	22690	107401.00	21.13	36931	214682.00	17.20
2041	14575	108506.00	13.43	23147	108531.00	21.33	37721	217037.00	17.38
2042	14913	109711.00	13.59	23605	109633.00	21.53	38518	219344.00	17.56
2043	15255	110900.00	13.76	24065	110716.00	21.74	39320	221616.00	17.74
2044	15601	112071.00	13.92	24526	111776.00	21.94	40127	223847.00	17.93
2045	15951	113217.00	14.09	24988	112810.00	22.15	40939	226027.00	18.11
2046	16305	114345.00	14.26	25451	113818.00	22.36	41755	228163.00	18.30
2047	16662	115455.00	14.43	25915	114800.00	22.57	42577	230255.00	18.49
2048	17023	116543.00	14.61	26380	115757.00	22.79	43403	232300.00	18.68
2049	17388	117601.00	14.79	26847	116683.00	23.01	44235	234284.00	18.88
2050	17757	118640.00	14.97	27315	117588.00	23.23	45072	236228.00	19.08

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.4C:
Most likely scenario, DM cases, 1998-2050, Registered First Nations population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998	3085	50376	6.12	5251	50858.00	10.32	8336	101234	8.23
1999	3271	51996	6.29	5573	52490.00	10.62	8843	104486	8.46
2000	3468	53658	6.46	5914	54130.00	10.93	9383	107788	8.70
2001	3675	55317	6.64	6270	55791.00	11.24	9946	111108	8.95
2002	3893	56806	6.85	6643	57242.00	11.61	10535	114048	9.24
2003	4117	57980	7.10	7028	58448.00	12.02	11145	116428	9.57
2004	4349	59167	7.35	7425	59662.00	12.45	11774	118829	9.91
2005	4590	60375	7.60	7835	60898.00	12.87	12424	121273	10.24
2006	4840	61603	7.86	8257	62153.00	13.28	13097	123756	10.58
2007	5100	62853	8.11	8692	63433.00	13.70	13792	126286	10.92
2008	5371	64122	8.38	9141	64730.00	14.12	14512	128852	11.26
2009	5653	65421	8.64	9603	66050.00	14.54	15256	131471	11.60
2010	5946	66739	8.91	10079	67391.00	14.96	16025	134130	11.95
2011	6251	68080	9.18	10568	68752.00	15.37	16818	136832	12.29
2012	6567	69439	9.46	11069	70132.00	15.78	17635	139571	12.64
2013	6893	70822	9.73	11584	71526.00	16.20	18477	142348	12.98
2014	7231	72216	10.01	12111	72937.00	16.60	19341	145153	13.32
2015	7578	73618	10.29	12650	74352.00	17.01	20228	147970	13.67
2016	7936	75028	10.58	13202	75772.00	17.42	21137	150800	14.02
2017	8304	76444	10.86	13765	77198.00	17.83	22069	153642	14.36
2018	8681	77857	11.15	14340	78618.00	18.24	23021	156475	14.71
2019	9068	79270	11.44	14927	80041.00	18.65	23995	159311	15.06
2020	9464	80688	11.73	15525	81465.00	19.06	24988	162153	15.41
2021	9869	82093	12.02	16133	82876.00	19.47	26001	164969	15.76
2022	10281	83492	12.31	16752	84282.00	19.88	27033	167774	16.11
2023	10701	84886	12.61	17380	85677.00	20.29	28081	170563	16.46
2024	11128	86274	12.90	18017	87065.00	20.69	29145	173339	16.81
2025	11562	87648	13.19	18663	88436.00	21.10	30225	176084	17.17
2026	12004	89016	13.48	19317	89799.00	21.51	31320	178815	17.52
2027	12453	90375	13.78	19980	91147.00	21.92	32433	181522	17.87
2028	12911	91723	14.08	20652	92481.00	22.33	33563	184204	18.22
2029	13379	93064	14.38	21333	93801.00	22.74	34712	186865	18.58
2030	13858	94399	14.68	22024	95111.00	23.16	35881	189510	18.93
2031	14348	95726	14.99	22723	96410.00	23.57	37070	192136	19.29
2032	14849	97044	15.30	23432	97689.00	23.99	38280	194733	19.66
2033	15360	98355	15.62	24150	98962.00	24.40	39510	197317	20.02
2034	15883	99664	15.94	24878	100216.00	24.82	40760	199880	20.39
2035	16417	100956	16.26	25615	101455.00	25.25	42032	202411	20.77
2036	16963	102242	16.59	26363	102679.00	25.68	43326	204921	21.14
2037	17523	103521	16.93	27122	103889.00	26.11	44644	207410	21.52
2038	18097	104790	17.27	27893	105080.00	26.54	45989	209870	21.91
2039	18687	106042	17.62	28675	106250.00	26.99	47362	212292	22.31
2040	19293	107281	17.98	29470	107401.00	27.44	48763	214682	22.71
2041	19914	108506	18.35	30277	108531.00	27.90	50190	217037	23.13
2042	20549	109711	18.73	31093	109633.00	28.36	51641	219344	23.54
2043	21197	110900	19.11	31919	110716.00	28.83	53115	221616	23.97
2044	21858	112071	19.50	32754	111776.00	29.30	54611	223847	24.40
2045	22532	113217	19.90	33597	112810.00	29.78	56129	226027	24.83
2046	23220	114345	20.31	34448	113818.00	30.27	57667	228163	25.27
2047	23920	115455	20.72	35307	114800.00	30.76	59226	230255	25.72
2048	24633	116543	21.14	36174	115757.00	31.25	60806	232300	26.18
2049	25359	117601	21.56	37049	116683.00	31.75	62408	234284	26.64
2050	26098	118640	22.00	37933	117588.00	32.26	64031	236228	27.11

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.4D:
Rapid growth scenario, DM cases, 1998-2050, Registered First Nations population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998	3085	50376	6.12	5251	50858.00	10.32	8336	101234	8.23
1999	3271	51996.00	6.29	5573	52490.00	10.62	8843	104486.00	8.46
2000	3472	53658.00	6.47	5921	54130.00	10.94	9393	107788.00	8.71
2001	3688	55317.00	6.67	6290	55791.00	11.27	9978	111108.00	8.98
2002	3918	56806.00	6.90	6684	57242.00	11.68	10602	114048.00	9.30
2003	4161	57980.00	7.18	7097	58448.00	12.14	11258	116428.00	9.67
2004	4416	59167.00	7.46	7531	59662.00	12.62	11947	118829.00	10.05
2005	4686	60375.00	7.76	7985	60898.00	13.11	12671	121273.00	10.45
2006	4971	61603.00	8.07	8461	62153.00	13.61	13432	123756.00	10.85
2007	5273	62853.00	8.39	8958	63433.00	14.12	14231	126286.00	11.27
2008	5591	64122.00	8.72	9479	64730.00	14.64	15070	128852.00	11.70
2009	5928	65421.00	9.06	10022	66050.00	15.17	15949	131471.00	12.13
2010	6283	66739.00	9.41	10589	67391.00	15.71	16871	134130.00	12.58
2011	6657	68080.00	9.78	11179	68752.00	16.26	17835	136832.00	13.03
2012	7049	69439.00	10.15	11792	70132.00	16.81	18841	139571.00	13.50
2013	7461	70822.00	10.54	12428	71526.00	17.38	19889	142348.00	13.97
2014	7892	72216.00	10.93	13087	72937.00	17.94	20979	145153.00	14.45
2015	8342	73618.00	11.33	13770	74352.00	18.52	22111	147970.00	14.94
2016	8810	75028.00	11.74	14475	75772.00	19.10	23284	150800.00	15.44
2017	9297	76444.00	12.16	15202	77198.00	19.69	24499	153642.00	15.95
2018	9803	77857.00	12.59	15953	78618.00	20.29	25755	156475.00	16.46
2019	10326	79270.00	13.03	16726	80041.00	20.90	27052	159311.00	16.98
2020	10869	80688.00	13.47	17521	81465.00	21.51	28389	162153.00	17.51
2021	11428	82093.00	13.92	18337	82876.00	22.13	29765	164969.00	18.04
2022	12005	83492.00	14.38	19174	84282.00	22.75	31179	167774.00	18.58
2023	12598	84886.00	14.84	20031	85677.00	23.38	32629	170563.00	19.13
2024	13208	86274.00	15.31	20907	87065.00	24.01	34114	173339.00	19.68
2025	13833	87648.00	15.78	21803	88436.00	24.65	35635	176084.00	20.24
2026	14474	89016.00	16.26	22718	89799.00	25.30	37191	178815.00	20.80
2027	15133	90375.00	16.75	23651	91147.00	25.95	38784	181522.00	21.37
2028	15812	91723.00	17.24	24604	92481.00	26.60	40415	184204.00	21.94
2029	16511	93064.00	17.74	25576	93801.00	27.27	42086	186865.00	22.52
2030	17232	94399.00	18.25	26567	95111.00	27.93	43799	189510.00	23.11
2031	17976	95726.00	18.78	27579	96410.00	28.61	45554	192136.00	23.71
2032	18743	97044.00	19.31	28609	97689.00	29.29	47351	194733.00	24.32
2033	19532	98355.00	19.86	29659	98962.00	29.97	49190	197317.00	24.93
2034	20342	99664.00	20.41	30727	100216.00	30.66	51069	199880.00	25.55
2035	21176	100956.00	20.98	31815	101455.00	31.36	52991	202411.00	26.18
2036	22034	102242.00	21.55	32923	102679.00	32.06	54956	204921.00	26.82
2037	22918	103521.00	22.14	34050	103889.00	32.78	56967	207410.00	27.47
2038	23829	104790.00	22.74	35198	105080.00	33.50	59027	209870.00	28.13
2039	24770	106042.00	23.36	36366	106250.00	34.23	61135	212292.00	28.80
2040	25738	107281.00	23.99	37555	107401.00	34.97	63293	214682.00	29.48
2041	26733	108506.00	24.64	38762	108531.00	35.72	65495	217037.00	30.18
2042	27753	109711.00	25.30	39984	109633.00	36.47	67737	219344.00	30.88
2043	28796	110900.00	25.97	41219	110716.00	37.23	70014	221616.00	31.59
2044	29860	112071.00	26.64	42465	111776.00	37.99	72325	223847.00	32.31
2045	30946	113217.00	27.33	43722	112810.00	38.76	74667	226027.00	33.03
2046	32053	114345.00	28.03	44988	113818.00	39.53	77041	228163.00	33.77
2047	33181	115455.00	28.74	46263	114800.00	40.30	79443	230255.00	34.50
2048	34329	116543.00	29.46	47547	115757.00	41.07	81875	232300.00	35.25
2049	35497	117601.00	30.18	48841	116683.00	41.86	84337	234284.00	36.00
2050	36685	118640.00	30.92	50144	117588.00	42.64	86829	236228.00	36.76

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.5A:

Prevention scenario, DM cases, 1998-2050, Non-Registered First Nations population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998	23655	512681.00	4.61	23751	528550.00	4.49	47406	1041231.00	4.55
1999	24637	511748.00	4.81	24751	527536.00	4.69	49389	1039284.00	4.75
2000	25563	513123.00	4.98	25697	528991.00	4.86	51260	1042114.00	4.92
2001	26438	512845.00	5.16	26590	529026.00	5.03	53028	1041871.00	5.09
2002	27270	512849.00	5.32	27437	529318.00	5.18	54707	1042167.00	5.25
2003	28068	513933.00	5.46	28240	529502.00	5.33	56309	1043435.00	5.40
2004	28837	514886.00	5.60	29005	529611.00	5.48	57842	1044497.00	5.54
2005	29577	515764.00	5.73	29737	529709.00	5.61	59315	1045473.00	5.67
2006	30291	516588.00	5.86	30440	529831.00	5.75	60731	1046419.00	5.80
2007	30979	517326.00	5.99	31117	529944.00	5.87	62097	1047270.00	5.93
2008	31644	518015.00	6.11	31771	530096.00	5.99	63415	1048111.00	6.05
2009	32284	518620.00	6.22	32403	530245.00	6.11	64687	1048865.00	6.17
2010	32899	519176.00	6.34	33013	530414.00	6.22	65912	1049590.00	6.28
2011	33489	519671.00	6.44	33603	530591.00	6.33	67092	1050262.00	6.39
2012	34053	520068.00	6.55	34172	530742.00	6.44	68226	1050810.00	6.49
2013	34590	520398.00	6.65	34722	530890.00	6.54	69312	1051288.00	6.59
2014	35098	520619.00	6.74	35250	530994.00	6.64	70348	1051613.00	6.69
2015	35577	520754.00	6.83	35756	531074.00	6.73	71333	1051828.00	6.78
2016	36023	520788.00	6.92	36239	531109.00	6.82	72263	1051897.00	6.87
2017	36437	520676.00	7.00	36698	531051.00	6.91	73135	1051727.00	6.95
2018	36815	520437.00	7.07	37130	530913.00	6.99	73945	1051350.00	7.03
2019	37156	520062.00	7.14	37534	530673.00	7.07	74691	1050735.00	7.11
2020	37459	519498.00	7.21	37909	530280.00	7.15	75369	1049778.00	7.18
2021	37722	518766.00	7.27	38252	529743.00	7.22	75975	1048509.00	7.25
2022	37945	517819.00	7.33	38562	529014.00	7.29	76507	1046833.00	7.31
2023	38125	516688.00	7.38	38837	528120.00	7.35	76963	1044808.00	7.37
2024	38264	515368.00	7.42	39076	527046.00	7.41	77340	1042414.00	7.42
2025	38359	513827.00	7.47	39277	525760.00	7.47	77637	1039587.00	7.47
2026	38413	512101.00	7.50	39441	524282.00	7.52	77854	1036383.00	7.51
2027	38425	510188.00	7.53	39565	522610.00	7.57	77990	1032798.00	7.55
2028	38396	508070.00	7.56	39650	520721.00	7.61	78046	1028791.00	7.59
2029	38328	505785.00	7.58	39696	518643.00	7.65	78024	1024428.00	7.62
2030	38222	503310.00	7.59	39704	516352.00	7.69	77926	1019662.00	7.64
2031	38081	500694.00	7.61	39674	513888.00	7.72	77755	1014582.00	7.66
2032	37906	497940.00	7.61	39607	511259.00	7.75	77513	1009199.00	7.68
2033	37699	495033.00	7.62	39505	508442.00	7.77	77205	1003475.00	7.69
2034	37464	492011.00	7.61	39370	505478.00	7.79	76835	997489.00	7.70
2035	37203	488884.00	7.61	39202	502376.00	7.80	76406	991260.00	7.71
2036	36919	485632.00	7.60	39005	499121.00	7.81	75924	984753.00	7.71
2037	36614	482303.00	7.59	38780	495751.00	7.82	75394	978054.00	7.71
2038	36291	478871.00	7.58	38529	492248.00	7.83	74821	971119.00	7.70
2039	35954	475377.00	7.56	38254	488653.00	7.83	74208	964030.00	7.70
2040	35604	471828.00	7.55	37958	484967.00	7.83	73562	956795.00	7.69
2041	35244	468207.00	7.53	37643	481191.00	7.82	72887	949398.00	7.68
2042	34877	464558.00	7.51	37310	477355.00	7.82	72188	941913.00	7.66
2043	34504	460886.00	7.49	36963	473470.00	7.81	71468	934356.00	7.65
2044	34128	457180.00	7.46	36604	469534.00	7.80	70733	926714.00	7.63
2045	33751	453472.00	7.44	36234	465577.00	7.78	69985	919049.00	7.61
2046	33372	449739.00	7.42	35856	461582.00	7.77	69228	911321.00	7.60
2047	32993	446023.00	7.40	35470	457592.00	7.75	68464	903615.00	7.58
2048	32616	442315.00	7.37	35079	453609.00	7.73	67695	895924.00	7.56
2049	32239	438594.00	7.35	34683	449613.00	7.71	66923	888207.00	7.53
2050	31864	434884.00	7.33	34284	445636.00	7.69	66149	880520.00	7.51

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.5B:

Status quo scenario, DM cases, 1998-2050, Non-Registered First Nations population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998	23655	512681.00	4.61	23751	528550.00	4.49	47406	1041231.00	4.55
1999	24637	511748.00	4.81	24751	527536.00	4.69	49389	1039284.00	4.75
2000	25590	513123.00	4.99	25723	528991.00	4.86	51313	1042114.00	4.92
2001	26518	512845.00	5.17	26667	529026.00	5.04	53186	1041871.00	5.10
2002	27428	512849.00	5.35	27590	529318.00	5.21	55018	1042167.00	5.28
2003	28329	513933.00	5.51	28491	529502.00	5.38	56820	1043435.00	5.45
2004	29223	514886.00	5.68	29376	529611.00	5.55	58600	1044497.00	5.61
2005	30113	515764.00	5.84	30250	529709.00	5.71	60364	1045473.00	5.77
2006	30999	516588.00	6.00	31116	529831.00	5.87	62116	1046419.00	5.94
2007	31882	517326.00	6.16	31977	529944.00	6.03	63859	1047270.00	6.10
2008	32761	518015.00	6.32	32833	530096.00	6.19	65595	1048111.00	6.26
2009	33637	518620.00	6.49	33687	530245.00	6.35	67324	1048865.00	6.42
2010	34507	519176.00	6.65	34538	530414.00	6.51	69046	1049590.00	6.58
2011	35371	519671.00	6.81	35387	530591.00	6.67	70759	1050262.00	6.74
2012	36228	520068.00	6.97	36233	530742.00	6.83	72462	1050810.00	6.90
2013	37074	520398.00	7.12	37076	530890.00	6.98	74151	1051288.00	7.05
2014	37909	520619.00	7.28	37915	530994.00	7.14	75824	1051613.00	7.21
2015	38728	520754.00	7.44	38747	531074.00	7.30	77476	1051828.00	7.37
2016	39530	520788.00	7.59	39571	531109.00	7.45	79102	1051897.00	7.52
2017	40311	520676.00	7.74	40385	531051.00	7.60	80696	1051727.00	7.67
2018	41069	520437.00	7.89	41185	530913.00	7.76	82254	1051350.00	7.82
2019	41800	520062.00	8.04	41970	530673.00	7.91	83770	1050735.00	7.97
2020	42501	519498.00	8.18	42736	530280.00	8.06	85237	1049778.00	8.12
2021	43170	518766.00	8.32	43480	529743.00	8.21	86650	1048509.00	8.26
2022	43804	517819.00	8.46	44199	529014.00	8.35	88004	1046833.00	8.41
2023	44400	516688.00	8.59	44891	528120.00	8.50	89292	1044808.00	8.55
2024	44957	515368.00	8.72	45553	527046.00	8.64	90510	1042414.00	8.68
2025	45473	513827.00	8.85	46181	525760.00	8.78	91655	1039587.00	8.82
2026	45947	512101.00	8.97	46775	524282.00	8.92	92722	1036383.00	8.95
2027	46378	510188.00	9.09	47330	522610.00	9.06	93709	1032798.00	9.07
2028	46766	508070.00	9.20	47848	520721.00	9.19	94614	1028791.00	9.20
2029	47112	505785.00	9.31	48325	518643.00	9.32	95438	1024428.00	9.32
2030	47417	503310.00	9.42	48763	516352.00	9.44	96180	1019662.00	9.43
2031	47681	500694.00	9.52	49159	513888.00	9.57	96841	1014582.00	9.54
2032	47907	497940.00	9.62	49516	511259.00	9.69	97423	1009199.00	9.65
2033	48096	495033.00	9.72	49832	508442.00	9.80	97929	1003475.00	9.76
2034	48250	492011.00	9.81	50110	505478.00	9.91	98361	997489.00	9.86
2035	48373	488884.00	9.89	50351	502376.00	10.02	98724	991260.00	9.96
2036	48466	485632.00	9.98	50556	499121.00	10.13	99022	984753.00	10.06
2037	48532	482303.00	10.06	50725	495751.00	10.23	99258	978054.00	10.15
2038	48576	478871.00	10.14	50863	492248.00	10.33	99439	971119.00	10.24
2039	48598	475377.00	10.22	50970	488653.00	10.43	99569	964030.00	10.33
2040	48604	471828.00	10.30	51048	484967.00	10.53	99653	956795.00	10.42
2041	48595	468207.00	10.38	51101	481191.00	10.62	99696	949398.00	10.50
2042	48574	464558.00	10.46	51130	477355.00	10.71	99704	941913.00	10.59
2043	48543	460886.00	10.53	51138	473470.00	10.80	99681	934356.00	10.67
2044	48506	457180.00	10.61	51127	469534.00	10.89	99633	926714.00	10.75
2045	48464	453472.00	10.69	51099	465577.00	10.98	99563	919049.00	10.83
2046	48417	449739.00	10.77	51058	461582.00	11.06	99475	911321.00	10.92
2047	48368	446023.00	10.84	51003	457592.00	11.15	99371	903615.00	11.00
2048	48316	442315.00	10.92	50938	453609.00	11.23	99255	895924.00	11.08
2049	48262	438594.00	11.00	50864	449613.00	11.31	99127	888207.00	11.16
2050	48205	434884.00	11.08	50782	445636.00	11.40	98988	880520.00	11.24

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.5C:

Most likely scenario, DM cases, 1998-2050, Non-Registered First Nations population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998	23655	512681.00	4.61	23751	528550.00	4.49	47406	1041231.00	4.55
1999	24637	511748.00	4.81	24751	527536.00	4.69	49388	1039284.00	4.75
2000	25618	513123.00	4.99	25750	528991.00	4.87	51368	1042114.00	4.93
2001	26602	512845.00	5.19	26748	529026.00	5.06	53350	1041871.00	5.12
2002	27595	512849.00	5.38	27751	529318.00	5.24	55346	1042167.00	5.31
2003	28607	513933.00	5.57	28758	529502.00	5.43	57365	1043435.00	5.50
2004	29640	514886.00	5.76	29776	529611.00	5.62	59416	1044497.00	5.69
2005	30695	515764.00	5.95	30807	529709.00	5.82	61502	1045473.00	5.88
2006	31774	516588.00	6.15	31856	529831.00	6.01	63630	1046419.00	6.08
2007	32878	517326.00	6.36	32925	529944.00	6.21	65803	1047270.00	6.28
2008	34005	518015.00	6.56	34016	530096.00	6.42	68021	1048111.00	6.49
2009	35156	518620.00	6.78	35130	530245.00	6.63	70286	1048865.00	6.70
2010	36329	519176.00	7.00	36266	530414.00	6.84	72595	1049590.00	6.92
2011	37522	519671.00	7.22	37427	530591.00	7.05	74949	1050262.00	7.14
2012	38733	520068.00	7.45	38610	530742.00	7.27	77343	1050810.00	7.36
2013	39961	520398.00	7.68	39815	530890.00	7.50	79776	1051288.00	7.59
2014	41201	520619.00	7.91	41041	530994.00	7.73	82242	1051613.00	7.82
2015	42451	520754.00	8.15	42285	531074.00	7.96	84736	1051828.00	8.06
2016	43707	520788.00	8.39	43546	531109.00	8.20	87253	1051897.00	8.29
2017	44964	520676.00	8.64	44821	531051.00	8.44	89785	1051727.00	8.54
2018	46220	520437.00	8.88	46106	530913.00	8.68	92326	1051350.00	8.78
2019	47468	520062.00	9.13	47397	530673.00	8.93	94865	1050735.00	9.03
2020	48706	519498.00	9.38	48692	530280.00	9.18	97398	1049778.00	9.28
2021	49928	518766.00	9.62	49985	529743.00	9.44	99913	1048509.00	9.53
2022	51130	517819.00	9.87	51273	529014.00	9.69	102403	1046833.00	9.78
2023	52309	516688.00	10.12	52551	528120.00	9.95	104860	1044808.00	10.04
2024	53461	515368.00	10.37	53815	527046.00	10.21	107276	1042414.00	10.29
2025	54582	513827.00	10.62	55060	525760.00	10.47	109642	1039587.00	10.55
2026	55671	512101.00	10.87	56284	524282.00	10.74	111955	1036383.00	10.80
2027	56724	510188.00	11.12	57482	522610.00	11.00	114206	1032798.00	11.06
2028	57741	508070.00	11.36	58651	520721.00	11.26	116392	1028791.00	11.31
2029	58721	505785.00	11.61	59789	518643.00	11.53	118510	1024428.00	11.57
2030	59664	503310.00	11.85	60894	516352.00	11.79	120558	1019662.00	11.82
2031	60569	500694.00	12.10	61965	513888.00	12.06	122534	1014582.00	12.08
2032	61439	497940.00	12.34	63000	511259.00	12.32	124439	1009199.00	12.33
2033	62273	495033.00	12.58	63999	508442.00	12.59	126272	1003475.00	12.58
2034	63075	492011.00	12.82	64962	505478.00	12.85	128037	997489.00	12.84
2035	63845	488884.00	13.06	65890	502376.00	13.12	129735	991260.00	13.09
2036	64588	485632.00	13.30	66783	499121.00	13.38	131371	984753.00	13.34
2037	65305	482303.00	13.54	67642	495751.00	13.64	132947	978054.00	13.59
2038	66000	478871.00	13.78	68469	492248.00	13.91	134469	971119.00	13.85
2039	66677	475377.00	14.03	69265	488653.00	14.17	135942	964030.00	14.10
2040	67338	471828.00	14.27	70033	484967.00	14.44	137371	956795.00	14.36
2041	67987	468207.00	14.52	70774	481191.00	14.71	138761	949398.00	14.62
2042	68627	464558.00	14.77	71491	477355.00	14.98	140118	941913.00	14.88
2043	69261	460886.00	15.03	72188	473470.00	15.25	141449	934356.00	15.14
2044	69892	457180.00	15.29	72866	469534.00	15.52	142758	926714.00	15.40
2045	70522	453472.00	15.55	73528	465577.00	15.79	144050	919049.00	15.67
2046	71152	449739.00	15.82	74176	461582.00	16.07	145328	911321.00	15.95
2047	71782	446023.00	16.09	74813	457592.00	16.35	146595	903615.00	16.22
2048	72414	442315.00	16.37	75440	453609.00	16.63	147854	895924.00	16.50
2049	73046	438594.00	16.65	76060	449613.00	16.92	149106	888207.00	16.79
2050	73678	434884.00	16.94	76673	445636.00	17.21	150351	880520.00	17.08

* 1998 DM case counts are actual observed counts, not projected counts

Table 7.5D:
Rapid growth scenario, DM cases, 1998-2050, Non-Registered First Nations population

	Males			Females			Total		
YEAR	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100	DM Cases	Population	Cases/100
1998	23655	512681.00	4.61	23751	528550.00	4.49	47406	1041231.00	4.55
1999	24637	511748.00	4.81	24751	527536.00	4.69	49389	1039284.00	4.75
2000	25657	513123.00	5.00	25787	528991.00	4.87	51444	1042114.00	4.94
2001	26718	512845.00	5.21	26861	529026.00	5.08	53579	1041871.00	5.14
2002	27829	512849.00	5.43	27976	529318.00	5.29	55806	1042167.00	5.35
2003	29000	513933.00	5.64	29137	529502.00	5.50	58137	1043435.00	5.57
2004	30235	514886.00	5.87	30346	529611.00	5.73	60581	1044497.00	5.80
2005	31536	515764.00	6.11	31611	529709.00	5.97	63148	1045473.00	6.04
2006	32906	516588.00	6.37	32936	529831.00	6.22	65843	1046419.00	6.29
2007	34347	517326.00	6.64	34324	529944.00	6.48	68671	1047270.00	6.56
2008	35859	518015.00	6.92	35779	530096.00	6.75	71638	1048111.00	6.83
2009	37443	518620.00	7.22	37303	530245.00	7.04	74746	1048865.00	7.13
2010	39098	519176.00	7.53	38897	530414.00	7.33	77996	1049590.00	7.43
2011	40825	519671.00	7.86	40563	530591.00	7.64	81388	1050262.00	7.75
2012	42620	520068.00	8.20	42302	530742.00	7.97	84922	1050810.00	8.08
2013	44483	520398.00	8.55	44113	530890.00	8.31	88597	1051288.00	8.43
2014	46410	520619.00	8.91	45997	530994.00	8.66	92408	1051613.00	8.79
2015	48398	520754.00	9.29	47953	531074.00	9.03	96352	1051828.00	9.16
2016	50443	520788.00	9.69	49978	531109.00	9.41	100422	1051897.00	9.55
2017	52541	520676.00	10.09	52070	531051.00	9.81	104612	1051727.00	9.95
2018	54685	520437.00	10.51	54226	530913.00	10.21	108912	1051350.00	10.36
2019	56870	520062.00	10.94	56442	530673.00	10.64	113313	1050735.00	10.78
2020	59090	519498.00	11.37	58713	530280.00	11.07	117804	1049778.00	11.22
2021	61339	518766.00	11.82	61035	529743.00	11.52	122374	1048509.00	11.67
2022	63611	517819.00	12.28	63401	529014.00	11.98	127012	1046833.00	12.13
2023	65899	516688.00	12.75	65805	528120.00	12.46	131704	1044808.00	12.61
2024	68196	515368.00	13.23	68243	527046.00	12.95	136439	1042414.00	13.09
2025	70498	513827.00	13.72	70706	525760.00	13.45	141204	1039587.00	13.58
2026	72799	512101.00	14.22	73189	524282.00	13.96	145988	1036383.00	14.09
2027	75094	510188.00	14.72	75686	522610.00	14.48	150780	1032798.00	14.60
2028	77381	508070.00	15.23	78191	520721.00	15.02	155572	1028791.00	15.12
2029	79655	505785.00	15.75	80699	518643.00	15.56	160355	1024428.00	15.65
2030	81916	503310.00	16.28	83206	516352.00	16.11	165122	1019662.00	16.19
2031	84160	500694.00	16.81	85707	513888.00	16.68	169868	1014582.00	16.74
2032	86389	497940.00	17.35	88200	511259.00	17.25	174589	1009199.00	17.30
2033	88601	495033.00	17.90	90681	508442.00	17.84	179283	1003475.00	17.87
2034	90797	492011.00	18.45	93150	505478.00	18.43	183948	997489.00	18.44
2035	92980	488884.00	19.02	95605	502376.00	19.03	188585	991260.00	19.02
2036	95149	485632.00	19.59	98043	499121.00	19.64	193193	984753.00	19.62
2037	97309	482303.00	20.18	10046	495751.00	2.03	197775	978054.00	20.22
2038	99462	478871.00	20.77	10287	492248.00	2.09	202333	971119.00	20.84
2039	10161	475377.00	2.14	10526	488653.00	2.15	206871	964030.00	21.46
2040	10375	471828.00	2.20	10763	484967.00	2.22	211393	956795.00	22.09
2041	10590	468207.00	2.26	10999	481191.00	2.29	215902	949398.00	22.74
2042	10806	464558.00	2.33	11234	477355.00	2.35	220401	941913.00	23.40
2043	11022	460886.00	2.39	11467	473470.00	2.42	224898	934356.00	24.07
2044	11239	457180.00	2.46	11700	469534.00	2.49	229394	926714.00	24.75
2045	11456	453472.00	2.53	11932	465577.00	2.56	233893	919049.00	25.45
2046	11675	449739.00	2.60	12163	461582.00	2.64	238394	911321.00	26.16
2047	11894	446023.00	2.67	12394	457592.00	2.71	242894	903615.00	26.88
2048	12113	442315.00	2.74	12625	453609.00	2.78	247396	895924.00	27.61
2049	12333	438594.00	2.81	12856	449613.00	2.86	251895	888207.00	28.36
2050	12552	434884.00	2.89	13086	445636.00	2.94	256386	880520.00	29.12

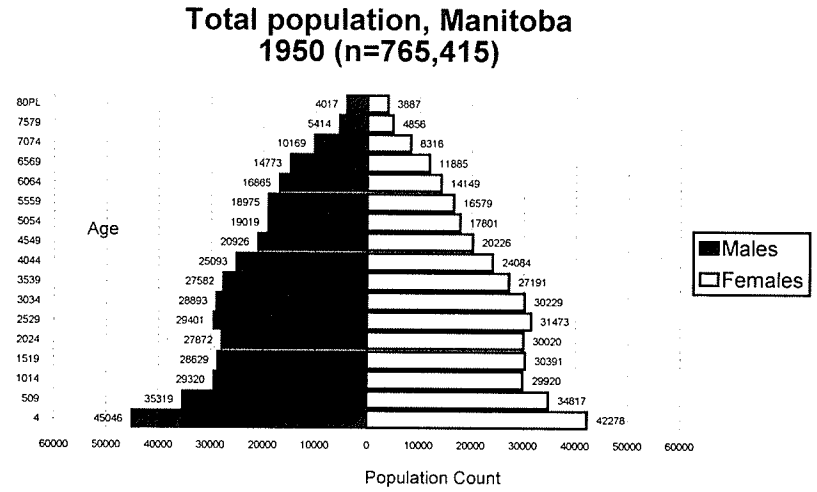
* 1998 DM case counts are actual observed counts, not projected counts

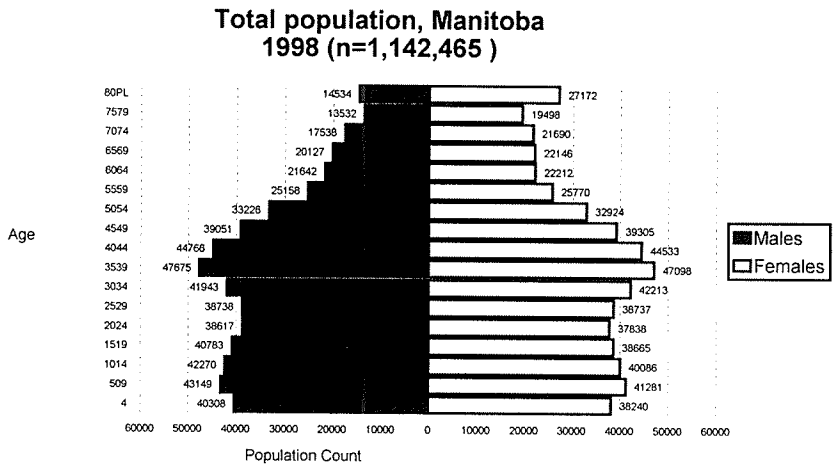
Table 7.6:
Projections of DM cases, Manitoba, 1998 - 2050, by Registered First Nation Status, age and sex

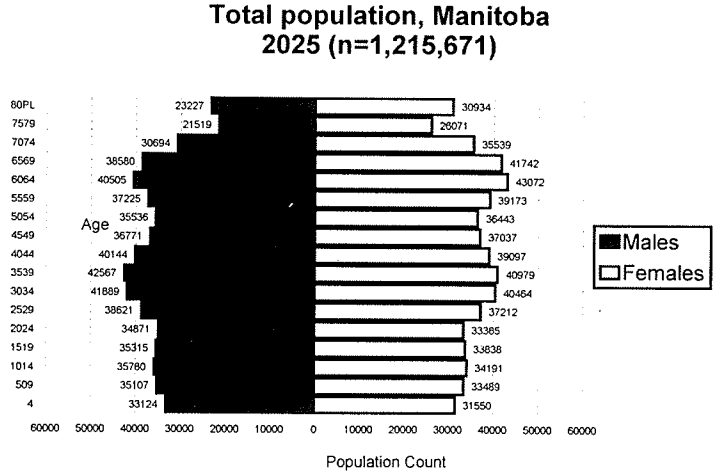
	1998 (observed) Cases			2025 (projected) cases			2050 (projected) cases		
Age	Male	Female	Total	Male	Female	Total	Male	Female	Total
Non-RFN									
0-19 yrs	402 (0.28)	387 (0.3)	789 (0.28)	313 (0.3)	355 (0.35)	668 (0.32)	321 (0.42)	366 (0.51)	687 (0.47)
20-24 yrs	205 (0.60)	224 (0.6)	429 (0.6)	257 (0.9)	339 (1.3)	596 (1.1)	269 (1.2)	359 (1.7)	628 (1.5)
25-29 yrs	276 (0.8)	395 (1.1)	671 (0.97)	468 (1.5)	674 (2.2)	1142 (1.8)	483 (2.0)	702 (3.0)	1185 (2.5)
30-34 yrs	459 (1.2)	657 (1.7)	1116 (1.5)	786 (2.2)	1093 (3.2)	1880 (2.7)	811 (3.1)	1137 (4.6)	1948 (3.8)
35-39 yrs	707 (1.6)	982 (2.3)	1689 (1.9)	1178 (3.2)	1578 (4.5)	2756 (3.8)	1246 (4.6)	1674 (6.5)	2920 (5.5)
40-44 yrs	1107 (2.6)	1191 (2.8)	2298 (2.7)	1758 (5.0)	2109 (6.1)	3867 (5.6)	1968 (7.3)	2335 (8.9)	4304 (8.1)
45-49 yrs	1714 (4.6)	1394 (3.7)	3108 (4.2)	2695 (8.3)	2821 (8.6)	5516 (8.5)	3250 (12.1)	3318 (12.8)	6569 (12.4)
50-54 yrs	2360 (7.4)	1942 (6.2)	4302 (6.8)	4021 (12.8)	3837 (11.9)	7858 (12.4)	5169 (17.1)	4791 (16.1)	9961 (16.4)
55-59 yrs	2555 (10.4)	1899 (7.7)	4454 (9.2)	5761 (17.3)	5260 (15.1)	11020 (16.2)	7367 (22.3)	6568 (20.1)	13935 (21.2)
60-64 yrs	2836 (13.6)	2247 (10.6)	5083 (12.0)	7609 (20.4)	6745 (17.1)	14354 (18.7)	9254 (27.9)	8165 (24.5)	17419 (26.2)
65-69 yrs	3112 (15.9)	2676 (12.5)	5788 (14.1)	8414 (12.1)	7559 (19.4)	15973 (21.2)	10017 (33.1)	9153 (28.8)	19169 (30.9)
70-74 yrs	3093 (18.1)	3054 (14.4)	6147 (16.0)	7846 (26.7)	7294 (21.6)	15140 (23.9)	9748 (38.5)	9372 (32.6)	19120 (35.4)
75-79 yrs	2452 (18.5)	2950 (15.4)	5402 (16.6)	5977 (28.7)	6094 (24.4)	12071 (26.4)	8360 (41.0)	8942 (36.3)	17301 (38.5)
80pl yrs	2377 (16.7)	3753 (14.0)	6130 (14.9)	7499 (33.1)	9302 (31.0)	16801 (31.9)	15414 (43.9)	19792 (42.1)	35206 (42.8)
Total	23655 (4.61)	23751 (4.49)	47406 (4.55)	54583 (10.62)	55061 (10.47)	109643 (10.5)	73678(16.94)	76673(17.21)	150351(17.08)
RFN									
0-19 yrs	24 (0.09)	65 (.28)	89 (0.18)	79 (0.22)	166 (0.5)	245 (0.362)	129 (0.352)	271 (0.78)	400 (0.56)
20-24 yrs	45 (1.0)	117 (2.7)	162 (1.8)	131 (1.8)	316 (4.5)	447 (3.11)	228 (2.5)	545 (6.3)	773 (4.3)
25-29 yrs	109 (2.6)	255 (5.7)	364 (4.1)	322 (4.4)	646 (9.2)	968 (6.8)	574 (6.3)	1124 (12.9)	1698 (9.6)
30-34 yrs	211 (5.1)	383 (9.4)	594 (7.0)	614 (8.9)	1039 (15.3)	1654 (12.0)	1128 (12.6)	1838 (21.4)	2966 (16.9)
35-39 yrs	279 (8.1)	535 (14.3)	814 (11.1)	613 (9.9)	1370 (22.5)	1983 (16.2)	1192 (14.0)	2563 (31.2)	3755 (22.5)
40-44 yrs	349 (13.9)	560 (20.3)	909 (16.6)	896 (17.9)	1628 (34.1)	2524 (25.9)	1875 (24.0)	3228 (42.7)	5103 (33.2)
45-49 yrs	434 (23.6)	660 (31.3)	1094 (27)	1238 (28.5)	1869 (43.3)	3107 (35.9)	2657 (36.8)	3729 (53.7)	6386 (45.1)
505-54 yrs	483 (33.6)	641 (41.5)	1124 (35.7)	1540 (37.2)	2237 (50.9)	3777 (44.2)	3273 (46.7)	4287 (62.7)	7560 (54.6)
55-59 yrs	333 (34.2)	605 (47.1)	938 (40)	1664 (42.3)	2383 (54.9)	4047 (48.9)	3499 (53.9)	4456 (62.7)	7955 (60.8)
60-64 yrs	313 (37.1)	512 (53.9)	825 (45)	1567 (48.8)	2214 (59.0)	3782 (54.3)	3343 (60.2)	4092 (71.2)	7435 (65.8)
65-69 yrso	208 (39)	358 (53.5)	566 (43)	1192 (55.1)	1787 (62.6)	2979 (59.4)	2726 (65.6)	3394 (79.1)	6120 (72.5)
70-74 yrs	131 (31.5)	234 (51.4)	365 (41.9)	797 (62.4)	1300 (70.7)	2098 (67.4)	2086 (65.5)	2826 (79.0)	4912 (72.7)
75-79 yrs	86 (30.9)	159 (49.7)	245 (41)	450 (62.1)	829 (76.5)	1279 (70.7)	1422 (59.5)	2244 (73.6)	3666 (67.4)
80pl yrs	80 (26.8)	167 (42)	247 (35.7)	458 (78.9)	877 (86.4)	1336 (83.7)	1966 (76.8)	3335 (86.3)	5301 (82.5)
Total	3085 (6.11)	5251 (10.32)	8336 (8.23)	11562 (13.1)	18663 (21.1)	30225 (17.17)	26098 (22.0)	37933 (32.26)	64031 (27.11)
A Manitobans									
Total	26740 (4.75)	29002 (5.0)	55742 (4.88)	66,145(10.9)	73724 (12.0)	139868 (11.5)	99776(18.02)	114606 (20.3)	214382 (19.1)

Note: Cell values are counts; values in parentheses are covariate specific prevalence expressed as a percentage.

Fig. 7.1A:
Population structure, total Manitoba population, 1950 - 2050







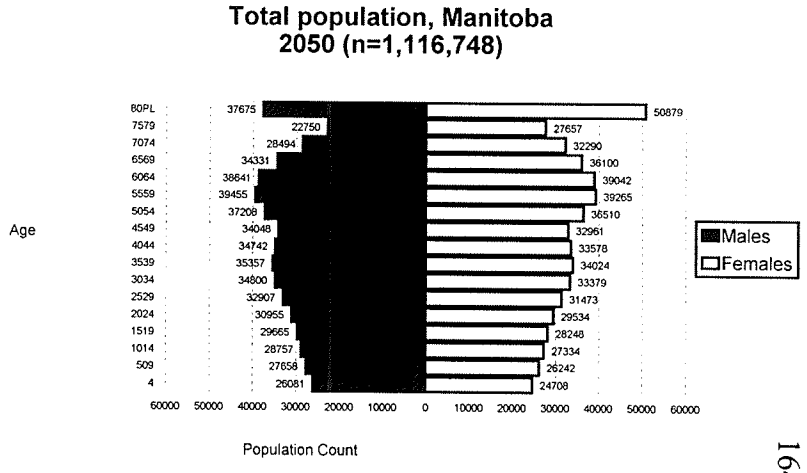
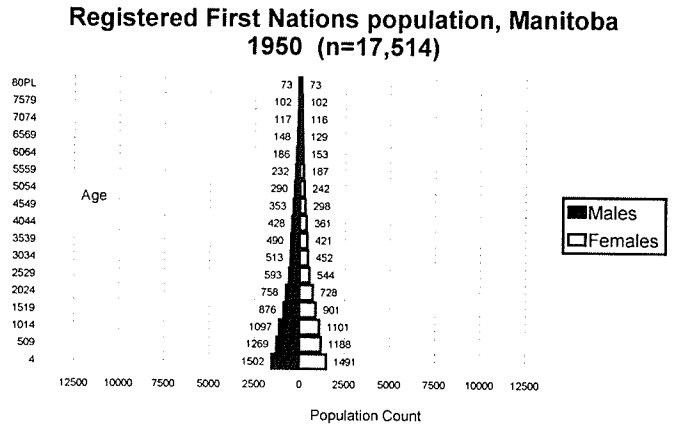
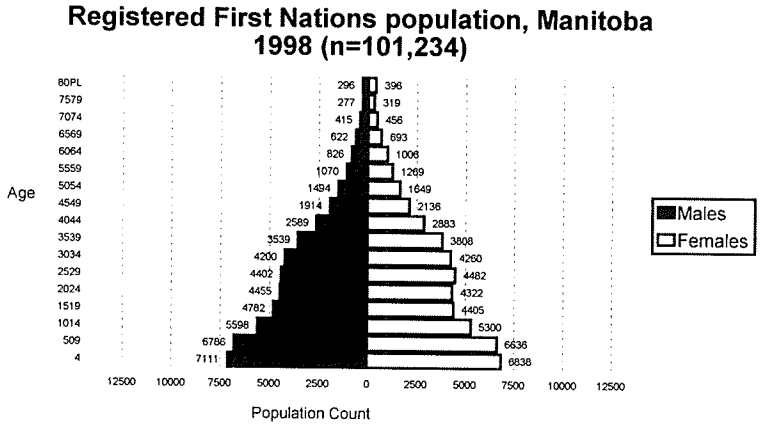
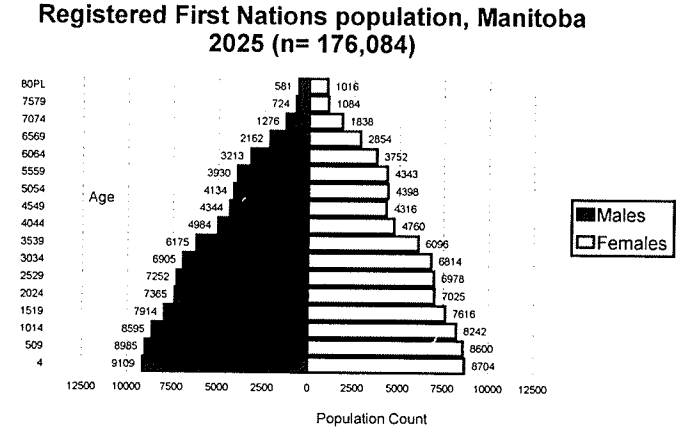


Fig. 7.1B:
Population structure, Registered First Nation population, 1950 to 2050







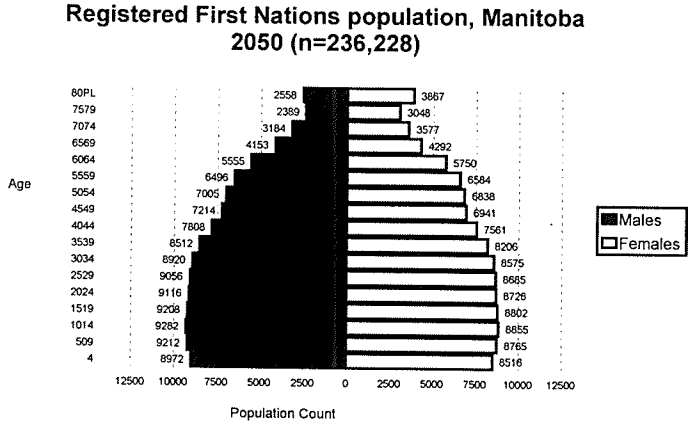
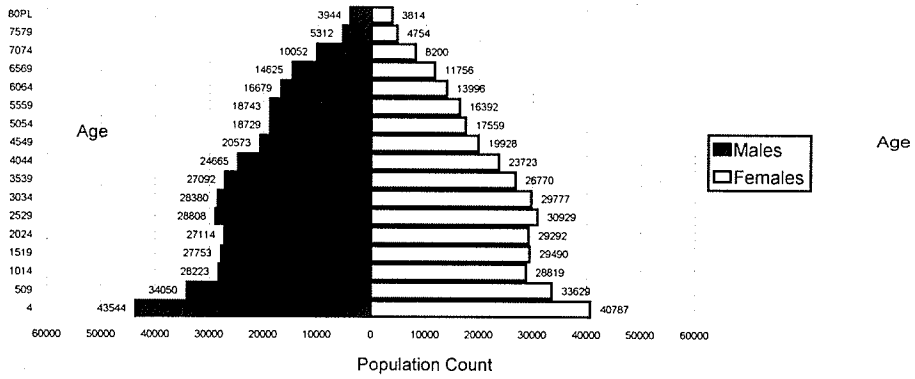
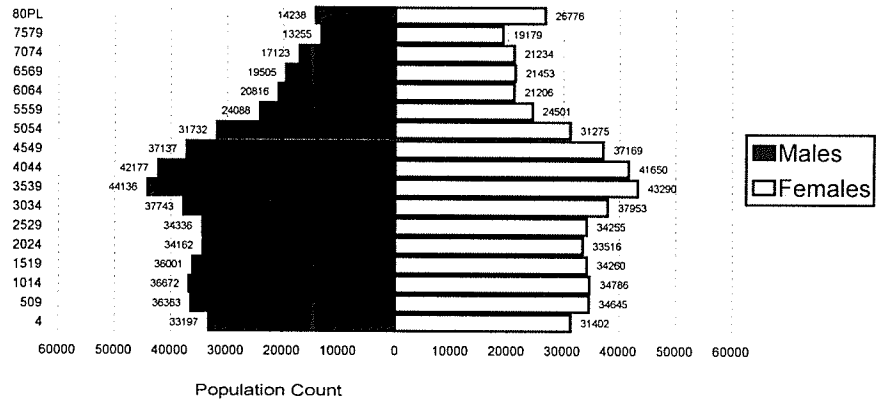


Fig. 7.1C:
Population Structure, Non-Registered First Nations population, 1950 - 2050

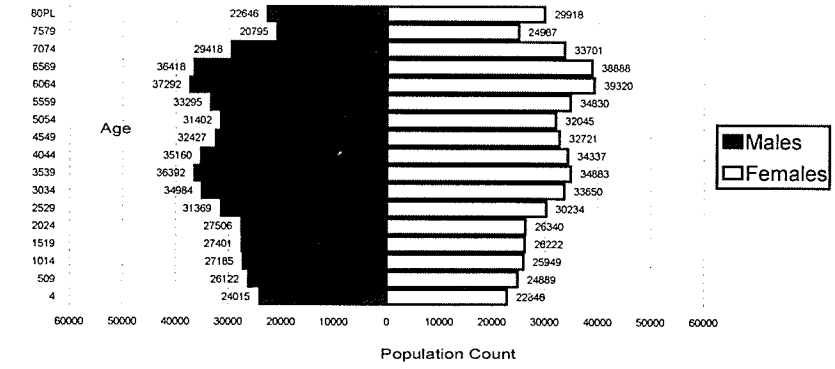
Non-Registered First Nations population, Manitoba 1950 (n=747,901)



Non-Registered First Nations population, Manitoba 1998 (n=1,041,231)



Non-Registered First Nations population, Manitoba 2025 (n=1,039,587)



Non-Registered First Nations population, Manitoba 2050 (n=880,520)

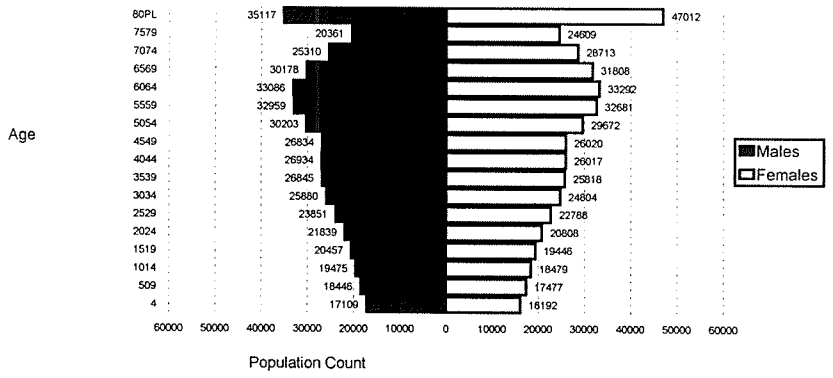


Fig. 7.2A:
Projected Growth in DM cases in Manitoba, total Manitoba population, 1950 - 2050

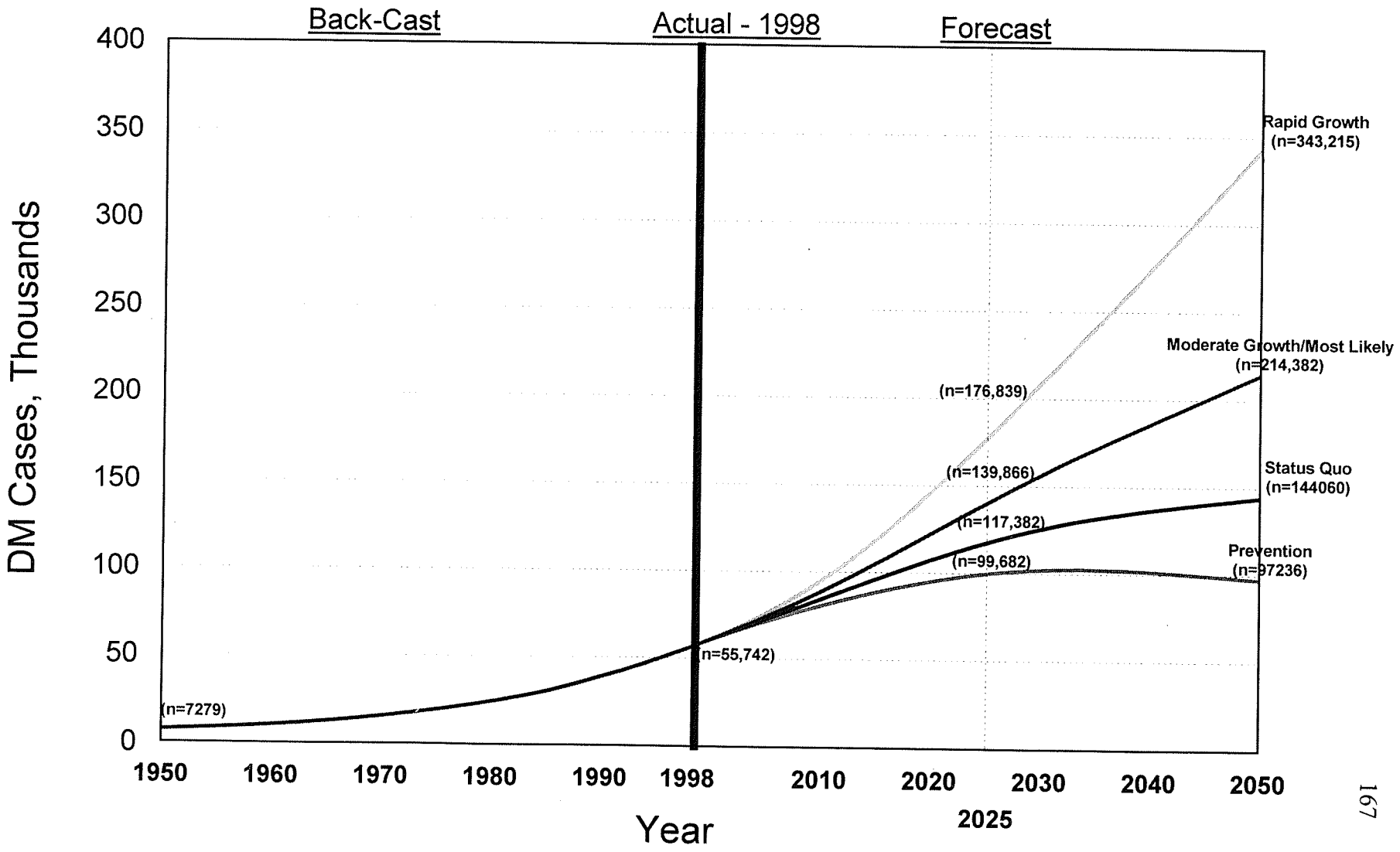


Fig. 7.2B:
Projected growth in DM cases in Manitoba, Registered First Nations, 1950 - 2050

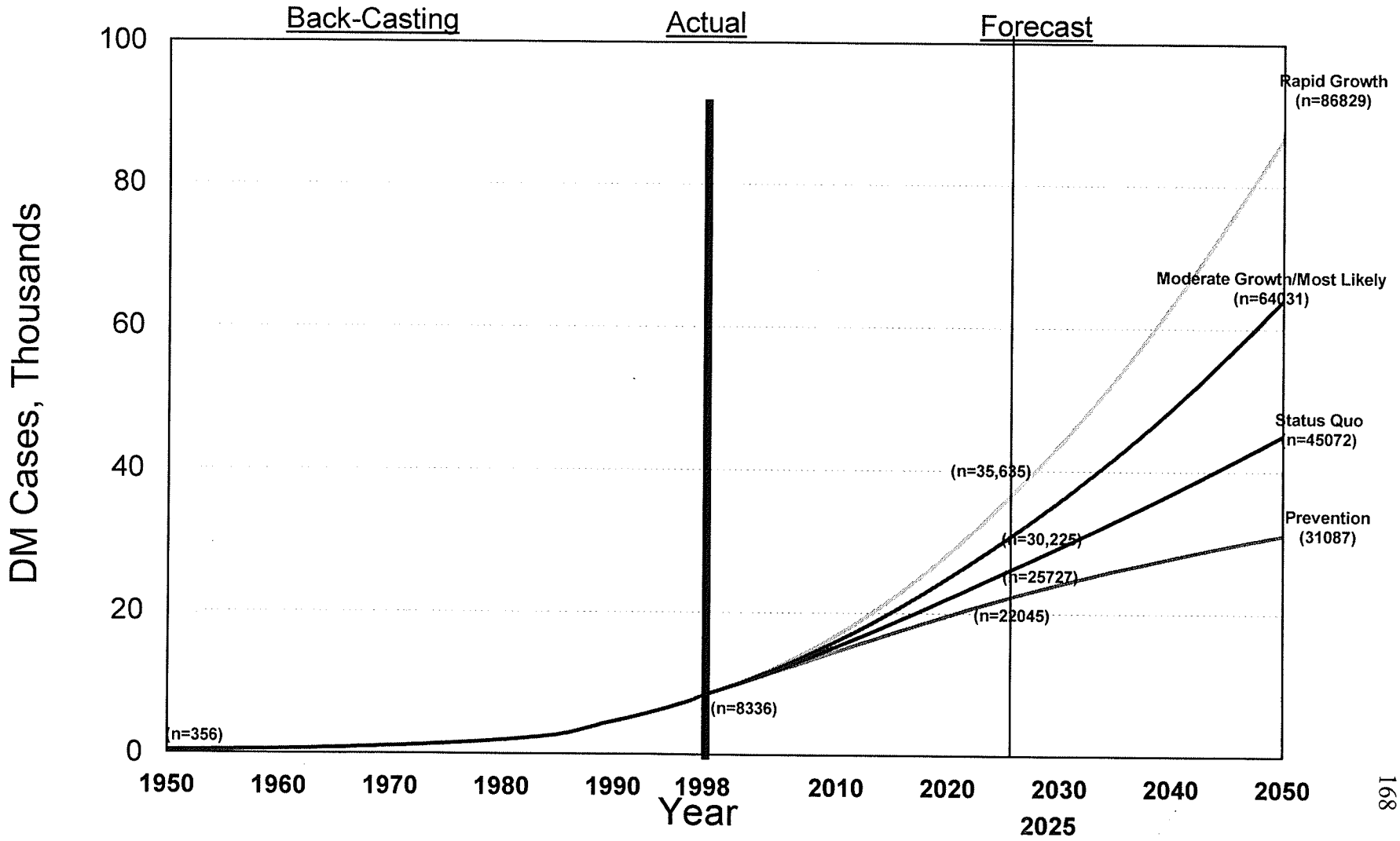


Fig. 7.2C:
Projected growth in DM cases in Manitoba, Non-Registered First Nations, 1950 - 2050

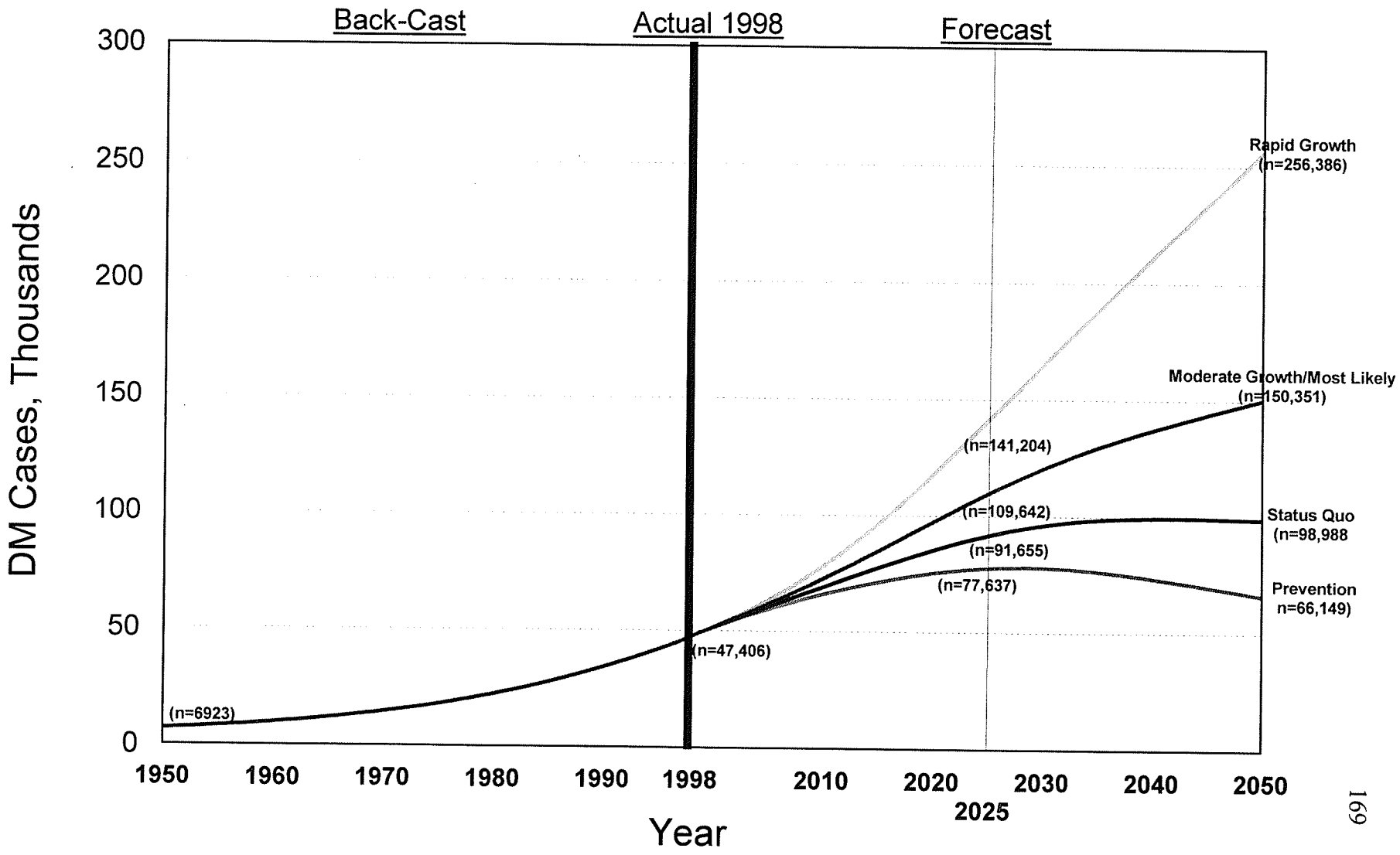
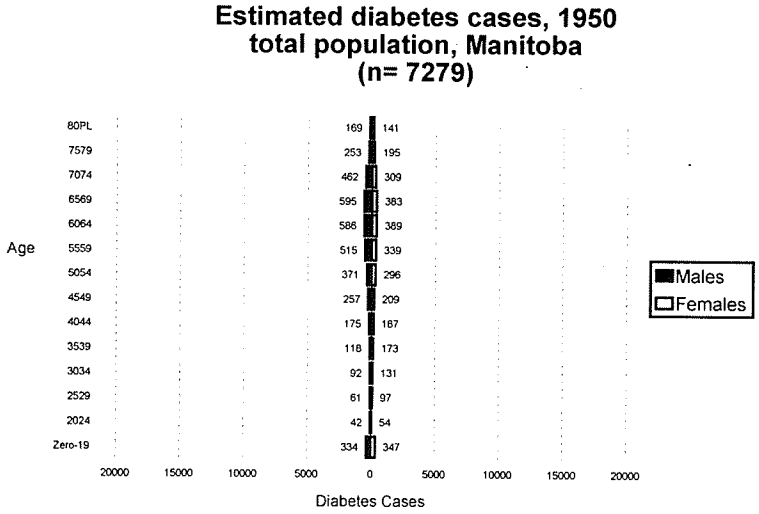
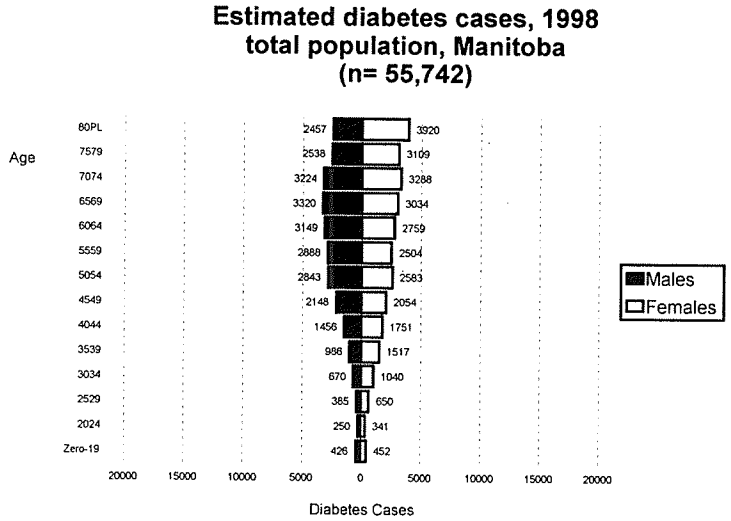
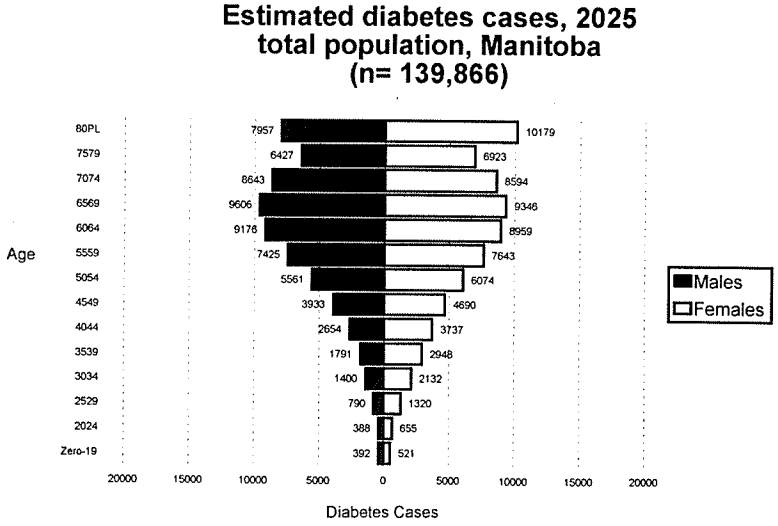


Figure 7.3A:
Age structure of DM cases, total Manitoba population, 1950 - 2050







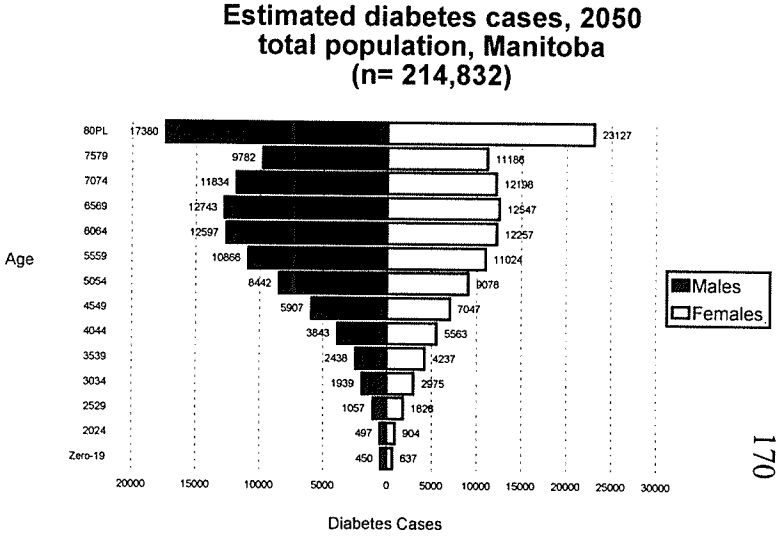
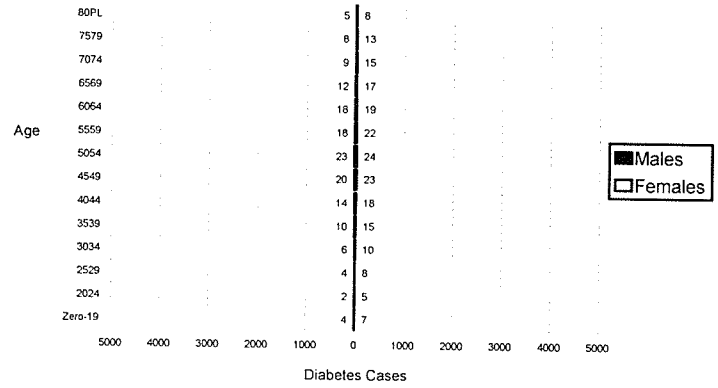
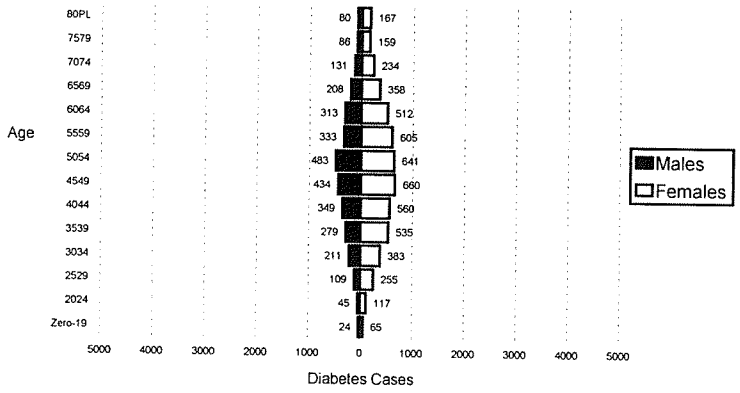


Figure 7.3B:
Age structure of DM cases, 1950 to 2050, Registered First Nations population

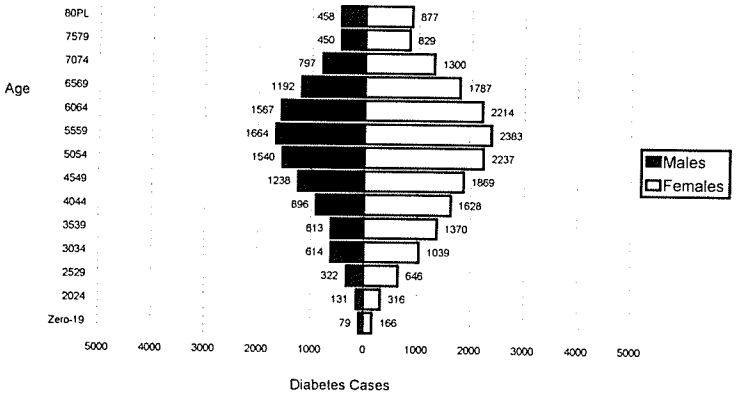
Estimated diabetes cases, 1950
Registered First Nations population, Manitoba
(n= 356)



Estimated diabetes cases, 1998
Registered First Nations population, Manitoba
(n= 8,333)



Estimated diabetes cases, 2025
Registered First Nations population, Manitoba
(n= 30,225)



Estimated diabetes cases 2050
Registered First Nations population, Manitoba
(n= 64,031)

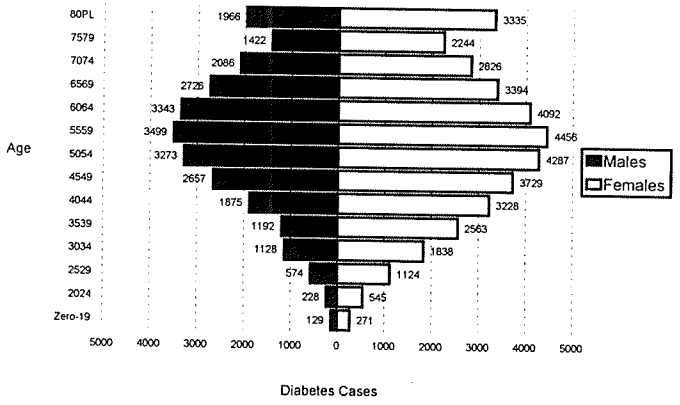
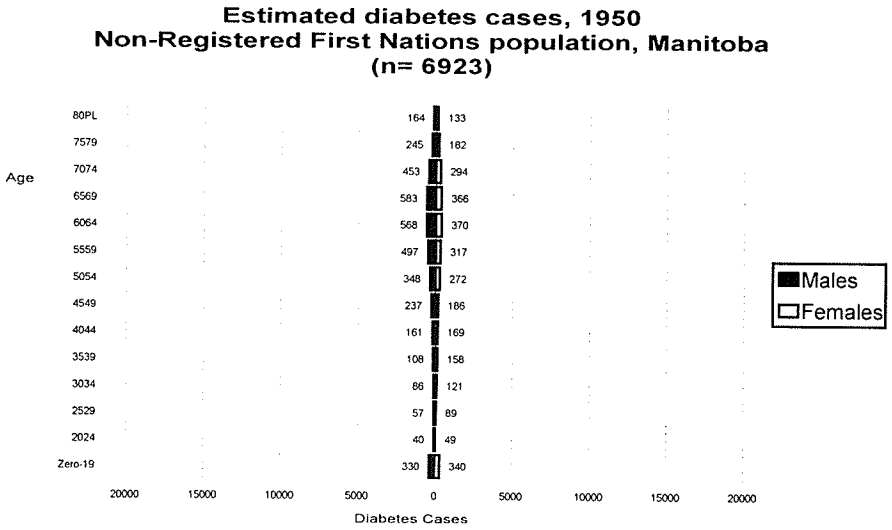
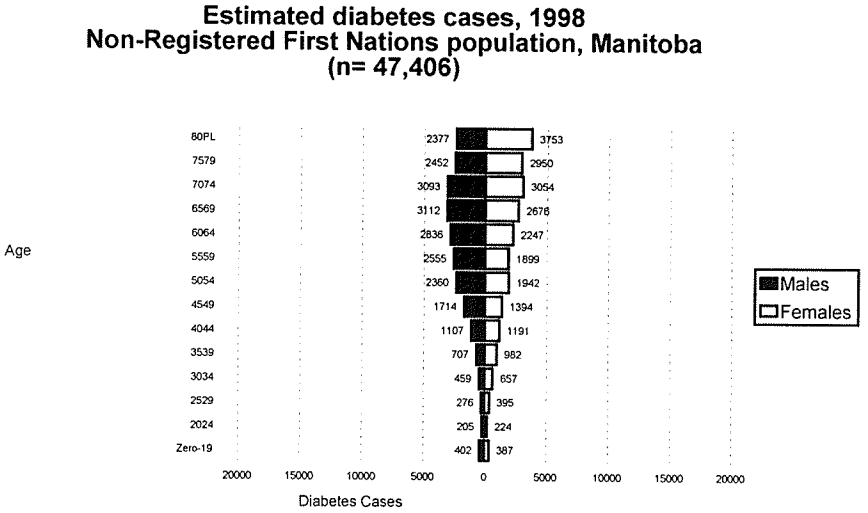
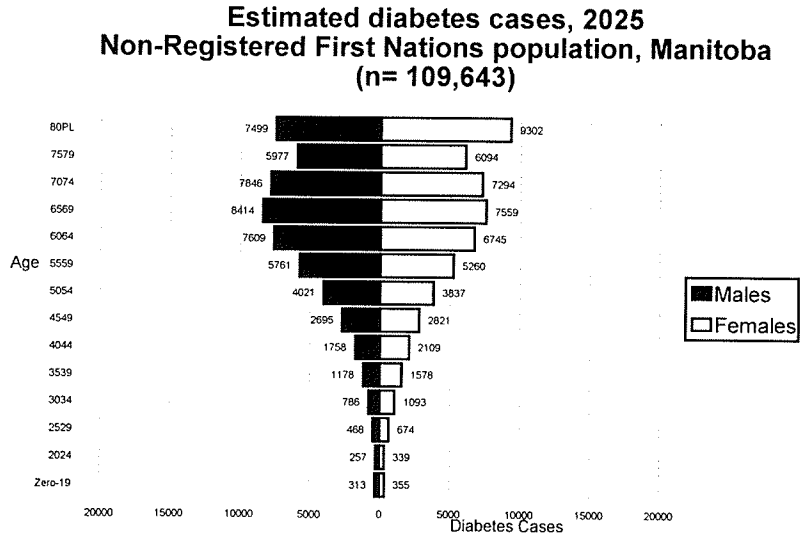


Figure 7.3C:
Age structure of DM cases, 1950 to 2050, Non-Registered First Nations population







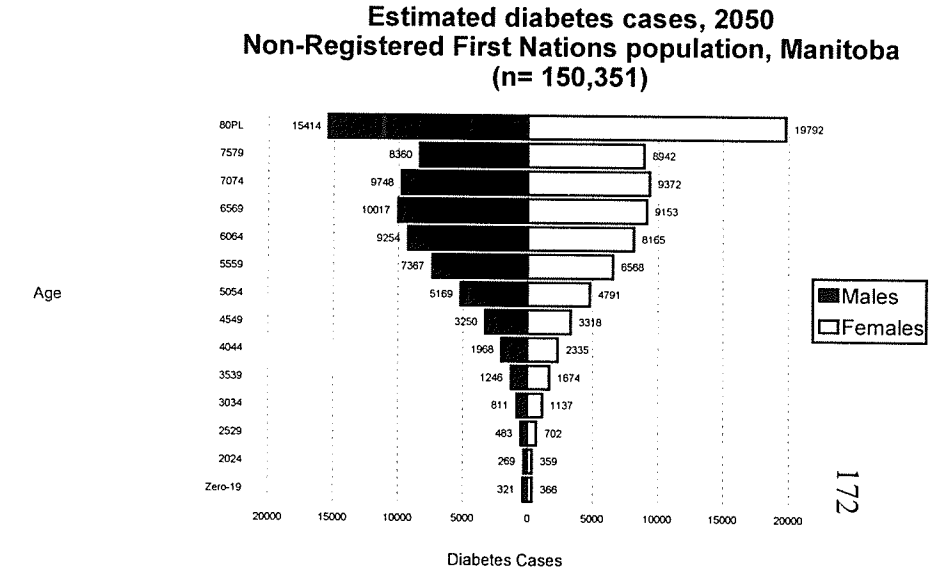
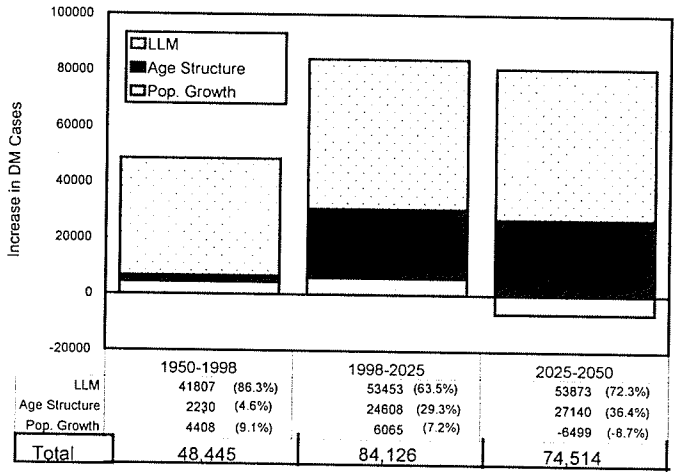


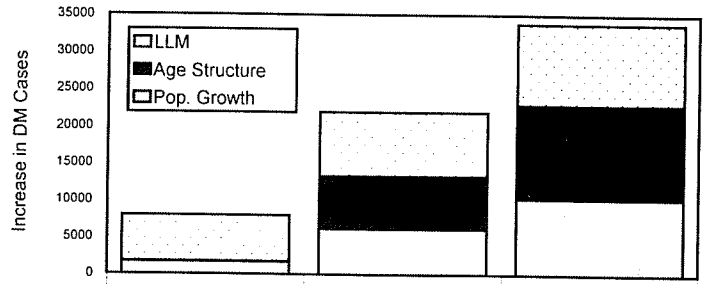
Figure 7.4:
Increase in DM Cases attributable to changes in demography and LLM, 1950 - 2050

Increase in DM Cases Attributable to Changes in Demography and LLM, All Manitobans, 1950 - 2050



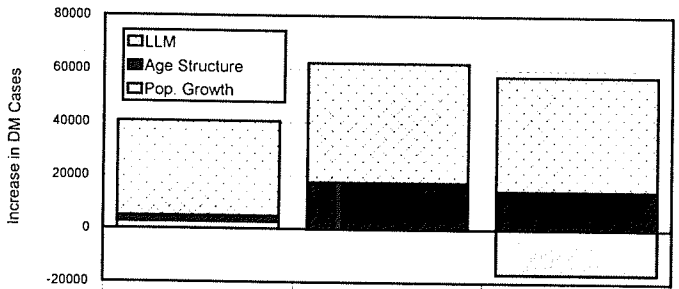
	1950-1998	1998-2025	2025-2050
LLM	41807 (86.3%)	53453 (63.5%)	53873 (72.3%)
Age Structure	2230 (4.6%)	24608 (29.3%)	27140 (36.4%)
Pop. Growth	4408 (9.1%)	6065 (7.2%)	-6499 (-8.7%)
Total	48,445	84,126	74,514

Increase in DM Cases Attributable to Changes in Demography and LLM, Registered First Nations, 1950 - 2050



	1950-1998	1998-2025	2025-2050
LLM	6252 (78.3%)	8645 (39.4)	10927 (32.32%)
Age Structure	27 (0.3%)	7084 (32.3%)	12603 (37.28%)
Pop. Growth	1701 (21.3%)	6160 (28.14%)	10276 (30.39%)
Total	7,980	21,889	33,806

Increase in DM Cases Attributable to Changes in Demography and LLM, Non-Registered First Nations, 1950 - 2050



	1950-1998	1998-2025	2025-2050
LLM	35555 (87.8%)	44808 (72.0%)	42946 (105.%%)
Age Structure	2203 (5.4%)	17524 (28.1%)	14537 (35.7%)
Pop. Growth	2707 (6.7%)	-95 (-0.2%)	-16775 (-41.2%)
Total	40,465	62,237	40,708

Chapter 8

Summary

The main purpose of this thesis research was to describe the spatial and temporal trends in the epidemiology of diabetes mellitus (DM) in Manitoba and their relationship to individual and population level characteristics. This was accomplished through the application of a number of descriptive epidemiological, spatial and demographic techniques. The four papers which make up the body of this thesis have concluded that the number of DM cases in Manitoba have increased dramatically over the past 50 years, and will continue to rise into the foreseeable future as a result of population growth and aging and increasing risk. These papers have also demonstrated that rates of DM vary significantly by a number of individual, geographic and population level characteristics. The highest rates of DM were observed to occur in older age groups, in Aboriginal/First Nation populations, in northern remote and rural First Nation communities, in the central core of the City of Winnipeg, and in populations of low socio-economic status. Despite the gradient in DM risk observed across population groups in this study, all population groups were identified as being at significant risk for developing the disease. The implications of the spatial and temporal trends in DM identified in this study for program, policy and further research are summarized below.

This research provides important insights into the causes of the DM epidemic and the policy, program and research responses required to effectively prevent and manage the disease at the population level. The observations in this research that:

a.) there exists a low level of inequality in DM rates across the majority of geographically defined population groups (low Gini coefficient),

b.) the majority of DM cases have occurred and are anticipated to occur in the non-First Nation population, a population traditionally considered at low risk for the development of DM, and

c.) there is rapid growth of DM cases in all population groups beyond that attributable to the impact of population growth and aging,

collectively suggest that the causes of the DM epidemic are distributed through all segments of Manitoba society.

These observations are consistent with the emerging globalization perspective on chronic diseases which argues that large scale global forces of the late 20th century / early 21st century are leading to an increasing homogenization of ways of life around the world (urbanization, fast and processed food, sedentary lifestyle) and that this is leading to a rapid proliferation of obesity and chronic disease in all population groups (Ritzer, 2003; Sobal, 2001). The process of homogenization is facilitated by increasingly global governmental structures and trade agreements, global corporations, global media and global food systems which are increasingly able to structure and influence the characteristics of local geographic areas and the their populations.

Applied to the Manitoba situation, the globalization perspective suggests that while there may be particular high risk groups (low SES and First Nations groups) less able to maintain good health in the face of deleterious global forces, no segments of society are immune to the obesogenic and diabetogenic effects of the modern western

environment. This suggests that explanatory paradigms which emphasize either genetic predisposition or socio-economic inequalities as the primary cause of the DM epidemic are incomplete explanations for the disease. This is because their more individual and local level focus does not take into consideration powerful forces and trends operating at wider global scales of influence. These explanatory paradigms therefore do not provide a solid basis for preventative program and policy development since they emphasize interventions which focus primarily on high risk individuals and communities. This research has shown that preventative efforts focusing only on populations living in the highest risk geographic areas for DM would have a very limited potential for reducing the overall population incidence of DM, while prevention approaches which aim to reduce the risk of DM in all population groups would have much greater impact.

Alternatively, the globalization paradigm provides a more complete and useful perspective on the causes of the DM epidemic since it has the potential to acknowledge the complex ways in which individual and local level "causes" of DM are embedded within and structured by larger global forces and trends. The globalization paradigm suggests that neither the high risk nor the population based approaches to the prevention of the DM epidemic are likely to be successful in decreasing DM prevalence at either the local or population levels if they target only individual and local level causes, and if they ignore how the DM epidemic is inter-woven into the fabric of everyday life at many scales. This implies that effective population based prevention efforts need to be informed by further research utilizing diverse historical, geographical and anthropological research methods which can identify the range of forces affecting DM risk, and the types and scales of

interventions which may be required simultaneously at individual, local, regional, national, and even international levels to effectively deal with the DM epidemic.

Sobering is the observation in this study that DM cases will still almost double in number by the year 2025 in Manitoba even if prevention efforts are successfully put into place. Given that the "prevention" scenario described in this study is likely overly optimistic given the continued rapid increase in child-hood and adult obesity in Canada (Canadian Population Health Inititiative, 2004), it appears that Manitoba is going to have to prepare for a future in which there will be an extremely high burden of illness from DM and its complications. This means that both the public and policy makers are going to have to come to terms with how to organize and pay for the supports and medical interventions that are going to be required to deal with the serious complications associated with increasing rates of DM. These include kidney failure, limb amputation, loss of vision, and cardiovascular disease. This will be especially challenging in the emerging context of an aging population in which there will be fewer persons of working age generating the wealth required to pay for medical and community support programs.

Over the next 25 years specific programs and policies will need to be put into place which specifically address the impacts of DM and its complications in the Registered First Nation and Aboriginal populations of Manitoba. As described in this study, the burden of illness from DM in the Registered First Nations population is significantly higher than in the general Manitoba population. The results of this study suggest that unless aggressive preventative interventions are put into place soon, the number of cases of DM in the this population will increase 3 to 4 fold by the year 2025. Delivery of primary and secondary

prevention services to the Aboriginal population living in First Nation communities will be especially challenging because of issues related to remote geography and often poorly developed community and health services infrastructure. As well, interventions will need to be designed in such a way that acknowledges the unique cultural background and challenges facing this population (Abonyi, 2001; Young, Reading, Elias & O'Neil, 2000;).

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Appendices

Appendix A: Approvals

- Health Information and Privacy Committee, Manitoba Health
- Ethics, University of Manitoba
- Permission from publishers for use of published manuscripts in thesis document
    - Elsevier (Social Science and Medicine)
    - American Diabetes Association (Diabetes Care)

Appendix B: Statement of contribution to published manuscripts used in thesis document

Appendix C: Copies of Published Papers

- Geographic analysis of diabetes prevalence in an urban area
- The epidemiology of diabetes in the Manitoba Registered First Nation population: Current patterns, comparative trends

Appendix A

Approvals

1. Health Information and Privacy Committee, Manitoba Health
2. Ethics, University of Manitoba
3. Permission from publishers for use of published manuscripts in thesis document
    - Elsevier (Social Science and Medicine)
    - American Diabetes Association (Diabetes Care)

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HEALTH INFORMATION PRIVACY COMMITTEE

June 9, 2003

File No.: 2003/2004 - 01

Mr. C. Green

Dear Mr. Green:

Re: **The epidemiology of diabetes and its complications: An exploration through time and space**

The Health Information Privacy Committee would like to thank you for providing evidence that HIRC is aware of this study and a copy of the ethics approval. This study is now *approved.*

Please note that any significant changes to the proposed study design should be reported to the Chair for consideration.

If you have any questions regarding the Committee's decision, please contact Leonie Stranc, Committee Coordinator at 786-7204.

Yours truly,

Dr. R.Walker
Chair

Please quote the file number on all correspondence

cc. L. Barre

APPROVAL CERTIFICATE

29 May 2003

TO: **Chris Green** (Advisor R. Hoppa)
Principal Investigator

FROM: **Wayne Taylor, Chair**
Joint-Faculty Research Ethics Board (JFREB)

Re: **Protocol #J2003:086**
"The Epidemiology of Diabetes and its Complications in Manitoba: An Exploration through Time and Space"

Please be advised that your above-referenced protocol has received human ethics approval by the **Joint-Faculty Research Ethics Board**, which is organized and operates according to the Tri-Council Policy Statement. This approval is valid for one year only.

Any significant changes of the protocol and/or informed consent form should be reported to the Human Ethics Secretariat in advance of implementation of such changes.

Please note that, if you have received multi-year funding for this research, responsibility lies with you to apply for and obtain Renewal Approval at the expiry of the initial one-year approval; otherwise the account will be locked.

5 January 2005

Our ref: HG/SS/Jan 05/J005

Chris Green
University of Manitoba

Dear Dr Green

SOCIAL SCIENCE AND MEDICINE, Vol 57, No 3, 2003, pp 551-560, Green et al, "Geographic analysis of..."

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>>> Chris Green
I would like permission to use the manuscript I submitted to Diabetes Care as one of the chapters in my PhD thesis.

Citations is:

Green,C, J F Blanchard, T K Young, J Griffith, 2003a, The epidemiology of diabetes in the Manitoba-registered First Nation population: current patterns and comparative trends: Diabetes Care, v. 26, p. 1993-1998

Thanks

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Appendix B

Statement of Contribution to Published Manuscripts used in Thesis Document

Two previously published manuscripts are included in this thesis document. These are:

Chapter 4: Green,C., Hoppa,R.D., Young,T.K., & Blanchard,J.F. (2003). Geographic analysis of diabetes prevalence in an urban area. *Soc.Sci.Med. 57*(3), 551-560.

Chapter 5: Green,C., Blanchard,J.F., Young,T.K., & Griffith,J. (2003). The epidemiology of diabetes in the Manitoba-registered First Nation population: current patterns and comparative trends. *Diabetes Care 26*(7), 1993-1998.

My contribution to both of these manuscripts was:

a. Research design
b. Data extraction and preparation
c. Data analysis
d. Manuscript preparation
e. Coordination of feedback and input from co-authors
f. Submission to publisher

Appendix C

Copies of Published Manuscripts Used in Thesis Document

Green,C., Hoppa,R.D., Young,T.K., & Blanchard,J.F. (2003). Geographic analysis of diabetes prevalence in an urban area. *Soc.Sci.Med. 57*(3), 551-560.

Green,C., Blanchard,J.F., Young,T.K., & Griffith,J. (2003). The epidemiology of diabetes in the Manitoba-registered First Nation population: current patterns and comparative trends. *Diabetes Care 26*(7), 1993-1998

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Geographic analysis of diabetes prevalence in an urban area

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Abstract

The objective of this research is to identify the sociodemographic, environmental, and lifestyle factors associated with the geographic variability of Diabetes Mellitus (DM) prevalence in the City of Winnipeg, Manitoba in Canada. An ecological regression study design was employed for this purpose. The study population included all prevalent cases of DM in 1998 for Winnipeg. Predictor and outcome data were aggregated for analysis using two methods. First, the spatial scan statistic was used to aggregate study data into highly probable diabetes prevalence clusters. Secondly, predictor and outcome data were aggregated to existing administrative health areas. Analysis of variance and spatial and non-spatial linear regression techniques were used to explore the relationship between predictor and outcome variables. The results of the two methods of data aggregation on regression results were compared. Mapping and statistical analysis revealed substantial clustering and small-area variations in the prevalence of DM in the City of Winnipeg. The observed variations were associated with variations in socioeconomic, environmental and lifestyle characteristics of the population. The two methods of data aggregation used in the study generated very similar results in terms of identifying the geographic location of DM clusters and of the population characteristics ecologically correlated to those clusters. High rates of DM prevalence are strongly correlated with indicators of low socioeconomic status, poor environmental quality and poor lifestyles. This analysis further illustrates what a useful tool the spatial scan statistic can be when used in conjunction with ecological regression to explore the etiology of chronic disease.
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Keywords: Diabetes epidemiology; Spatial analysis; Spatial scan statistic; Spatial regression; Racial covariate; Canada

1. Introduction

Type 2 Diabetes Mellitus (T2DM) is one of the most common non-communicable diseases in the world today (Amos, McCarty, & Zimmet, 1997). It is projected that the number of cases of T2DM around the world will increase rapidly over the next 25 years, from 154 million estimated cases in 2000 to 300 million cases in 2025 (King, Aubert, & Herman, 1998). There is great debate about the cause of the T2DM epidemic (Swinburn, 1996). Although there is general consensus that T2DM has both genetic and social roots, there is little consensus on the relative contribution of these factors (Carter, Pugh, & Monterrosa, 1996; Fujimoto, 1996; Haffner, 1998; Hales & Barker, 1992; Hales, Desai, & Ozanne, 1997; McDermott, 1998; Ozanne & Hales, 1998).

This study used two spatial techniques to explore the geographic variability of Diabetes Mellitus (DM) prevalence in the City of Winnipeg, Manitoba. Since 95% of all cases of DM are estimated to be T2DM (Harris, 1995), DM prevalence was used in this study as a proxy for T2DM. A common problem in geographic epidemiology is that observed rates, especially in low incidence or prevalence situations, can often be artefacts of the areal geographic units to which individual events are aggregated for analysis. This can have the effect of rendering invisible small geographic areas that have

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significantly elevated rates of disease (Meade & Earickson, 2000). The spatial scan statistic, the first geographic method used in this study, avoids this problem by iteratively creating a number of statistically significant high- and low-rate cluster areas from small geographic regions. The spatial scan has been used in a number of recent studies to identify spatial clusters of cancer (Hjalmars & Gustafsson, 1999; Hjalmars, Kulldorff, Gustafsson, & Nagarwalla, 1996; Hjalmars, Kulldorff, Wahlqvist, & Lannering, 1999; Jemal, Devesa, Kulldorff, Hayes, & Fraumeni, 2000; Kulldorff, Athas, Feurer, Miller, & Key, 1998a; Kulldorff, Feuer, Miller, & Freedman, 1997; Kulldorff & Nagarwalla, 1995), child-hood mortality (Sankoh, Ye, Sauerborn, Muller, & Becher, 2001), aviation crashes (Grabowski & Li, 2001) and acute respiratory disease in cattle(Norstrom, Pfeiffer, & Jarp, 1999). This spatial scan method is compared to the more traditional approach of aggregating event data to pre-existing and large geographic administrative areas.

Materials and methods

Study setting

The study was conducted in the City of Winnipeg, Manitoba, Canada. Winnipeg has a population of 632,000 and is the only large metropolitan city in the province. Over the past 20 years, Winnipeg has experienced significant social and physical deterioration of its central core and downtown area, paired with rapid growth of its peripheral suburbs. The majority of the population historically have been of European descent. However, because of in-migration from rural communities and natural population increases, an increasing percentage of the population is of Aboriginal descent. Manitoba has a universal health insurance plan and all residents of the province are eligible to receive health care services without cost.

Data sources

Sociodemographic data including self-reported Aboriginal status were obtained from the 1996 Census Canada microdata files. Data on the quality of the physical and social environment in 1999 were obtained from the City of Winnipeg. Data on smoking rates and DM prevalence data for 1998 were obtained from the Manitoba Health Epidemiology Unit. Definitions for the sociodemographic, lifestyle and environmental predictor variables are included in Table 2. The methodology used to generate population-based DM prevalence estimates has been described previously (Blanchard et al, 1996). This method used the standardized case definition of either one hospital visit or two physician visits with a DM diagnosis (ICD 250) within a 2-year period in order to generate DM incidence and prevalence estimates from hospital and physician claims data. This methodology was unable, however, to distinguish between T2DM and DM. Hospital and physician claims data were available for all residents of Manitoba since the entire population is covered by a universal health care program. Population denominator data were obtained from the Manitoba Health population registry of all citizens insured for health services in the province. All data were aggregated initially to the neighborhood level ($n=230$) using the geocoding functionality within the GIS software Arc-View 3.2 (Environmental Systems Research Institute, 1999).

Spatial methods

Two methods were used to explore the geographic variability and clustering of DM within the City of Winnipeg and to identify, using ecological methods, the social and environmental factors associated with variability in DM. The first is the spatial scan method and the second is the pre-existing regions method. Linear regression and analysis of variance were used with both these methods to identify the socioeconomic, environmental and lifestyle factors ecologically associated with this variability. These factors included self-reported Aboriginal status, education, income, family structure, unemployment, housing conditions, crime and smoking rates.

Spatial scan method:

The spatial scan statistic was used to test for the presence of clusters of DM and to identify their approximate location. The open domain software Statscan distributed by the National Cancer Institute was employed for this purpose (Kulldorff, Rand, Gherman, Williams, & DeFrancesco, 1998b). The spatial scan statistic, which works by aggregating together the unique combinations of small-area geographies which have a high probability of being clusters is an especially powerful tool to use in low-prevalence and low-incidence situations. Traditional epidemiological approaches which require that rare events be aggregated to pre-existing higher-level geographies can often mask the existence of real clusters. The statistic assumes the number of cases in each geographic region to be Poisson distributed. The method tests the null hypothesis that within any age and gender group, the risk of having DM is the same as in all regions combined. This means that the expected age and gender-adjusted prevalence rate is constant over the whole area.

The spatial scan statistic places a circular window of varying size on the map surface and allows its center to move so that at any given position and size, the window includes different sets of adjacent neighborhoods. If the

window contains the centroid of a neighborhood, then the whole neighborhood is included in the window. As the window is placed at each neighborhood centroid, its radius is varied continuously from zero up to a maximum radius which never includes more than 50% of the total population. The method creates a large number of distinct circular windows, each containing a distinct set of adjacent neighborhoods, and each a possible candidate for containing a cluster of prevalent diabetes cases. For each window, the method uses a Monte Carlo simulation to test the null hypothesis that there is an elevated risk of DM prevalence. The Statscan software allows any number of covariates to be implemented into the model, and calculates indirectly standardized rates. Details of how the likelihood function is maximized over all windows under the Poisson assumption have been described elsewhere (Kulldorff et al., 1997).

In this study, the Statscan software was applied to the 230 Winnipeg neighborhoods in order to generate possible clusters of DM prevalence. Age and gender were applied as covariates. Two iterations were undertaken. The first iteration used the default setting within the Statscan software which maximizes the cluster size at 50% of the total study population. The second iteration set the maximum generated cluster size at 10% of the total study population. Smaller maximum cluster sizes result in a larger number of smaller clusters with more extreme values. The Monte Carlo simulation used to test significance was set at 999 iterations. The software was set to generate both high and low clusters. Only statistically significant clusters were retained for analysis. Non-cluster areas were aggregated together into one cluster area and assumed to have a relative risk of 1.0. Clusters were initially mapped using Arc-View 3.2 (Environmental Systems Research Institute, 1999) in order to identify their physical location. Social and environmental predictor variables were then aggregated to the cluster areas in order to identify their possible relationship to DM prevalence. Where appropriate, analysis of variance and non-spatial and spatial linear regression techniques were used to formally explore the relationship between predictor variables and DM prevalence.

Pre-existing regions method

In the second method, DM prevalence and predictor data were further aggregated to the 23 Health regions used to organize the delivery of services within the City of Winnipeg. DM prevalence was directly standardized by age and gender to the 1998 Winnipeg population. Chloropleth maps of all variables were generated to visually examine their spatial distribution. Spatial clustering of all variables were assessed using the Moran's I statistic. Non-spatial and spatial regression techniques were used to explore the relationship between predictor variables and the standardized DM prevalence.

Analysis of variance and regression analysis

In both the spatial scan and pre-existing regions methods, variables were log transformed when necessary in order to ensure that the regression assumptions of normality and heteroskedascity were not violated. Regression analyses were undertaken using the S-PLUS Spatial Statistics extension for Arc-View 3.2 (Mathsoft, 1999). A simultaneous autoregressive model was used for spatial regressions. Analysis of variance was undertaken using NCSS (Hintz, 2000).

Results

In Winnipeg in 1998 there were 29,885 prevalent cases of DM, resulting in an overall DM prevalence rate of 47.3 cases/1000 population. Prevalence rates of DM were observed to be higher in men than in women and to increase rapidly in the age 65 and over age group.

Spatial scan results

Table 1 shows the results applying the spatial scan statistic. With the maximum cluster size set at 50% of the total study population, two significant ($P<0.001$) clusters were generated with relative risks of 1.3 and 0.84. Fig. 1 shows that the high relative risk cluster is located in the central and northern core of the City of Winnipeg, while the low relative risk cluster is located in the southern suburbs of the City. The high relative risk cluster is the most likely one, with a log likelihood ratio of 291.56.

With the maximum cluster size set at 10% of the total study population, the spatial scan statistic generated 10 significant ($P<0.009$) high and low clusters. Relative risks ranged from 0.69 to 1.45. Fig. 2 shows that the high relative risk clusters are again all located in the central and northern core of the City of Winnipeg, while the low relative risk clusters are located in the southern suburbs of the city. The most likely cluster, with a relative risk of 1.45, and a log likelihood ratio of 282.79 is located in the central core of the city.

Table 2 shows the predictor variables aggregated to cluster areas generated by the spatial scan method when the maximum cluster size was set to 50% of the total population. This table illustrates that high diabetes prevalence is clustering in those areas of the City of Winnipeg which have a high percentage of Aboriginal population, low educational levels, low family income, a high percentage of lone parent families, high levels of unemployment, high numbers of vacant and placarded houses, high levels of crime, and high rates of smoking.

Table 1
DM prevalence analysis, City of Winnipeg, Manitoba, 1998, using the spatial scan statistic

Max. cluster size	Cluster type[a]	Cases	Expected	RR[b]	LLR[c]	p value
A 50%	High	7335	5644	1.3	291.56	<0.001
	Non	12782	n/a	1.0	n/a	n/a
	Low	9768	11578	0.84	236.17	<0.001
B 10%	High	4101	2825	1.45	282.79	<0.001
	High	912	778	1.171	11.07	<0.009
	High	2056	1820	1.13	15.64	<0.001
	Non	17527	n/a	1.0	n/a	n/a
	Low	2457	2916	0.84	42.16	<0.001
	Low	677	840	0.81	17.47	<0.001
	Low	1799	236	0.76	78.66	<0.001
	Low	199	272	0.73	11.09	<0.009
	Low	362	509	0.71	23.96	<0.001
	Low	464	659	0.70	33.07	<0.001
	Low	243	353	0.69	19.74	<0 001

[a]Cluster type: High—cluster with relative risk > 1, Non—aggregation of non-clustered population, Low—cluster with relative risk < 1.
[b]RR: Relative risk—Observed DM prevalence/expected DM prevalence.
[c]LLR: Log likelihood ratio.

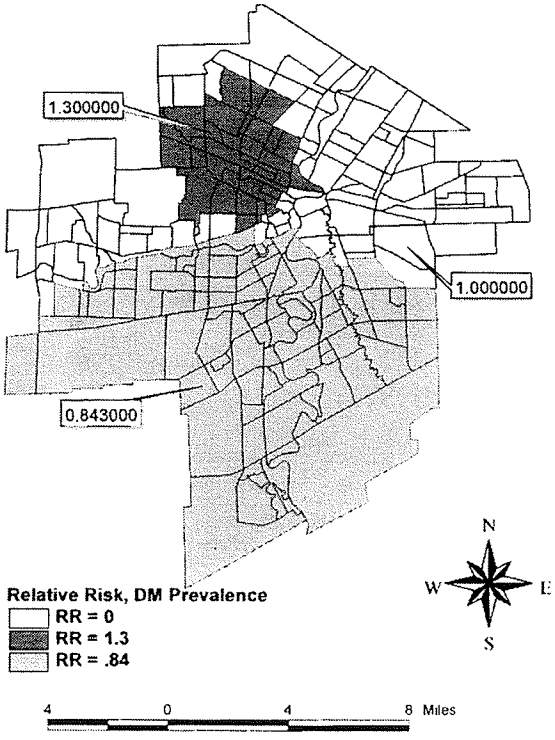

Fig. 1. DM Prevalence Analysis, City of Winnipeg, 1998, using the Spatial Scan Statistic, Maximum Cluster Size set at 50% of Study Population.

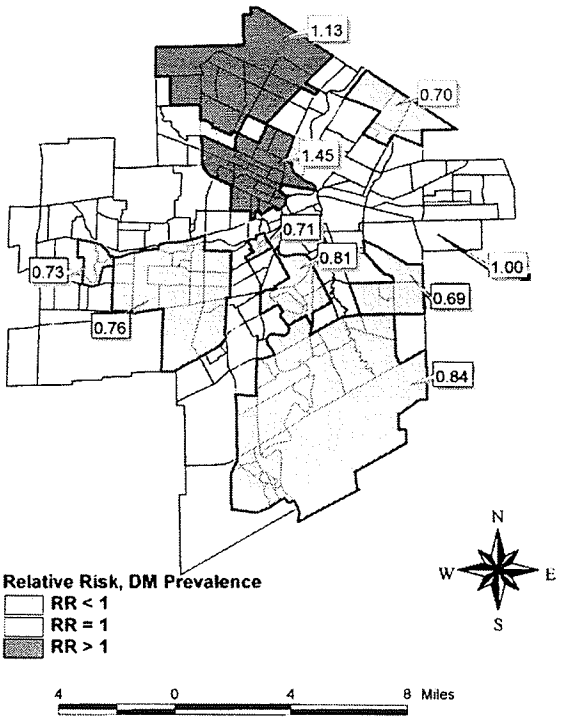

Fig. 2. DM Prevalence Analysis, City of Winnipeg, 1998, using the Spatial Scan Statistic, Maximum Cluster Size set at 10% of Study Population.

Analysis of variance was undertaken for all predictor variables. In all cases, the between cluster variance was significant at the $P<0.005$ level.

Regression analysis of predictor variables against the relative risk of the cluster areas generated when the maximum cluster size was set to 10% produced similar

Table 2
Analysis of variance, predictor variables aggregated to spatial scan generated cluster areas for DM prevalence, maximum cluster size set to 50%

Predictor[a]	Clusters			Analysis of variance
	Low cluster ($RR^b = 0.84$)	Non-clustered ($RR^b = 1.0$)	High cluster ($RR^b = 1.30$)	
Aboriginal status	3.8	5.5	16.9	$p<0.005$
Less than grade 9	5.3	8.9	17.3	$p<0.005$
Average family income	62994	50810	37392	$p<0.005$
Lone parent	13.7	16.1	23.6	$p<0.005$
Unemployment	6.1	7.7	14.5	$p<0.005$
Vacant House	0.6	0.8	15.1	$p<0.005$
Crime	56.6	88.5	157.6	$p<0.005$
Smoking	18.1	26.7	35	$p<0.005$
DM cases by cluster	$n = 9768$	$n = 12,782$	$n = 7335$	
Study population by cluster	$n = 245528$	$n = 258849$	$n = 127623$	

[a] Aboriginal status—% of the population reporting aboriginal status; Less than grade 9—% of the population 15 yr + reporting less than grade 9 education; Average family income—average family income; Lone parent—% of families reporting being headed by a lone-parent; Unemployment—% of the population 15+ in the labor force that is unemployed; Vacant house—no. of houses/1000 residential properties that are vacant or placarded; Crime—no. of crimes against property and persons/1000 population; Smoking—% of mothers of newborns smoking on discharge from hospital.
[b] RR: Relative risk—observed DM prevalence/expected DM prevalence.

Table 3
Regression analysis of DM prevalence relative risk vs. predictor variables, using spatial scan generated cluster areas, maximum cluster size set to 10%

Predictor[a]	Non-spatial regression			Spatial regression		
	R^b	Regression coefficient	p value	Regression coefficient	p value	Residual spatial autocorrelation[c]
Aboriginal Status	0.90	0.034	<0.001	0.0398	<0.001	N.S
Less Than Grade 9	0.97	0.0452	<0.001	0.0456	<0.001	N.S.
Average Family Income	−0.93	−0.0000139	<0.001	−0.000013	<0.001	N.S.
Lone Parent	0.89	0.0342	<0.001	0.0240	<0.001	N.S.
Unemployment	0.93	0.0504	<0.001	0.0558	<0.001	N.S.
Vacant House	0.69	0.0202	<0.001	0.0079	<0.098	$p<0.05$
Crime	0.90	0.00433	<0.001	0.0049	<0.001	N.S.
Smoking	0.88	0.218	<0.001	0.023	<0.001	N.S.

[a] For definitions, refer to footnote a of Table 2.
[b] R Pearsons R.
[c] Spatial autocorrelation of regression residuals. Significance is based upon the Moran's I statistic.

results (Table 3). Non-spatial regression resulted in very high Pearson R values ranging from 0.69 to 0.97, with all regressions significant at the $P<0.001$ level. Education had the greatest predictive value. Spatial regression, which accounts for the spatial clustering of variables in a regression equation, did not appreciably change either the non-spatial regression coefficients or significance levels. With one exception, regression equations generated using spatial regression techniques did not result in any residual spatial autocorrelation, indicating that the regression model was successful in fully accounting for any spatial correlation in the DM prevalence rates.

Pre-existing regions method

When aggregated to the 23 health regions, all variables used in the model were highly spatially clustered. Visual inspection of chloropleth maps showed clustering of DM prevalence in the central core of the City of Winnipeg (Fig. 3), associated with a larger Aboriginal population, low education, low family income, lone parent families, high unemployment, poor housing stock, high crime rates, and high rates of smoking. This visual impression was confirmed by significant Morans I values ($P<0.001$) for all values.

Standardized DM prevalence rates ranged from 37.7/1000 to 78.8/1000 and were significantly different from the mean in all but one region.

Regression of predictor variables against the standardized diabetes prevalence rates for the 23 health regions within the City of Winnipeg generated results similar in strength and direction to the spatial scan analysis. Non-spatial regression again resulted in very high Pearson R values (Table 4). All regression models were significant at the $p<0.001$ level. Unemployment had the greatest predictive value. Spatial regression did not appreciably change either the non-spatial correlation coefficients or significance levels and did not result in any residual spatial autocorrelation.

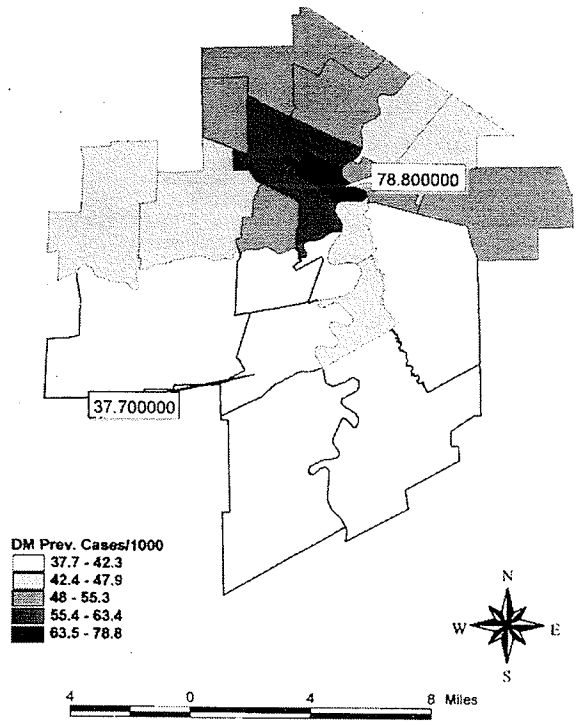

Fig. 3. Standardized Diabetes Prevalence Rates, City of Winnipeg, 1998, by Health Region.

Multiple regression of predictor variables against diabetes prevalence resulted in a model incorporating family income and unemployment, with a Pearsons R value of 0.944. Spatial regression analysis of these predictor variables did not result in any appreciable change in either the regression coefficients or significance levels and did not result in any residual spatial autocorrelation. In this model, diabetes prevalence was positively associated with unemployment and negatively associated with family income. Additional variables could not be incorporated into the model because of the high level of multi-collinearity of predictor variables (Table 5).

Discussion

This study has demonstrated substantial clustering and small-area variations in the prevalence of DM in the City of Winnipeg, and that these variations are associated with variations in socioeconomic, environmental and lifestyle characteristics of the population. This study has also demonstrated that two distinct approaches to spatial analysis, the spatial scan statistic and the pre-existing regions method generate very similar results in terms of identifying the geographic location of DM clusters and of the population characteristics ecologically correlated to those clusters. Finally, our results have shown that when high levels of

Table 4
regression analysis, age standardized DM prevalence rates vs. predictor variables, using existing health boundaries for the City of the Winnipeg, Manitoba

Predictor[a]	Non-spatial regression			Spatial regression		
	R^b	Regression coefficient	p value	Regression coefficient	p value	Residual spatial autocorrelation[c]
Aboriginal Status	0.90	1.009	<0.001	0.969	<0.001	N.S.
Less Than Grade 9	0.90	1.584	<0.001	1.586	<0.001	N.S.
Avg. Family Income	−0.89	−0.0006	<0.001	−0.0006	<0.001	N.S.
Lone Parent	0.86	1.156	<0.001	1.153	<0.001	N.S.
Unemployment	0.92	1.682	<0.001	1.76	<0.001	N.S.
Vacant House	0.74	0.480	<0.001	0.4008	<0.001	N.S.
Crime	0.80	0.110	<0.001	0.1026	<0.001	N.S.
Smoking	0.84	0.729	<0.001	0.766	<0.001	N.S.

[a] For definitions, refer to footnote a of Table 2.
[b] Pearsons R.
[c] Spatial autocorrelation of regression residuals. Significance is based upon the Moran's I statistic.

Table 5
Covariance of predictor variables, Pearsons R, using existing health boundaries for the City of Winnipeg

	Abor	LTG9	Income	Lparent	Unemp	Vacant	Crime	Smoking
Abor	1.0	0.90	−0.82	0.94	0.98	0.83	0.86	0.89
LTG9	0.90	1.0	−0.87	0.87	0.90	0.74	0.77	0.86
Income	−0.82	−0.87	1.0	−0.89	−0.86	−0.78	−0.82	−0.87
Lparent	0.94	0.87	−0.89	1.0	0.96	0.85	0.90	0.93
Unemp	0.98	0.90	−0.86	0.96	1.0	0.83	0.90	0.88
Vacant	0.83	0.74	−0.78	0.85	0.84	1.0	0.89	0.79
Crime	0.86	0.77	−0.82	0.90	0.90	0.89	1.0	0.78
Smoking	0.89	0.86	−0.87	0.93	0.87	0.79	0.79	1.0

For definitions, refer to footnote a of Table 2.

non-spatial correlation exist between predictor and dependent variables, spatial regression approaches do not appreciably change the strength or direction of the regression coefficients.

The study has a number of methodological limitations. First, it has relied exclusively on data derived from administrative databases in order to estimate the DM prevalence rates. Since cases have not been individually verified, this approach could result in either an overestimate or underestimate of prevalence rates. However, we have previously studied the accuracy of this approach and found that the specificity is high when compared to local registries of DM (Blanchard et al., 1996). It is possible that some of the small-area variations that we observed are due to variability in health care access and diagnostic practices. This is unlikely, however, since Manitoba provides universal health care to its residents so restricted access to physician and hospital services is not likely.

Secondly, the administrative databases from which the diabetes prevalence rates were derived cannot distinguish between T2DM and Type 1 insulin-dependent diabetes. However, given that it is estimated that approximately 95% of all DM cases are T2DM it is likely that the variability in DM prevalence observed in this study reflects primarily the impact of T2DM (Harris, 1995).

Thirdly, the small number of observations used in regression analysis within both the spatial scan and pre-existing regions approaches means that regression results must be used with some caution. Tests for normality and heteroscadiscity may not have been sensitive to violations of regression assumptions because of the small number of observations. However, given the strength of the direction and significance of the generated correlation coefficients, and their consistency between the two spatial methods, the observed correlations are likely real and significant.

Fourthly, the ecological approach used in this study has been frequently criticized as being a weak design and commits what is known as the ecological fallacy. The ecological fallacy suggests that it is a mistake to apply characteristics measured at the scale of the population or geographic level to individuals living within those geographies or populations (Morgenstein, 1982, 1995). The ecological design used in this study therefore restricts us to making statements about the characteristics of the populations living in specific geographies. Statements made about individuals living within those geographies can only be made with caution. However, given the arguments by Rose and others (Rose, 1985, 1992; Wilkinson, 1996, 1999) on the primary importance of population and geographic level factors on population health, this study design legitimately provides important clues to the etiology of DM at the population level. It suggests that DM prevalence at the population level is powerfully graded by socioeconomic status, environmental quality, and lifestyle.

Finally, the study used only one lifestyle variable, smoking in mothers of newborns on discharge from hospital, as a proxy for overall lifestyle quality. Given that this variable may be a relatively weak proxy for lifestyle attributes relevant to DM, caution must be taken in concluding that lifestyle is associated with diabetes prevalence at the ecological level. Lifestyle measures more directly related to DM prevalence such as diet, exercise and obesity were not available at the geographic levels required for this study.

The high level of consistency between the results of the spatial scan statistic and the pre-existing regions method in identifying etiological factors associated with DM is encouraging. Previous studies that have used the spatial scan statistic to identify cancer clusters have not attempted to systematically explore possible etiological factors associated with clusters using analysis of variance and linear regression (Hjalmars et al., 1996, 1997, 1999; Jemal et al., 2000; Kulldorff et al., 1998a; Kulldorff and Nagarwalla, 1995; Sankoh et al., 2001; Walsh & Fenster, 1997). This study suggests that the spatial scan statistic in conjunction with analysis of

variance and linear regression may be a useful tool in exploring the etiology of cancer and other chronic diseases.

The relationship observed between DM prevalence and low levels of socioeconmic status, environmental quality and lifestyle at the geographic level is consistent with previous studies (Auslander W F, Haire-Joshu, Houston, & Fisher E B, 1992; Hanis, Chakraborty, Ferrell, & Schull, 1986; Hazuda & Monterrosa, 1992; Hendricks & Haas, 1991; Leonetti, Tsunehara, Wahl, & Fujimoto, 1992; Marshall et al., 1993) . This study provides some of the strongest evidence to date of this relationship, with DM prevalence estimates based upon diabetes prevalence estimates covering the whole population of Winnipeg. Previous studies have not been population based and were often restricted in scope to limited surveys of specific sub-populations.

This study demonstrates that the highest rates of DM are occurring in geographic areas that have the highest concentration of Aboriginal people. It has been hypothesized that populations of Aboriginal, Black, and Mexican American origin are genetically predisposed to develop T2DM supposedly due to the high frequency of the "thrifty gene" in their respective population gene pools. The thrifty gene, it is proposed, conferred an adaptive advantage in historical times of feast and famine. However, in modern conditions of relative plenty, the thrifty gene predisposes individuals to the development of obesity and increased frequency of DM (McDermott, 1998; Neel, 1962, 1982, 1999). In this study, it was observed that the geographic areas with the highest prevalence of DM also had the lowest socioeconomic status, the poorest lifestyles, and the lowest levels of environmental quality. Regression analyses demonstrated that broad neighborhood characteristics such as education and income were more predictive of DM prevalence than Aboriginal status. Once family income and unemployment were used in the regression analysis as predictors, Aboriginal status lost all of its significance as a predictor of DM. This suggests that it may be more the impact of low socioeconomic status that is putting populations at risk of DM in Winnipeg rather than genetic background. This also suggests that population-based studies using race as a covariate need to critically question their use of racial constructs by examining the social and physical circumstances in which particular racially defined groups find themselves. These studies may need to examine whether it is these circumstances which are predisposing these groups to disease and ill-health rather than something inherent in their "race" (King, 1997). There may indeed be a genetic component that confers some variability in DM between individuals, but at the level of the population it appears that larger socioeconomic and environmental factors are more important. Further studies which stratify the analysis by Aboriginal status in order to explore whether the socioeconomic and environmental gradients in DM prevalence observed in this study apply to the non-Aboriginal population alone would add strength to these conclusions.

The high level of multi-collinearity observed between predictor variables also suggests that attempts at disentangling the independent relationships between these variables and DM prevalence may be counter-productive since all predictor variables may in fact be measuring aspects of the same phenomena (Evans & Barer, 1994; Hertzman, Frank, & Evans, 1994; Marmot, 1999; Wilkinson, 1996, 1999). This phenomenon is likely related to social position, access to real life choices, and a sense of personal empowerment. This suggests that ecological studies utilizing socioeconomic predictors should start to locate their analyses within well-developed perspectives on how the social position of particular groups become established, spatially concentrated, reproduced over time, and results in poor health outcomes. The specific pathways by which low social position becomes translated into poor health outcomes through the generalized stress response, poor lifestyle practices, and reduced opportunities are becoming increasingly recognized (Baum, Garofalo, & Yali, 1999; Cohen, 1999; Kawachi, 1999; Lundberg, 1999; McEwen & Seeman, 2001; Pickering, 1999; Williams, 2001).

This study raises questions about how we need to better understand the powerful and predictable impact that place has on the health of populations. This study has documented that geographies in the central core of Winnipeg are associated with high levels of DM. These core area neighborhoods are places that seemed to have emerged as gathering places for individuals low on the social hierarchy with few social choices. This has likely occurred as a result of historical, political, and economic forces. The result has been the transformation of the physical and social fabric of these geographies into places of risk with ecological characteristics having strong association with health status. In order to more fully understand how this has happened over time, the unique history of how these high-risk geographies have evolved over time have to be explored more carefully through the use of diverse historical, political, economic and ethnographic methods.

Finally, the results of this study suggest that high rates of DM are tightly embedded within a context of poverty and disempowerment. Population-based prevention programs which focus only on lifestyle modification would likely not be successful. As illustrated by this study, lifestyle quality indicators like smoking are highly correlated with income and education. This suggests that DM prevention programs, to be successful, would require comprehensive policy interventions above and beyond lifestyle modification. These interventions have

to address the socioeconomic resources and opportunities available to individuals.

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The Epidemiology of Diabetes in the Manitoba-Registered First Nation Population

Current patterns and comparative trends

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OBJECTIVE — This study provides an overview of the epidemiology of diabetes in the Manitoba First Nation population.

RESEARCH DESIGN AND METHODS — The study uses data derived from the population-based Manitoba Diabetes Database to compare the demographic and geographic patterns of diabetes in the Manitoba First Nation population to the non–First Nation population.

RESULTS — Although the prevalence of diabetes rose steadily in both the First Nation and the non–First Nation populations between 1989 and 1998, the epidemiological pattern of diabetes in these two populations differed significantly. The First Nation population was observed to have age-standardized incidence and prevalence rates of diabetes up to 4.5 times higher than those found in the non–First Nation population. The sex ratio and the geographic patterning of diabetes incidence and prevalence in the two study populations were reversed.

CONCLUSIONS — The results of the study suggest that diabetes prevalence will likely continue to rise in the Manitoba First Nation population into the foreseeable future, and that the impact of this rising diabetes prevalence can only be effectively managed through a population-based public health approach focusing on primary and secondary prevention. The dramatically higher rates of diabetes in Manitoba First Nation population as compared with the non–First Nation population highlight the urgency of this activity. These prevention efforts need to be supported by further research into the reasons for the unique epidemiological patterns of diabetes incidence and prevalence in the First Nation population observed in this study. These include investigating why First Nation populations living in the Northern areas of the province seem to be protected from developing high rates of diabetes and why First Nation women experience much higher rates of the disease.

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Diabetes is increasingly responsible for substantial morbidity and mortality in Canada's First Nation populations. Planning and implementing effective primary and secondary intervention programs to deal with this disease and its devastating effects in First Nation people requires accurate population-based data on the temporal trends and geographic distribution of diabetes (1).

This study uses Manitoba Health administrative databases to examine trends in the incidence and prevalence of diabetes among Registered First Nation adults in Manitoba from 1989 to 1998. Comparisons are made to the non–First Nation adult population to highlight the magnitude of the diabetes epidemic in First Nation people. The geographic variation in diabetes rates across Manitoba is also examined.

The study was conducted in the Canadian province of Manitoba. Manitoba has a population of 1.14 million people, of whom more than one-half (645,000) reside in the City of Winnipeg, the provincial capital. The majority of Manitobans are of European descent, whereas ~10% of the population is self-identified as having Aboriginal ancestry (2). Manitoba has a universal health insurance plan, and all residents of the province are eligible to receive health care services with no payments required at the time of service.

RESEARCH DESIGN AND METHODS

Data sources

The data used for this study were derived from the Manitoba diabetes database (MDD), which has been described previously (3). This database contains a longitudinal record for Manitoba residents of all physician contacts and hospital separation records that cited a diagnosis of diabetes (ICD-9-CM code 250) between 1 April 1984 and 31 March 1999. Individuals are categorized as having diabetes if they have had at least two separate physician contacts for diabetes within 2 years of each other or at least one hospital separation for diabetes. Cases of gestational diabetes are excluded from the MDD. The sensitivity of this database for detecting clinically diagnosed cases of diabetes has been demonstrated, and the validity of the methodology has been discussed previously (3). This methodology is unable to distinguish between type 2 and type 1 di-

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Abbreviations: CCA, community characterization area; MDD, Manitoba diabetes database; RHA, regional health authority.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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abetes. Individuals who move from the province subsequent to being identified as having diabetes are included in the MDD only for the years that they are residents in the province.

Manitoba's First Nation population is comprised of individuals who are registered under the Indian Act of Canada and living in Manitoba. To identify people belonging to Manitoba's First Nation population, the Manitoba Health population registry was used. As part of the process for registration within Manitoba Health's insurance system, an attempt is made to verify whether new registrants are registered as Indian. If so, their First Nation affiliation is recorded. It is important to note that for a variety of complex historical and political reasons many individuals of Aboriginal ancestry are not eligible for registration under the Indian Act of Canada. These individuals are therefore not identified as First Nation in this study. Also, approximately only 70% of the registered First Nation population living in the province of Manitoba is identified as being First Nation in the Manitoba Health population registry.

Estimating incidence and prevalence

The diagnosis date of incident cases was defined based on the first physician contact for a diagnosis of diabetes, which was followed within 2 years by a subsequent physician contact or by the hospitalization for diabetes, whichever came first. The annual incidence rates were calculated using the mid-year population at risk based on the Manitoba population registry. The population at risk is the population at mid-year without diabetes. The average annual incidence rates for the years 1994–1998 were computed by cumulating all incident cases and summing the mid-year population at risk for those years. The point prevalence at December of each year was estimated by determining the number of cases that had previously been diagnosed who had neither died nor left the province at that time. This was done using the Manitoba Health population registry, which is routinely updated and closely matches census population estimates. The average annual prevalence rates for 1994–1998 were computed by cumulating the point prevalent cases for each of the 5-year periods and summing the mid-year populations for those years. Average annual incidence and prevalence rates for 1994–1998 were calculated to maximize rate stability when making geographic comparisons. To facilitate comparisons across time and with other published reports and studies, age-adjusted rates were computed for both incidence and prevalence by the direct method using the 1991 Canadian population as the standard.

To examine the geographic variations in cases of diabetes, the province was divided into 22 regions. The City of Winnipeg was divided into 12 community characterization areas (CCAs). CCAs are administrative areas used by the Winnipeg Regional Health Authority to deliver health services. CCAs were used in this study because >60% of the province's population resides within Winnipeg. Rural Manitoba was divided into 10 regional health authority (RHA) areas. The two northern RHAs of Burntwood and Churchill were combined for this study to maintain the population size required to ensure stable rate calculations. The average population size of each region was ~50,000. Age-adjusted incidence and prevalence rates for each region were calculated using the 1991 Canadian population as a standard.

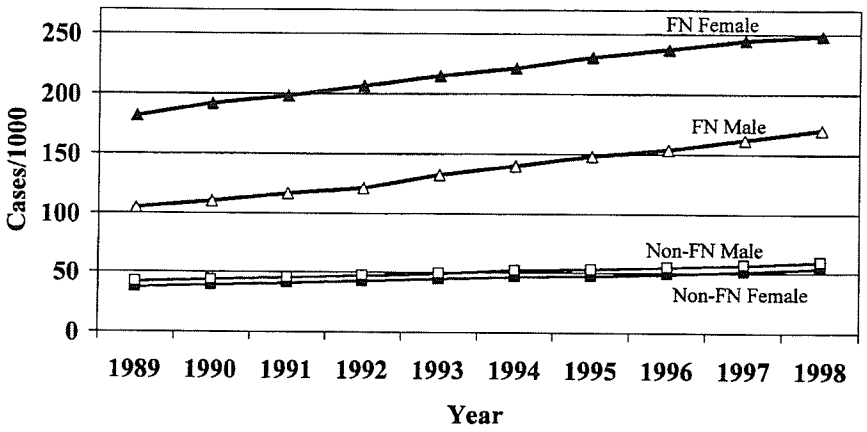

Figure 1—*Point prevalence of diabetes in the Manitoba population. Aged ≥20 years; standardized to the 1991 Canadian population. FN, First Nation.*

RESULTS

Time trends

The age-adjusted prevalence of diabetes among individuals aged ≥20 years increased steadily between 1989 and 1998 for First Nation and non–First Nation men and women (Fig. 1). Among First Nation women, the age-adjusted prevalence increased by 37% from 181.6/1,000 in 1989 to 248.7/1,000 in 1998. Among First Nation men, there was a 1.6-fold increase from 104.2/1,000 in 1989 to 170/1,000 in 1998. There were also increases in prevalence among non–First Nation men (1.4-fold, from 41.9 to 59.63/1,000) and women (1.4-fold, from 37.1 to 53.5/1,000). Throughout the study period, First Nation men had an average diabetes prevalence rate 2.5 times higher than non–First Nation men, whereas First Nation women had an average diabetes prevalence rate 4.5 times higher than non–First Nation women.

The age-adjusted incidence of diabetes among individuals aged ≥20 years increased slightly for First Nation men and Non–First Nation men and women between 1989 and 1998. The incidence in First Nation women did not change during this period. Among First Nation men, there was 1.4-fold increase from 15.3/1,000 in 1989 to 21.1/1,000 in 1998. There were also increases in incidence among non–First Nation men (1.25-fold, from 5.44/1,000 to 6.8/1,000) and women (1.2-fold, from 4.7/1,000 to 5.7/1,000). Throughout the study period, First Nation men had an average diabetes incidence rate three times higher than non–First Nation men, whereas First Nation women had an average diabetes incidence rate 3.7 times higher than non–First Nation women.

Age- and sex-specific incidence and prevalence

Age-specific incidence and prevalence of diabetes were substantially higher among the First Nation population as compared

Table 1—*Age-specific and age-adjusted prevalence (per 1,000 adults) of diagnosed diabetes in Manitoba, 1998, First Nation population compared with non–First Nation population*

	First Nation population		Non–First Nation population		Comparison
	Cases	Prevalence per 1,000	Cases	Prevalence per 1,000	Prevalence ratio*
Sex and age-group (years)					
Women					
20–24	78	26.79	261	7.47	3.58
25–29	173	57.99	433	12.23	4.74
30–34	262	94.14	743	19.468	4.83
35–39	343	143.81	1,129	25.14	5.72
40–44	368	203.20	1,335	31.09	6.53
45–49	393	313.39	1,666	43.30	7.22
50–54	410	415.82	2,135	64.77	6.41
55–59	333	471.67	2,145	84.27	5.59
60–64	316	539.25	2,406	110.06	4.89
65–69	216	535.9	2,836	133.47	4.01
70+	359	509.21	10,249	150.95	3.37
Total (crude)	3,251	185.63	25,338	62.68	2.96
Age adjusted†	—	248.70	—	53.54	4.64
Men					
20–24	28	9.93	210	5.90	1.68
25–29	73	26.85	305	8.55	3.14
30–34	133	51.01	481	12.56	4.06
35–39	184	80.98	788	17.35	4.66
40–44	232	139.33	1,157	26.77	5.20
45–49	302	236.86	1,778	46.57	5.08
50–54	351	336.20	2,454	74.30	4.52
55–59	262	340.25	2,617	104.95	3.24
60–64	218	371.37	2,864	135.63	2.73
65–69	162	390.36	3,214	163.72	2.38
70+	217	317.25	8,307	184.73	1.71
Total (crude)	2,162	128.27	24,175	63.62	2.01
Age adjusted†	—	170.01	—	59.38	2.86
Total (crude)	5,413	157.50	49,513	63.13	2.34
Age adjusted†	—	209.70	—	56.06	3.74

*Ratio of First Nation prevalence to non–First Nation prevalence. †Adjusted to the 1991 Canadian population.

with non–First Nation people in all age-groups. Whereas the incidence and prevalence among non–First Nation individuals increased steadily with age, among the First Nation population both incidence and prevalence peaked in the 60- to 69-year-old age-group and began to fall in the group aged ≥70 years. The highest prevalence difference between First Nation and non–First Nation women occurred in the 45- to 49-year-old age-group, with a prevalence ratio of 7.2 (Table 1). In men, the highest prevalence difference occurred in the 40- to 44-year-old age-group (prevalence ratio of 5.2).

In the First Nation population, both incidence and prevalence of diabetes were higher among women than in men. The opposite trend was observed in the non–First Nation population.

Distribution by geographic region

Although the incidence and prevalence of diabetes was substantially higher among the First Nation than the non–First Nation population in all geographic areas, the geographic patterns differed (Fig. 2). In the First Nation population, the highest prevalence of diabetes was among those who lived in the southwestern rural area of the province (290/1,000) and in the eastern and western sections of the City of Winnipeg. The lowest prevalence was among those living primarily in the more remote northern rural areas of the province and in the southern section of the City of Winnipeg. In contrast, among non–First Nation populations, the highest rates were found in the more remote northern rural areas of the province and in the central downtown area of the City of Winnipeg. Similar patterns were observed for diabetes incidence.

Relationship among incidence, mortality, and increasing prevalence

Figure 3 illustrates that the annual incidence of diabetes exceeded annual mortality in the First Nation population with diabetes between 1989 and 1998, leading to net annual increases in diabetes cases over the entire study period.

CONCLUSIONS— The prevalence of diabetes in First Nation population observed in this study are comparable with those observed in other studies. Harris (4), using data collected through an intensive community-wide prevalence survey, reported age standardized prevalence

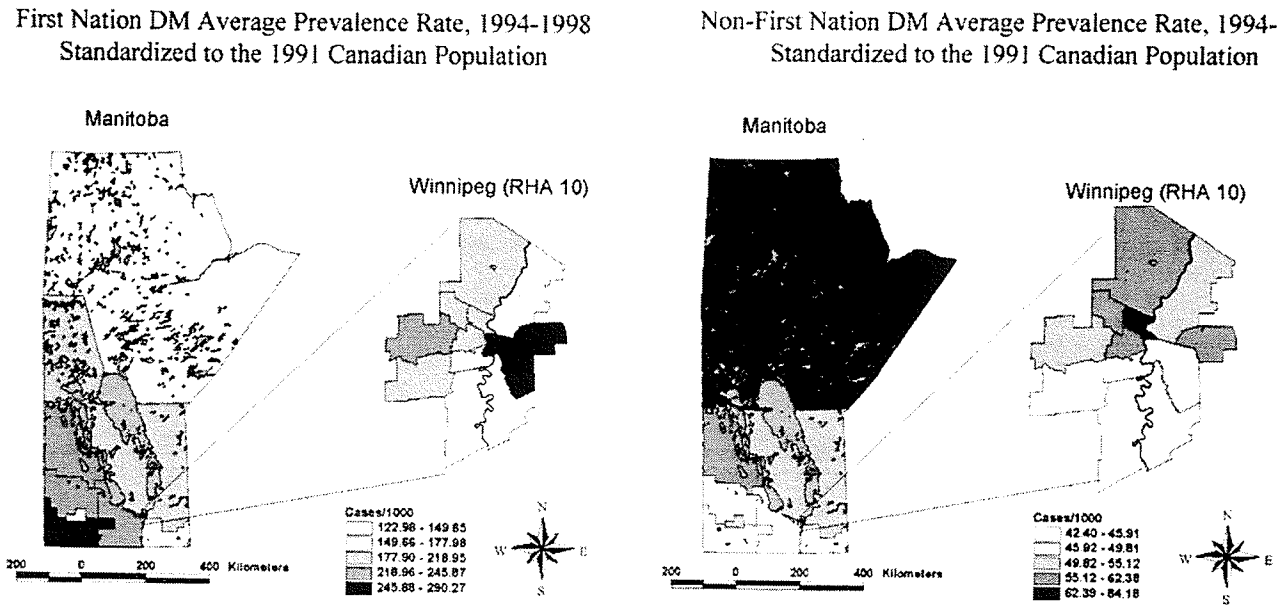

Figure 2—*Geographic variation in diabetes (DM) prevalence. First Nation population compared with non–First Nation population.*

rates (standardized to the 1991 Canadian population) of diabetes in the Sandy Lake First Nation population of 280/1,000 and 242/1,000 for women and men, respectively. This compares with rates of 248/1,000 and 209/1,000 for First Nation women and men observed in this study. Schraer et al. (5) and Burrows et al. (6), drawing upon data derived from a variety of patient registries, observed somewhat lower age-specific prevalence rates in American Indians and Alaska Natives between 1990 and 1997. Consistent with this study, however, both Schraer et al. (5) and Burrows et al. (6) observed increasing prevalence of diabetes over time.

It is important to note that prevalence comparisons between these studies are difficult because of differences in data capture methodologies, age ranges, time frames, and age standardization techniques used.

This study has a number of methodological limitations that must be kept in mind when interpreting its results. First, it has relied exclusively on data derived from administrative databases to estimate diabetes prevalence and incidence rates. Since this approach depends upon diabetes cases being recognized, diagnosed, and recorded through routine interaction with the health care system, it most certainly underestimates the actual incidence and prevalence of diabetes. As described by Young and Mustard (8) and Harris et al. (9), up to one-third of diabetes cases are undiagnosed. Some of the increasing prevalence of diabetes observed in this study may actually have been due to movement of individuals from the undiagnosed diabetes pool as a result of increased screening by health care providers. Also, as a result of the recent lowering of the cutoff point for diagnosing diabetes in Canada from a fasting plasma glucose level of 7.8 to 7 mmol/l, the pool of undiagnosed individuals with diabetes has increased. Movement of individuals from the undiagnosed to the diagnosed diabetes pool may be an important factor in driving future increases in observed diabetes prevalence. Despite likely underestimation of diabetes incidence and prevalence, the specificity of our approach is high when compared with existing registries of type 2 diabetes (3) and to abstracted patient charts of randomly selected physicians (7).

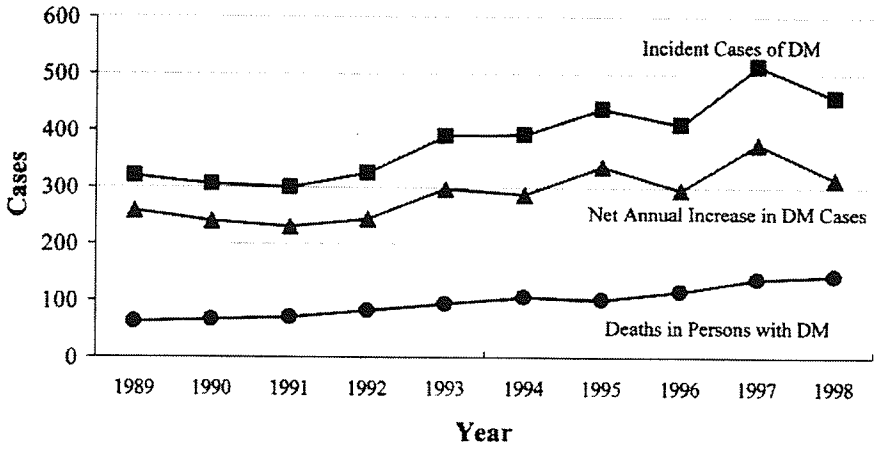

Figure 3— *Incident cases of diabetes (DM), diabetes-related deaths, and net annual increase in diabetes cases, registered First Nation, Manitoba, 1989–1998.*

Second, the restriction of First Nation designation in this study to those individuals registered under the Indian Act of Canada and the under-recording of First Nation status in the Manitoba Health Registry likely diminishes the observed differences between the First Nation and the non–First Nation population. This is probably not a major issue because those

individuals identified as First Nation are highly likely to be of First Nation ancestry, whereas those missed and thus classified as non–First Nation are unlikely to affect the rate in the much larger non–First Nation population.

Third, this study does not differentiate between type 1 and type 2 diabetes because the case definition for diabetes used in the generation of MDD from administrative databases is based upon ICD-CM-250. No subclassifications exist in these administrative databases, which would allow differentiation between type 1 and type 2 diabetes. However, given that type 2 diabetes makes up ~90–95% of all diabetes cases, the use of diabetes in this study is likely a valid proxy for type 2 diabetes (10).

The results of this study have two important implications for First Nation diabetes prevention and management programs. First, it appears that diabetes prevalence rates will almost certainly continue to rise in the Manitoba First Nation population over the next two decades. As illustrated in Fig. 2, as long as the number of incident cases of diabetes exceeds the number of deaths in individuals with diabetes, diabetes prevalence will continue to rise. The rapid aging of the currently very young First Nation population into high-risk older age-groups, paired with emerging life-prolonging diabetes treatments, will maintain the spread between incidence and mortality into the foreseeable future. Even if incidence rates were flat or declining due to a breakthrough in diabetes prevention, prevalence rates would continue to rise as incidence outpaces mortality. This observation is consistent with others (11,12) who are predicting worldwide increases in diabetes prevalence over the next several decades in all population groups.

As a result, the health burden due to all types of diabetic complications will likely continue to rise in the Manitoba First Nation population. This means that the health care and social service systems should start preparing now to provide the secondary prevention and support services and systems a large number of First Nation adults with diabetes are going to require to maintain quality of life. These include diabetes-screening programs, foot-care programs, accessible dialysis services, dietary counseling services, and enhanced infrastructure at the community level to facilitate independent living by adults with limited mobility and eyesight.

Second, "upstream" population-based primary prevention programs need to be aggressively implemented to ensure that diabetes incidence among the First Nation population begins to decrease in the future. The dramatically higher rates of diabetes in the Manitoba First Nation population as compared with the non–First Nation population highlight the urgency of this activity. Because diabetes appears to be closely related to the adoption by First Nation people of many aspects of the modern lifestyle including diet and low levels of physical activity, prevention programs that draw upon Aboriginal traditions and ways of life and that focus on the lifestyle habits of Aboriginal youth need to be implemented (13,14). Currently, almost 50% of the First Nation population is <20 years of age and still in the process of forming lifelong lifestyle habits that will affect their future susceptibility to developing diabetes and its complications. A number of very promising primary prevention programs that draw upon Aboriginal traditions and ways of life have been implemented across Canada (15–21).

The results of this study are also suggestive of a number of future research priorities. First, the observation in this study of lower diabetes prevalence in more northern and remote areas of the province suggests that living in these areas has a protective effect on diabetes. Further research is required to determine if this is due to a greater adherence to traditional lifestyle practices, such as hunting, fishing, and consumption of wild game, to broader community level factors, or to genetic factors.

Second, the reason for the higher prevalence of diabetes in First Nation women observed in this study also needs to be better understood. The relationship to earlier episodes of gestational diabetes should be investigated as one possible pathway that increases the susceptibility of First Nation women to diabetes.

In conclusion, this study has demonstrated the value of having accurate population-based information on the epidemiology of diabetes in the First Nation population. By providing information on the trajectory and the geography of the diabetes epidemic in the First Nation population and the intensity of the epidemic in comparison with non–First Nation populations, it provides important clues as to the magnitude and structure of the primary and secondary intervention programs that will be required to effectively manage this disease in First Nation people. It also highlights the important need to undertake further research into the community and individual-level factors that appear to place some First Nation population groups at lower risk for developing diabetes.

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