Diabetes in the James Bay Cree Communities of Québec, Canada

by

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Faculty of Graduate Studies and Research

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The text of the above shall be cited in full in the introductory sections of any theses to which it applies.

Abstract

Using chronic disease registers, a list of physician-diagnosed diabetics was drawn up for the 8 James Bay Cree communities (total population 8840) Medical records were then consulted to ascertain the clinical histories of the patients The World Health Organization definition of diabetes was applied to diagnosed cases and those not meeting the biochemical criteria were eliminated. A total of 235 persons with diabetes were found, giving a total prevalence of 2.7%. Prevalence of type II was 5.2% in the 20 years and older age group Prevalence varied from 1.9% in the northernmost village to 9.0% in the southernmost Cree community. The Cree diabetic population is characterized by high levels of obesity and low rates of macrovascular diseases. Duration of disease and poor glycemic control (as reflected by the type of medication needed) influences the presence of microangiopathies

Résumé

Une liste des cas de diabète chez les Cris de la Baie James fut obtenue à partir des régistres de malades chroniques. Les dossiers médicaux furent consultés afin de recueillir l'histoire clinique. Les cas ne remplissant pas les critères biochimiques de l'Organisation Mondiale de le Santé furent éléminés. 235 diabétiques furent découverts dans cette population de 8840 individus donnant une prévalence de 2.7%. La prévalence du diabète de type II est de 5.2% chez les 20 ans et plus. Celle-ci varie du village le plus au nord avec 1.9%, à celui le plus au sud avec 9.0%. Le diabétique Crie est caractérisé par un taux d'obésité élevé et un faible taux de maladies macrovasculaires. Les microangiopathies sont associées à l'accroissement de la durée de la maladie de même que avec la faible qualité du contrôle glycémique telle que reflétée par le type de médication reçu.

Introduction

Within the last 40 years, diabetes mellitus prevalence rates have increased steadily in many native populations of Canada with rates now being 2-5 times higher than in all other Canadians (1, 2, 3). There is a general agreement that non-insulin dependent diabetes mellitus (NIDDM), the most frequent form of diabetes, has a genetic basis, but environmental factors have also been recognized as important in contributing to the increasing rate. Indeed, prior to the 1940's, diabetes was rare in native populations of Canada, then sedentarization occurred accompanied by a decrease in physical activity, increased carbohydrate and caloric intake and high rates of obesity and all these factors favored the emergence of NIDDM (4).

The James Bay Cree have not eluded this modernization process and are now undergoing a social, economic and demographic transition that reveals an increasing emergence of chronic disease such as cancer, heart disease, hypertension and diabetes compared to a decline of more traditional health problems like infectious diseases (5).

For the Cree population of Northern Québec, the rate of hospitalisation for diabetes is twice that of the total population of Québec (6). Concomitantly, great concern emerged from the communities and the medical care personnel as they sensed an increasing number of diabetic cases in their communities. In response to this concern, the ongoing round table on community health on which representatives of the CCSSSBJ, the MNQ, and the two CLSC coordinators were present, decided to emphasise diabetes research in Region 10-B for 1989-90. Thus, it was to obtain a better view of the extent of the disease in the Cree population that this research project was conceived, planned and executed.

The general objective was to conduct a comprehensive descriptive study of diabetes among the James Bay Cree in order to add to a body of already existing knowledge for other native populations of Canada. Specific goals were to: a) obtain prevalence rates of the two major types of diabetes; b) describe socio-demographic-anthropometric and biochemical characteristics of the diabetic individuals; c) identify and describe resulting medical

complications; d) describe medical care practices directed towards the diabetics; e) identify risk factors associated with the presence of complications, more particularly microvascular complications which are the most specific complications of diabetes.

This information, we hope, would increase our understanding of the disease thereby allowing more effective prevention by means of appropriate intervention programs.

This study will provide baseline data on Cree diabetics and could serve two purposes. first, provide a data base for planning interventions or needs for medical resources planification, secondly, it could also provide baseline indicators to evaluate potential prevention activities or programs.

This thesis will use manuscripts of papers to be submitted for publication as its format It will also include a review of pertinent literature. An overview will emphasize the original contribution of the thesis to the overall body of knowledge and will describe limitations of the study. Each of these sections, as well as this introduction, will include its own bibliography

The first chapter (paper) consists of the prevalence study. It describes the study population and case finding procedures. The second chapter (paper) focuses on clinical features of the diabetic patients. It tells us who Cree diabetics are with their anthropomorphic, biochemical and life-style characteristics. It also describes how they were diagnosed, what medication they are taking, how the glycemic follow-up is performed, type and rate of medical complications and how often clinical acts pertaining to general care of a diabetic patient are carried out. Finally, the third chapter (paper) focuses on factors associated with obesity, microvascular complications and glycemic control.

In order to overcome a certain brevity associated with publishing requirement, some chapters (papers) will include appendices to allow the reader a more precise judgment as to the scope of the research reported.

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Review

History

Diabetes mellitus is a clinically and genetically heterogeneous group of disorders that have one common feature; abnormally high levels of glucose in the blood.

Diabetes was originally considered to be a disorder characterized by the passage of large amounts of sweet urine leading to death over the course of a few weeks or months. The name "diabetes" was introduced by Aretaeus, a Roman physician of the first century A.D.(1). Interest in the disease increased in the latter part of the nineteenth century. In 1875, Bouchardat (1) recognized that, clinically, at least two forms of diabetes existed in man, one characterized by obesity and the other occurring usually in younger persons regardless of their weight. Further developments in clinical diabetes awaited the ability to measure the blood sugar or blood glucose level in a systematic and reliable manner. The subsequent discovery and isolation of insulin in 1921 (2) and its successful application to reduce hyperglycemia and prolong life in human diabetes, strengthened the concept that the underlying lesions were in the pancreas.

In the latter part of the 1930's, diabetic retinopathy and diabetic nephropathy were recognized as specific complications, not only of subjects with diabetes who had received insulin therapy for a number of years, but also of persons with less severe degrees of hyperglycemia who had never received insulin (1). During the next two decades there was increasing recognition that both insulin-treated and non-insulin-treated diabetics frequently suffered from these and other less specific forms of vascular disease, and that vascular complications caused much more disability and premature death among diabetics than non-diabetics. In the 1950's and 1960's interest in the earlier recognition of the disease, with the hope that earlier treatment would prevent development of the complications, led to the widespread adoption of the glucose tolerance test to detect the disease at an early stage and before the appearance of lasting hyperglycemia. This test led to the identification of

asymptomatic subjects with abnormally high one- or two-hour post-load glucose levels, but with normal fasting values. This was defined as impaired glucose tolerance (IGT).

Also, the potential significance of impaired glucose tolerance in pregnancy became widely recognized (3). It was found that impaired glucose tolerance, sometimes manifest only during the third trimester of pregnancy, could lead to increased perinatal mortality and morbidity (4). The term gestational diabetes was used to describe such circumstances.

Meanwhile, observations also led to the concept that diabetes could result from resistance to the action of insulin, as well as from a lack of insulin (5). This finding contributed to the concept of heterogeneity of diabetes. Association between diabetes and certain genetic markers in the human lymphocytes antigen (HLA) system, and subsequently with islet cell and other autoantibodies, led to further characterization of insulin-dependent diabetes (6). These markers were not found in non-insulin-dependent diabetes; thus a further criterion for the separation of the two most frequent types of the disease became available. A large number of specific genetic syndromes accompanied by hyperglycemia were also described, and diabetes became a recognized complication of a number of therapeutic agents (6).

Classification

It became apparent, therefore, that the growth in knowledge of the etiology and pathogenesis demanded revision of both the classifications and the diagnostic criteria for diabetes. In 1979, the American Workgroup of the National Diabetes Data Group (NDDG) suggested revised classification and diagnostic criteria (7) which subsequently were endorsed by the Expert Committee on Diabetes of the World Health Organization (WHO) (7). In addition, they were reviewed by professional members of the American, British, Australian and Canadian diabetes societies and the European Association for the Study of Diabetes, in addition to other organizations (8). Table 1 serves as a model for categorizing patients and as a general guide for determining appropriate treatment modalities for individuals who have been diagnosed as having diabetes. In addition, the classification is being used as a framework for

clinical and epidemiological research, so that comparative data can be obtained on the various forms of diabetes and other classes of glucose intolerance. Table 1 presents the different clinical presentations and genetic and environmental etiologic factors that permit discrimination among the types of diabetes.

Diagnosis

Diabetes may present with one or more of the classical symptoms such as thirst, polyuria, and weight loss, or sometimes coma. Glucose will be present in the urine and the diagnosis can be made on the basis of a blood glucose estimation without provocative tests. The WHO recommendation is that a random plasma glucose level greater than 11.1 mmol/L, or a fasting venous plasma glucose concentration of 7.8 mmol/L or over on more than one occasion, are themselves sufficient to establish the diagnosis. If the fasting glucose level does not consistently exceed 7.8 mmol/L or if doubt remains as to whether or not the subject has diabetes, the only way to establish the diagnosis is to perform an oral glucose tolerance test (OGTT). There is universal agreement that the diagnosis should not be based on the presence of glucosuria alone, nor can it be excluded on the basis of urinalysis, because of the low sensitivity of the test. In the past, the interpretation of diagnostic tests for diabetes was hindered by a lack of uniformity in procedures and in agreement on criteria for abnormality (1). Now a standardized method of performing the OGTT has emerged as the only formal provocative test recommended for the diagnosis of diabetes. Never theless, the OGTT is not necessary for diagnosis in the majority of patients. The difficulty in arriving upon agreed criteria occurs in large part because in most populations the distribution of glucose levels, fasting or following a glucose load, is unimodal with some skewing towards higher values. Consequently, no obvious cut-off point to distinguish between those with and without diabetes was apparent. However, several populations have been described with high frequencies of diabetes in which bimodal frequency distributions of glucose concentrations are seen and provided appropriate cut-off points to distinguish between normal and diabetic subpopulations (9). Further studies on populations with unimodal glucose distribution confirmed earlier

inference made on the basis of the bimodal frequency distribution, that only individuals falling in the hyperglycemic component were at risk of developing complications (10, 9).

In general, it is believed that venous plasma provides the best specimen for determination of glucose values, although whole blood or capillary blood is acceptable provided the differences in the results are accounted for in interpreting the test. Whole-blood glucose determination (except in anemia) are approximately 15% lower than the corresponding plasma values; the capillary values are about 8% higher than the whole-blood values after glucose loading (1).

Insulin-dependent diabetes mellitus (IDDM), sometimes termed type 1 diabetes, is generally characterized by the abrupt onset of symptoms, insulinopenia, dependence on exogenous insulin to sustain life, and proneness to ketosis even in the basal state. In contrast to IDDM, patients with non-insulin dependent diabetes mellitus (NIDDM) are not dependent on exogenous insulin for preventing ketonuria and are not prone to ketosis. However, they may require insulin for correction of fasting hyperglycemia if this cannot be achieved with the use of diet or oral agents, and they may develop ketosis under special circumstances such as severe stress precipitated by infections or trauma. Although onset in most patients who develop NIDDM is in adult years, the disease also occurs in young persons who do not require insulin and are not ketotic and hence could not be considered to have IDDM. Although some NIDDM patients may be treated with insulin, this alone does not indicate that they should be classified as having developed IDDM. The development of complete insulin deficiency, characteristic of persons with IDDM, in patients with NIDDM appears to be only about 3 percent (11). Persons with NIDDM may be relatively asymptomatic for years and show only very slow progression of the disease. However, typical chronic complications may also occur, including micro- and macroangiopathy, such as renal and retinal lesions, neuropathy, accelerated atherosclerosis, peripheral vascular disease, and amputations. NIDDM etiology, as for IDDM, remains unclear.

Natural History

In general, a number of stages relating to the course and development of diabetes may be defined which apply to both common types of the disease. The first is genetic susceptibility. Second is the state of potential abnormality of glucose tolerance, a statistical risk class, including subjects who have normal glucose tolerance but who by virtue of other characteristics have a substantially increased risk of the development of diabetes. Third is impairment of glucose tolerance, a phase in the development of NIDDM, and also recognized in IDDM. However, not all people with impaired glucose tolerance progress to diabetes. Fourth is diabetes mellitus without complications, a stage in which chronic hyperglycemia is present and symptoms attributable to hyperglycemia may or may not be present. Fifth is diabetes with vascular complications, but without associated symptomatology or disability. Last is diabetes with disability, a stage in which complications of diabetes lead to functional impairment. Depending upon the particular complications this may be end stage renal disease, blindness, cerebrovascular disease, peripheral vascular disease or coronary heart disease. Potentially, intervention at any of these stages may prevent progression to a later stage (12).

Prevalence

About 2.7% of the U.S. population (estimates are the same for Canada) declared through a self-report survey having diabetes in 1987 (13, 14). NIDDM is by far the most prevalent type of diabetes, comprising about the 90 to 95% of all diagnosed cases of diabetes. IDDM constitutes about 5 to 10 percent (7).

NIDDM was rare in many populations, particularly traditional-living, but with progressive modernization of life-style, NIDDM has become prevalent and almost endemic in certain American Indians and Pacific Island populations (15). A number of problems arise in attempting comparisons of the prevalence of NIDDM between and within populations over time or in different geographic locations in the same period. The principal problem is the use of different diagnostic criteria. Also, apparent trends may be confounded by local practices of

population screening. Furthermore, mortality rates or migration can influence prevalence estimates. Data on NIDDM prevalence arise from two sources: identification of already known cases or from epidemiologic surveys identifying previously and newly diagnosed diabetics. Both methods have their problems in interpretation. In the first, there is no certain way of estimating underdiagnosis due to subjects not presenting to medical clinics, nor is there any way of estimating either under or overdiagnosis due to inappropriate criteria. Furthermore, a variable proportion of insulin-treated patients could be considered part of the NIDDM category. Prevalence rates for diabetes based on cases of known diabetes from health interviews or chronic registry almost certainly underestimate the true situation. The magnitude of this estimate will vary according to the screening procedures done in many diverse population groups. It is thought that for every known diabetic, there are at least one or two undiagnosed cases (16, 14). Recently, in a western Canada Indian community, 29% more cases were found following screening compared to the existing physician diagnosed cases (17). As for epidemiologic surveys, until recently (16) they lacked agreed-upon diagnostic criteria and often failed to test a representative sample.

Nonetheless, knowledge of the prevalence of diabetes in the population provides important information that could serve several purposes. Prevalence can be used to estimate the required resources in hospital and outpatient facilities, nutritional and patient education services, and medical specialists required to care for diabetics. Prevalence data can also be used to assess the risk of harbouring diabetes, to explore hypotheses about causal factors, and to plan programs for control and prevention of the disease. Prevalence data can also be used to estimate the community impact of diabetes and to place this disease in perspective with other competing priorities, and to determine the appropriate allocation of resources for diabetes.

The highest diabetes prevalence rates reported in the literature are those of the Pima Indians with 35% of the Pima aged 15 years or more (15). Prevalence rates are also high in a number of other American Indian populations (18), other Pacific Island populations (19), Australian aborigines (20), and migrant Indian communities (21). High prevalence rates in

North American Indians are a relatively modern phenomenon. Indeed, in these populations, diabetes was an uncommon diagnosis until the 1950's (21).

Rural-urban and migrant studies clearly indicate that there are factors relating to modernization which precipitate diabetes. Diabetes prevalence rates are much lower in rural areas (19). Yet the same ethnic groups when urbanized show high diabetes prevalence rates (22).

Apart from rural-urban comparisons, migration studies have provided useful epidemiological data on NIDDM. Indeed, Japanese, Polynesians, Jewish, Asians and Mexican Americans are migrant populations with a higher risk of NIDDM than the original population (15, 24, 22, 23).

In each of these situations, the role of environmental factors appears very important in the development of diabetes. While high diabetes prevalence rates have been reported in a number of populations which have undergone rapid acculturation, direct evidence of a secular change in prevalence rates is lacking, apart from the Pima Indians (15).

Risk Factors

Risk factors for NIDDM have been reviewed extensively by the Canadian Diabetes Association (14) and more recently by Jarret (22) and Barrett-Connor (25). The prevalence of NIDDM increases with age. Women seem to be at higher risk of contracting the disease but reports are not consistent. Prevalence studies conducted on Mexican Americans as well as in the Pima Indians show that the levels of disease prevalence are directly proportional to the degree of inheritance supporting genetic contribution to the etiology of diabetes. High concordance rate in pairs of monozygous twins and the higher risk of first degree relatives of patients with NIDDM only confirm heredity as a precursor of NIDDM.

Modernization of traditional ways of living of American Indians have led to the reduction of physical activity with increased sedentarization and change in traditional diet reflected in ingestion of more lipids and refined sugars, and the emergence of obesity. These

factors seem to favor the emergence of NIDDM in the presence of genetic susceptibility. The risk of developing NIDDM is much increased if glucose tolerance is impaired. About 11% of subjects with impaired glucose tolerance (IGT) become diabetic within 5 years and 21% are diabetic after 10 years (26). But, notably, glucose tolerance improved in 53% of those originally identified with IGT. Thus, progression to diabetes is far from inevitable. Hyperlipidemia, particularly hypertriglyceridemia has also been associated with an increased risk of developing NIDDM (27)

Complications

The public health concern with NIDDM is predominantly its associated morbidity and increased mortality. In addition to metabolic abnormalities, diabetes is also characterized by a greatly increased risk of subsequent vascular and neuropathic disease. Epidemiological studies have focused primarily on the vascular lesions (28). Retinopathy and neuropathy are the result of microvascular disease highly specific for diabetes mellitus. The risk of such lesions is strongly dependent on the duration of diabetes and perhaps dependent on the level of hyperglycemia (29, 30, 22). Microvascular lesions are found in all forms of diabetes and across many ethnic groups (28). Macrovascular disease manifested as coronary, peripheral vascular, or cerebrovascular disease is also significantly associated with diabetes and is the most frequent complication. About 75 to 80% of diabetics die from consequences of atherosclerosis (14) compared to about a third of North Americans. The Framingham study also showed increased mortality and morbidity related to atherosclerosis in diabetic patients (31). Macrovascular disease is a much less specific outcome of diabetes than microvascular disease, sharing other risk factors such as hyperlipidemia, hypertension, obesity or tobacco use. Moreover, macrovascular disease is not clearly related to duration of diabetes nor severity of hyperglycemia and is not frequently found among diabetics from societies with low rates of atherosclerosis (32). In addition, there is more and more evidence supporting the thesis that an increased risk of coronary heart disease precedes the development of significant hyperglycemia

(22, 33). Indeed Haffner et al (33) have recently showed that pre-diabetic subjects have an atherogenic pattern of risk factors (especially hyperinsulinemia), which may be present for many years and may contribute to the risk of macrovascular disease as much as the duration of clinical diabetes itself.

Treatment

Until recently it was thought that medical treatment would reduce the incidence of complications and hence reduce morbidity and mortality. The largest controlled clinical trial to date, the University Group Diabetes Program Study (34), revealed no benefit of improved glycemic control over periods of 10 to 14.5 years of follow-up. No corresponding significant decreases in retinopathy or cardiovascular mortality or morbidity were found. No recent work contradicts these findings (12). A major trial is now in progress to help determine whether improved blood glucose control is beneficial in preventing or delaying chronic diabetic complications in IDDM (35). Furthermore, several recent reviews of screening policy have concluded that screening for diabetes in the non-pregnant adult is not justified (12, 36). Indeed, available, adequate and standardized follow-up procedures and demonstration of efficacy of treatment are needed before screening can be considered beneficial.

In diabetes, a number of suggested methods of prevention, treatment and care are available but relationship between care process and patient outcome, as reflected by their glucose levels, are poorly correlated (37, 38, 39). The effect of other "softer" variables such as health beliefs and social support on regime adherence are now being considered as important as proper medical treatment in community based multidisciplinary approaches to diabetes (40, 41, 42).

Conclusion

With the recognition by the scientific community of the NDDG and WHO diagnostic criteria for diabetes the result of properly standardized epidemiologic study worldwide should

have an important bearing on many aspects of diabetes including etiology, natural history, treatment and prevention of diabetes. The definition of the main risk factors for diabetes and its complications may influence, through appropriate public health intervention, the impact and cost of diabetes in many societies, particularly for populations in which NIDDM is already or becoming very common, such as American Indians.

Table 1

Classification of the Types of diabetes and Other Categories of Glucose Intolerance

CLASS NAME	FORMER TERMINOLOGY	CHARACTERISTICS
	Diabetes Mellitus	
Insulin-dependent diabetes mellitus (IDDM, Type 1)	Juvenile diabetes Juvenile-onset diabetes (JOD) Ketosis-prone diabetes Brittle Diabetes	Low or absent levels of circulating endogenous insulin and dependent on injected insulin to prevent ketosis and sustain life Onset predominantly in youth but can occur at any age Associated with HLA D3 and D4 Abnormal immune response and islet cell antibodies are frequently present at diagnosis Etiology probably only partially genetic, as only 35% of monozygotic twins are concordant
Non-insulin-dependent diabetes mellitus (NIDDM, Type II)	Adult-onset diabetes Maturity-onset diabetes (MOD) Ketosis-resistant diabetes Stable diabetes	Insulin levels may be normal, elevated, or depressed Not insulin-dependent or ketosis-prone under normal circumstances, but may use insulin for treatment of hyperglycemia or during stress conditions
Subtype obese Subtype nonobese		Onset predominantly after age 40, but can occur at any age Approximately 60 per cent of patients are obese Hyperinsulinemia and insulin resistance characterize some patients Etiology probably strongly genetic as almost 100% of monozygotic twins are concordant
Gestational diabetes (GDM)	Gestational diabetes	Glucose intolerance that has its onset during pregnancy virtually all patients return to normal glucose tolerance following parturition Conveys increased risk for progression to diabetes
Other types of diabetes, including or secondary to those associated with pancreatic disease, hormonal disease, drugs or chemical exposure, insulin receptor abnormalities, certain genetic syndromes	Secondary diabetes	In addition to the presence of the specific condition, hyperglycemia at a level diagnostic of diabetes is also present Causes of hyperglycemia are known for some cases, e.g., pancreatic disease, in other cases an etiologic relationship is suspected
	Other Categories of Glucose Intolerance	
Impaired glucose tolerance (IGT)	Borderline, chemical subclinical, asymptomatic, latent diabetes	Nondiagnostic fasting glucose levels and OGTT values between normal and diabetes Confers higher risk for development of diabetes In some individuals, may represent only normal variation of glucose tolerance
Previous abnormality of glucose tolerance (PrevAGT)	Latent diabetes, prediabetes	Current normal glucose tolerance but previous diabetes of IGT spontaneously or in response to an identifiable stimulus
Potential abnormality of glucose tolerance (PotAGT)	Prediabetes, potential diabetes	Normal glucose tolerance but a higher risk for diabetes due to family history, genetics, HLA type, islet antibodies, obesity, physical inactivity, etc.

From National Diabetes Data Group. Classification and diagnosis of diabetes and other categories of glucose intolerance, *Diabetes*, 28. 1039-1057, 1979

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Chapter 1

Prevalence of Diabetes Mellitus Amongst the James Bay Cree Indians, Québec, Canada

Introduction

The pattern of health and disease among native people in Canada has undergone substantial changes since the second World War. This seems to be the result of rapid social and cultural changes due to Euro-Canadian influence on Indian lifestyle (1). The extent of this influence is often related to the geographical location of the communities. Chronic diseases such as diabetes, hypertension, ischemic heart disease and stroke have become important and are replacing infectious diseases as the major source of mortality and morbidity (2, 3).

Published information on the extent of diabetes in Canadian Native populations reveals that prevalence rate varies widely and seems to vary within and between linguistic and cultural groups (4).

This study was undertaken in response to growing concern from the James Bay Cree community, a region of Québec where there is no data on the extent of this disease. We present a population based profile of the prevalence of diabetes in the James Bay Cree Indians and attempt comparison with observations made on other northern populations.

Methods

Study Population

The James Bay Cree are part of the algonkian language family and the sub-arctic culture area of the North American Indians (5). The total Cree population beneficiaries of the James Bay agreement residing on the territory as of January 1989 numbered 8,840. The Cree villages are located in the boreal forest on relatively flat land close to waterways. They live in eight communities: the Coasters who pursue their livelihood from the resources along the James Bay coast, and the Inlanders who have inland hunting territories (Figure 1).

Villages vary in population (from 384 inhabitants for Eastmain to 2,419 for Chisasibi) and accessibility. The five coastal communities and the inland community of Nemaska are relatively isolated and can be defined in terms of geographical category as remote. Transport in and out of these six communities is by airplane, although Chisasibi and Nemaska are accessible by dirt roads built for the James Bay hydro-electric project. The other inland communities, Mistissini and Waswanipi, lie further south and are easily accessible by road from larger urban centers such as Val d'Or and Chibougamau. These two communities can be considered as rural compared to the others. More than 52% of the total population of the James Bay Cree is less than 25 years old compared to 31% for Québec (6). From a traditional hunter-gatherer economy, the Cree, like many other First Nations, have undergone tremendous social and cultural pressures over the last 20 years (2, 7) through the influence of sedentarization and population aggregation, increased consumption of purchased foods and more opportunity for earning a living through wage labour (1).

Case Finding

A list of physician diagnosed diabetes mellitus cases was obtained during July and August 1989. To do so we used chronic diseases registers and diabetic clinic lists kept at each community clinic for follow-up of patients in the communities. Any potential missing case was also discussed and documented with the local nurses and interpreters. The clinical histories of the identified patients were obtained from their medical records. The biochemical criteria of the World Health Organization (WHO) were used to confirm the diagnosis of diabetes mellitus (8). Those not meeting the criteria were eliminated from the study. We also excluded cases defined as gestational diabetes, secondary diabetes or impaired glucose tolerance. Dead patients identified because they were still listed on a chronic disease registry were not included. Diagnoses of type I diabetes were accepted only when the following criteria were found: type I or juvenile onset as noted by a primary physician, continuous management with insulin, and a history of either ketoacidosis or a labile glycemic response to insulin consistent with type I

diabetes. The term insulin-dependent diabetes was not accepted as equivalent to type I because in many cases this term referred to a type II diabetic patient on insulin. Potential cases had to usually reside in the communities and be of Cree origin and status. The principle investigator with the help of a research assistant collected data in each respective community, using a clear operational definition of the inclusion-exclusion criteria and as standard a procedure as possible in order to minimize inter-observer variations (Addenda 1 – 3). The extent of bush living in diabetics was obtained from the Cree Hunter & Trapper Association which administers the income security programs which provide a guaranteed income to individuals spending more than 120 days per year in the bush. Only diabetics who had fulfilled this requirement for the previous two years were considered as living a fair portion of their time in a traditional lifestyle.

Prevalence rates were calculated for the entire James Bay Cree Community. As the age structure between native and non-native populations of Canada is different, Canadian comparisons are made using the direct method of age standardization using the 1985 Canadian population as a standard. A confidence interval was added to the obtained crude and age-standardized rates (9). The crude rates are used for comparisons with other native populations because we expect roughly the same age-distribution amongst native populations of Canada.

Results

As of August 1989, a total of 235 diabetics were found for a crude overall prevalence of 2.7%. Only 5 cases of Type I diabetes, 3 men and 2 women (Addenda 4), were clearly identified for an overall prevalence of 0.06% (Table 1). Crude prevalence (Addenda 5) for non-insulin dependent diabetes mellitus (NIDDM) was 5.2% in the 20 years and over age group and 11.8% in the 40 to 69 age group. Women's rates were consistently higher than those for men and peaked in the 60 to 69 age group compared to the 50 to 59 group for men (Figure 2) (Addenda 6). Women to men ratio using the absolute numbers is 2.4:1 although if we distinguish between rural and remote communities, the ratios are respectively 1.8:1 and 4.9:1.

Over 53% of the diabetes cases were diagnosed in the last 5 years (Figure 3). The NIDDM rate standardized to the 1985 Canadian population was 6.6% for the 20 years and over age group.

A clear north-south gradient is observed. Rates are higher in southern latitudes compared to northern ones. Indeed, prevalence for NIDDM varied from 1.9% in the northernmost Cree community to 9.0% in the southernmost village (Figure 4). The rural communities (Waswanipi, Mistissini) versus the 6 remaining communities defining the remote category shows a significantly higher rate of NIDDM, respectively 8.8% and 3.4% ($\chi^2 = 57.1$, $p < 0.001$). Furthermore the proportion of diabetics going to the bush thus living in a more traditional manner is not significantly different ($\chi^2 = 1.64$, $p > 0.05$) between the rural (55%) and the remote communities (45%).

Discussion

Our findings are consistent with other studies of diabetes amongst native populations of North America making this disease a true continental phenomenon (10, 11, 12, 13, 14, 15, 16).

Fortunately, Type I diabetes is still unfrequent in native populations (13, 15, 17) and it accounts for only 2% of the diabetes cases found in the James Bay Cree. We believe we classified our Type I cases appropriately although we have not confirmed the diagnosis with HLA type or autoinsulin antibody titre.

Whev compared to other studies who used the same method of case ascertainment, ours revealed that the James Bay Cree have a higher rate than the Aleuts, Indians and Inuits of Alaska (13), but are very similar with Cree and Ojibwa nations of Northern Ontario (14, 15) and the Mohawks near Montréal for the same age group (12) (Addenda 7). Our results are almost identical to those reported by Young *et al* (4) on other native populations of Québec based on a federal chronic disease registry with no case validation. Our crude prevalence is also very similar to those reported for the same linguistic-culture area of Canada (4).

An increasing trend with age is noticeable with an alarming overall prevalence rate for NIDDM of 11.8% in the 40-69 age group. In addition, rates based on medical chart review most likely underestimate the true prevalence rate as many cases in the communities which had not come to medical attention, diagnosed or not, may have been missed. Surveys have suggested that for every known diabetic, there is at least one undetected case (18). The magnitude of the underestimation for the James Bay Cree would have to be evaluated but is probably very similar to the magnitude in another study which compared chart diagnosis to mass screening in a Canadian Native population (10). In this other study, the investigators found a 30% increase in prevalence rate using the latter method.

Nevertheless, more than 53% of the diabetes diagnoses have been made in the last 5 years, confirming previous observations in another northern tribe (14) and probably indicating a high incidence rate. Our rates are also higher than a 1982-84 study reported for the James Bay Cree using a random sample blood survey methodology in approximately 20% of the 15 years and older age group (19). Their estimated prevalence rate for NIDDM was 2.9% . In comparison, our results only support the recent increase (last five years) of NIDDM diagnoses. A series of prevalence studies in this population might be helpful in estimating the magnitude of this incidence, as baseline rates are now available.

Our findings on sex ratio are consistent with those of Zimmet (20) who reports that in partly urbanized Pacific populations, the female to male ratio is often as high as 3:1, but in the fully urbanized populations, this ratio changes towards unity. West (21) points out that deviations of the sex ratio from unity can be explained by differences in the degree of obesity and frequency of testing. It is generally accepted that both the degree and the duration of overweight increases the risk of diabetes (22). In our study women are more obese than men (23) and tend to use the health care facilities more often. Indeed, 72.5% of the visits recorded to the local health clinics in order to participate in community health programs devoted to chronic diseases are made by women (6). These tendencies could explain the sex differences observed in our prevalence study.

The prevalence of diabetes has been associated with urbanization in other high risk populations (24, 25). It appears that certain populations have a certain genetic susceptibility to diabetes which is more likely to express itself when changes from traditional life-style occur (26, 27). Our results show a clear increase in prevalence concomitantly with a decrease in the latitude of the community reflecting to a certain degree alterations of traditional life-styles. The southernmost communities are more urbanized because of increased availability by roads and frequent contact with nearby urban centers. Young *et al* (4) have recently reported similar findings on a nationwide scale between native groups of different cultural and language. It has been suggested (1, 27, 28) that traditional ways of living seem to protect against the acculturation process. We then expected a higher proportion of diabetics going to the bush in the remote communities which harbour the lowest prevalence of NIDDM . We did not find such a trend. Our definition of traditional life-style might be too simple and would probably need more details about physical activity and food consumption in order to further explore sex ratio and prevalence rate differences between remote and rural communities.

In comparison with non-natives, our study shows that age adjusted overall prevalence of diabetes for the 20 years and older age group in the James Bay Cree Indians is about two times the self-reported rates of diabetes of the 1978 Canada health survey (29). Furthermore, environmental factors are important as shown by the north-south gradient and the rural-remote dichotomy. Better understanding of these environmental factors is the next step for the James Bay Cree Indians.

Table 1

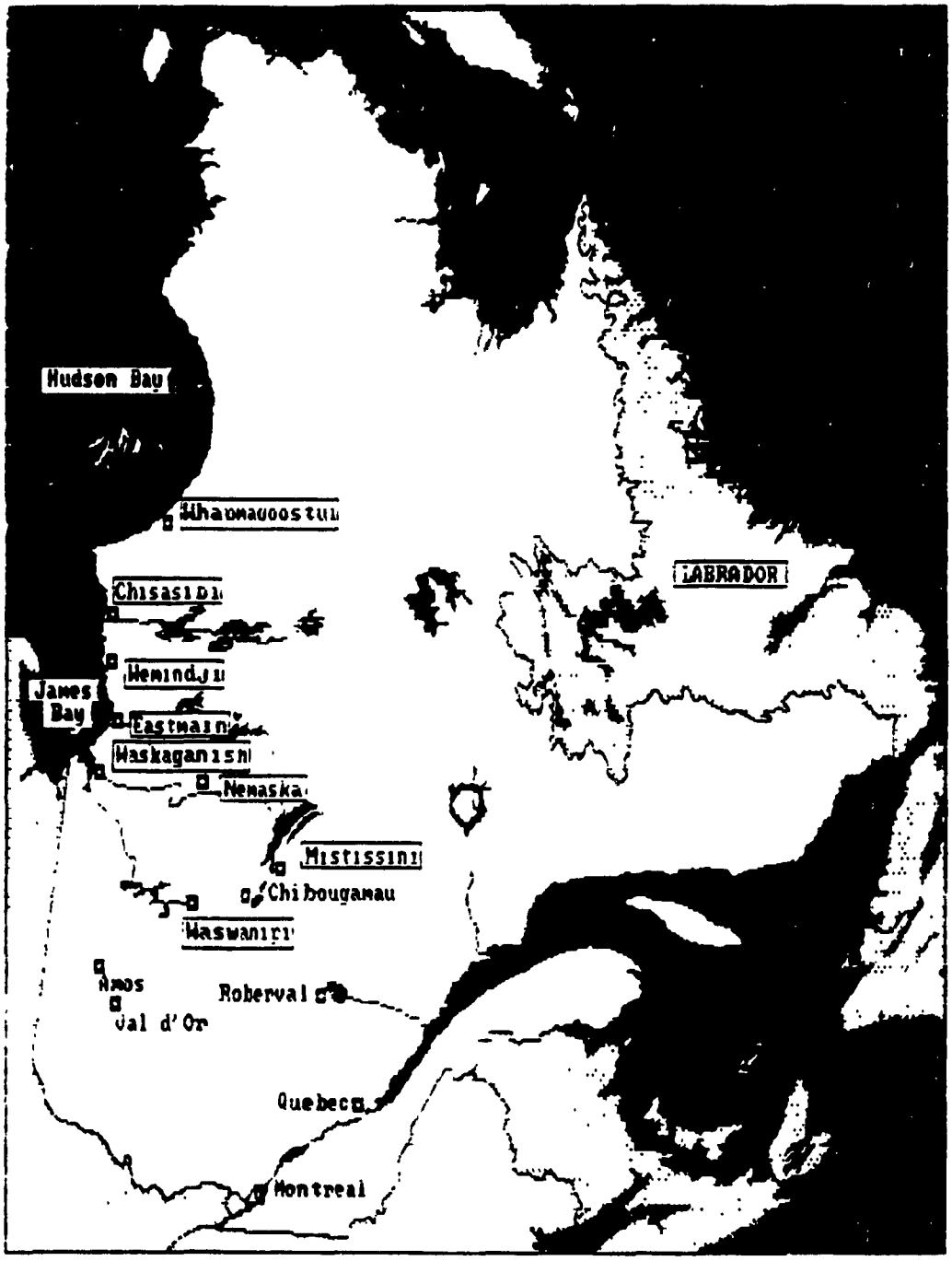
Diabetes Mellitus Prevalence Amongst The James Bay Cree Indians, Québec , 1989.

Type of Diabetes			Prevalence Rate Men	Women	Overall	±95% C.I.*	Age Group
I	Crude	%	.07	.04	0.06	0.05	All
		n	4422	4418	8840		
I & II	Crude	%	1.6	3.7	2.7	0.3	All
		n	4422	4418	8840		
II	Crude	%	7.7	15.8	11.8	1.8	40-69
		n	702	728	1430		
II	Crude	%	3.0	7.2	5.2	0.7	20+
		n	2195	2220	4415		
II	Standardized†	%	3.7	9.7	6.6	0.7	20+
		n	2195	2220	4415		

* 95% Confidence Interval

† To the 1985 Canadian Population

Figure 1: Geographical Location of the Eight Different Cree Communities.



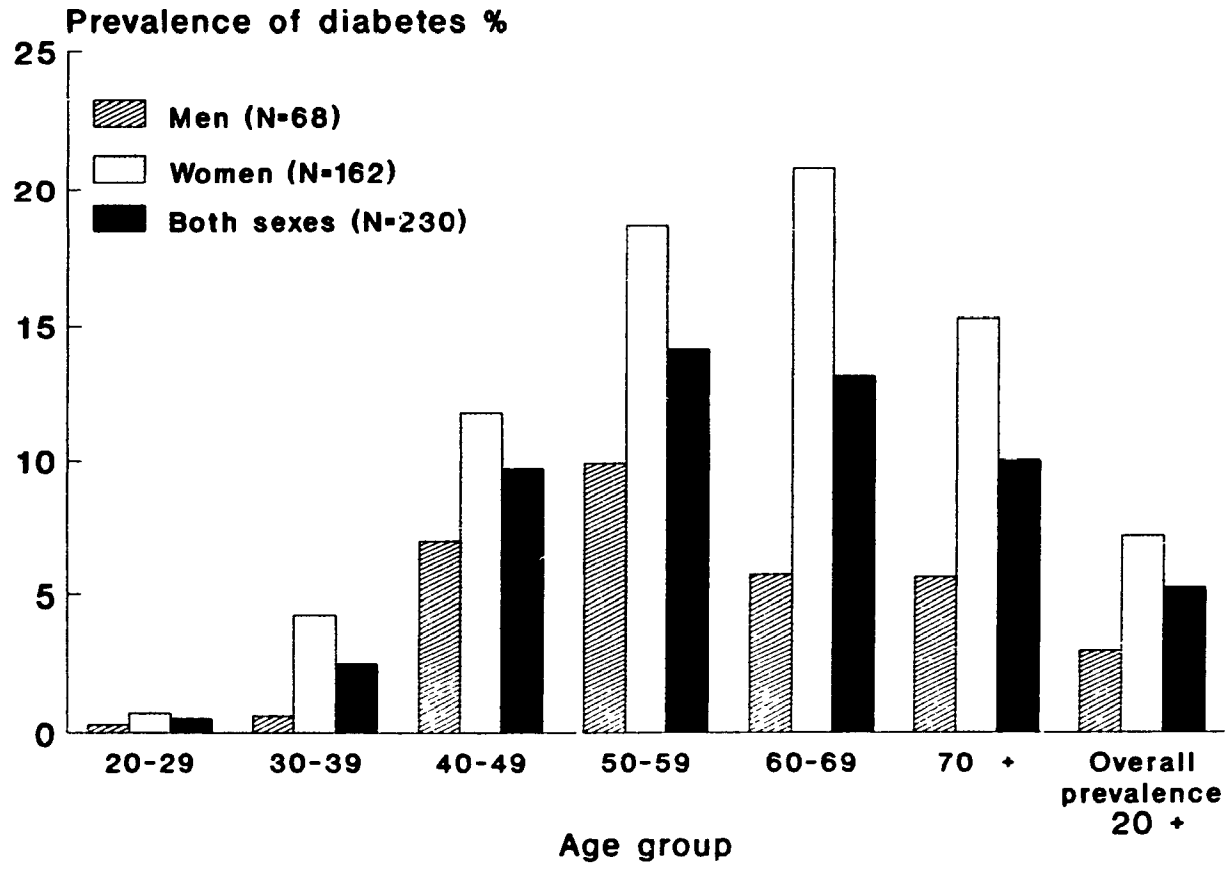


Figure 2. Crude prevalence (%) of type II diabetes in The James Bay Cree community, 1989.

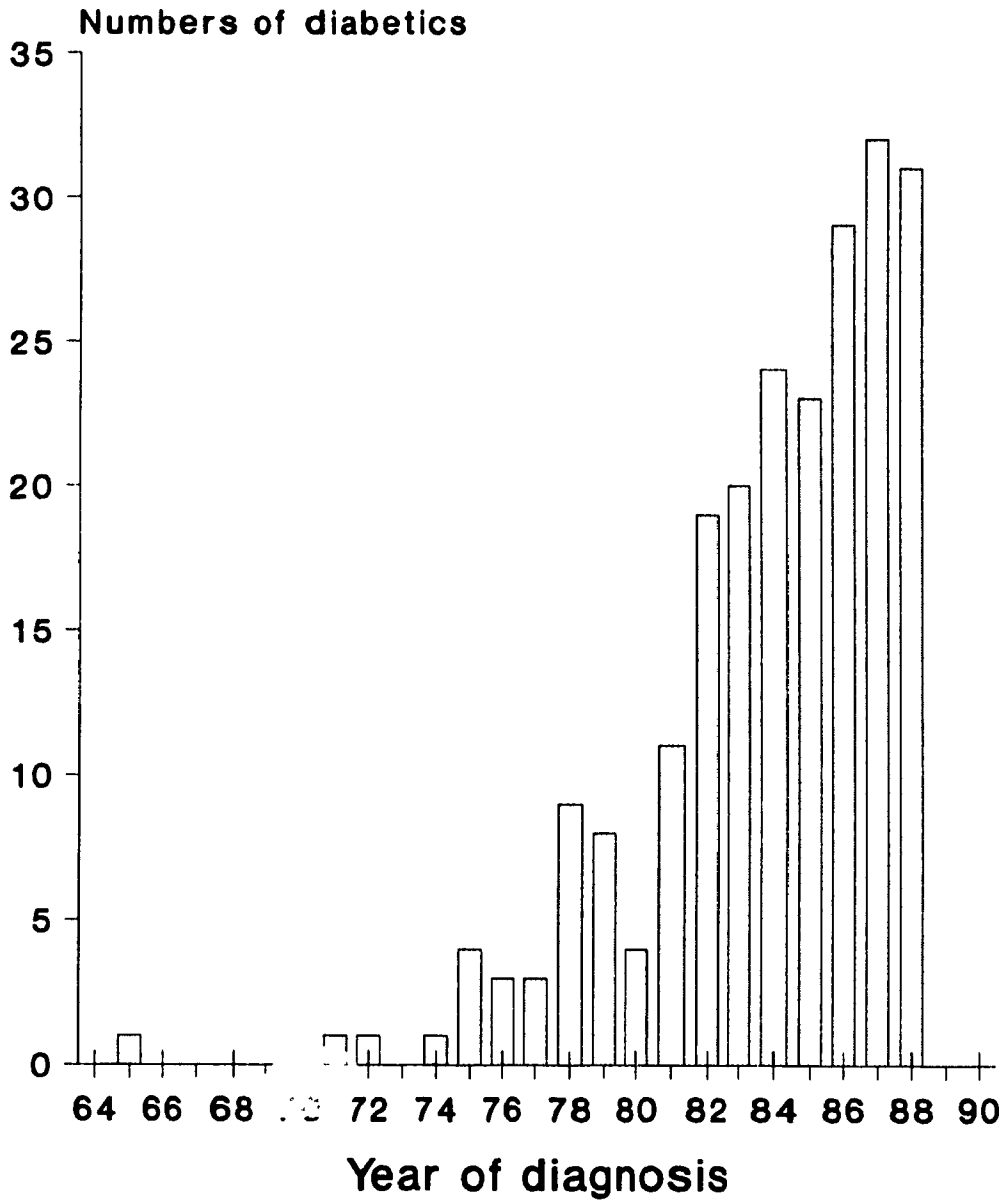


Figure 3. Year of diagnosis of diabetes in the James Bay Cree community, 1989.

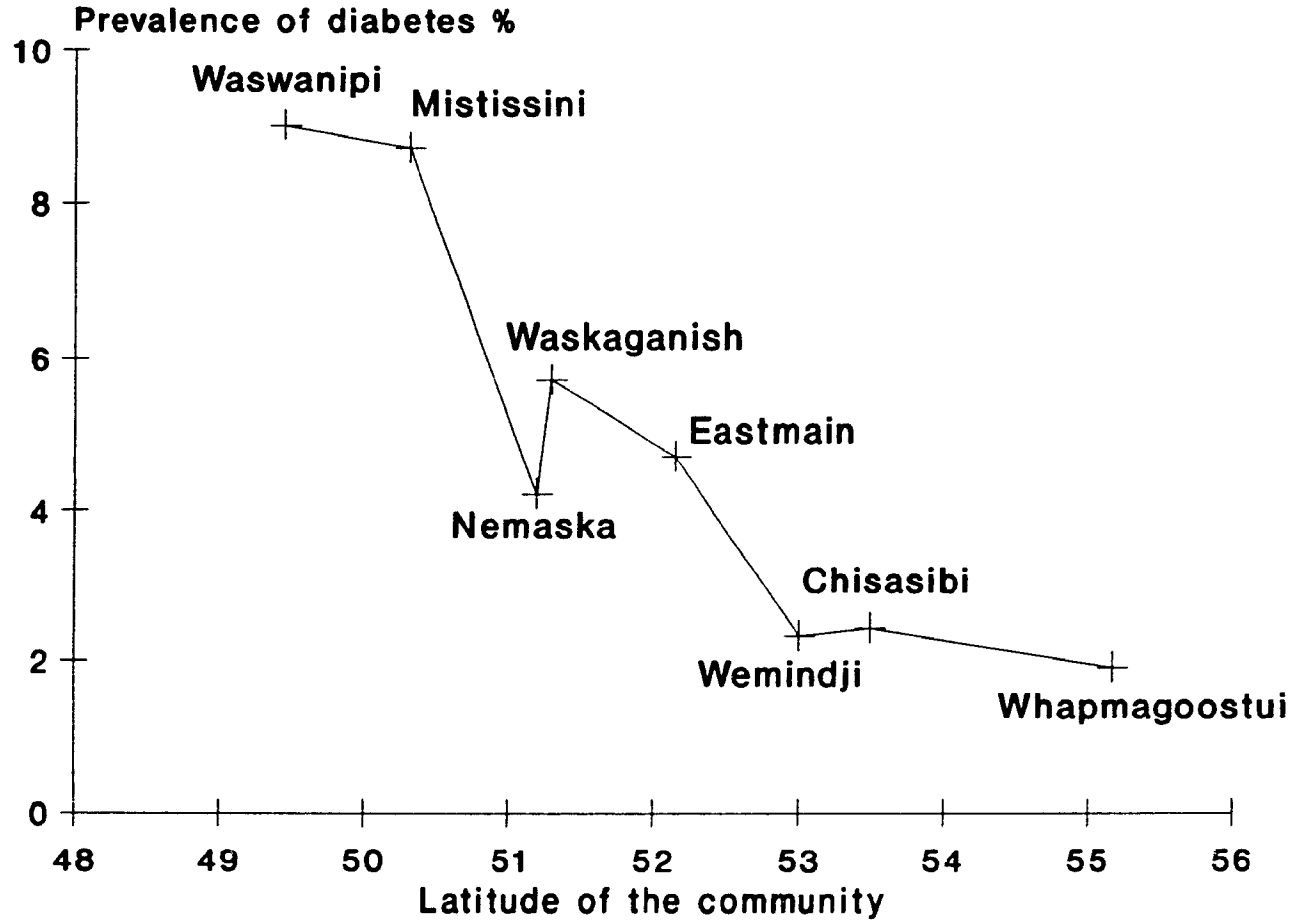


Figure 4. Prevalence of type II diabetes, persons 20 years of age and older in 8 James Bay Cree communities, by latitude, 1989.

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Chapter 2

Clinical Description of Type II Diabetes in The James Bay Cree Indians of Québec

Introduction

The James Bay Cree Indians have previously been described as having as high a prevalence of non-insulin dependent diabetes mellitus (NIDDM) as in other native populations of Canada (1, 2, 3, 4). These prevalence rates for native groups seem to vary according to geographical distribution and genetic background (5), but there may be characteristics of the diabetic individual that might contribute to these differences. Such features as type and rate of complications, life-style, anthropomorphic and metabolic descriptors as well as clinical care practices can be useful to determine similarities or differences between different native diabetic groups, and help define regional differences. This paper describes such features and attempts comparison with similar published data.

Methods

A cross-sectional study was performed during the summer of 1989 among the James Bay Cree Indian community. This community is composed of eight different settlements numbering 8840 individuals (1). Two of those communities, Mistissini and Waswanipi, are located inland and can be considered rural compared to the six remaining remote communities which are more isolated and mainly scattered along the James Bay coast. NIDDM subjects were identified through medical chart review of physician diagnosed diabetes mellitus cases upon which the biochemical criteria of the World Health Organisation was applied to confirm the diagnosis (6). Those not meeting the criteria were not considered. Current data extracted from the medical files included sex, presence and type of complications, hypertension, smoking, mode of treatment, family history, glycemic follow-up procedures, bush living, body mass index (BMI), waist to hip circumference ratio (WHR), and length of illness. Body Mass Index defined as weight in kilograms/height in meters squared, was used to separate overweight

(BMI ≥ 26) from obese (BMI>30) individuals (7). Truncal obesity (upper body obesity) was considered for those with a waist to hip circumference ratio of over 0.99. Macrovascular disease was defined as a physician's written chart diagnosis of ischemic heart disease, cerebral vascular disease or peripheral vascular disease. Microvascular disease was defined as either diabetic retinopathy if written in the chart by an ophthalmologist or diabetic nephropathy as chart diagnosis. Peripheral neuropathy and hypertension were also written chart diagnoses. No specific, standardized examinations or verifications were done to confirm these diagnoses but complications had to follow in time diagnosis of diabetes.

Diabetics who spent more than 120 days per year for the last two years in traditional native activities such as hunting, trapping and fishing were considered to be living in the bush. Family history of NIDDM was considered for those cases with first degree relatives harbouring the disease.

The medical record of each subject was also retrospectively reviewed over a period of 30 months (January 1987 to June 1989) and mean values of all fasting plasma glucose (FPG), glycosylated hemoglobin (HbA_1), total cholesterol, and triglycerides obtained at diabetic clinic visits over those 30 months were recorded. Indices were obtained from these mean values and compared with the expected normal values from the clinical chemistry laboratory which performed the analyses for FPG and HbA_1. Guidelines recommended by the Canadian Consensus Conference on Cholesterol were used for the lipoprotein levels (8). Age, diagnostic criteria used and presence of symptoms, all at time of diagnosis as well as the routine clinical care received by the diabetics from time of NIDDM diagnosis and relevant to the evolution of the illness were also retrospectively evaluated from medical chart review.

Results

A total of 230 Type II diabetics were identified for a crude prevalence rate of 5.2% for the 20 years and older age group (1). Mean age at diagnosis was 48.3 years and mean duration of illness as of July 1989 was 60.4 months (Table 1). No differences in mean weight were

noted from diagnosis to actual time of study either expressed by total weight or by the body mass index. According to our current definition, 77.3% were overweight (BMI≥26) and 65.4% were obese (BMI>30) and both characteristic are found more frequently in women than in men. Fat distribution as measured by the waist/hip ratio is predominantly truncular in 47% of men and close to 23% of women. Men also show higher rates of family history of diabetes, presence of symptoms at time of diagnosis, self-reported cigarette smoking, hypertension and bush living. The female to male ratio of diabetes is 2.4:1.

In the James Bay area microvascular disease is the most frequent group of complications (19.6%, Table 2). Macrovascular disease is second in importance and reveals a predominance of ischemic heart disease (10.0%, Addenda 8). Overall complication rate is 28.7% and is related to duration of illness. Indeed, among the patients who had the disease for 5 years or less, the rate of complications is 18.5% compared to 43% if duration of illness is more than 5 years. Nephropathy is the most frequent individual complication which affects 10.5% of the diabetics (Addenda 9). Unfortunately, no gradation of the severity of nephropathy was documented. In men, peripheral neuropathy is the predominant individual complication (17.6%). No trends in complication rate (Addenda 10) or in duration of illness according to geographical location could be found for the different communities taken individually or in remote (coastal) versus rural (inland) settings.

In our diabetics, HbA_1 and FPG are highly correlated with a Pearson's Correlation Coefficient (r) of 0.64. However, in the low range of FPG levels, HbA_1 values are more scattered than at the higher "uncontrolled" range. The correlation coefficient for subjects with fasting glucose less than 7.8 mmol/L is weaker ($r = 0.20$) and non-statistically significant compared to the higher range ($r = 0.51$, $p < 0.001$). Insulin treated NIDDM patients showed a weaker correlation coefficient ($r = .43$) although still statistically significant compared to those on oral medication ($r = .65$) or on diet only ($r = .68$). Furthermore, 76.4% of the Cree diabetics showed poor glycemic control using FPG as an index compared to less than 20% with our HbA_1 cut-off point (Table 3). Taken individually, neither sex nor age influenced the glucose or

lipid profiles in the diabetics except age for total cholesterol ($F = 2.3$, $p < 0.05$). Total cholesterol and triglycerides were above recommended levels in respectively 9.3% and 25% of the diabetics.

Fasting plasma glucose was used preferentially (55.7%) as a diagnostic procedure (Addenda 11). Oral glucose tolerance test with 75 g of glucose was used to diagnose diabetics in 4.8% of the cases. Moreover, 24.8% of cases of NIDDM do not have any documented diagnostic criteria. Nonetheless, evolution of these cases was consistent with adequate diagnosis.

Retrospectively it was found that 61.7% of the type II diabetics had not reported any symptoms at time of diagnosis. The most frequent symptoms, when present, were fatigue and weakness in 18% of the cases (Addenda 12).

Diet combined with oral hypoglycemic medication is the current treatment of choice (Figure 1). Insulin is used in 18.4% of the cases although these patients can not be considered insulin dependent. Overwhelmingly (85.2%), glycemic follow-up is performed in more than one way (Addenda 13). These include fasting plasma glucose, self-monitoring device, and/or glycosylated hemoglobin . This monitoring is mainly performed within the medical clinics. We unfortunately have no idea of the importance of self-monitoring at home, although we suspect it is negligible.

Table 4 describes the mean number of consultations with respective medical specialties, physical examination performed by general practitioners with and without neurological assessment and of specific clinical acts relating to diabetes care from time of diagnosis. Since the mean duration of diabetes mellitus in our study population was 5 years, we can approximate the mean number of performed acts per year. Thus, using this technique, urinalysis was done 1.6 times a year per patient, since diagnosis of NIDDM.

Discussion

The James Bay Cree diabetics are of higher weight compared with those reported in other Canadian studies on native diabetic populations (2,3, 9 ,10) and are more obese than the

overall Canadian and Cree popu'ation (11, 12) (Addenda 14). Furthermore, there was no difference between current weight and weight at time of diagnosis indicating poor results of the dietary interventions recorded in 97% of the diagnosed diabetics through their current mode of treatment. There are few referrals to a dietician for consultation. There is only one non-native dietician for all eight communities and she is located in one of the coastal communities (Chisasibi). This situation creates cultural, linguistic, and accessibility barriers that hinder acceptance of the service.

Obesity alone is believed to produce diabetes if the degree and duration of obesity are severe and long enough (13, 14) The obese children of diabetic parent(s) have a much higher risk of developing Type II diabetes than obese people with non-diabetic parents (15, 16, 17, 18). Thus, genetic susceptibility and obesity seems to act synergistically. In our study family history of diabetes was recorded in 38.6% of patients and truncal obesity which is a known risk factor for diabetes and atherosclerotic disease (19, 20), was more frequent in men than women with respectively rates of 47% and close to 23%, with an overall rate of 29.8%.

Our results show that the mean age at time of diagnosis of NIDDM is lower than for the Mohawks of Kahnawake (9) but higher than the Oneida of southwestern Ontario (3). Furthermore, our female to male ratio is similar to the Cree-Ojibwa of northern Ontario (2) and much higher than the more southern and urbanized Mohawks and Oneida. The latter have the highest rate (75%) of symptoms at time of diagnosis (3).

Macrovascular complication rate for the James Bay Cree are much lower than the reported rates for other comparable native populations of Canada (2, 9, 10, 21). Direct comparison of these rates with other studies is valid since all these studies involved medical record review without special standardized examination to confirm the diagnosis. There is no reason to believe any difference in medical care practices from one study to another, although the lack of uniformity in the definitions of diabetic complications could preclude any accurate comparisons of its prevalence. Furthermore, there was probably more underreporting of microangiopathy and neurological complications than macrovascular disease, the latter being

more obvious. Nonetheless, microvascular disease was the most prevalent complication recorded in our study and could be a peculiarity of the James Bay Cree Indian diabetics. Length of illness has to be addressed in further comparison as it has been shown to influence complication rates (22, 23, 24). The James Bay Cree showed lower duration of illness as well as lower rates of hypertension, hypercholesterolemia, and smoking than the diabetic Mohawks near Montréal (9). These findings could explain our relatively low level of macrovascular disease (14.4%) compared to the latter native group (48% of ischemic heart disease), but does not preclude the development of such high rates in the Crees. Indeed, impairment of metabolic variables versus recommended values (Table 3) was present in most NIDDM Cree diabetics. Over 75% of the known diabetics still had fasting hyperglycemia using FPG as an indicator. This confirms clinical observations that biochemical control among native diabetics is often difficult to achieve and supports another study on northern Ontario native groups (25). Although closely correlated, the discrepency observed in rates of adequate metabolic control between FPG and HbA_1 levels might reflect the instability of FPG as an individual glucose control measure under present medical treatment such as for unstable insulin treated Type II diabetics in whom blood sugars vary markedly from day to day (26). Insulin treatment was recorded in 18% of the patients and reflects the failure of the initial dietary and oral therapy. The nature of these indices is also different; HbA_1 measures long-term (4-6 weeks) glucose status in the body under normal physiological conditions. On the other hand, FPG level is measured at a single point in time and prior to schedule appointments, subject to changes in usual habits, dietary or otherwise or non-compliance to fasting regulations, so that metabolic control appears worse than it actually is. It is generally recognized that HbA_1 reflects integrated glycemia over its respective period of time (27, 28, 29, 30, 31) and is a more reliable method of follow-up than individual plasma glucose readings. Our study showed that hyperglycemia is not a unique feature in our diabetics but is also accompanied by lipid impairments. Furthermore, recent literature highlights a lack of awareness and treatment from primary care physicians of hyperlipidemia in NIDDM patients (32, 33).

A medical practice profile like the one we produced for the James Bay Cree (Table 4) might provide a comparison basis with recommended baseline care (34). Obvious reasons come to mind in interpreting this profile. First, rareness of primary care physicians and second, a high turnover rate of those who agree to practice in the James Bay area. Long-term involvement is required for adequate follow-up of these diabetic patients and it has not been the regular practice so far in the north. Availability of medical specialties has also to be addressed and discrepancies in service have to be looked upon accordingly as it has varied from year to year and from community to community. Overall, such a profile might be of interest to plan health care needs and to the medical personnel in order to formulate a protocol for standards of diabetic care.

For NIDDM, a more critical appraisal of needs, health care practices and organisation involving the communities would be useful in promoting community-based services directed towards the real needs. With time, and as the cultural transition is intensifying (35, 36), obesity, its metabolic consequences leading among others to NIDDM and its vascular and neurological complications will increase. This will put an additional burden on the population and health care facilities. Health promotion strategies are of some urgency if we want to contribute to a certain control of NIDDM.

Table 1

Characteristics of Type II Diabetics by Sex, James Bay Cree, 1989.

	Men		Women		TOTAL	
Variable	**Mean**	**S.D.†**	**Mean**	**S.D.†**	**Mean**	**S.D.†**
Age at Diagnosis	48.8	(12.3)	48.0	(13.2)	48.3	(12.9)
Length of Illness (months)	56.8	(39.9)	61.9	(49.2)	60.4	(46.7)
BMI at Diagnosis *	34.8	(4.4)	38.5	(6.4)	37.5	(6.2)
Current BMI	34.6	(5.8)	37.4	(6.4)	36.7	(6.3)
Family History %	43.9		37.6		38.6	
Symptoms at Diagnosis %	45.6		35.2		38.3	
Smoking %	22.7		13.6		16.2	
Hypertension %	43.9		43.2		43.4	
Bush Living %	53.0		38.9		43.0	
Truncal Obesity % ‡	47.0		22.8		29.8	
Excess Weight (BMI ≥ 26) %	71.2		80.2		77.3	
Obesity (BMI > 30) %	56.1		69.1		65.4	

* BMI Body Mass Index - Weight (kg)/Height Squared (m)

† Standard Deviation

‡ From Waist/Hip Ratio > .99

Table 2

Prevalence (%) of Diagnosed Type II Diabetic Complications Per Sex, James Bay Cree, 1989

	Men n = 68	Women n = 162	Total n = 230
Microvascular disease *	19.1	19.7	19.6
Macrovascular disease †	16.2	13.6	14.4
Peripheral neuropathy	17.6	6.2	9.6
Total‡	35.3	25.9	28.7

* Retinopathy, nephropathy

† Ischemic heart disease, cerebro-vascular disease, peripheral vascular disease

‡ With at least one complication

Table 3

Glucose and Lipid Profile Among the James Bay Cree Type II Diabetics
Recorded over a 30 month period (January 1987 to June 1989)

Metabolic Characteristics	Men	Women	Total
Mean Fasting Plasma Glucose (SD)*	10.4 (3.2)	10.3 (2.6)	10.4 (2.9)
% > 7.8 mmol/L	72.7	77.8	76.4
N	68	155	223
Mean Glycosylated Hemoglobin (SD)*	8.2 (2.0)	7.7 (1.5)	7.9 (1.9)
% > 9%	25.8	16.7	19.3
N	57	119	176
Mean Total Cholesterol (SD)*	4.9 (1.0)	5.0 (1.2)	5.0 (1.1)
% > 6.2 mmol/L	6.1	10.5	9.3
N	54	114	168
Mean Triglycerides (SD)*	2.3 (1.9)	1.8 (1.0)	1.9 (1.4)
% > 1.7 mmol/L	33.3	21.6	25.0
N	55	113	168

* Standard Deviation

Table 4

Clinical Care Received From Time of NIDDM Diagnosis, James Bay Cree, 1989.

Characteristics	Mean Number of Exams (Standard Deviation)						
	Men		Women		Total		Total Per* Year Since Diagnosis
By Primary Care Physician							
Physical Examination	3.2	(3.1)	4.0	(3.8)	3.8	(3.7)	0.8
Physical Examination Including Neurological Assessment	0.79	(1.1)	1.0	(1.7)	0.9	(1.6)	0.2
Number of Urinalysis	5.4	(6.2)	9.1	(9.3)	8.0	(8.6)	1.6
Number of Serum Creatinine	3.9	(4.1)	4.3	(4.3)	4.2	(4.2)	0.8
Number of Funduscopy	0.9	(1.3)	1.1	(1.6)	1.1	(1.5)	0.2
ECG Number†	1.2	(2.2)	0.9	(1.3)	1.0	(1.6)	0.2
By Specialist							
Number of Consultations Performed:							
- Neurology	0.12	(0.3)	0.04	(0.2)	0.06	(0.2)	0.01
- Cardiology	0.13	(0.4)	0.1	(0.3)	0.1	(0.4)	0.02
- Nephrology	0.03	(0.2)	0.06	(0.4)	0.05	(0.3)	0.01
- Dietician	0.3	(0.8)	0.6	(1.0)	0.5	(0.9)	0.1
- Endocrinology	1.6	(1.6)	1.4	(1.9)	1.5	(1.8)	0.3
- Ophthalmology	1.6	(1.8)	2.4	(2.1)	2.2	(2.0)	0.4

* Based on average illness duration of 5 years.

† 12 lead electrocardiogram

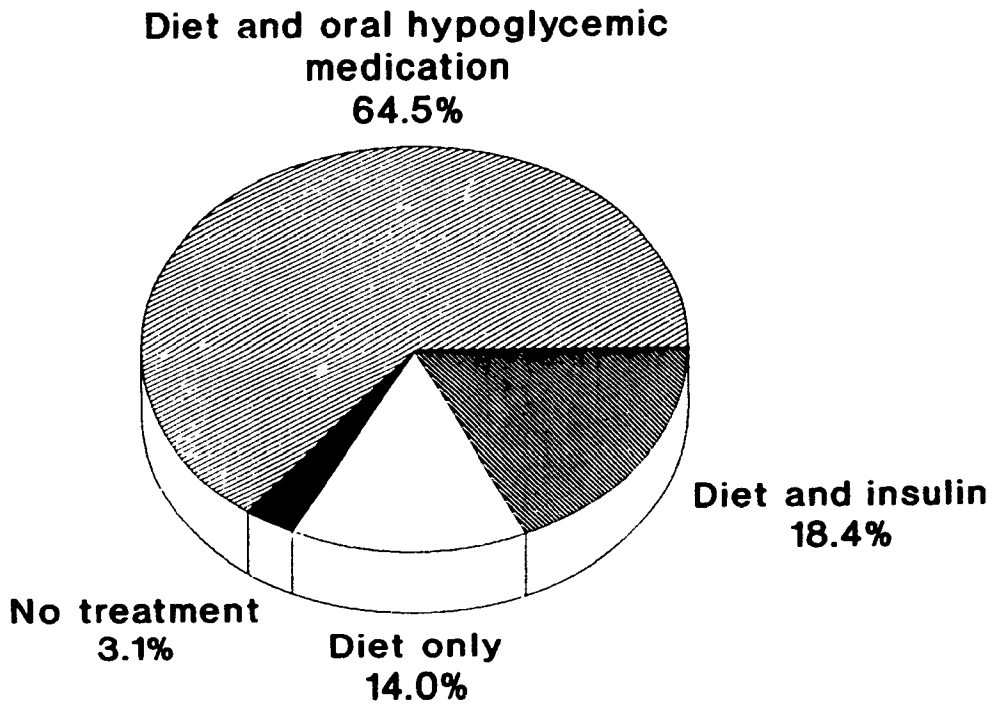

Figure 1. Current treatment of 230 type II diabetics in 8 James Bay Cree communities, 1989.

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Chapter 3

Plasma Glucose Level, Obesity and Microvascular Complications: Associated Factors Among James Bay Cree Indian Diabetics.

Introduction

Non-insulin dependent diabetes mellitus (NIDDM) is a major burden amongst the James Bay Cree Indians of Québec (1) and other native populations of Canada (2, 3, 4, 5). From a public health point of view it is important to discover the role and effect of various risk factors leading to the development of NIDDM or its vascular complications within the Indian population. Interventions on these factors may prevent progression to a later stage within the natural history of the disease.

Genetic susceptibility and family history as risk factors for NIDDM are not modifiable but factors affecting both obesity, which is the best recognized risk factor for NIDDM, and microangiopathies, which is the most specific and frequent complication in the James Bay Cree, may be (6). Furthermore, process of care for diabetes in several practice settings seems to be unrelated to metabolic control of the disease (7, 8, 9). Identification of these factors could provide a rationale for a community-based diabetic education program. It could improve medical care and help to focus interventions on recognized and modifiable components. The present study was undertaken to increase our knowledge about the importance of these potential risk factors in the James Bay Cree Indian Diabetics.

Methods

Case Findings and Data Collection

A cross-sectional study was performed during the summer of 1989 among the eastern James Bay Cree Indians of Québec. This northern community is composed of eight different settlements numbering 8840 individuals. NIDDM subjects were identified through medical chart review of physician diagnosed diabetes mellitus cases to which we applied the biochemical criteria of the World Health Organization to confirm the diagnosis (10). Those not

meeting the criteria were not considered. We also excluded subjects defined as gestational diabetes, secondary diabetes or impaired glucose tolerance. This study was indexed by all eligible diabetics prevalent during July and August of 1989. Current data collected from the medical files during that time span included sociodemographic, anthropometric, and care process information as well as family history and lifestyle factors (1, 6). No assessment of physical activity or dietary profile was carried out. In addition, a retrospective data collection during the prior 30 months period spanned by January 1987 to June 1989 was also performed from medical charts to obtain glycemic and lipid profiles.

Variable Definition

Overall obesity and fat distribution indices were constructed from measurements of height - weight and waist and hip circumferences. Overweight was defined as a Body Mass Index (BMI; weight (kg) divided by height in meters squared) of over or equal to 26. Obesity was considered for those with a BMI > 30 (11). Waist-hip ratio (WHR) was used an an index of fat distribution. Waist circumference was measured in the abdomen midway between the lower rib margin and the iliac crest. Hip girth was measured posteriorly at the maximum protrusion of the gluteal area and anteriorly at the symphysis pubis according to the Canadian Guide for anthropometric and nutritional assessment (12). Truncal obesity was considered for those with a WHR > 0.99. A microvascular complication was defined as diabetic nephropathy and/or retinopathy from a written diagnosis by a physician in the reviewed chart without special standardized examination or verification and had to be recorded after the time of diagnosis of NIDDM. A person with no history of physician diagnosed hypertension or use of antihypertensive drugs was considered normotensive.

Diabetics who spent more than 120 days per year for the last two years in traditional native activities such as hunting, trapping and fishing were categorized as bush living. Glycemic follow-up was defined as the use by health professionals or by the diabetic person himself of either one or more than one follow-up technique for blood or urine sugar, such as fasting plasma glucose, glycosylated hemoglobin, self monitoring device and urinalysis. Mode

of treatment was divided into three categories: diet, diet with oral hypoglycemic medication, and diet with insulin. Family history of Type II diabetes was considered for those cases with first degree relatives harbouring the disease.

Other variables included age, sex, current smoking, and length of illness from time of diagnosis.

The retrospective variables, over the period of 30 months prior to current data collection were: mean values of all fasting plasma glucose (FPG), glycosylated hemoglobin (HbA_1), total cholesterol and triglyceride obtained at diabetic clinic visits over those 30 months. We considered these indices as an integrated reflection of the current glycemic and lipidic situation. The number of visits to the medical clinic for diabetic follow-up and the number of respective blood tests performed in order to obtain these different metabolic measurements was also recorded within the same time interval. The frequency of visits was calculated for a yearly basis in order to adjust for differences in time of diagnosis. A retrospective assessment was also needed in order to assess the presence of symptoms at time of diagnosis as recorded in the medical chart.

Data Analysis

Analysis of data was done in two steps. First, bivariate associations were evaluated either with t-test, chi-square, analysis of variance or simple linear regression. Choice of test depended on the measurement level of the variables being compared (13). Based on associations observed in bivariate analyses, multivariate models were constructed. Multiple linear regression (14) was used with the dependent continuous variables BMI and glucose level (FPG and HbA_1). The principal independent variable of interest for glucose level were current mode of treatment, mean number of visits to clinics per year in the prior 30 months, and glycemic follow-up. Multiple logistic regression was used to determine factors associated with the presence of microvascular complications, controlling for potentially confounding factors such as age, sex, smoking, hypertension and BMI level. All data entry, management, and

analysis were performed on a microcomputer with the software SYSTAT (Version 3.2, 1988, Evanston, IL, U.S.A.). All probabilities reported are for two-tailed tests at the 0.05 % level.

Results

A total of 230 Type II diabetics were identified for a crude prevalence rate of 5.2 % for the 20 years and older age group (1). Mean age at diagnosis was 48.3 years and mean duration of illness as of July 1989 was 60.4 months (6).

Obesity

According to our current definition, 77.3 % of the Type II diabetics were overweight (BMI $\geq$ 26) and 65.4 % were obese (BMI $\geq$ 30). Overall level of BMI was higher in women than in men ($p < 0.01$) and age was not associated with BMI level. The obesity of this population of diabetics was predominantely of the central type. Table 1 shows that centrality (WHR) measures increase as the level of BMI increases. A higher proportion of obese men were of the central type even if obesity was more frequent in women.

Table 2 presents the results of the multiple linear regression analysis with only the most statistically significant factors. The resulting model shows that female sex, the presence of a positive family history of Type II diabetes among first degree relatives and current smoking are associated with higher BMI level. Age, bush living and hypertension were not associated with BMI level.

We also examined metabolic determinants of increasing levels of BMI. No associations with BMI were found for total cholesterol, triglycerides, HbA_1 or FPG levels when considering the effect of sex, family history and current smoking.

Glucose Level

Initial bivariate analyses compared mean levels of the two glycemic indices (FPG, HbA_1) between different categories of potential influential factors (Table 3). There were no sex differences and no increase with age was recorded for both FPG and HbA_1. Significantly higher levels were observed for both indices when insulin was the current mode of treatment. Length of

illness was positively associated with increased levels of FPG and HbA_1. The presence of at least one symptom at the time of diagnosis was associated with higher level of mean HbA_1.

To investigate which of the principal independent variables of interest related to process of care were significant determinants of glucose levels considering other independent variables as covariates or potential confounders, multivariate linear regression was performed (Table 4). Of the principal variable of interest, only current mode of treatment was a determinant of both glycemic measures. Total cholesterol and triglyceride levels were also identified as significant predictors ($p < 0.05$) for HbA_1, while age and total cholesterol were for FPG. Sex itself was not a significant predictor in multivariate analyses.

Squared multiple r values which reflect the portion of the overall variance explained by the regression model were low and range between 18 and 24%. In all covariance models, the observed realtionships were homogeneous as no significant interactions were found between our variables of interest and the selected covariates.

The mean number of visits to the medical clinics for diabetic follow-up in our 30 months time frame, calculated on a yearly basis to account for different times of diagnosis, did not influence significantly the levels of glycemic measures. Although the number of test readings performed in order to obtain these mean glycemic values was taken into account as it could influence the resulting index, it did not influence the final mean figure in bivariate and multivariate analysis.

Microvascular Complications

Overall microvascular complication rate was 19.6 %. Longer duration of illness, and insulin as the mode of treatment were significantly related to the presence of microvascular complications in NIDDM patients. Other metabolic variables such as total cholesterol, triglyceride levels, HbA_1 and FPG were also positive determinants (Table 5). In considering the independent effects of different variables on microvascular complication status, a multiple logistic regression model was constructed to compare the risk of harbouring microangiopathies

as a function of age, sex, smoking, hypertension and BMI level. The net effect of this multivariate approach is seen by comparing the crude and adjusted odds-ratio (OR) estimates for the selected factors. Retinopathy and nephropathy are 2.7 times (95% confidence interval : 1.1, 6.7) more frequent in those with long duration of diabetes (more than 5 years), and respectively 4.5 (2.0, 9.9) and 3.0 (1.2, 7.8) times more frequent for above normal levels of triglycerides and for insulin as current mode of treatment (Table 6). Several variables significantly associated with microvascular complications in the univariate analyses, such as FPG and HbA_1 levels, as well as total cholesterol were found not to bear any significant relationship to the presence of microangiopathies in the multivariate analysis. Age and sex were of no significant contribution. Another model composed of the three main effect variables and their interaction was also constructed. None of the interaction terms met the 0.05 significance level.

Discussion

Obesity

Obesity and heredity have both been implicated independently in the etiology of NIDDM. Their relative importance in this relation is not well established and the possible interaction of these factors is even less clear. In a cross-sectional design such as ours, where temporal sequences cannot be ascertained, the designation of certain factors as determinants and others as effects is often subject to debate. Nonetheless, our study suggests that obesity promotes diabetes in the genetically susceptible and our data supports much more detailed studies on the intricate effect of obesity and family history as causes of NIDDM (15, 16, 17, 18). Furthermore, BMI levels in our diabetic population were not associated with age nor duration of disease. This finding would indicate that in our population, obesity develops at a young age and stays relatively stable thereafter. Knowler *et al* (19) showed that the rate of development of diabetes in the most obese is greatest at younger ages, while in less obese

subjects, the risk of diabetes increases with age. This would imply that the most obese who do not develop diabetes early are much less genetically susceptible.

This study also provides cross-sectional evidence of the adverse health impact of obesity by increase truncal obesity with increasing BMI levels. Obesity by itself (17, 20, 21, 22,) and truncal obesity (upper body obesity) have been implicated in the development of ischemic heart disease, stroke, diabetes, hypertension and all cause mortality in both men and women. The association for truncal obesity is independent of overall adiposity (17, 23, 24, 25, 26). Furthermore, truncal obesity was found to be related to potentially modifiable behavioral factors such as smoking, alcohol consumption and exercise (23, 27). In our study, current smoking was also positively associated with BMI levels. Unfortunately, other pertinent life-style determinants were not assessed. Hyperlipidemia and carbohydrate disorders have been commonly associated with obesity and these metabolic derangements might lead to the development of obesity-associated diseases such as cardiovascular diseases (24, 28). In our study, we did not find such association after adjustment for sex, smoking and family history. The effect of treatment might have reduced the association between BMI and its metabolic features by enhancing the control of the latter in our study group.

Glycemic Control

Bivariate analysis of glycemic control of NIDDM in the James Bay Cree Indians reveals an association between HbA_1 levels and the presence of symptoms at time of diagnosis as if these individuals will be the ones in which adequate control would be the most difficult to achieve. Furthermore, age which was not significantly associated with glucose levels in the bivariate analysis is more influential than duration of illness for FPG in the multivariate analysis. Both lipids measurements contribute independently to higher levels of HbA_1 and only triglyceride levels was associated with FPG. BMI and WHR were of no significant contribution to our multivariate models.

From our three variables of interest concerning medical care, only mode of treatment comes out consistently as an independent influential factor. This observation may reflect the

common clinical practice of placing patients in poorer control on more aggressive therapies rather than reflecting the competing hypothesis that patients treated with more aggressive therapies have worse metabolic control. However, our cross-sectional study design does not permit speculation as to whether more aggressive treatments improved, or is a reaction to, degree of glycemic control. Indeed, process of medical care, as we measured it, is only related to glycemic control to a limited extent. A substantial proportion of the variance in glycemic control is not explained by the independent variables measured in this study as squared multiple r varies from 18 to 24%. Our data is consistent with others' (29, 30, 31, 32, 33, 34) who concluded that differences in the process of care may not be associated with differences in glycemic outcome amongst diabetic patients. Social and psychological factors may be more strongly associated with metabolic outcomes than medical factors (35, 36).

Microvascular Complications

The question of glycemic control as the etiology of vascular complications is still debated (37, 38). Beside metabolic control, environmental, genetic, and other factors such as hypertension, have been proposed as risk factors for the development of complications (39).

In our study, the three most powerful risk factors for microangiopathies after adjustment for potential confounders such as age, sex, current smoking, hypertension and BMI, were above normal level of triglycerides (OR = 4.5), need of insulin therapy (OR = 3.0) and duration of diabetes of over 5 years (OR = 2.7). These relationships are independent of the effect of other variables. Risk of microvascular complications was also related in bivariate analyses with mean HbA_1 and FPG levels. Since mode of treatment in the form of a need for insulin therapy is strongly associated with poorer glucose status it was a stronger predictor of microangiopathies than FPG or HbA_1 levels in the multivariate analysis. Note that in a cross-sectional study the odds-ratios (relative risks) refer to the risk of having the disease and not of developing the disease. Our findings are consistent with other studies (2, 7, 40, 41, 42) which also looked at determinants of microvascular complications in diabetic native populations.

Conclusion

Overall, our study shows an association between microangiopathies and duration of disease and it supports the hypothesis linking microvascular complications to the quality of glycemic control. Furthermore, our data shows a tendency to a lower complication prevalence in diabetic patients treated by diet and oral medication compared to those treated by diet and insulin. This trend is consistent with the differences in severity of the metabolic abnormality. Moreover, hyperglycemia as expressed by our FPG and HbA_1 levels is not an isolated chemical abnormality as it is accompanied by hyperlipidemia. High levels of obesity in this population (6) combined with a predominance of truncal fat distribution which is consistent with a previous study on another Canadian native population (43) only enhances the fact that intervention on this and the above intricate factors is of some urgency if we want to contribute to a certain control and decrease of the overall mortality or morbidity in Canadian Indians from such chronic diseases as diabetes and coronary heart disease which share the same risk factors.

Reversion to a traditional way-of-life has been shown to reverse the metabolic abnormalities of diabetes, and resulted in weight reductions in a study of Australian Aborigines (44), indicating the reversibility of the risk factors for non-communicable diseases that relate to the modern lifestyles.

Our results also show that a better understanding is needed of how social and psychological factors are related to metabolic outcomes. Factors such as social network, social support, cultural understanding and importance of the illness and family function (45, 46) are amenable to interventions that could reduce cost of care (29) while maintaining or improving outcome (35).

For the James Bay Cree Indians, this was a first step. A community based diabetic education program is now underway and hopefully will improve the quality of life of many diabetic patients.

TABLE 1

RELATION OF BODY MASS INDEX (BMI) TO CENTRALITY INDEX

BMI	Proportion (%) with WHR* > 0.99	
	Men	Women
< 26	5.3	0.0
26 - 30	55.5	22.2
> 30	65.8	29.5
Total	46.9	22.9
p†	< 0.001	0.002

* WHR: Ratio of waist to hip circumferences.
† p value by chi-square test.

TABLE 2

PREDICTORS OF BODY MASS INDEX (BMI)*

SIGNIFICANT PREDICTOR† (comparison group)	β	95% C.I.‡	P value
Positive family history of type II diabetes (yes, no)	0.44	(0.22 - 0.66)	< 0.001
Current cigarette smoking (yes, no)	0.42	(0.15 - 0.69)	0.004
Sex (women, men)	0.27	(0.03 - 0.51)	0.02
N	230		
Total r^2	0.12		

* From multiple linear regression model.

† Other variables tested but not found to be statistically significant were. age, bush living, mode of treatment and hypertension.

‡ 95% confidence interval.

TABLE 3

MEAN GLYCOSYLATED HEMOGLOBIN AND FASTING PLASMA GLUCOSE BY RISK FACTORS AMONG JAMES BAY CREE INDIAN TYPE II DIABETICS

RISK FACTOR	GLYCOSYLATED HEMOGLOBIN (%)		FASTING PLASMA GLUCOSE (MMOL/L)	
	Number of subjects	Overall mean (p value)	Number of subjects	Overal mean (p value)
Age		(.87)		(.73)
20-39	21	8.0	30	11.0
40-59	100	8.1	119	10.4
60 +	55	7.9	71	10.3
Sex		(.24)		(.32)
Men	56	8.2	56	10.6
Women	120	7.9	154	10.2
Mode of treatment		(.001)		(.001)
Diet	34	6.9	38	8.2
Diet and oral med.	107	8.2	143	10.4
Diet and insulin	35	8.4	39	12.1
Glycemic follow-up		(84)		(.14)
Single technique	14	8.1	29	11.1
Multiple technique	162	7.9	191	10.3
Symptoms at time of diagnosis		(002)		(.14)
Yes	65	8.5	82	10.7
No	111	7 6	138	10.1
Positive family history of type II diabetics		(72)		(.86)
Yes	66	7 9	87	10.4
No	110	8.0	133	10.3
Current cigarette smoking		(.10)		(.88)
Yes	28	8.5	37	10 3
No	148	7.9	183	10.4
Hypertension		(.80)		(.20)
Yes	74	8.0	95	10.6
No	102	7.9	125	10.1
Bush living		(80)		(.12)
Yes	79	7.9	97	10.0
No	97	8.0	123	10.6
Length of illness		(.001)		(.001)
< 5 years	101	7.5	137	9.7
≥ 5 years	75	8.4	88	11.3

TABLE 4

PREDICTOR OF MEAN GLYCOSYLATED HEMOGLOBIN AND MEAN FASTING PLASMA GLUCOSE*

	GLYCOSYLATED HEMOGLOBIN		FASTING PLASMA GLUCOSE	
INDEPENDENT VARIABLE†	β	95% C.I.‡	β	95% C.I.‡
Mode of treatment				
- diet and insulin	0.74	(0.18 - 1.30)	1.9	(1.00 - 2.80)
- diet and oral medication	0.05	(0.01 - 0.09)	0.04	(0.02 - 0.06)
- diet only (referent)				
Mean total cholesterol	0.27	(0.02 - 0.52)	0.53	(0.20 - 0.86)
Mean triglyceride	0.25	(0.02 - 0.48)	----	----
Age	----	----	0.03	(0.01 - 0.05)
N	139		159	
Total r^2	0.18		0.24	

* From multiple linear regression model.

† Other variables tested but not found to be statistically significant were; sex, glycemic follow-up, mean number of visits to medical clinics, duration of illness, bush living, current smokers, waist-hip ratio and body mass index

‡ 95% confidence interval.

TABLE 5

MICROVASCULAR COMPLICATIONS BY RISK FACTORS
AMONG JAMES BAY CREE INDIAN TYPE II DIABETICS

RISK FACTOR	% with complications	Odds ratio	95% C.I.*	P†
Age				.16
20-39	6.3	1.0	referent	
40-59	16.9	3.2	(0.7 - 14.3)	
60 +	20.8	4.1	(0.9 - 19.1)	
Sex				.69
Men	18.2	1.0	referent	
Women	16.0	0.9	(0.4 - 1.8)	
Mode of treatment				<.001
Diet	2.5	1.0	referent	
Diet and oral med.	14.9	6.7	(0.9 - 51.2)	
Diet and insulin	35.7	21.1	(2.6 - 168.6)	
Current cigarette smoking				.68
No	16.2	1.0	referent	
Yes	18.9	1.2	(0.5 - 2.9)	
Hypertension				.11
No	13.2	1.0	referent	
Yes	21.2	1.5	(0.8 - 3.0)	
Length of illness				<.001
< 5 years	8.9	1.0	referent	
≥ 5 years	27.9	4.0	(1.9 - 8.4)	
Triglyceride level				<.001
≤ 1.7 mmol/l	10.6	1.0	referent	
> 1 7 mmol/l	34.5	4 4	(2.1 - 9.2)	
Total cholesterol level				.05
≤ 6.2 mmol/l	15.0	1.0	referent	
> 6.2 mmol/l	31.8	2.6	(1.0 - 6.9)	
Glycosylated hemoglobin level				.01
≤ 9%	13.6	1.0	referent	
> 9%	29.5	2.7	(1.2 - 5.7)	
Fasting plasma glucose level				01
≤ 7.8 mmol/l	5.5	1.0	referent	
> 7 8 mmol/l	20.1	4.3	(1.3 - 14.4)	
Body mass index				.06
< 26	5.8	1.0	referent	
26-30	22.2	4.6	(1.0 - 19.9)	
> 30	19.3	3.8	(1.1 - 13.2)	

* 95% confidence interval.
† p value by chi-square test.

TABLE 6

PREDICTORS OF MICROVASCULAR COMPLICATIONS*

VARIABLES† (Comparison group)	Crude odds ratio	Adjusted‡ odds ratio	95% confidence interval
Mean triglyceride level (> 1.7 mmol/l, ≤ 1.7 mmol/l)	4.4	4.5	(2.0 - 9.9)
Lenght of illness (≥ 5 years, < 5 years)	4.0	2.7	(1.1 - 6.7)
Mode of treatment§ (Diet and insulin, diet and oral medication)	3.9	3.0	(1.2 - 7.8)

* From multiple logistic regression model.

† Other variables included were; mean total cholesterol, mean fasting plasma glucose, and mean glycosylated hemoglobin.

‡ For age, sex, current smoking, hypertension and body mass index.

§ Grouped into two categories.

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Overview

As of August 1989, 235 diabetics were found in the eight different Cree communities. 98% of these cases were type II diabetics. The magnitude of the underestimation of the real number of diabetics would have to be ascertained although we think it is close to 30% as reported in another Canadian native group (1). Nonetheless, our prevalence rates fell within the range observed for the Algonkian-subartic culture area of Canada as described in a nationwide report on native diabetic prevalence rate (2). Female prevalence rates are consistently higher than those for males and increase with age for both sexes with a decrease around 60 years old. This decrease has been observed in other Canadian native populations (3, 4) and might reflect a survival effect by an overrepresentation of non-diabetics in older age groups derived from their better health conditions in comparison to diabetics. It could also be the manifestation of the emergence of a new phenomenon that has not yet reached the older age group. Indeed, 53% of the Cree diabetics have been diagnosed in the last five years and this fact tends to support the latter hypothesis.

Environment seems to be an important factor for the emergence of non-insulin dependent diabetes mellitus (NIDDM) as reflected by the clear north-south gradient and rural-remote variation in prevalence rates. The nature of this environmental influence still has to be elucidated. Traditional living has been shown to protect against the acculturation process and emergence of NIDDM (5). We were not able to show this phenomenon in our study by looking at the extent of bush living among the diabetics in their respective geographical locations. We did not record important variables such as physical activity and food consumption which might have helped in answering the question.

The Cree diabetics are more obese, but have lower rates of hypertension, hypercholesterolemia, smoking and duration of illness than the Mohawk diabetics near Montréal. These findings could explain our relatively low level of macrovascular disease (14.4%) compared to the Mohawks (48% of ischemic heart disease) (6). This goes along with a

low circulatory disease death rate among the general Cree population (7) as well as its low cholesterol level (8). Furthermore, smoking is very frequent among young people, but overall, Crees are light smokers (9). The question is, will these characteristics increase in the future and should health promotion focus on these factors which are common to such chronic diseases as diabetes and coronary heart disease?

Moreover, duration of disease (more than 5 years) and poor glycemic control as reflected by the type of medication needed as well as hypertriglyceridemia influences the presence of microangiopathies. The latter are the most frequent complications reported in the Cree diabetics (19.6%). Process of care as we measured it (current mode of treatment, mean number of visits to clinics per year and glycemic follow-up) do not contribute markedly to glycemic control. Therefore, features such as social support and psychological factors relating to diabetes as well as cultural understanding and importance of the illness for the Cree population should be considered as important as proper medical treatment in community based multidisciplinary approaches to diabetes (10 - 12)..

The data for our study is based on clinical records (medical files) and certain limitations associated with such a procedure have to be addressed. First, there are problems of reliability and validity. The information has usually been collected by a number of different health professionals, who may have used different definitions of our variables of interest or different methods of obtaining and recording data. There is also a possibility that error had occurred when the information was extracted from the records. Sources of unreliability (inconsistent information) were difficulties reading handwriting and when the required information had to be hunted for because it was not recorded in the standard place or was buried in long passages of prose. Secondly, since the records were not kept for research purposes, the information required was recorded either patchily or not at all. Weight and height, family history and current smoking were seldom recorded. When the presence of a symptom at time of diagnosis was not recorded, was it because it was absent, or that no attempt was made

to establish its presence, or that its presence was established but not recorded, whether by oversight or because it was regarded as unimportant or irrelevant?

Despite these drawbacks, the medical files were the only existing and convenient source of information at our disposal and can be nevertheless considered adequate for the type of study we performed.

This baseline study showed that the James Bay Cree Indians harbour a higher rate of diabetes than the Caucasians of Canada even if this illness is relatively recent among the communities As the acculturation process intensifies, obesity and its metabolic consequences (among other NIDDM) will put an additional burden on the population and health care facilities. In response to this situation, a community-based diabetic education program is now underway and hopefully will improve the quality of life of many diabetic patients.

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Lexicon

BMI — Body Mass Index

CCSSSBJ — Conseil Crie de la Santé et des Services Sociaux de la Baie James

CLSC — Centre Local de Services Communautaires

FPG — Fasting Plasma Glucose

HbA_1 — Glycosylated homoglobin

HLA — Human lymphocyte antigen

IDDM — Insulin-dependent diabetes mellitus or Type I diabetic

IGT — Impaired glucose tolerance

MNQ — Module du nord Québecois

NDDG — National Diabetes Data Group

NIDDM — Non-insulin-dependent diabetes mellitus or Type II diabetic

OGTT — Oral Glucose Tolerance Test

WHO — World Health Organization

WHR — Waist to hip circumference ratio

Addenda

We extracted from the medical files, pertinent anthropomorphic, biological and environmental data. Other variables related to life-style and clinical care (see Chapter 2) were also recorded . A list and definition of these variable and the record form used to collect the information can be consulted in Addenda 1 and 2. The relationship between those variables is described in Chapter 3. A pre-test to ascertain the feasibility of obtaining the various variables of interest from the medical files was performed in June of 1989 in Mistissini. Modifications were then brought to the original record form to render it as simple, informative and easy to use as possible.

Pertinent missing information from the medical files was noted in almost all cases found. In order to improve our information retrieval, a pre-coded sheet was left in the respective medical files with a pre-paid return envelope in order to be completed by a nurse or physician during the next routine visit by the patient (Addenda 3). A response rate of over 70% was obtained for these record sheets through excellent collaboration from the different community clinics.

We obtained permission to conduct our study from the Cree health board and the Chisasibi hospital Director of Professional Services.

Addenda 4 - 7 correspond to additional information referred to in Chapter 1 as well as Addenda 8 - 14 for Chapter 2.

ADDENDA 1

Définition des variables

FACTEURS BIOLOGIQUES

Variable	Définition	Unité de mesure
Age	L'âge au moment du diagnostic de diabète type I ou II. Sera obtenu par la date de naissance et la date du diagnostic.	An, mois, jour
Sexe	Masculin, féminin	1. Masculin 2. Féminin
Histoire familiale	Présence de diabète NIDDM chez les parents, enfant ou fratrie du sujet à l'étude	0. Aucun 1. Parents 2. Enfants 3. Frère, soeur
Cholestérol	Incluant la forme estérifiée et libre, telle que déterminée par analyse de ponction veineuse.	M.MOL/L.
Triglycérides	Tels que déterminés par ponction veineuse après un jeûne de 12 heures.	M.MOL/L.
Hémoglobine glycosylée	Glucose lié à l'hémoglobine, déterminée à partir d'une ponction veineuse.	% de l'hémoglobine totale
Glycémie à jeun	Niveau de glucose dans le sang, ou dans le sérum après un jeûne de 12 heures. Déterminé par ponction veineuse.	M.MOL/L.
Pression artérielle	Tel qu'indiqué par un diagnostic médical et noté au dossier.	0. Oui 1. Non

Variable	Définition	Unité de mesure
Indice de masse corporelle (IMC)	Rapport du poids au moment du diagnostic en Kg sur la taille en mètre^2. Si disponible le IMC actuel.	Index pur du rapport $\frac{\text{Poids (kg)}}{\text{Taille (m}^2\text{)}}$
Distribution centripète de la masse pondérale	Rapport de la mesure en cm du diamètre de la taille sur celui en cm de celui des hanches. Selon les mensurations actuelles.	Ratio pur du rapport: $\frac{\text{Taille (cm)}}{\text{Hanche (cm)}}$

Complications (doivent être apparues après le diagnostic)

1. Microvasculaire
- Rétinopathie
- Néphropathie
- Neuropathie

0. Oui
1. Non

2. Macrovasculaire

A Maladie coronarienne
- Infarctus du myocarde
- Angine
- Pontage coronarien
- et/ou dilatation

Si le diagnostic médical est posé et noté au dossier, aucune vérification, examen physique ou dépistage ne sera fait.

B Vasculaire périphérique
- Pied de homard
- Claudication intermittente
- Amputation (non reliée au trauma)

0. Oui
1. Non

Variable	Définition	Unité de mesure
C Cérébrale ischémique . Accident cérébro-vasculaire . Amorose fugace . Attaque d'ischémie transitoire	Si le diagnostic médical est posé et noté au dossier, aucune vérification, examen physique ou dépistage ne sera fait.	0. Oui 1. Non

ENVIRONNEMENT

Variable	Définition	Unité de mesure
Village	Endroit de résidence principale	Un des 8 villages cris de la Baie James

HABITUDES DE VIE

Variable	Définition	Unité de mesure
Activités traditionnelles	Mode de vie traditionnel avec des activités hors-village tels: chasse, pêche, trappe. Se situe en opposition avec le mode de vie dans les villages cris.	Si plus de 120 jours par an pour les 24 derniers mois
Tabagisme	Tel que noté au dossier médical.	0. Oui 1. Non

SYSTÈME DE SOINS

Variable	Définition	Unité de mesure
Symptômes	Symptômes notés au dossier médical au moment du diagnostic.	0. Aucun 1. Perte de poids 2. Soif 3. Polyurie 4. Polyphagie 5. Autres

Variable	Définition	Unité de mesure
Visites	Présence lors des cliniques de diabétiques offertes dans les villages, ou lors d'autres visites où il est question de contrôle et/ou suivi de diabète.	. Nombre de visites par an
Critères diagnostiques	Critères cliniques utilisés lors du diagnostic de diabète selon les notes au dossier médical.	0. Aucun de noté 1. Analyse d'urine 2. Glycémie à jeun 3. Glycémie à jeun et analyse d'urine 4. Test d'intolérance au glucose 5. Hémoglobine glycosylée 6. Autre
Traitement	Méthode utilisée actuellement pour le contrôle de la glycémie.	0. Aucune 1. Diète 2. Insuline seulement 3. Hypoglycémiants oraux seulement 4. Diète et insuline 5. Diète et hypoglycémiants oraux
Suivi glycémique	Méthode utilisée pour évaluer la qualité de contrôle glycémique	0. Aucun 1. Glucomètre 2. Analyse d'urine 3. Hémoglobine glycosylée 4. Glycémie à jeun 5. Autres, spécifiez
Suivi des complications	Nombre d'examens effectués pour évaluer l'apparition ou le développement des complications tels: analyse d'urine, ECG, examen neurologique, ... depuis le diagnostic de diabète.	Nombre par an pour chacun des examens requis

ADDENDA 2

INSTRUMENT DE CUEILLETTE DES DONNÉES AU DOSSIER

A - Caractéristiques de base

Ne rien écrire ici

1. Nom du patient _____

2. Numéro de bande : 0. Oui |_|1
   1. Non
   2. W.S. (white status)

3. Numéro d'identification: _|_|_ |_2|_|_|_|

4. Sexe : 0. M |_5|
   1. F

5. Village : 1. Chisasibi |_6|
   2. Poste de la Baleine (Whapmagoostui)
   3. Wemindji
   4. Eastmain
   5. Rupert (Waskaganish)
   6. Nemaska
   7. Waswanipi
   8. Mistassini
   9. Ne sais pas

6. Date de naissance : ___|___|___ |_7|_|_|_|_|_|_|
   An Mois Jour

7. Diagnostic : 0. Type I |_13|
   1. Type II

Ne rien écrire ici

8. Date de diagnostic : ___|___|___ (An Mois Jour) |__|__|__|__|__|__| 14

(Age au diagnostic): __|__ |__|__| 20

(Durée de la maladie en mois): jusqu'à juin 89 __|__|__ |__|__|__| 22

9. Histoire familiale de NIDDM : 0. Aucune |__| 25
1. Parents
2. Enfants
3. Frère/Soeur

10. Tabagisme : 0. Oui |__| 26
1. Non
2. Ne sais pas

B - Au moment du diagnostic

Ne rien écrire ici

11. Critère de diagnostic utilisé :
    0. Aucun noté
    1. Glycémie à jeun
    2. Analyse d'urine
    3. Glycémie à jeun et analyse d'urine
    4. Test d'intolérance au glucose 50g
    5. Test d'intolérance au glucose 75g
    6. Hémoglobine glycosylée
    7. Autre, spécifiez _____ |__|27

12. Symptômes notés au moment du diagnostic :
    0. Aucun
    I. Perte de poids
    2. Soif
    3. Polyurie
    4. Polyphagie
    5. Autre, spécifiez _____ |__|28

13. Poids : __|__ kg |__|__|29

14. Taille : __|__ cm |__|__|31

    __|__ (IMC) |__|__|33

C - Dans les derniers deux ans

Ne rien écrire ici

15. Cholestérol total (M.MOL/L) _____ 1987 |__|__|__| 35

_____ 88 |__|__|__| 38

_____ 89 |__|__|__| 41

16. Triglycérides (M.MOL/L) _____ 1987 |__|__|__| 44

_____ 88 |__|__|__| 47

_____ 89 |__|__|__| 50

17. Hémoglobine glycosylée (%) _____ 1987 |__|__|__| 53

_____ 88 |__|__|__| 56

_____ 89 |__|__|__| 59

18. Glycémie à jeun (M.MOL/L) _____ 1987 |__|__|__| 62

_____ 88 |__|__|__| 65

_____ 89 |__|__|__| 68

19. Fructosamine (M.MOL/L) _____ 1987 |__|__|__| 71

_____ 88 |__|__|__| 74

_____ 89 |__|__|__| 77

20. Glucomètre (M.MOL/L) _____ 1987 |__|__|__| 80

_____ 88 |__|__|__| 83

_____ 89 |__|__|__| 86

Ne rien écrire ici

21. Poids (Kg) _____ 1987 |__|__|__| 89

_____ 88 |__|__|__| 92

_____ 89 |__|__|__| 95

22. Visites faites _____ 1987 |__|__|__| 98

_____ 88 |__|__|__| 101

_____ 89 |__|__|__| 104

23. Activités traditionnelles: 0. Ne sais pas |__| 107
1. Non
2. Oui

Si oui, nombre de jours: En 1987-88 __|__|__ |__|__|__| 108

En 1988-89 __|__|__ |__|__|__| 111

D - Actuellement

Ne rien écrire ici

24. Médication actuelle : 0. Aucune — |__| (114)
1. Diète
2. Insuline seulement
3. Hypoglycémiants oraux seulement
4. Diète et insuline
5. Diète et hypoglycémiants oraux

25. Méthode de suivi glycémique : 0. Aucun — |__| (115)
1. Glucomètre
2. Analyse d'urine
3. Glycémie à jeun
4. Hémoglobine glycosylée
5. Fructosamine
6. Autre, spécifiez _____

26. Hypertension : 0. Ne sais pas — |__| (116)
1. Oui
2. Non

27. Diamètre hanche (crête iliaque) : __|__ cm — |__|__| (117)

28. Diamètre taille (ombilic) : __|__ cm — |__|__| (119)

(ratio 28/27) : __|__ — |__|__| (121)

29. Poids : __|__ kg — |__|__| (123)

30. Taille : __|__ cm — |__|__| (125)

__|__ (IMC) — |__|__| (127)

Ne rien écrire ici

31. Complications microvasculaires :

1. Rétinopathie 0. Oui 1. Non |__| 129

Si oui, indiquer la date du diagnostic

__|__|__ An Mois Jour |__|__|__|__|__|__| 130

2. Néphropathie 0. Oui 1. Non |__| 136

__|__|__ An Mois Jour |__|__|__|__|__|__| 137

3. Neuropathie périphérique 0. Oui 1. Non |__| 143

__|__|__ An Mois Jour |__|__|__|__|__|__| 144

32. Complications macrovasculaires

A Coronarienne :

1. Infarctus du myocarde 0. Oui 1. Non |__| 150

__|__|__ An Mois Jour |__|__|__|__|__|__| 151

2. Angine 0. Oui 1. Non |__| 157

__|__|__ An Mois Jour |__|__|__|__|__|__| 158

3. Pontage coronarien et/ou dilatation 0. Oui 1. Non |__| 164

__|__|__ An Mois Jour |__|__|__|__|__|__| 165

Ne rien écrire ici

B Vasculaire périphérique :

4. Pied de homard — 0. Oui 1. Non — |__|$_{171}$
   ___|___|___ An Mois Jour — |__|__|__|__|__|__|$_{172}$
5. Claudication intermittente — 0. Oui 1. Non — |__|$_{178}$
   ___|___|___ An Mois Jour — |__|__|__|__|__|__|$_{179}$
6. Amputation (non reliée au trauma) — 0. Oui 1. Non — |__|$_{185}$
   ___|___|___ An Mois Jour — |__|__|__|__|__|__|$_{186}$

C Cérébrale ischémique :

7. Accident cérébro-vasculaire — 0. Oui 1. Non — |__|$_{192}$
   ___|___|___ An Mois Jour — |__|__|__|__|__|__|$_{193}$
8. Amorose fugace — 0. Oui 1. Non — |__|$_{199}$
   ___|___|___ An Mois Jour — |__|__|__|__|__|__|$_{200}$
9. Ischémie cérébrale transitoire — 0. Oui 1. Non — |__|$_{206}$
   ___|___|___ An Mois Jour — |__|__|__|__|__|__|$_{207}$

E - Autres

Ne rien écrire ici

33. Depuis le diagnostic de diabète, indiquer par le médecin généraliste :

1. Nombre d'analyse d'urine	__|__	|__|__| 213
2. Nombre de créatine sérique	__|__	|__|__| 215
3. Nombre d'examen des fundis oculaires	__|__	|__|__| 217
4. Nombre d'électrocardiogramme (12 voies)	__|__	|__|__| 219
5. Nombre d'examen physique	__|__	|__|__| 221
6. Nombre d'examen physique et neurologique (incluant sens vibratoire et sensibilité)	__|__	|__|__| 223

34. Depuis le diagnostic de diabète, indiquer le nombre de consultation en:

1. Ophtalmologie	__|__	|__|__| 225
2. Neurologie	__|__	|__|__| 227
3. Cardiologie	__|__	|__|__| 229
4. Néphrologie	__|__	|__|__| 231
5. Diététique	__|__	|__|__| 233
6. Endocrinologie	__|__	|__|__| 235

ADDENDA 3

A qui de droit,

Lors de notre passage pour l'étude du diabète chez les communautés cries de la Baie James, nous n'avons pu obtenir certaines données utiles à notre recherche. Si cela est possible, nous aimerions que vous complétiez cette partie du questionnaire lors de la prochaine visite de ce patient et que vous nous le renvoyiez en utilisant l'enveloppe affranchie ci-jointe.

Merci de votre collaboration.

Paul Brassard _____

A - Caractéristiques de base

Ne rien écrire ici

1. Nom du patient _____ |__|$_{1}$

3. Numéro d'identification __|__|__ |__|__|__|$_{2}$

5. Village : 1. Chisasibi |__|$_{6}$
   2. Poste de la Baleine (Whapmagoostui)
   3. Wemindji
   4. Eastmain
   5. Rupert (Waskaganish)
   6. Nemaska
   7. Waswanipi
   8. Mistassini
   9. Ne sais pas

9. Histoire familiale de NIDDM : 0. Aucune |__|$_{25}$
   1. Parents
   2. Enfants
   3. Frère/Soeur

10. Tabagisme : 0. Oui |__|$_{26}$
    1. Non
    2. Ne sais pas

D - Actuellement

Ne rien écrire ici

27. Diamètre hanche (crête iliaque) : ___|___ cm |__|__| 117

28. Diamètre taille (ombilic) : ___|___ cm |__|__| 119

(ratio 28/27) : ___|___ |__|__| 121

29. Poids : ___|___ kg |__|__| 123

30. Taille : ___|___ cm |__|__| 125

___|___ (IMC) |__|__| 127

Autre(s) :

Addenda 4

Characteristics of Type I Diabetics at Time of Diagnosis, James Bay Cree Community

Sex	Age at Diagnosis	Year of Diagnosis	Village	Diagnostic Criteria	*Symptoms at Diagnosis	Continuous Insulin Medication	Body Mass Index†
F	21	1989	Waswanipi	Ketoacidosis	Yes	Yes	29
F	4	1984	Waswanipi	Ketoacidosis	Yes	Yes	N/A
M	37	1981	Mistissini	Ketoacidosis	Yes	Yes	27
F	21	1983	Mistissini	Ketoacidosis	Yes	Yes	28
M	25	1988	Nemaska	Ketoacidosis	Yes	Yes	N/A

* One or more of the following: stupor, apathy, polyuria, polydipsia, polyphagia.

† Defined as weight in kilograms over height in meters squared, at time of diagnosis.

Addenda 5

Type II Diabetics (WHO Criteria on Chart Diagnosis) per Communities, James Bay Cree, 1989

Village	Number	% Of All Type II Diabetics
Chisasibi	30	13.0
Whapmagoostni	6	2.6
Némaska	8	3.5
Waswanipi	42	18.3
Mistissini	87	37.8
Wemindji	10	4.3
Eastmain	10	4.3
Waskaganish	37	16.1
Total	**230**	**100.0**

Addenda 6

Type II Diabetes Prevalence Rate (%) by Age, Sex and Community, James Bay Cree, 1989

	20 - 29		30 - 39		40 - 49		50 - 59		60 - 69		70+		All ages (20 +)		
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M + F
Chisasibi	0	0	0	3 3	3	6.2	1.9	8 2	4	12.8	0	2 9	1.0	3.6	2.4
Whapmagoostui	0	0	0	4 0	0	0	6.6	12 5	0	10 0	0	0	0 8	2 9	1.9
Wemindji	0	0	0	3 6	2 8	5.2	0	4.8	0	5 8	6.2	25.0	0.9	3 9	2.3
Eastmain	0	2 7	0	3.4	16.6	14 3	0	18.2	0	12 5	0	11.1	1 9	7 5	4 7
Waskaganish	0	0.7	0	6.5	2 2	19 0	12.0	33.3	10.7	38.9	0	5.5	2.0	9.9	5.7
Némaska	0	2 7	0	7 1	8.3	7.1	20.0	0	0	10 0	12.5	0	3.2	5 0	4 2
Waswanipi	0	3 8	0	6.4	25.0	23.0	24.0	25.8	6.2	14.3	20 0	11.8	6 6	11.6	9 0
Mistissini	1.5	0.5	0.9	3.4	11.4	17.8	15.5	29.8	14.8	40 5	12.1	40.7	5.9	11.4	8.7
All Villages	0.3	0.7	0.6	4.3	7.0	11.8	9.9	18.6	5.7	20.8	5.6	15.3	3.0	7.2	
All Villages - Two sexes combined	0 5		2 5		9.7		14.1		13.1		10				5.2

Addenda 7

Some Reported Prevalence Studies Among Indians and Caucasians of Canada and Alaska

Origin	Ref. (From Ch 1)	Year	Prevalence	M	F	Overall	*±95% C.I.	Age Gp. Denominato r	Type	Diagnostic Criteria	Nation
Brassard *et al*		1989	Crude	0 07	0 04	0 06	0 05	All	I		
			Crude	1 6	3 7	2 6	0 3	All	I & II	WHO Criteria	James Bay
			Crude	3 0	7 2	5 2	0 7	20+	II	applied to chart	Cree, Quebec
			Standardized†	3 7	9 7	6 6	0 7	20+	II	diagnosis	
			Crude	7 7	15 8	11 8	1 8	40 - 69	II		
Young	10	1987	Crude	-		8 2		All	I & II	Chart diagnosis	Cree & Ojibwa
		1987	Crude	-	-	10 6		All	I & II	screening with WHO criteria	Northern Ontario & Manitoba
Montour	12	1985	Crude	-	-	12 0		45 - 64	II	NDDG criteria applied to chart diagnosis	Mohawks of Kannawake, Que
Evers	11	1985	Crude	10 0	7 7	9 8		5+	I & II	Chart diagnosis	Oneida, Chippawa
			Standardized†	13 9	15 5	14 7		5+		(No criteria applied)	and Muncy of Southern Ontario
Schraer	13	1985	Crude	-	-	02		All	I	WHO criteria	Aleuts Indians
			Crude	-	-	83		All	I & II	applied to	and Inuits of
			Standardized‡			1 57				chart diagnosis	Alaska, USA
Young	14	1983	Crude	-	-	2 8		All	I & II	NDDG criteria	Cree and Ojibwa
	15	1983	Crude	-	-	9 9		45 - 64		applied to chart diagnosis	from Northern Ontario and Manitoba
Young	4	1987	Crude	1 9	3 9	2 9		All	I & II	Chronic Disease	Other Native
			Standardized†	-	-	4 8				Registry (No Case Validation)	communities of Québec except Mohawks and Crees
Gillis	16	1980	Crude	-	-	3 4		20+	I & II	Chronic disease registry based on chart diagnosis	Saskatchewan Indians
Prince Edward Island (PEI)	30	1988	Crude	-	-	2 5		All	I & II	Chronic disease registry based on drug dispensing units	Caucasians of PEI
Evers	11	1985	Crude	2 2	1 3	2 0		5+	I & II	Chart diagnosis	Caucasians of South Western Ontario
			Standardized†	2 2	2 3	2 2					
Canada Health Survey	29	1978-1979	Crude	1 7	2 5	2 1		15+	I & II	Self reported	Caucasians of Canada

*95% Confidence Interval † To 1985 Population of Canada ‡ To 1980 Population of USA

Addenda 8

Proportion of Diagnosed Macrovascular Complications Per Sex,
James Bay Cree Indian Diabetics, 1989.

	Males		Females		TOTAL	
Type	Number	%	Number	%	Number	%
Ischemic Heart Disease *	7	10.3	16	9.9	23	10.0
Cerebral Vascular Disease †	3	4.4	5	3.1	8	3.5
Peripheral Vascular Disease ‡	1	1.5	1	0.6	2	0.9

* Myocardial Infarction, Angina, bypass and/or dilation

† Cerebro-vascular disease, amaurosis fugax, transient cerebral ischemia

‡ Amputation

Addenda 9

Proportion (%) of Diagnosed Type II Diabetics With Complications By Sex, James Bay Cree, 1989.

	Males	Females	TOTAL
Retinopathy	10.3	8 6	9 2
Nephropathy	8.8	11 1	10.5
Peripheral Neuropathy	17.6	6.2	9.6
Cataracts	6.1	5.6	5.7
Myocardial Infarction	7.6	3 7	4.8
Angina	7.6	8.0	7.9
Bypass - Dilation	0	0.6	0.4
Amputation	1.5	0.6	0.9
Cerebro-vascular Disease	3.0	1.2	1.8
Amaurosis fugax	0	0.6	0 4
Transient Cerebral Ischemia	1.5	1.2	1.3
TOTAL*	35.3	25.9	28.7

* With at least one complication

Addenda 10

Proportion (%) of Diagnosed NIDDM Complications Per Community, James Bay Cree, 1989

	Number*	%
Mistissini	28	32.2
Waswanipi	13	30.9
Waskaganish	11	29.7
Chisasibi	7	24.1
Eastmain	3	30.0
Némaska	0	0
Wemindji	3	30.0
Whapmagoostui	1	20.0
TOTAL	66	28.7

* With at least one complication.

Addenda 11

Diagnostic Criteria Used for NIDDM, James Bay Cree.

Criterion	Number	%
Fasting Plasma Glucose	128	55.7
Fasting Plasma Glucose and Urinalysis	18	7.8
Oral Glucose Test (75 g)	11	4.8
Others *	57	24.8
More Than One Type †	16	6 9
Total	230	100.0

* Not available.

† Can include glycosylated hemoglobin or fructosamine above those already mentioned.

Addenda 12

Frequency of Reported Symptoms at Time of Diagnosis of NIDDM, James Bay Cree.

Symptoms	Number	%
None	142	61.7
Weight Loss	2	0.9
Polydypsia	8	3.5
Polyuria	4	1.7
Others *	41	17.8
More than One †	33	14.3
Total	230	100.0

* Can include: fatigue, visual disturbance, headache, weakness.

† More than one of the enumerated symptoms.

Addenda 13

Glycemic Follow-Up for NIDDM, James Bay Cree, 1989.

Type	Number	%
None *	4	1.7
Self-Monitoring Device	24	10.4
Urinalysis	1	0.4
Fasting Plasma Glucose	3	1.3
Glycosylated Hemoglobin	2	0.9
More Than One Method †	196	85.2
Total	230	100.0

* Could not be found in either medical or nursing notes nor in any laboratory reports.

† Includes more than one method described.

Addenda 14

Comparison of Some Clinical Characteristics of Type II Diabetics From Different Native Populations of Canada

	James Bay Cree Québec	Mohawks Kahnawake Québec	Oneida Southwestern Ontario	Cree-Ojibwa Northern Ontario
Reference (From Chapter 2)		(9, 21)	(3)	(2, 10)
Number of diabetics studied	230	82	116	191
Mean age at diagnosis	48.3			
Male	48.8	51.6	44.2	
Female	48.0	54.1	40.8	
Mean duration of diabetes (years)	5.0			
Male	4.7	7.1	8.4	
Female	5.2	5.8	9.8	
Female to Male ratio	2.4:1	1.4:1	0.9:1	2.5:1
% Family history of diabetes	38.6		51.5	
Mean body mass index	36.7			
Male	34.6	30.0	29.2	28.4
Female	37.4	33.0	29.5	30.3
% with symptoms at diagnosis	38.3	31.7	75.0	42.0
% with hypertension	43.4	71.0		36.3
% with hypercholesterolemia	9.3	16.0		
% with hypertriglyceridemia	25.0			
% who smoked	16.2	29.0		
% with microvascular disease	19.6	18.3		7.8
% with macrovascular disease	14.4	63.0		25.0
% with peripheral neuropathy	9.6	6.0		6.3