THE EPIDEMIOLOGY AND CONTROL OF NON-INSULIN DEPENDENT DIABETES MELLITUS IN TWO REMOTE ABORIGINAL COMMUNITIES

BY

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THE EPIDEMIOLOGY AND CONTROL OF NON-INSULIN DEPENDENT DIABETES MELLITUS IN TWO REMOTE ABORIGINAL COMMUNITIES

BY

DOUGLAS QUARRY

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ABSTRACT

In 1994, data on the epidemiology, degree of obesity, and metabolic control was gathered from the medical records of 153 diabetics identified in the Island Lake area of Northern Manitoba. The success of drug versus non-drug regimens in attaining glycemic control was determined by comparing "intervals of therapy", defined as periods of four or more months in which patients used one particular treatment regimen (no-drug, sulphonylurea, metformin, or insulin). The crude prevalence of diabetes was 45/1000, with a maximum prevalence of 372/1000 in men aged 55-64 years. Thirty percent of diabetics had retinopathy, 19% had peripheral neuropathy, 19% had peripheral vascular disease, 3.9% had had a lower limb ulcer, and 2.9% had a lower limb amputation. A total of 190 intervals of therapy were identified. Over 70% of the intervals of therapy associated with each of the four treatment regimens showed compromised glycemic control (HBA_1C > 9.8%). There was no significant difference in the mean level of glycemic control between the treatments. The high prevalence of complications and the lack of success in achieving glycemic control regardless of treatment regimen suggest that diabetes programs on isolated Indian Reservations should focus on the prevention and management of the complications of NIDDM rather than the manipulation of hypoglycemic drug therapy.

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1. INTRODUCTION

The relatively recent onset, and even more recent recognition of the epidemic of non-insulin dependent diabetes (NIDDM) sweeping Canadian aboriginal peoples has led to a surge of epidemiological research. In some communities the prevalence of NIDDM has been shown to be higher than the Canadian average and this prevalence seems to be increasing with time. NIDDM is now a major cause of morbidity and mortality for aboriginal people. Physicians, nurses, community health workers and diabetes educators suspect that, in general, the control of NIDDM in remote aboriginal communities is very poor. The publication of management guidelines[1,2] does not seem to have improved the control of NIDDM nor lead to uniform treatment; in fact therapy varies widely. There are no published studies which carefully examine the determinants of metabolic control of NIDDM on remote aboriginal communities.

This apparent poor control of diabetes has left many health-care professionals confused about how best to manage diabetic patients. Often the manipulation of hypoglycemic drug therapy is the main focus of management. Other aspects of the management of NIDDM, especially strict control of blood pressure, regular ophthalmological examination (with laser photocoagulation

therapy when required), and foot care, are often foregone or undertaken in a haphazard manner.

This study reviews the literature in relation to NIDDM in Canadian aboriginal communities and examines what is known about the prevention of the complications of NIDDM. It determines the prevalence of NIDDM and its complications on two isolated aboriginal communities. It examines the metabolic control of NIDDM in relation to the various methods of treatment (both drug and non-drug), and documents the degree of obesity of the diabetic patients and weight changes which occur with the various forms of therapy.

Specific policy suggestions are made about the achievement of metabolic control and the prevention of complications.

2. REVIEW OF THE LITERATURE

2.1 EPIDEMIOLOGY OF NIDDM IN CANADIAN NATIVES

Information on the prevalence of diabetes among Canadian Indians prior to 1940 is limited. Chase[3] reported in 1937 that "Indians are not subject to diabetes" after medical examination of over 1500 native Saskatchewan people failed to reveal any cases. In 1935, Urquhart[4] noted the absence of glycosuria among the Athapaskin Indians in Aklavic, Northwest Territories. West[5] concluded that diabetes was rare, if not absent, in American Indian tribes before the 1940's. He extensively reviewed studies of urine testing, anecdotal stories and Indian Health Service records and concluded that the current epidemic of diabetes among Amerindians "began in the 1940's in a few tribes, reached substantial proportions of geographic scope and severity in the 1950's and became massive in the 1960's".

In 1985, Montour[6] noted that 12% of the 544 Mohawk Indians resident in Kahnawake aged 45-65 were diabetic (having venous whole blood glucose greater than 6.7 mmol/l before meals or greater than 10 mmol/l two hours after meals). In the same year, Young, Dooley et al[7] described the epidemiology of NIDDM among Indians in north-western Ontario and north-eastern Manitoba. The overall prevalence was 28/1000. The prevalence for those aged 15-64 was 46/1000, and was 96/1000 for those aged 65 years and over. At that time the overall Canadian

figures from the Health Canada Survey[8] were 15/1000 for those aged 15 to 64 and 67/1000 for those aged 65 and over. The female/male ratio in the study groups was 2.5:1 compared to the Canadian figure of 1.5:1.

In 1987, Evers et al[9] reported that the age-adjusted prevalence of known diabetes amongst the Indian residents of the Carodac Reserve and Oneida Settlement was 14.7% whereas the rate among Caucasians was 2.2% (which was similar to the rate of 1.95% reported for registered diabetics of all ages in Prince Edward Island).[10]

In 1988, Dean and Moffatt[11] reported on 15 confirmed cases of diabetes amongst Indian children in Manitoba. They concluded that 6 of the 15 cases had IDDM (diagnosed by way of having had ketoacidosis), 5 cases definitely had NIDDM and 4 cases probably had NIDDM. Seven of the cases were concentrated amongst the 1,243 children of the Island Lake area (the area in which this study was undertaken), giving a prevalence rate of 5.6/1000, comparable to the rate in Pima Indians.[12] Studies are currently under way to examine the genealogy of these cases.[13]

Young, Szathmary et al[14] produced the most comprehensive summary of the geographical distribution of diabetes among the native population of Canada. The prevalence of diagnosed

diabetes was determined for 76% of the registered Indian and Inuit population of Canada from case registers maintained by Medical Services Branch, Department of Health and Welfare. A total of 5,324 cases of NIDDM were identified. The age-sex adjusted rate varied among the Indians from a low of 0.8% in the Northwest Territories to a high of 8.7% in the Atlantic region. Most cases occurred in middle aged or older individuals, with a higher prevalence among Indian (but not Inuit) females. A clear north-south gradient was observed in the prevalence rates of NIDDM with diabetes rates decreasing with increasing northern location of the population. However the degree of geographical isolation did not seem to have any independent effect on diabetes prevalence. In most areas of Canada, the age-sex adjusted rates for diabetes amongst natives was 2-5 times higher than in all other Canadians. There was a wide variation in the prevalence of NIDDM in Canadian native populations with the age-adjusted rate among the Indians in the Atlantic region being 22 times higher than the rate of the Inuit.

In southern Saskatchewan, two prevalence surveys five years apart in the same two communities indicated a rise in prevalence of NIDDM from 1.9% in 1980 to 3.8% in 1985.[15,16]

In 1993 Brassard et al[17] described the epidemiology of the 230 non-insulin dependent diabetics identified in the James Bay

Cree population. Mean age at diagnosis was 48.3 years and the mean duration of illness at the time of the study was 60.4 months. Seventy-seven percent of diabetics were overweight (BMI > 26 kg/m^2) and 65.4% obese (BMI > 30 kg/m^2). Microvascular disease (diabetic nephropathy or retinopathy) occurred in 19.6% and 76.4% showed poor control (fasting plasma glucose > 7.8 mmol/l). Dietary interventions were said to have been unsuccessful as the body weight of 97% of the diabetics had not changed from the time of diagnosis until the time of the study.

Young and Harris[18] examined the risk of clinical diabetes in a Northern Native Canadian population. A cohort of 630 non-diabetic adults were followed for 46-63 months. During this period, 22 new cases were identified, giving an incidence density of 8.0/1000 person years. BMI and fasting plasma glucose were significant predictors for the development of diabetes.

Fox, Harris and Whalen-Brough have recently described that in aboriginal communities in the Sioux Lookout Zone of Northwestern Ontario there has been an apparent increase in the crude prevalence of NIDDM from 28/1000 to 38/1000 over the last ten years.[19]

It is likely that all prevalences described here represent an

underestimate of the true prevalence of NIDDM as, according to the United States National Health and Nutrition Examination Survey (1976-80), for every known case of diabetes there is at least one undiagnosed case.[20]

2.2 EPIDEMIOLOGY OF THE COMPLICATIONS OF NIDDM IN AMERICAN AND CANADIAN NATIVES

The complications of diabetes include nephropathy, retinopathy, neuropathy, and vascular disease (peripheral, coronary and cerebral).

2.2.1 Retinopathy

In North America, diabetic retinopathy is the leading cause of new blindness in adults.[21] Lee et al[22] reexamined a cohort of Oklahoma Indian diabetics 12 years after initial ophthalmological screening and found the overall prevalence of proliferative retinopathy to be 18.6%. Of those who had background retinopathy at the initial screening, 45% had developed proliferative retinopathy on review. The prevalence of proliferative retinopathy among Pima diabetics is 18%.[23] On multi-variate analysis, the incidence of retinopathy among the Pima was positively associated with duration of diabetes, age, hypertension, hypercholesterolemia and treatment with insulin. No case of proliferative retinopathy was found in subjects aged less than 35 years of age.[24]

Ross and Fick[25] reported a high prevalence of serious and untreated retinopathy among native diabetics in southern Alberta. The study suggested that exogenous insulin therapy itself may be a risk factor for the development of diabetic retinopathy.

2.2.2 Nephropathy

Diabetes can lead to nephropathy and eventually to end-stage renal failure (ESRF). Diabetic nephropathy is defined as the onset of persistent proteinuria with raised blood pressure in patients with chronic diabetes mellitus.[26] The life expectancy in such patients is reduced by a high risk of renal failure or cardiovascular disease, or both.[27] Pettit et al[28] found that, among the Pima, when controlled for age, sex, and duration of diabetes, diabetic subjects with proteinuria had a death rate of 3.5 times as high as those without proteinuria. Heavy proteinuria (> 1 gm/day) is strongly associated with the development of renal insufficiency.[29]

Bennett and Miller[30] found nephropathy to be present in 43% of Pima who had diabetes for ten years. In diabetic Mohawk at Kahnawake the overall prevalence of nephropathy is 4%; this rises to 11% in those who had diabetes for 10 years or more.[31] The incidence of ESRF among Pima with diabetes exceeds that of the general U.S. population by a factor of ten.[32] The Zuni have 10.6 times the prevalence of ESRF of New Mexico whites.[33]

Young et al[34] have shown that compared to the Canadian population, native Canadians have 2.5 times the standardized incidence rate of ESRF. Dyck and Tan[35] found that in Saskatchewan, the prevalence of ESRF is 16.2 times higher in the native population than the non-native population. Even when controlled for the higher rate of diabetes among the native people, native people were seven times as likely to have ESRD. The James Bay Cree of northern Ontario have an ESRD rate of 3.2 times the national average and the annual incidence rate from 1981-1989 was 1.6 times the national rate.[36]

2.2.3 Vascular Disease and Amputations.

Over 60% of Mohawk diabetics have had at least one major vascular complication (ischemic heart disease, stroke or peripheral vascular disease). The adjusted risk for having these complications was 6 times that of non-diabetics.[37]

An excessive incidence of lower limb amputations (LLA) attributable to NIDDM was found in the Pima[38] and Ghodes[39] has noted that as many as 88% of all LLA's among certain American Indian populations were related to diabetes. Among the Pima, the age-sex adjusted rate of LLA was almost four times the rate in the six U.S. states from which population estimates were obtained.

2.3 ETIOLOGY OF NIDDM

2.3.1 Genetics

The etiology of NIDDM has a genetic component; evidence supporting this includes familial aggregation in populations with a high prevalence of NIDDM[40] and identical twin studies.[41] Also, NIDDM is more prevalent in some populations including Micronesians, Polynesians, Mexican Americans, American Indians, Asian Indians, and Australian Aborigines.[42,43]

Studies in the U.S. have shown that Indian ancestry is an important risk factor in the development of NIDDM. Gardner et al[44] suggested that the degree of Indian admixture has a dose-response relationship with the presence of diabetes. Similar studies have not been performed in Canada as native Canadians are not categorized according to their "blood quantum". In the Island Lake region of Manitoba, the inheritance of NIDDM in an extended family is currently being investigated.[13]

An explanation of the increased susceptibility of former hunter-gatherer populations to NIDDM was advanced by Neel[45] 30 years ago when he proposed the "thrifty" genotype hypothesis. Neel suggested that these populations had evolved a mechanism, a "quick insulin trigger", which gave them an advantage under the traditional conditions of unstable food supply. This allowed quicker storage of fat in periods of relative plenty. Now that food is both regular and abundant and often combined

with a sedentary life-style, this mechanism may result in hyperinsulinemia, itself causing insulin resistance, upper body obesity, and NIDDM. In 1991 Wendorf and Goldfine[46] refined this theory suggesting that the thrifty genotype may initially cause insulin resistance in muscle tissue. This resistance would blunt the hypoglycemia associated with fasting and allow energy to be stored in fat cells and the liver during feeding. Halea and Barker[47] then advanced the "thrifty phenotype" hypothesis, postulating that poor fetal and early post-natal nutrition may affect the development of B-cells in the pancreas. Later in life, and possibly in conjunction with other factors (such as obesity, physical inactivity, and other factors leading to insulin resistance), a lack of insulin leads to hyperglycemia. They argue that over the years many studies, failing to control for the effects of obesity and unable to distinguish insulin from pro-insulin, have falsely concluded that a proportion of those with NIDDM have hyperinsulinemia. They propose that NIDDM is universally associated with hypoinsulinemia. In 1994, McCance et al, noting a "U" shaped association between birth weight and NIDDM, suggested that those low birth weight babies with a genetic predisposition to insulin resistance are more likely to survive than those without and named the postulate the "surviving small baby syndrome".

However, the suggestion that following Westernization, some

groups may be irrevocably destined to develop NIDDM is countered by the finding that the major metabolic abnormalities of NIDDM were either greatly improved or completely normalized when a group of urbanised Australian aboriginal diabetics temporarily reverted to a traditional lifestyle.[48]

2.3.2 Obesity

The role of obesity in the pathogenesis of NIDDM is complex and subject to the confounding influence of several related factors including possible hyperinsulinemia, physical inactivity, and the centralized distribution of fat.[43]

Longitudinal studies among the Pima Indian tribe of Arizona have shown that obesity, measured by body-mass index (BMI), is highly predictive for the development of diabetes. However, the prevalence of diabetes is not associated with concurrent obesity, possibly because once diabetes develops, weight loss usually occurs.[49]

Degree, duration, and distribution of obesity have been investigated. Degree of obesity appears to predict the early onset of NIDDM.[40] Szathmary has demonstrated that "central" fat distribution is associated with a higher prevalence of diabetes in several populations.[50] Everhart and associates demonstrated an association between duration of obesity and

the incidence of diabetes (adjusted for age, sex and BMI) among the Pima.[51] Duration of obesity in Pima children born to diabetic mothers acts synergistically with genetic susceptibility to increase the risk of the child developing NIDDM.[40]

Obesity appears to be a common but not necessary precursor to NIDDM. Even among Pima with a low BMI (20-25), the age-sex adjusted incidence rate of diabetes is still eight times that of whites. It may be that the etiology of obese NIDDM differs from that of non-obese NIDDM.[52] Conversely, obesity may prove to be a common effect rather than cause of diabetes. It may be that both are manifestations insulin resistance with hyperinsulinemia being the intervening mechanism.[53]

2.3.3 Exercise

The effect of exercise on carbohydrate metabolism in both diabetic and non-diabetic subjects suggests that exercise may be an important protective factor against the development of NIDDM. It has been shown that physically trained insulin-resistant obese subjects can decrease their plasma insulin values by approximately 50% without decreasing body fat.[54] Many studies show that physically active men have a lower prevalence of NIDDM (independent of age, obesity and urban living) than inactive men.43

There has been concern among epidemiologists about the lack of standardized approaches in assessing physical activity levels.[55] Kriska et al. developed a comprehensive questionnaire to assess physical activity in Pima Indians.[56] With this questionnaire it was demonstrated that a history of physical activity was inversely associated with NIDDM (after controlling for BMI, sex, age, and parental diabetes status).[57]

It seems likely that the role of physical inactivity as a major risk factor for NIDDM has been underestimated because many investigators have focused their work on obesity.[43]

2.3.4 Prenatal environment

The prenatal environment has been shown to predict subsequent diabetes, independent of genetic inheritance. Pettitt et al[58] showed that at age 20-24, the prevalence of diabetes was 45% among those whose mothers were diabetic during the pregnancy, 9% among those whose mothers were pre-diabetic during pregnancy, and only 1.4% among those whose mothers were not diabetic during pregnancy.

2.4 PRIMARY PREVENTION OF NIDDM

The development of successful public health policy relating to chronic diseases has three phases: 1) observational epidemiological studies, 2) intervention trials, and 3) public health action. Diabetes epidemiology has only just entered the second phase, in contrast to cardiovascular disease which is well into the third phase. Stern[59] warns against making premature policy decisions as "in the absence of a solid scientific base, it is difficult to marshall the necessary commitment of societal resources for a sustained public health effort."

Little is known about the primary prevention of NIDDM.[60] There is general consensus that improved dietary practices and increased physical activity are the most promising interventions to explore.[55] The Sioux Valley Diabetes Primary Prevention Project, Manitoba Canada[61], is assessing the impact of a sustained and comprehensive health promotion program (emphasizing both dietary practices and physical activity) on the prevalence of hyperglycemia (measured by glycated hemoglobin) and obesity (measured by BMI).

2.5 MANAGEMENT OF NIDDM

The Expert Committee of the Canadian Diabetes Advisory Board[1] states that the goals of diabetes care are:

- To relieve the patient's symptoms.
- To prevent and treat acute and long-term complications.
- To promote self-care when appropriate.
- To treat accompanying disorders.
- To improve the quality of the patient's life.
- To reduce morbidity and mortality associated with diabetes.

The Canadian Diabetes Association expert committee[62,63] states that unless a patient is very symptomatic, diet and exercise (without drugs) will control the diabetes in a significant percentage of patients. When metabolic control does not result, the choice is between oral hypoglycemic agents and insulin. In those in whom diet and exercise therapy have failed, oral hypoglycemic medication is recommended if fasting plasma glucose is less than 13 mmol/l, with a change to insulin if control is not achieved. For those with fasting plasma glucose of 13 mmol/l or more, insulin is recommended as the initial drug of choice.

2.5.1 Diet therapy

Obesity associated with diabetes increases the risk of mortality. The mortality ratio for patients with diabetes who are 20-30% above average body weight is 2.5-3.3 times higher than diabetics of normal weight, and 5.2-7.9 times higher among diabetics with body weights more than 40% above ideal.[64]

The Expert Committee guideline[1] states that in obese individuals with NIDDM "an adequate trial of diet alone is essential prior to the use of oral hypoglycemic agents or insulin" and that "a modest weight reduction (5-10 kg) may be effective in achieving marked improvement in or total disappearance of glucose intolerance." The CDA guideline advises that diets with energy deficits of 2100-4200 kJ (500-1000kcal)/day should produce a weight loss of 0.25-1.0 kg/week and but notes also that the exogenous administration of insulin may make weight loss more difficult.

The utility of dietary change for reversal of early NIDDM was determined by the United Kingdom Prospective Diabetes Study (UKPDS - 1990)[65], which followed 3,044 newly diagnosed, obese diabetic patients through a 3 month dietary intervention. On average, patients with a fasting plasma glucose of 10-12 mmol/L needed to lose 28% (18 kg) of ideal body weight to attain near-normal fasting plasma glucose concentrations. The study confirmed the value of dieting and weight loss in

reducing hyperglycemia in newly diagnosed diabetics but emphasized the large weight loss and large reduction in energy intake required to attain these ends with diet alone.

Sustaining weight loss is an obstacle to the dietary therapy of NIDDM. Although some health care professionals claim that 70-80% of persons with NIDDM can control their blood glucose levels by bringing their weight to normal and maintaining that weight[66], successful control through diet may be more ideal than real for many patients. There may be a biological basis for the difficulty sustaining weight loss presents for obese NIDDM patients.[67] There is also growing concern that repeated cycles of weight loss and regain may have adverse metabolic consequences for NIDDM.[68]

Eckerling and Kohrs[69] examined the problem of the low compliance with diabetic dietary regimens and concluded that the often asymptomatic nature of NIDDM may foster apathy in the patient toward modifying established dietary behaviour. Compliance may hinge on the patient's health belief model and knowledge of diabetes, extent of social support, and the degree to which modified behaviour disrupts family and social habits. Cultural sensitivity in dietary modification strategies would be likely to help the outcome as dietary behaviour is deeply rooted in social norms.

It has been reported that dietary interventions have not been successful in the Cree of James Bay as the weight of 97% of the diabetics had not changed from the time of diagnosis until the time of follow-up, a median of 30 months later.[17]

2.5.2 Exercise Therapy

A major project among North American Indians to study the effect of exercise on NIDDM, the Zuni Diabetes Project[70,71] demonstrated that after two years of follow-up, participants in the exercise program compared with non-participants experienced weight loss, a drop in fasting blood glucose values, and a reduction in the use of hypoglycemic medications. Thirty percent of participants developed normal blood glucose in contrast to only 9% of non-participants. In a weight-loss competition, 45% of enrolles finished and lost $\geq$ 2.3 kg. It was suggested that this study demonstrated that 1) participation in a community-based exercise program can produce significant weight-loss and improvement in glycemic control in Zuni Indians with NIDDM, and 2) weight-loss competitions appear to be an important public health model for health behaviour change in communities similar to that of the Zuni.

The Expert Committee[1] recommends that with appropriate precautions, exercise therapy should be part of the treatment plan for persons with NIDDM.

2.5.3 Oral Hypoglycemic Agents

Oral hypoglycemic agents have been shown to be useful in augmenting basic dietary and exercise therapy although many patients fail to respond (primary failure) or lose sensitivity to their effects over time (secondary failure).

The sulfonylurea group of drugs increase insulin secretion from the pancreas.[72] The increased portal flow of insulin into the liver reduces postprandial hepatic glucose output. The sulphonylurea glyburide has been shown also to enhance the responsiveness of the B-cell to glucose.[73]

Side effects occur in 5% of those using sulfonylurea agents and cause discontinuation of therapy in 1-2%. The most common side effects of sulphonylureas include hypoglycemic episodes, and gastrointestinal and cutaneous complaints.[74] Rosenstock[75] (1987) compared the efficacy and safety of low-dose insulin regimen with the sulphonylurea glipizide in 79 patients with NIDDM. Glycemic status was at least as good using glipizide. The rate of hypoglycemic reactions corrected for duration of treatment in 135 patients was 0.32 event per patient-month of therapy with insulin compared to 0.12 event per patient-month of therapy with glipizide.

Metformin has also been found to be an effective hypoglycemic agent and, because it causes less weight gain than

sulphonylureas or insulin, is often recommended as the first line drug to be used in obese diabetics when a trial of diet and exercise has failed.[1,76] The antihyperglycemic effects of metformin are primarily due to the suppression of hepatic glucose production, not stimulation of peripheral glucose uptake.[77] While metformin causes less hypoglycemic episodes than sulphonylurea drugs, it is often associated with dyspeptic symptoms. To avoid this, treatment starts at a lower dose and builds up to the final therapeutic dose over several weeks.

2.5.4 Insulin

Insulin treatment is often seen as the final treatment option in a hierarchy of interventions of increasing severity, initiated when treatment goals are not met by diet or oral agents. Insulin is often also recommended as the initial therapy in normal weight or slightly obese patients who have severe hyperglycemia when diagnosed.

In NIDDM, insulin reduces hyperglycemia by suppressing hepatic glucose production (in both the fasting and post-prandial states) rather than increasing the disposal of glucose; this has been shown to secondarily improve insulin secretion.[78] While approximately one-third of all patients with NIDDM are treated with insulin[79], the beneficial long-term effects of insulin therapy on morbidity and mortality have not been

proved and the possible role of hyperinsulinemia as a predisposing factor for atherosclerosis remains unresolved.[78] Some authors suggest that if sufficient amounts of insulin are given, the blood glucose level can be controlled in nearly all patients with NIDDM.[72] Scarlett and associates[80] (1982) showed that the use of exogenous insulin significantly improved glycemic control, mean glycated haemoglobin level, and urinary glucose excretion in patients with NIDDM. In 1988 Harris and Davidson demonstrated that exogenous insulin therapy slowed weight loss in patients with NIDDM.[81]

The United Kingdom Prospective Diabetes Study (UKPDS) recently released its 13th report[82] and concluded that hypoglycemic drugs (sulphonylureas, metformin, and insulin) had similar glucose lowering efficacy.

2.6 PREVENTION AND MANAGEMENT OF COMPLICATIONS

The Diabetes Control and Complications Trial (D.C.C.T.)[83] has recently demonstrated that, under ideal conditions, intensive insulin treatment can delay the onset and slow the progression of nephropathy, retinopathy and neuropathy in patients with insulin dependent diabetes mellitus (IDDM). There is no equivalent study in NIDDM - the UKPDS may, in the future, determine if there is a link between control of NIDDM and the complications of diabetes. While it has not been demonstrated that control of NIDDM effects the onset or progression of

complications, it has been shown that appropriate medical management of several of the complications has a beneficial effect on the outcome.

2.6.1 Retinopathy

All persons with diabetes are at risk of retinal complications, although persons with insulin dependent diabetes (IDDM) face a greater danger of severe vision loss than persons with non-insulin dependent diabetes (NIDDM)[84]. However, patients with NIDDM are more numerous and comprise a larger proportion of those affected with diabetic retinopathy. By 10 years after the onset of disease, at least 70% of those with NIDDM have some form of retinopathy. The risk of developing retinopathy continues throughout life. Lee et al[22] found that duration of diabetes, the level of metabolic control, and the systolic blood pressure were predictors of the development of proliferative diabetic retinopathy.

Retinopathy has two stages:the non-proliferative stage, in which intraretinal microaneurisms, hemorrhages, and soft and hard exudates, typically occur well before the more serious proliferative stage, which is characterised by neovascularisation and fibrovascular growth from the retina or optic nerve. Macular edema, a serious development, can occur in either stage. Untreated neovascularization and/or macular edema are the two major retinal complications which can lead

to blindness.

Advances in the medical care of diabetes and the surgical treatment of diabetic retinopathy have substantially reduced the risk of vision loss. The Diabetes Control and Complications Trial (DCCT)[84] demonstrated that in IDDM, the microvascular complications of diabetes, including retinopathy, can be markedly reduced with good control of serum glucose. Three prospective randomised clinical trials - the Diabetic Retinopathy Study (DRS)[85], the Early Treatment Diabetic Retinopathy Study (ETDRS)[86], and the Diabetic Retinopathy Vitrectomy Study (DRVS)[87] - have shown that severe vision loss can be avoided with appropriate laser surgery or vitrectomy surgery. Patients being considered for laser therapy must be informed that there is a possibility of a decrease of visual acuity of one or more lines and constriction of peripheral visual fields but that the risk of severe visual loss without therapy outweighs these side-effects.

The Expert Committee of the Canadian Diabetes Advisory Board[1] recommends that, in the absence of organized screening programs, the following groups should be referred to an ophthalmologist for a detailed dilated fundoscopic examination:

- All patients who have had NIDDM for five or more years.
- All patients aged 30 or more when they are first diagnosed to have diabetes.
- All diabetic patients with minimal background retinopathy or other ocular pathology.
- All women with diabetes in the first trimester of pregnancy.

See **ATTACHMENT A** for classification of diabetic retinopathy and schedule of ophthalmologic follow-up examinations for patients with diabetes.

2.6.2 Nephropathy

Hypertension can contribute to a progressive deterioration of renal function in patients with diabetic nephropathy.[88] The treatment of the hypertension has been shown to reduce the rate of deterioration of renal function[89] and more recently the use of captopril and enalapril, drugs of the angiotensin-converting enzyme inhibitor (ACE) group, have been shown to protect against the deterioration of renal function in insulin

dependent diabetics more effectively than blood pressure control alone.[90,91] These drugs have also been shown to prevent the development of nephropathy in normotensive diabetics with persistent micro-albuminuria, possibly due to the reduction of intraglomerular pressure.[92,93] Captopril has been shown to have a significant antiproteinuric effect in patients with nephrotic proteinuria accompanied by an arrest in the progression of renal insufficiency.[94]

The Expert Committee[1] recommends that for diagnostic and therapeutic purposes, the systolic pressure should be less than 140 mmHg and the diastolic pressure less than 90 mmHg however, a recent unpublished study has found that lowering blood pressure from 140/90 mmHg to 125/75 mmHg in patients with nephrotic level proteinuria slows the progression of renal deterioration by as much as 50-60%.[95]

2.6.3 Peripheral Vascular Disease / Peripheral Neuropathy / Ulceration / Amputation.

Peripheral neuropathy, independently or in combination with peripheral vascular disease, can increase the risk of lower limb ulceration, gangrene, or both. These can lead to lower-limb amputation (LLA). Diabetics are 2.94 times as likely to develop foot ulceration as non-diabetics. Duration of diabetes, absence of light touch, impaired pain perception, absence of dorsalis pedis pulse, and the presence of diabetic

retinopathy have been shown to be significant predictors of diabetic foot ulceration.[96] The absence of sensation, measured by a mono-filament technique (see below), together with foot deformity is positively correlated with an increased risk of LLA.[97] Pecoraro et al found that 72% of LLAs were associated with a causal sequence of minor trauma, cutaneous ulceration, and subsequent failure of wound healing.[98]

In a recent detailed review article, Grunfield[99] notes that despite its clinical importance, the methods of treatment of this condition are largely the product of subjective impressions rather than of objective studies. In attempting to "separate the wheat from the chaff", Grunfield makes the following points:

- The Semmes-Weinstein mono-filament technique of diagnosing neuropathy has been well tested. This technique is simpler and more reliable than alternative thermal or vibratory techniques.

- Pressure, structural and biomechanical factors, and vascular disease are the main factors involved in the development of ulceration.

- The development of pressure induced ulceration is proportional to both the amount and timing of the

pressure applied. Common causes of ulceration include: continuous pressure (e.g., that which occurs on the top or side of the feet due to ill-fitting shoes), intermittent pressure (e.g., that which occurs on the bottom of the feet during walking), objects inadvertently left in shoes which, because of neuropathy, cannot be felt and cause areas of pressure necrosis, and even holes in shoes allowing the foot to come into contact with the ground. It has also been shown that increased pressure occurs during the normal course of walking in patients with severe diabetic peripheral neuropathy.

- Structural and biomechanical factors increase the pressure and reduce "cushioning" in the feet of diabetics. These factors include: thinning of muscle and fat pads, hammer toe and claw foot deformities, Charcot joints, bunions, callus, deceased ankle and sub-talar range of motion, and an inability to sense pain and change gait. Thus hammer toes, in addition to neuropathy, are major factors in the development of ulceration under the metatarsal heads.

- Adequate clinical criteria to determine at what level a patient is at risk for ulceration from vascular disease have not been identified. Grundfield's group estimates that 60-70% of diabetics with foot ulcers have neuropathy

without vascular disease, 15-20% have some sign of vascular disease and 15-20% have both neuropathy and vascular disease.

- Infection is a common and important cause for ulcers not to heal, however, swabs taken directly from ulcers are heavily contaminated with skin flora. Culture following debridement and curettage more accurately reflects the infecting pathogens; these are commonly penicillinase-producing staphylococci, drug-resistant gram-negative rods, anaerobes, and enterococci.

- The first step in the treatment of diabetic foot ulcers is the removal of necrotic tissue and the prompt control of infection. Studies conducted so far have not adequately tested the relative effectiveness of different antibiotic regimes in the treatment of infected diabetic foot ulcers; the design limitations have included: methods of culture, distinguishing ulcers complicated by osteomyelitis, and lack of randomization.

- There are no clear-cut guidelines for vascular intervention in the treatment of diabetic foot ulcers. This comes partly from the experience that a significant number of ulcers do not heal despite normal blood flow. A practical solution is to arrange vascular work-up in

patients in whom the ulcer is not healing and the pulses are not bounding. This investigation can include the measurement of the ankle/brachial pressure index, angiography (with care to prevent dye induced renal damage), and the transcutaneous measurement of tissue oxygenation.

- In a randomized study, Mueller et al[100] found that "total contact casting" (immobilizing the foot in a knee-to-toe cast to direct pressure more evenly over the foot and away from the ulcer) was associated with a significantly higher percentage of ulcer healing than conventional treatment; however more studies of this modality are required. Studies are also required to show the relative effectiveness of various topical agents claimed to accelerate ulcer healing.

- Grunfeld suggests that all patients with a history of ulceration and those at risk from neuropathy or vascular disease should be entered into intensive education and examination programs. Multidisciplinary clinics for the referral of high-risk patients should be available. There is sufficient reason to suspect that proper footwear fitted correctly will reduce the incidence of ulceration. This should be available to patients at risk of ulceration.

Chantelau et al have shown that up to 85% of amputations can be prevented with intensive foot care, training and education.[101] The Expert Committee[1] recommends appropriate patient education in foot care and regular examination of the feet by qualified health personnel.

3. METHODS

3.1 OBJECTIVES

The objectives of this study are to:

1. Describe the epidemiology of NIDDM and its complications in two remote Canadian aboriginal communities.
2. Describe the degree of obesity of those with NIDDM on these communities.
3. Measure the success of drug versus non-drug regimens in achieving glycemic control and any relationship to patient's weight, sex and morphology.
4. Develop specific recommendations for the management of NIDDM on remote aboriginal communities such as Garden Hill and Red sucker Lake.

3.2 PATIENT POPULATION AND LOCATION

The Chiefs and Councils of the Garden Hill and Red Sucker Lake First Nation Communities agreed for this study to be conducted in their communities. They felt that this would improve their knowledge of NIDDM, and that this was important as both communities are considering transfer to local control of health services.

The Garden Hill First Nation Community is located 350 km northeast of Winnipeg and the Red Sucker Lake First Nation Community 80 km to the north-east of Garden Hill. Members of these two communities are peoples of the Cree/Ojibwa

aboriginal culture who share language and culture with other Cree/Ojibwa communities in the Island Lake area and in northwestern Ontario. Both communities are very isolated having road access for only two months of the year (during the winter). Access at other times is by aeroplane.

Medical Services Branch of Health Canada operates a nursing station on both communities. Garden Hill nursing station is staffed by 5 full-time nurse practitioners, 1 full-time public health nurse, 1 "fly-in" primary-care physician (3 days per week) and 3 community health representatives, one of whom has had some training in diabetic dietary instruction. Red Sucker Lake nursing station is staffed by two full time nurse practitioners and one community health representative who has no training in diabetic dietary instruction. A physician visits for 1 day per week. The author (D.Q.) was the physician visiting these two communities from October 1992 until July 1994.

There has been no broad community based education in relation to NIDDM at either Garden Hill or Red Sucker Lake. Any knowledge of the disease and treatment modalities gained by each patient is a result of interactions with the nurses, doctors or community health representatives at the nursing station or from attendance at one of several diabetes education centers in Winnipeg.

3.3 STUDY DESIGN

The study design involves a cross-sectional survey of diabetics from the Garden Hill and Red Sucker Lake communities with data being mainly collected by chart review.

3.3.1 Data Collection

With the permission of the Medical Services Branch, Health Canada, a survey of medical records was undertaken by D.Q. in May, June and July of 1994. Information was entered onto the data entry form (**ATTACHMENT B**). If any information was found to be missing from the chart, the patient was telephoned and asked for the required information or asked to attend at the nursing station for examination or testing.

The chronic disease registries maintained by the nursing stations in the two communities were used to identify the persons with diabetes. It was assumed that all patients on these registries suffered from non-insulin dependent diabetes (NIDDM) as during the period in which DQ worked at these communities, no patients had presented with diabetic keto-acidosis, the hallmark of insulin dependent diabetes mellitus (IDDM).

To be included in the study, persons listed on the chronic disease registries were required to fulfil one of the diagnostic criteria of the Expert Committee of the Canadian

Diabetes Advisory Board (as modified below)[1] which are:

(1) **F.B.S. (overnight)** - plasma glucose > 7.8 mmol/L on at least **two** separate occasions

OR

(2) **A random plasma glucose concentration** (RBS) > 11.1 mmol/L in a subject with the common symptoms of diabetes (thirst, polyuria, polydipsia, weight loss, blurred vision

OR

(3) **Following a 75 gram glucose load** in a fasting subject,plasma glucose > 11.1 mmol/L at 2 hr.

- To ensure that all diabetics were included in the study, results of tests taken on capillary blood were accepted. It is possible that this may have allowed a small number of non-diabetics to enter the study.

- While one of the above criteria (measured by venous or capillary blood) was required for acceptance into the study, the first measurement of FBS > 7.8 mmol/l, or RBS > 11.1 mmol/l when associated with symptoms, was accepted as the date of diagnosis of NIDDM.

Only diabetics registered with the Garden Hill or Red Sucker Lake Bands were included in the prevalence calculations. All diabetics were included in control and complication

calculations.

3.3.2 Variables

Data on the following variables was gathered:

1. Date of birth.
2. Gender.
3. Treaty status.
4. Location of abode.
5. Year and method of diagnosis and weight at diagnosis.
6. Current weight, height, body mass index (BMI) and waist/hip ratio (WHR).
7. Smoking history.
8. History of drug and non-drug treatment of NIDDM and parameters of glycemic control and weight change for each type of treatment.
9. Peripheral vascular disease.
10. Nephropathy.
11. Retinopathy.
12. Peripheral neuropathy.
13. Ischemic heart disease and cerebro-vascular disease.
14. Hypertension and the methods and success of blood pressure control.

3.3.3 Types of treatment and "Intervals of Therapy"

Five main types of treatment of NIDDM were identified. These are:

1. Treatment without drug therapy (In the literature this group is often referred to as the "diet only" group. Given that there has been very little dietary instruction at GH and RSL, in this study this group is referred to as the "no-drug" group).
2. Treatment with drugs of the sulphonylurea class.
3. Treatment with metformin.
4. Treatment with insulin.
5. Treatment with combined oral hypoglycemic medications (sulphonylurea and metformin).

To measure the level of glycemic control achieved by any particular type of treatment, periods of four or more months of continuous therapy with that type of treatment were considered to be representative of the overall level of glycemic control achieved by that type of treatment. Four months is sufficient time to allow the glycated hemoglobin level to be measured meaningfully (see glycemic control below). These four month periods were defined as "intervals of therapy" (IOTs) and the glycemic control for each was determined.

Many diabetics had been treated with more than one type of treatment (at different times) and thus there are more "intervals of therapy" recorded than diabetics enrolled in the study.

3.3.4 Glycemic control

Data available on the level of glycemic control of each "interval of therapy" varied. For some "intervals of therapy" glycated hemoglobin was available, for others fasting blood sugar level was available, and for others both measurements were available.

The glycated hemoglobin test (also known as HBA_1C) measures the amount of glucose attached to hemoglobin molecules. This measurement accurately reflects the average blood glucose level over the preceding six weeks with lower levels of HBA_1C representing lower average blood glucose levels (and better control) and vice versa.[102]

Any one glycated hemoglobin level measured at least eight weeks from the beginning of an "interval of therapy" was accepted as representative of the glycated haemoglobin level for that "interval of therapy". If more than one glycated hemoglobin level was recorded, the mean of all glycated haemoglobin levels was taken. For fasting blood sugar levels to be accepted as representative of the level of glycemic

control during an "interval of therapy", two or more values must have been obtained. In these cases, the mean of all values recorded during that "interval of therapy" was recorded.

As different data (HBA_1C or FBS) was available for different "intervals of therapy", a categorical ranking of glycemic control for each "interval of therapy" was determined according to a formula based on the recommendations of the Expert Committee of the Canadian Diabetes Advisory Board.[1]

Optimal control

Defined as HBA_1C of less than 110% of the normal range (< 7.7%)[1] or FBS < 6 mmol/l.

Acceptable control

Defined as HBA_1C of between 110% and 140% of the normal range (7.7 - 9.8%)[1] or FBS 6 - 10 mmol/l.

Compromised control

Defined as HBA_1C of greater than 140% of the normal range (> 9.8%)[1] or FBS > 10 mmol/l.

To determine the category of control, data was be used in a hierarchical manner. When available, glycated haemoglobin

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[1] Values for the Department of Biochemistry, Health Sciences Center, Winnipeg.

levels were used. If these were not available, fasting blood sugar levels were accepted.

3.3.5 Indices of obesity

It is known that people differ with respect to the location of fat deposition, and that the major complications of obesity, including cardiovascular disease, diabetes mellitus, hypertension, and hyperlipidemia, are associated with increased abdominal fat.[103]

Body Mass Index (BMI)

The Body Mass Index (also known as Quetelet's Index) is an anthropometric measure of body mass. Defined as body weight (kg) divided by height (meters) squared (weight/height^2), the BMI is accepted as being correlated with body fat and relatively unaffected by height. The "Canadian Guidelines for Healthy Weights"[104] classifies BMI as follows:

Less than 20	"Underweight"
20 - 24.9	"Healthy"
25 - 26.9	"Caution, potentially unhealthy"
27 - 34.9	"Obese"
35 or more	"Massively obese"

This classification is used to compare the study group with

Canadian averages, however the diabetes literature tends to use a different classification of BMI (healthy: BMI < 25 kg/m^2, overweight BMI 25-30 kg/m^2, obese: BMI > 30 kg/m^2) and this second classification is used for some comparisons.

Waist-Hip ratio (WHR)

The waist-hip ratio is a technique which estimates central fat from the ratio of the circumference of the of the waist or abdomen to that of the hips. The waist or abdominal circumference is the smallest circumference below the ribs and above the umbilicus, and the hips or gluteal circumference is taken as the largest circumference at the posterior extension of the buttocks. The accepted cut off figure for WHR is 0.9 for men and 0.8 for women.[103]

Data collection

Height and weight data had been collected as part of routine clinic visits and were recorded during the chart review, after which BMI and WHR were calculated. If current data was not available, the patient was asked to attend the nursing station to have the data collected.

3.3.6 Peripheral neuropathy/peripheral vascular disease/ulcers and amputation

As primary care physician to these two communities, DQ had examined most diabetic's feet and recorded the results in the

medical charts. The 10g monofilament was used to screen for peripheral neuropathy.[105] Testing was performed following the recommended technique (see **ATTACHMENT C)** touching all areas of the foot with the mono-filament with pressure to just bend the mono-filament. Palpation for the dorsalis pedis pulse was used to assess for peripheral vascular disease. During the chart review, information sensation and circulation, amputations and level, history of vascular surgery, the presence of ulcers (at the time of chart review), and attendance at specialist clinics was recorded. If relevant information was not in the medical record and the patient was known to be attending another doctor, that doctor was contacted and results recorded. Thus in some patients, other techniques (such as pin-prick) were used to test for peripheral neuropathy.

In this study, peripheral neuropathy was diagnosed if any part of the plantar or dorsal surfaces of either foot could not detect the 10g monofilament, or if another physician reported that the patient suffered peripheral neuropathy. Peripheral vascular disease was diagnosed if either dorsalis pedis pulse was absent to palpation, another physician reported that the patient suffered peripheral vascular disease, or if the patient had had vascular surgery to either leg.

3.3.7 Blood pressure

Patients were considered to have hypertension if they were or had ever taken antihypertensive medications or if the last two recorded diastolic blood pressure readings both exceeded 95 mmHg or the last two recorded systolic blood pressure readings recorded both exceeded 160 mmHg. The last blood pressure recorded and medications used for the control of hypertension were recorded.

3.3.8 Nephropathy

The presence and severity of nephropathy was be indicated either by the presence of any proteinuria as measured by "multistix" (Ames) on the last two urine-dip tests, a raised serum creatinine, the patient being on dialysis, or the patient having had a kidney transplant.

3.3.9 Retinopathy

Approximately 70% of the diabetics in this study had been evaluated by an ophthalmologist in the previous two years. Diabetic retinopathy deteriorates in a step-wise manner (background/pre-proliferative/proliferative - the complications of proliferative retinopathy including vitreous hemorrhage, retinal detachment and/or macular edema, any of which can cause blindness) and information on the presence and type of retinopathy was recorded. To ensure accuracy, data from the 30% of diabetics who had not been seen by an

ophthalmologist in the previous two years was excluded.

3.3.10 Ischemic heart disease and cerebro-vascular disease

These conditions were considered to be present if they were noted in the patients' charts. No further examinations or investigations were undertaken to verify these diagnoses.

3.3.11 Laboratory Methods

All blood samples were analyzed at the Department of Biochemistry, Health Sciences Center, Winnipeg. Serum and plasma glucose and creatinine levels were measured on an E 700 analyzer (Kodak) or a 717 analyzer (BMC). Glycated hemoglobin levels were measured using the Glyc-affin glycated hemoglobin test-kit (Isolab).

3.3.12 Statistical Analysis

Following the collection of the data, the entire data set was coded and entered into Epi Info, Version 5.[106] In this program, F-ratios are reported for two group tests, but the significance is reported as it is for the t-test. Bi-variate associations were evaluated either with the Chi-square, t-test, analysis of variance or simple linear regression, choice of the test depended on the measurement variables being compared. Associations between glycemic control, morphology, age, sex, type and duration of therapy used were tested.

3.4 ETHICS

The study was explained in detail to the Chiefs and Councils of the two bands and permission to undertake the study obtained in writing. A full explanation of the benefits of this study was given, including its contribution to the measurement of the burden of illness and the emphasis being given to the prevention of the complications of diabetes. The Chiefs and Councils were assured that the results of the study would be fully explained to them.

Permission was then sought, and received in writing, from Medical Services Branch - Health Canada, to extract the relevant information from the nursing station charts. The study proposal was approved by the Ethics Committee of the University of Manitoba.

The majority of the information required to conduct this study had been entered into the subjects' medical records as part of the researcher's function as primary care physician to these two communities. As data collection for this study mainly involved chart review, individual consent forms were not completed.

3.5 VALIDITY

3.5.1 Internal Validity

The use of standard published definitions and criteria for patient evaluation in this study is important to the internal validity and reproducibility of the results.

Possible sources of bias in this study include:

1) Selection bias

- It is possible that differential surveillance between the communities studied here and other communities may bias the prevalence data. The interest of the author in NIDDM increased the awareness of the staff and almost certainly increased case finding resulted. However the effect of this bias brings the prevalence closer to the true prevalence of NIDDM as it is known that for every diagnosed case of NIDDM there are usually two undiagnosed cases.

2) Information bias

- Diabetics who attended the nursing station regularly were more likely to have intervals of therapy included in the study than non-attenders. If attenders and non-attenders varied in respect to weight, metabolic control, etc., results may be biased.

- The method of determining glycemic control for each interval of therapy may be a source of bias. Any one glycated hemoglobin level (or average of all levels if more than one had been taken) or the average of two or more fasting plasma glucose levels were taken to indicate the glycemic level for the entire interval of therapy. As intervals of therapy varied in length (in particular, intervals using insulin or sulphonylurea drugs tended to be longer than those using metformin) and as these measurements tended to be more frequent in recent years, the study tends to measure the level of glycemic control towards the end of the longer intervals of therapy and may not be indicative of the level of control achieved earlier in those intervals.

- Compliance with therapy is another possible source of information bias. When extracting information from the charts, dispensing records were examined in an attempt to check that during any particular "interval of therapy", the appropriate medication was dispensed at appropriate intervals (this being suggestive of good compliance). The medical records had not always been maintained by trained staff and thus were difficult to follow at times.

- The date of diagnosis may be biased by the incomplete nature of the medical records. It is possible that some

diabetics had developed diabetes well before the first time the necessary criteria were recorded in the medical record.

3) Confounding

- It is possible that compliance with therapy may be a confounding factor in relation data on metabolic control. Three "intervals of therapy" were excluded from the study for obvious non-compliance with drug therapy. In these cases, the dispensing record clearly indicated that the patient could not have been taking the prescribed treatment during the "interval of therapy". It is likely however, that other "intervals of therapy" accepted into the study did not have good compliance either even though dispensing records appeared to be appropriate; it is possible that the degree of compliance with therapy directly effects the control data.

- It was noted that most patients who had been treated with insulin had infrequently had the dose of insulin changed. While this is probably an expression of the limited medical services available to these communities, it is possible that glycemic control may have improved if the insulin doses had been adjusted more frequently.

Because of the small sample size in this study, there is a

possibility that some actual between groups differences in relation to the control of NIDDM may not have been detected (B-error). The study's retrospective nature, and its reliance on data obtained from chart reviews, may also reduce its reliability. It is likely however, that the overall conclusions in relation to control of diabetes are correct.

3.5.2 External Validity

While selection bias may have increased the prevalence of NIDDM in Garden Hill and Red Sucker Lake in comparison to other communities, it is probable this is only a relatively small effect and that other isolated aboriginal communities would have a similar prevalence of NIDDM.

It is likely that the control of NIDDM found in this study is similar to the control found in other aboriginal communities as most aboriginal communities have similar socio-economic standards and medical services.

4. RESULTS

4.1 COMMUNITY SIZE AND PREVALENCE OF DIABETES

In July 1994 there were 2,746 Indians registered with the Garden Hill Band, 1,415 male and 1,331 female. The Red Sucker Lake Band had 566 registered Indians; 284 male and 282 female. The combined population of the two bands is 3,312; 1,700 male and 1,612 female.

A total of 153 persons fulfilling the Expert Committee criteria for diabetes were identified, 55 male and 98 female. One hundred and fifty of these, 54 male and 96 female, were members of either the Garden Hill or Red Sucker Lake Bands. Diabetics not registered with either of the bands were excluded from prevalence calculations but included in control and complication calculations. The crude prevalence of diabetes in Garden Hill and Red Sucker Lake is 45/1000.

Of the 119 members of the Garden Hill Band found to have diabetes; 107 lived in Garden Hill, 11 lived in Winnipeg and one at another community. A total of 110 diabetics were living in Garden Hill; 107 registered with the Garden Hill Band and 3 registered with other bands. Of the 31 diabetics living at Red Sucker Lake, 30 were registered with the Red Sucker Lake Band and one with another band. All diabetics registered with the Red Sucker Lake Band lived at Red Sucker Lake.

TABLE 1 shows the composition of the two bands by age-group and sex and the prevalence of diabetes in each age-group.

TABLE 1
AGE & SEX DISTRIBUTION OF NIDDM IN THE GARDEN HILL & RED SUCKER LAKE INDIAN BANDS.

	GARDEN HILL						RED SUCKER LAKE					
	MALE			FEMALE			MALE			FEMALE		
AGE GROUP	N	DM	PREV	N	DM	PREV	N	DM	PREV	N	DM	PREV
<15	543	0	0	501	1	0.2	84	0	0	83	0	0
15-24	309	0	0	262	3	11.4	87	1	11.4	76	0	0
25-34	248	6	24	229	7	30	48	0	0	49	4	82
35-44	140	7	50	127	8	63	26	1	39	29	6	207
45-54	92	14	152	99	20	202	20	0	0	25	7	280
55-64	45	17	377	61	22	361	6	2	333	9	3	333
65+	38	5	132	52	9	173	14	1	71	10	5	500
TOTAL	1415	49		1331	70		285	5		281	25	

* Prevalence / 1000

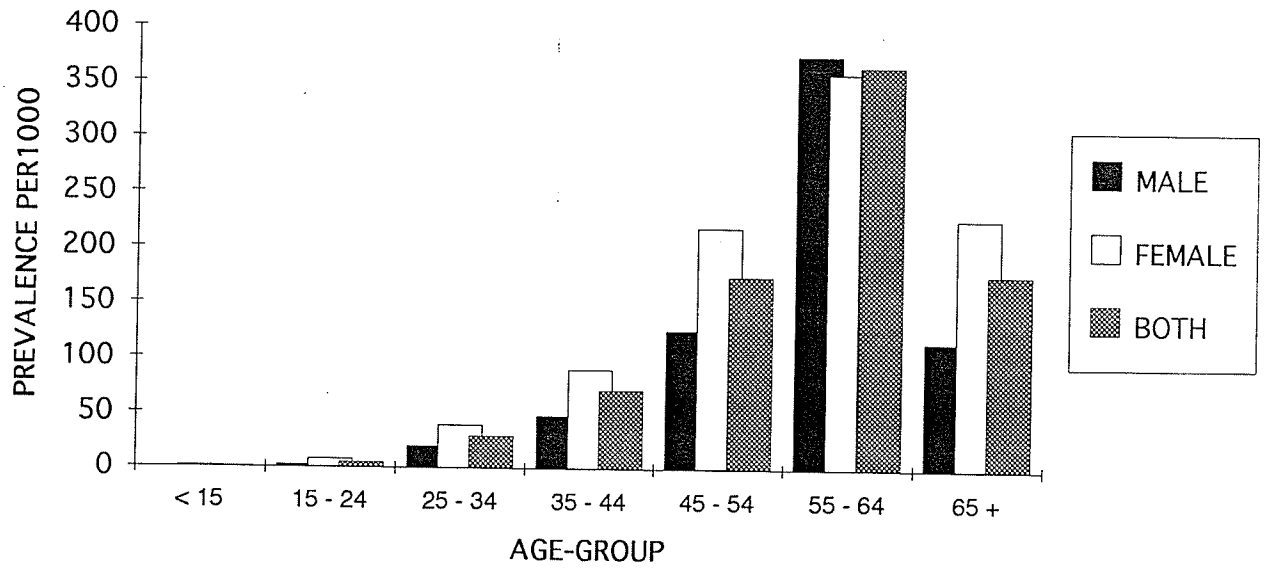
TABLE 2 and its graphical representation **FIGURE 1** show the prevalence of NIDDM in the combined population of Garden Hill and Red Sucker Lake Indian Bands. In both men and women, the prevalence of NIDDM increases steadily with age until the 55-64 age-group, after which it declines. Women have a higher

TABLE 2
PREVALENCE OF NIDDM ACCORDING TO AGE-GROUP

AGE GROUP	MALE			FEMALE			COMBINED		
	N	DM	PREV*	N	DM	PREV*	N	DM	PREV*
<15	627	0	0	584	1	1.7	1211	1	0.8
15-24	396	1	2.5	338	3	8	734	4	5.3
25-34	296	6	20.3	278	11	39.5	574	17	29.6
35-44	166	8	48	156	14	90	322	23	71
45-54	112	14	125	124	27	218	236	41	174
55-64	51	19	373	70	25	357	121	44	363
65+	52	6	115	62	14	226	114	20	176
TOTAL	1700	54	31.8	1612	95	58.9	3312	149	45

* Prevalence / 1000

FIGURE 1 PREVALENCE OF NIDDM ACCORDING TO AGE-GROUP



prevalence in all age-groups except 55-64. Men in that age-group have the highest prevalence of any age-group (373/1000), while women in the 55-64 age-group have a prevalence of 357/1000. The overall female/male ratio 1.78:1. Due to the different age structure between the native and non-native populations of Canada, the former being much younger, age-standardization by the "direct" method was performed, both for the whole study population and for those aged 15 years and over. The standard population used was the Canadian national population based on the 1991 census. The age adjusted prevalence for the study population is 89/1000 **(TABLE 3)** and for those 15 years and over it is 109/1000.

TABLE 3
CALCULATION OF AGE-ADJUSTED PREVALENCE OF NIDDM AT GH AND RSL.

AGE-GROUP	PREVALENCE IN GH & RSL *	MULT. FACTOR	SIZE OF STD POPULATION	EXPECTED NO. OF CASES
< 15	0.8	0.0008	5,695,743	4,556
15 - 24	8.1	0.0081	3,833,154	31,048
25 - 34	31.3	0.0313	4,831,934	151,239
35 - 54	115	0.115	7,315,386	841,269
55 - 64	363	0.363	2,375,475	862,297
65 +	176	0.176	2,942,350	517,856
* per 1000			26,994,045	2,408,262

$$\frac{\text{EXPECTED CASES}}{\text{STD POPULATION}} = \frac{2,408,262}{26,994,045} = 89.2/1000$$

4.2 AGE AT DIAGNOSIS / YEARS SINCE DIAGNOSIS

TABLE 4 gives the details of the mean age of diagnosis of NIDDM and the mean number of years since diagnosis for males and females.

TABLE 4
AGE AT DIAGNOSIS AND NUMBER OF YEARS SINCE DIAGNOSIS

	MALE DIABETICS	FEMALE DIABETICS
MEAN AGE AT DIAGNOSIS	44 years (SD:13)	43 years (SD:13)
MEAN NUMBER OF YEARS SINCE DIAGNOSIS	6.7 years (SD:6.2)	7.5 years (SD:5.3)

4.3 SMOKING

TABLE 5 gives details of tobacco smoking among diabetics. Data was collected on 51 (93%) of the male diabetics and 90 (92%) of the female diabetics.

TABLE 5
TOBACCO SMOKING AMONG DIABETICS

	MALE DIABETICS n = 51	FEMALE DIABETICS n = 90
CURRENT SMOKERS	21 (41%)	26 (29%)
NEVER SMOKED	10 (20%)	42 (47%)
EX-SMOKERS	20 (39%)	22 (24%)

Overall 31% of Canadian men and 28% of Canadian women smoke [107].

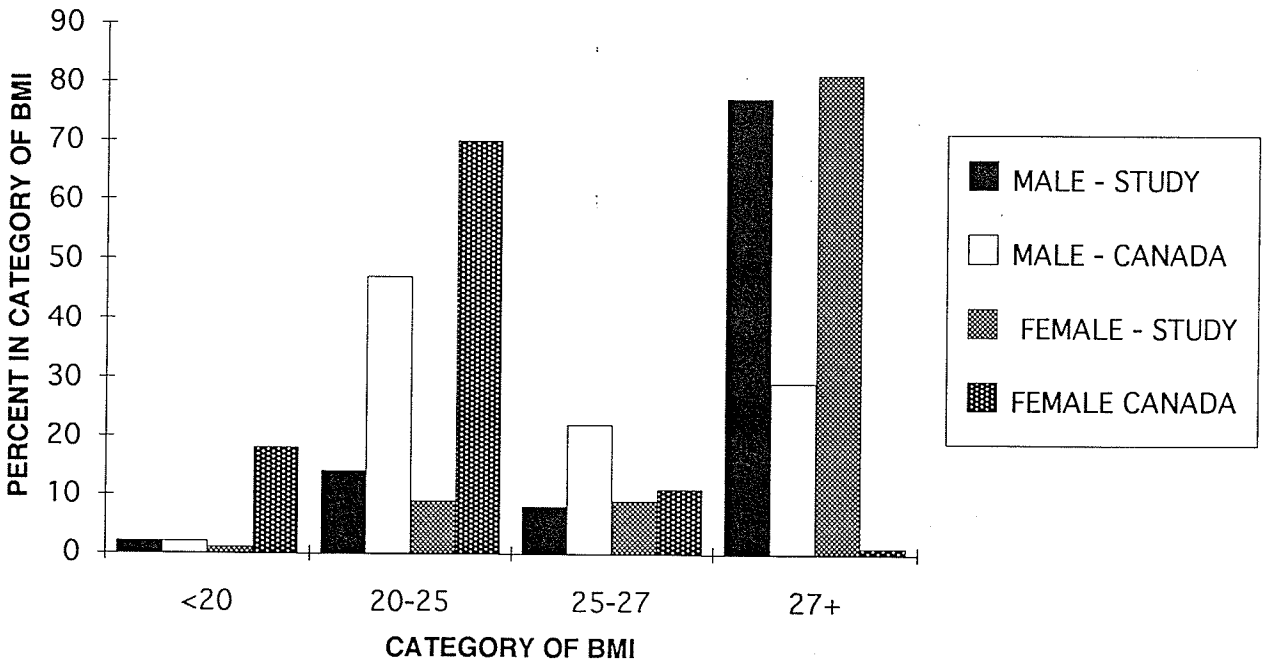
4.4 INDICES OF OBESITY

It was not possible to calculate the indices of obesity in all of the diabetics as: 1) not all diabetics attend the nursing station, 2) some diabetics live off-reserve, and 3) of those diabetics whose height and weight measurements were not recorded in the chart, several did not respond to requests that they attend for measurement.

4.4.1 Body Mass Index (BMI)

Fifty-one of the 55 male diabetics (92%) and 90 of the 98 female diabetics (91%) had their BMI measured. **FIGURE 2** shows

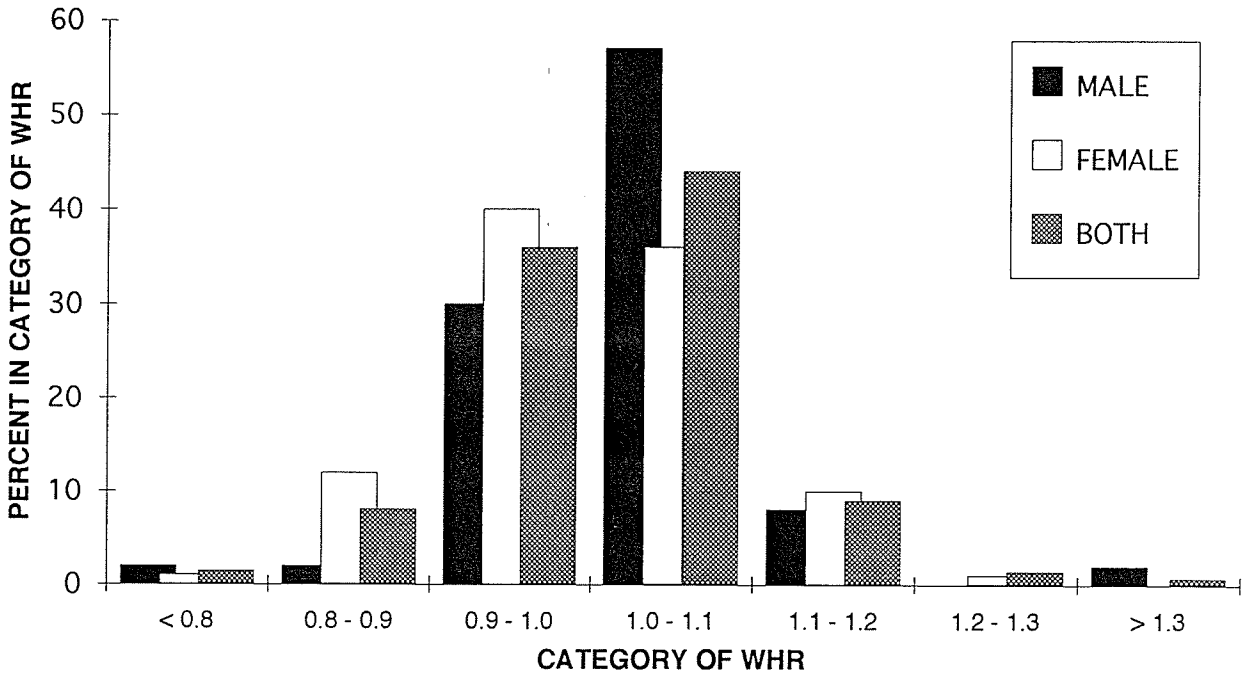
FIGURE 2 DISTRIBUTION OF BMI IN STUDY COMPARED TO CANADIAN AVERAGES



the distribution of BMI (of those measured) by gender and compares it to the Canadian average, taken from Canada's Health Promotion Survey[107] which combines the 27-35 kg/m^2 and 35+ kg/m^2 groups.

Of the male diabetics, 55% were obese (BMI 27-35 kg/m^2), and 22% were massively obese (BMI > 35 kg/m^2). The corresponding figures in female diabetics are 53% and 28%. The diabetics identified in this study have much higher BMIs than the Canadian average. No figures were available to compare this population with a non-diabetic Canadian aboriginal group.

FIGURE 3 DISTRIBUTION OF WHR AMONG DIABETIC SUBJECTS



4.4.2 Waist-Hip Ratio (WHR)

Fifty-one of the 55 male diabetics (92%) and 87 of the 98 female diabetics (88%) had their WHR measured. **FIGURE 3** shows the age/sex distribution of WHR on those measured. Forty-nine (96.1%) of the males had a WHR greater than 0.9 and 86 (98.9%) of the female diabetics had a WHR greater than 0.8, the accepted cut-off points. Thirty-five (68%) of the males and 42 (48.2%) of the females had a WHR greater than 1.

4.5 CONTROL OF DIABETES

A total of 190 four or more month "intervals of treatment" were drawn from the charts of 133 diabetics. **TABLE 6** shows the number of "intervals of treatment" and their mean duration for each type of treatment.

TABLE 6
NUMBER AND DURATION OF INTERVALS OF THERAPY

	NUMBER OF INTERVALS OF THERAPY USING TREATMENT TYPE			DURATION OF INTERVALS OF THERAPY (MONTHS)	
TREATMENT TYPE	MALE	FEMALE	BOTH	MEAN	SD
NO DRUGS	34	55	89	31	34
SULPHONYLUREA	11	32	43	54	45
METFORMIN	5	19	24	11	5
INSULIN	10	20	30	117	64
SULPHONYLUREA & METFORMIN	0	4	4	13	6
TOTAL	60	130	190		

4.5.1 Categories of control

One hundred and eighty two of the "intervals of therapy" (95.7%) used HBA_1C measurements to calculate the category of control. The other eight "intervals of therapy" used mean fasting blood sugar level to determine the category of control.

TABLE 7 shows the proportion of "intervals of therapy" in each category of control.

TABLE 7
PROPORTION OF INTERVALS OF THERAPY IN EACH CATEGORY OF CONTROL

	OPTIMAL HBA1C < 7.7%		ACCEPTABLE HBA1C 7.7-9.8%		COMPROMISED HBA1C > 9.8%	
TREATMENT TYPE	N	%	N	%	N	%
NO DRUGS	9	10%	11	12%	69	78%
SULPHONYLUREA	4	9%	9	21%	30	70%
METFORMIN	2	8%	3	13%	19	79%
INSULIN	1	3%	3	10%	26	87%
SULPHONYYLUREA & METFORMIN	0	-	2	50%	2	50%
TOTAL	16	8%	28	15%	146	77%

One hundred and forty-six of the total of 190 "intervals of therapy" (76.8%) showed "compromised" control (HBA_1C > 9.8%), 28 (14.7%) showed "acceptable control" (HBA_1C 7.7 - 9.8%), and 16 (8.4%) showed "optimal" control (HBA_1C < 7.7%). Except for "combined oral therapy", 70% or more of the "intervals of therapy" using each treatment type had "compromised control" (HBA_1C > 9.8%). Two of the four "intervals of therapy" using "combined oral therapy" had "acceptable control" (HBA_1C 7.7% - 9.8%), two had "compromised control" (HBA_1C > 9.8%).

4.5.2 Means of HBA_1C

TABLE 8 shows the mean HBA_1C of "intervals of therapy" stratified by type of treatment and BMI.

TABLE 8
STRATIFICATION OF MEAN HBA1C BY BMI AND TREATMENT TYPE

	BMI < 25			BMI 25-30			BMI > 30			ALL WEIGHTS		
TREATMENT TYPE	N	MEAN HBA1C	SD	N	MEAN HBA1C	SD	N	MEAN HBA1C	SD	N	MEAN HBA1C	SD
NO DRUGS	13	15.1	5.4	20	14.1	4.4	52	13.2	4.1	85	13.7	4.4
SULPHONYLUREA	5	10.7	4.7	12	12.6	3.1	22	12.9	3.5	40	12.5	3.5
METFORMIN	2	17.8	5.0	10	16.5	2.6	10	12.2	4.2	23	14.3	4.4
INSULIN	5	11.6	2.9	10	15.8	1.7	11	14.8	3.9	29	14.3	3.5
SULPHONYLUREA & METFORMIN	0	-	-	0	-	-	4	11.8	3.2	4	11.8	3.2

All sub-groups had a mean HBA_1C of above 9.8% and thus all would be classified as having "compromised control" and the overall mean HBA_1C was 13.6% In all categories except BMI > 30 kg/m^2 (i.e. BMI < 25, BMI 25-30, and the "all weights" category), drugs of the sulphonylurea class gave the lowest mean HBA_1C. When used in the BMI < 25 kg/m^2 category, sulphonylurea drugs gave the lowest mean HBA_1C of any category, 10.7%. In the category BMI > 30 kg/m^2, "no-drug therapy", sulphonylureas, and metformin afforded similar metabolic control (HBA_1Cs of 13.2%, 12.9%, and 12.2% respectively). In no category did insulin therapy give a lower mean HBA_1C than oral hypoglycemic medications. Excluding the effects of drugs, the level of control was not related to the BMI.

As only four "intervals of therapy" used "combined oral therapy" (a sulphonylurea and metformin), the effectiveness of this modality cannot be determined.

4.5.3 Changes of Treatment Resulting in Altered Category of Control

From the 190 "intervals of therapy" identified, 46 sequential pairs of "intervals of therapy" document the effect on control resulting from a change of type of treatment. Of these 46 changes in treatment, six resulted in an improvement of category of control, seven resulted in a worsening of the

category of control, and the remaining 33 changes of treatment did not alter the category of control. **TABLE 9** documents the changes of treatment which resulted in a change of category of control.

TABLE 9
CHANGES OF TREATMENT RESULTING IN CHANGE OF CATEGORY OF CONTROL

	NUMBER	CHANGE OF TREATMENT	
		FROM	TO
CONTROL IMPROVED	6	NO DRUG SULPH NO DRUG NO DRUG NO DRUG NO DRUG	to METFORMIN to METFORMIN to SULPH to METFORMIN to METFORMIN to SULPH
CONTROL WORSENED	7	SULPH SULPH SULPH SULPH INSULIN SULPH SULPH	to NO DRUG to METFORMIN to METFORMIN to METFORMIN to NO DRUG to NO DRUG to NO DRUG

Five of the six changes of treatment which improved the category of control involved starting oral medication in patients previously not taking medication. In six of the

seven changes of treatment resulting in a worsening of category of control, sulphonylurea treatment was changed to "no-drug" or metformin treatment. While this sample is small, these results suggest that treatment with oral hypoglycemics may provide better control than "no-drug" treatment, and that changing from sulphonylurea treatment to "no-drug" treatment or treatment with metformin may worsen control.

Altered HBA_1C

Smaller changes in metabolic control (most insufficient to change category of control) resulting from changes of treatment can be measured by assessing the change in HBA_1C. Twenty changes of treatment resulted in an improvement of the HBA_1C (mean change of HBA_1C -3.85%) and 23 changes resulted in a worsening of control (mean change of HBA_1C +3.18%). The 46 changes of type of treatment resulted in a minimal overall mean change of HBA_1C (-0.09%: range -13% to +7.4%).

Of the 20 changes of treatment which resulted in a decrease of HBA_1C (improved control):

- 12 involved a change from "no-drug" treatment to a type of hypoglycemic medication (7 to sulphonylurea, 4 to metformin, 1 to sulfonylurea and metformin)
- 4 involved a change from sulphonylurea (2 to metformin, 1 to "no-drug", 1 to insulin)
- 1 change was from metformin to "no drug" treatment

- 1 change was from sulphonylurea and metformin treatment to sulphonylurea treatment alone.

Thus the majority of changes of treatment which improved the metabolic control involved the starting of oral hypoglycemic treatment in diabetics previously on "no-drug" treatment.

Of the 23 changes of treatment which resulted in an increase of HBA_1C (worse control):

- 12 involved the cessation of sulphonylurea treatment (5 to "no-drug" and 7 to metformin)
- 1 involved changing from "no-drug" treatment to sulphonylurea
- 2 involved changing form metformin therapy to "no-drug" therapy
- 8 involved changing from insulin therapy (4 to "no-drug", 4 to metformin).

Thus the majority of the changes of treatment which worsened metabolic control involved the cessation of sulphonylurea or insulin treatment.

These results suggest that the use of hypoglycemic medication is associated with better metabolic control than "no-drug" treatment.

4.5.4 Comparison of diabetics subjects with "acceptable" and "compromised" control.

To determine if any attributes of diabetic subjects were associated with better control, two groups of diabetics were compared - those whose metabolic control was "acceptable" or better ($HBA_1C \leq 9.8\%$), and those whose metabolic control was "compromised" ($HBA_1C > 9.8\%$)[2]. See **TABLE 10**.

TABLE 10
COMPARISON OF DIABETICS WITH ACCEPTABLE AND COMPROMISED CONTROL

	HBA1C ≤ 9.8 %		HBA1C > 9.8%		P VALUE
NUMBER	42		140		
SEX	MALE	FEMALE	MALE	FEMALE	
PERCENT	23%	77%	34%	66%	0.28 #
MEAN AGE	54		50		0.049 *
MEAN YEARS SINCE DIAGNOSIS	6.3		8.0		0.076 *
MEAN BMI	31		31		0.77 *

* t test
\# Chi square test

[2] "Acceptable control" and "Compromised control" are as defined by The Expert Committee of the Canadian Diabetic Association.

The group of diabetics with worse metabolic control (HBA_1C > 9.8%) was significantly younger than those with better control ($HBA_1C \leq 9.8\%$). $p = 0.049$ This may indicate that younger diabetics have worse metabolic control than older diabetics. No statistical difference was found in the mean number of years since diagnosis or the mean BMI of the diabetics in the two categories of control. The Chi-square test found no significant difference in the proportion of men and women in the two groups of control.

4.5.5 Weight Change with each Treatment Type

TABLE 11 shows the mean weight change for IOTs of each treatment type.

TABLE 11
WEIGHT CHANGE WITH EACH TYPE OF TREATMENT

TREATMENT TYPE	MEAN WEIGHT CHANGE N	CHANGE (KG)	SD
NO DRUGS	74	- 3.0	6.8
SULPHONYLUREA	34	- 0.17	8.0
METFORMIN	24	- 5.0	4.8
INSULIN	25	- 4.1	7.3
SULPHONYLUREA & METFORMIN	3	+ 9.0	11.9

"Intervals of therapy" using a sulphonylurea class of drug were associated with very little mean weight change [mean -0.17 kg (SD:8.0 kg)]. Combined oral treatment was associated with a large mean weight gain [mean 9.0 kg (SD:11.9 KG)]. This result may be biased as there were only three eligible "intervals of therapy", one of which was associated with an unusually large weight gain of over 20 kg. The other treatment types (no-drug, metformin, and insulin) were associated with substantial weight loss, the maximum being metformin [mean -5.0 kg (SD:4.8 KG)].

4.6 COMPLICATIONS

4.6.1 Retinopathy

One hundred and seven of the 153 identified diabetics (70%) had had an ophthalmological examination in the previous two years. **TABLE 12** shows the prevalence of each category of

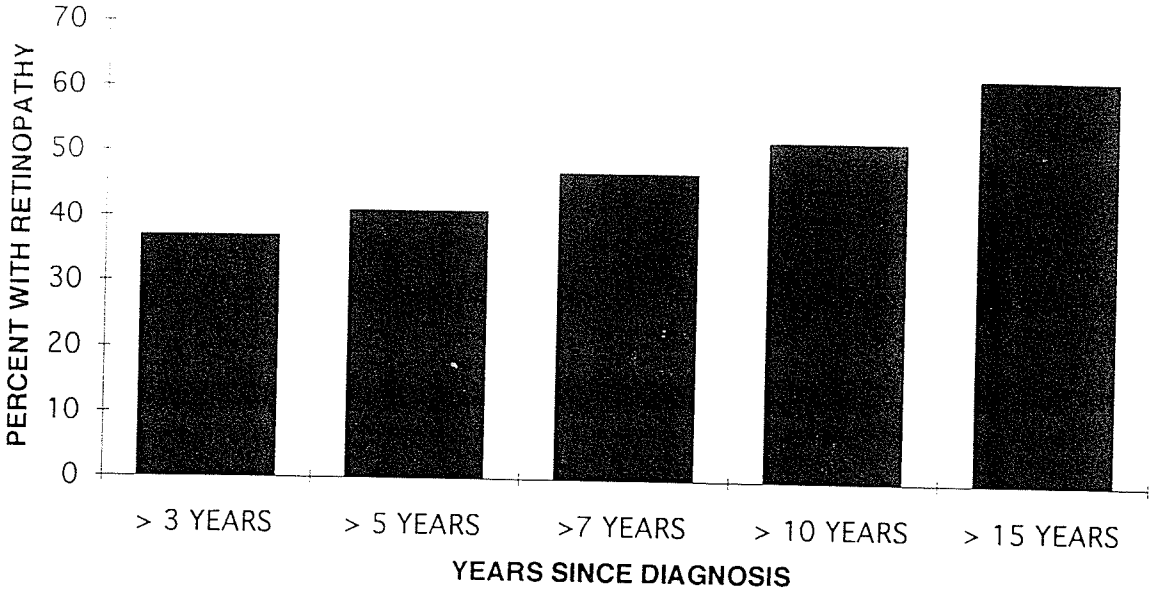
TABLE 12
RETINOPATHY

TYPE OF RETINOPATHY	N	%	YEARS SINCE DIAGNOSIS		AGE		LASER THERAPY
			MEAN	SD	MEAN	SD	
NIL	75	70%	7	5	49	13	0
BACKGROUND	27	25%	12	5	59	10	5
PROLIFERATIVE	2	2%	12	4	58	2	0
HEMORRHAGE	1	1%	20	-	59	-	1
DETACHMENT	1	1%	5	-	45	-	0
BLIND	1	1%	17	-	57	-	0
TOTAL	107						

retinopathy, the mean number of years since the diagnosis of diabetes, the mean age, and the number of diabetics treated with photo-coagulation. Of those examined, 32 (30%) had a form of retinopathy; 27 (25.2%) being background retinopathy, two (1.9%) being proliferative retinopathy, one (0.93%) had had a vitreous hemorrhage, one (0.93%) had had a retinal detachment and one (0.93%) was blind from macula scars associated with macula edema. The average number of years since diagnosis was 6.9 years (SD:5.1 years) for the 75 diabetics without retinopathy and 12.2 years (SD:4.9 years) for the 27 diabetics with background retinopathy. The difference was significant. $p = 0.00002$. Of the six patients with retinopathy who had laser treatment, five had background retinopathy and one had a vitreous hemorrhage.

FIGURE 4 shows the prevalence of any type of retinopathy stratified by number of years since the diagnosis of diabetes. Of the 81 diabetics who were diagnosed more than three years previous, 30 (37%) had retinopathy. Twenty-eight of the 67 diabetics (41.8%) diagnosed more than five years previous had retinopathy, and by ten years from diagnosis eight of the 13 (61.5%) had retinopathy.

FIGURE 4 PREVALENCE OF RETINOPATHY ACCORDING TO NUMBER OF YEARS SINCE DIAGNOSIS WITH NIDDM



4.6.2 Lower limb neuropathy, peripheral vascular disease, ulcers and amputations.

One hundred and thirty four of the 153 diabetics (86.7%) had had their lower limbs examined within the last two years. The results of these examinations and the mean number of years since diagnosis for groups with and without each complication are given in TABLE 13.

TABLE 13
PREVALENCE AND NUMBER OF YEARS SINCE DIAGNOSIS FOR LOWER LIMB COMPLICATIONS.

COMPLICATION	NEUROPATHY			PVD			ULCER			AMPUTATION		
	N	%	MEAN YEARS	N	%	MEAN YEARS	N	%	MEAN YEARS	N	%	MEAN YEARS
ABSENT	111	81	7	111	81	7.2	131	96.3	7	132	97	7.0
PRESENT	25	19	8.5	25	19	7.6	5	3.7	13.2	4	3	18
P VALUE			NS			NS			0.0157			0.0024

Neuropathy

Of the 134 diabetics who had had their feet tested, 129 (96.2%) were tested for peripheral neuropathy using the 10g monofilament standard stimulus. Other methods (pin/vibration) were used for the other five. Twenty-five of those tested (18.7%) had peripheral neuropathy. There was no significant difference in the mean number of years since diagnosis between the groups who did and did not have neuropathy. $p = 0.242$.

Peripheral vascular disease

Twenty-five of the 136 examined (18.7%) had had at least one dorsalis pedis pulse absent and were thus diagnosed to have peripheral vascular disease. There was no significant

difference in the mean number of years since diagnosis between the groups who did and did not have peripheral vascular disease. p = 0.73.

Lower limb ulcers

Five of the 136 examined (3.7%) currently had a lower limb ulcer; the mean number of years since diagnosis of this group was 13.2 years (SD:8.3 years) significantly more than the group who did not have ulcers, mean 7.1 years (SD:5.5 years). p = 0.016.

Amputations

Four of the 136 examined (2.9%) had had amputations; one had a below knee amputation on one side and a mid-tarsal amputation on the other side, and three diabetics had had toes amputated. The mean number of years since diagnosis was 18.0 years (SD:3.7 years) significantly more than the group without amputations, mean 7.0 years (SD:5.5 years). p = 0.002.

4.6.3 Ischemic Heart Disease

Records on the presence of ischemic heart disease were available for 148 (96.7%) of the 153 identified diabetics. Of these, seven (4.7%) had angina and three (2.0%) had had myocardial infarctions. There was no significant difference in the mean number of years since diagnosis for these groups. p = 0.89.

4.6.4 Cerebro-vascular disease

Records were available for 149 diabetics (91.9%). Six diabetics (4.0%) had had transient ischemic attacks (TIAs) and six (4.0%) had had a stroke. Again there was no significant difference in the mean number of years since diagnosis for these groups. $p = 0.51$.

4.6.5 Hypertension

Records of blood pressure were available for 146 of the diabetics (96.7%). Of these, 70 (47.9%) had hypertension. The most common treatment modalities were an angiotensin-converting-enzyme-inhibitor (ACE) (57.9%), and an ACE in combination with a calcium antagonist (23.7%). Of the 70 with hypertension, 37 (49.3%) had hypertension controlled sufficiently to have a diastolic blood pressure of 90 mmHg or less.

4.6.6 Nephropathy

One hundred and thirty two of the diabetics (86.3%) had had their urine tested for protein (Multistix: Ames) twice in the last two years. Forty five (34.1%) showed proteinuria on both tests. Nine of these (6.8%) showed 3.0 grams per liter protein or more and five (3.7%) showed 20 grams per liter or more.

One hundred and forty five diabetics (94.7%) had had their

serum creatinine level measured in the last two years. Eighteen of these (12.4%) had a creatinine above the normal range (< 110 mmol/l). One diabetic was receiving peritoneal dialysis and two had had renal transplants. One of the diabetics who had had a transplant was diagnosed as having glomerulonephritis, the other had diabetic nephrosclerosis.

5. DISCUSSION

5.1 PREVALENCE

The age-adjusted prevalence of NIDDM in the two communities studied (89/1000) is the highest reported in a native community in Canada; the highest prevalence previously reported in a native community being 87/1000 in the Atlantic region.[14] While the standard populations used to calculate the age-adjusted prevalence rates differ (in this study data from the 1991 Canadian census was used, in Young's study, data from the 1985 census was used), it is unlikely that the age-distribution of the Canadian population has changed sufficiently in the intervening five years to significantly effect the results. The age-adjusted prevalence documented in these communities is the same as the average age-adjusted prevalence in Indian populations in the United States (89/1000 -- prevalence ranges from 20/1000 to 154/1000[108]).

The crude prevalence of diabetes has apparently increased from 28/1000 (reported by Young et al. in a prevalence survey conducted in the same area in 1985[7] to a current value of 45/1000. Harris et al[18] have recently reported a similar increase in the prevalence of NIDDM among native communities in the Sioux Lookout Zone of Southwestern Ontario. Some of these increases may be explained by increased awareness of and testing for NIDDM among health professionals, however it is likely that this bias only partially explains the increase in

prevalence as a true increase in prevalence is seen among aboriginal people world-wide. While the highest prevalence is found in the 55-64 age-group, the prevalence is much less in both men and women in the 65+ age-group. This may be because this age-group contains a cohort whose lives precede the current epidemic of NIDDM; an alternative explanation is that selective mortality (i.e., higher death rate among diabetics) has reduced the prevalence of NIDDM in this group.

It is generally agreed that diabetes is associated with obesity, especially when there is a centralized distribution of fat. The diabetics in the communities studied here are, on average obese, and are much heavier than the Canadian average. Using the "Guidelines for Healthy Weights" classification[104] 54% of diabetics are obese (BMI 27-35 kg/m^2) and 26% are massively obese (BMI > 35 kg/m^2). Ninety seven percent of the diabetics had a waist/hip ratio (WHR) exceeding accepted healthy cut-off levels[103] confirming centrality of fat distribution.

5.2 CONTROL OF DIABETES

Very little has been written about the level of control of NIDDM among Canadian Indian populations. Brassard et al[17] described that 76.4% of the 230 diabetics in the James Bay Cree population had poor control of their diabetes (fasting plasma glucose > 7.8 mmol/l). Fox et al[19] recently reported

"unacceptable control" (FBS > 7.8 mmol/l) in 62.4% of diabetics in native communities in Sioux Lookout Zone.

The control of the diabetes in the Island Lake communities studied is poor. Using the Canadian Diabetic Association Expert Committee criteria[1] 77% of the 190 intervals of therapy (IOTs) measured had "compromised" control (HBA_1C > 9.8%), 14.7% had "acceptable" control (HBA_1C 7.8% - 9.8%), and only 8.4% had "optimal" control (HBA_1C < 7.7 mmol/l). The overall mean HBA_1C was 13.6% (SD:4.1%). Of interest was the finding that the control of diabetics with lower BMIs was, in general, no better than those with higher BMIs.

The control of diabetes in the two communities studied is on average, much worse than that reported in the United Kingdom Prospective Diabetes Study (UKPDS).[82] The UKPDS reported mean HBA_1Cs ranging from 6.8% to 7.6%, whereas this study found mean HBA_1Cs ranging from 12.5% to 14.3%. One possible explanation for this difference is that in the UKPDS, all enrolles received regular dietary education. In contrast, the majority of the diabetics studied here have had very little dietary education, and what education they have received was, in general, not been given by professionally trained staff nor was a curriculum designed for cross-cultural learning used. It is also possible that the population studied here is, on average, more obese than that enrolled in the UKPDS and this

has effected the control of the diabetes. In the UKPDS subjects were classified and randomized as obese if they were more than 20% above ideal weight. Taken together, these two possible explanations for the overall worse control of NIDDM found in these communities (than in the UKPDS) suggest that if there are no other factors involved (i.e. a metabolic reason for the worse control) that the effective alteration of diet and lifestyle has the potential to improve the control of NIDDM, at least to the level of the UKPDS.

In this study, diabetics had lost an average of 4.8 kg since being diagnosed with NIDDM. Often weight loss is seen by health-care professionals as a sign of a positive alteration of lifestyle however the weight loss found in this study may be a result of the diabetes itself rather than lifestyle change. This study found differing weight change in "intervals of therapy" using different types of treatment. "Intervals of therapy" using sulphonylurea were associated with virtually no weight change (mean loss of 0.17 kg), while "intervals of therapy" using "no-drug", insulin, and metformin were associated with weight loss (minus 3 kg, minus 4.1 kg, and minus 5 kg respectively). This suggests that drugs of the sulphonylurea group, in contrast to other hypoglycemics, prevent weight loss in diabetics.

The finding that diabetes control is poor, generally regardless of treatment type, has significant implications for the clinical management of diabetes on isolated Indian communities. Currently, health care providers can spend a significant amount of time manipulating hypoglycemic medications, hoping that glycemic control will be improved. While sometimes this is done to treat symptoms, more often it is undertaken in asymptomatic diabetics hoping that a reduction of diabetic complications may result. There is, however, no evidence that, in populations such as the one studied here, use and/or manipulation of hypoglycemic drug therapy dramatically improves glycemic control and thus the use of hypoglycemic drugs is unlikely to reduce the incidence of complications.

This study indicates that when used as mono-therapy, drugs of the sulphonylurea group seem to be give slightly better control of diabetes than "no-drug", metformin, or insulin therapy (no statistical significance was shown), however the use of sulphonylureas is associated with less weight loss than use of other types of treatment. This finding is consistent with the UKPDS which reported that in non-obese diabetics, metabolic control was significantly better in those treated with drugs of the sulphonylurea class or insulin, when compared to those treated by diet therapy alone; mean body weight also increased in this group. In obese subjects

metformin was as effective as the other drugs with no change in mean body weight. The report concluded that all of these drugs had similar glucose lowering efficacy, although most patients remained hyperglycemic.

There is insufficient data to comment on the control afforded by "combined oral therapy" (a sulphonylurea and metformin) except to say that the four "intervals of therapy" in this study using this combination of medication had the lowest mean HBA_1C level of any of the treatment types. In trying to ascertain if there are any other combinations of medications that may be more effective in achieving metabolic control than single drug therapy, Giugliano et al[109] have reported that the use of insulin combined with metformin significantly improved the control of a group of patients with secondary failure to sulphonylurea therapy (decrease in HBA_1C of 1.84%). Trischitta et al[110] have reported that the addition of metformin significantly improved the metabolic control of groups of patients who had failed to be controlled by sulphonylureas alone.

5.3 COMPLICATIONS

The more severe complications of NIDDM occur 10-20 years after the onset of the disease. While there does not appear to be a high prevalence of severe complications in the study population at present, four factors indicate that a worrying increase in these complications may occur in future years. Firstly, as discussed above, the prevalence of diabetes is increasing. Secondly, NIDDM appears to be effecting younger members of the population. Forty-five (30%) of the diabetics identified were aged 44 years or less. Because of the increased number of years of exposure to the disease, it is probable that those who develop diabetes at a younger age will develop more complications. Thirdly, this study examined the prevalence, not the incidence of complications. The study did not account for the premature mortality that may be associated with conditions such as renal failure, cerebrovascular disease and amputations. It is likely therefore that the incidence of severe complications is considerably higher than what is indicated by the prevalence figures reported here. Fourthly, the mean time since diagnosis of diabetes is 7.6 years, and thus the cohort of current diabetics may be expected to develop complications over the next few years.

5.4 LIFESTYLE, OBESITY AND EXERCISE

The degree of obesity found in the diabetics in this study can be, I feel, validly interpreted as a proxy indicator of

lifestyle. While diet and exercise levels have not been directly measured, the obesity found indicates that the diabetics (and probably the wider community) have a lifestyle in which caloric intake vastly exceeds caloric expenditure. It is the author's opinion that the excess caloric intake mentioned above mainly results from an excessive intake of fatty and fried foods such as bannock (a type of bread made from flour, water, and lard and then spread with lard, eggs and bacon, hamburger, french fries, fried pork chops, fried chicken, potato chips, etc.). The author has noticed that in the communities studied, the consumption of fat and physical heaviness are associated with a sense of well-being. It is unfortunate that the only dietary survey recently undertaken in remote communities in Northern Manitoba[111] did not measure the amount of fat in the diet. In terms of caloric expenditure, in general members of these communities tend to live a sedentary lifestyle using motor vehicles for most transport (cars, school buses, motor boats, snow mobiles, etc.) with very little other physical activity being undertaken. Actual exercise levels, however, have not been measured. Kriska et al[56] have produced a tool for measurement of activity levels in the Pima which, with appropriate modification, could be used for this purpose.

While there have been several reports of appropriate life-style modification improving the metabolic control of NIDDM in

aboriginal peoples (Australian aboriginal diabetics returning to a traditional hunter-gatherer lifestyle[48], the Zuni exercise project[70,71]), experience has shown those working on communities such as the two studied here that the small amount of counseling that the nurses and physician(s) are able to give is quite ineffective in bringing about behaviour change. Even the community health representatives trained in diabetic counseling frequently express feelings of despondency about their failure to convince diabetics (and other community members) to make healthy alterations to their lifestyle.

Community perceptions and understanding of diabetes must be probed if lifestyle modification is to occur. In 1989 Garro[112] undertook a study on an Ojibway reserve in southern Manitoba and reported on some of the cultural understandings which underlie people's talk about diabetes. Garro noted that although much information about the care and treatment of diabetes is conveyed by the physicians, nurses, and community health representatives, little is communicated that explains why diabetes has become so prevalent. Yet, the individuals interviewed readily provided explanations of how they, and other Indian people have come to have diabetes. These explanations included some which fit with the western model of illness; that diabetes was related to buying food from the store, to change away from the traditional diet, to overeating, to eating too much sugar, and to being overweight.

Other explanations were outside the western model; that diabetes was related to poisons, to chemicals, to the Chernobyl nuclear disaster, that diabetes was a sickness brought by the white man along with measles, tuberculosis, cancer, and hypertension, that "it's in all the grain, meat, and vegetables".

Hagey[113], a nurse anthropologist, in an attempt to adjust the native belief system, used a narrative entitled "Nanabush and the Pale Stranger" to communicate information on diabetes in an Ojibway community in southern Manitoba, Canada. The story tells of Nanabush, a legendary figure who represents the teacher in Ojibway culture and his first encounter with the personified character of Diabetes. No one can lie to Nanabush, so what Diabetes tells Nanabush about himself is taken to be the truth. Hagey claims that this method of information transfer helps to gain insight into the culturally specific logic surrounding illness and facilitates the coping process by adding meaning to medical information.

The utility of exploring belief systems and using this information to guide behaviour modification programs must be explored further.

6. HEALTH POLICY IMPLICATIONS

The goal of medical care is to improve the quality of life of patients. For diabetic patients living on isolated communities, strategies for the management of diabetes can be divided into two distinct groups: those which are known to be achievable and those, while being desirable, have proved difficult to achieve. In the first category are aspects of the reduction/prevention of several of the complications of diabetes, namely blindness from retinopathy, renal failure from nephropathy, and lower limb ulceration and amputation from the combined effects of neuropathy and vascular disease. In the second category are the desirable but difficult to achieve goals of improving glycemic control and helping diabetics, and the community in general, to modify lifestyle to reduce obesity and increase exercise levels.

6.1 HYPERTENSION AND NEPHROPATHY

There is considerable evidence that control of hypertension (especially using drugs of the angiotensin-converting enzyme inhibitor class) reduces the deterioration of renal function in diabetics (probably also reducing the chances of end-stage renal failure). Control of hypertension also reduces the risk of cerebro-vascular disease.

Suggested policy

The management of NIDDM should include the control of hypertension, possibly to blood pressures as low as 125/75, where practical. Drugs of the angiotensin-converting enzyme inhibitor class should be used where possible.

Recommendations for implementation of policy

- Physicians and nurses working on these communities should receive training in the latest research findings related to blood pressure control and its effect on renal function in diabetics.

- A register of diabetics with hypertension should be developed and kept up-to-date. A system to encourage attendance at the nursing stations and compliance with medications should be developed.

- The sub-group of diabetics with proteinuria of 1 gm/day or more and/or those with a raised creatinine should have careful follow-up, possibly being seen by an internist or nephrologist on a regular basis.

6.2 FOOT CARE

Peripheral neuropathy, peripheral vascular disease, and abnormal pressure, separately or in combination significantly increase the risk of lower-limb ulceration and amputation in

diabetics. Communities such as Garden Hill and Red Sucker Lake have limited medical services; nurses provide the majority of the primary care and the visiting physician sees cases by referral. Often both the nursing staff and visiting physicians are unaware of the importance of preventative foot care for diabetics and are unable to test for neuropathy. Both groups often feel uncomfortable trimming toe-nails. Knowledge of foot care among diabetics is generally low.

Suggested policy

The management of NIDDM should include high quality foot care.

Recommendations for implementation of policy

- Physicians and nurses working on these communities should receive detailed training on the detection of neuropathy, the prevention of amputation in "at-risk" diabetic feet, the treatment of ulcers, and post-amputation care.

- A community wide education program on general foot-care should be used to raise awareness of this issue.

- Community health representatives should be intimately involved in the processes of community education, the screening of diabetics for peripheral neuropathy, and the identification of diabetics who have ulcers.

- All diabetics should be screened annually for peripheral neuropathy - the 10g mono-filament is an appropriate tool for such screening.

- As there is no simple, reproducible technique to use for screening for peripheral vascular disease and as peripheral vascular disease is not as clearly associated with ulceration and amputation as peripheral neuropathy, only diabetics with non-healing ulcers should be screened for peripheral vascular disease.

- A register of diabetics with "at-risk" feet (those with history of ulceration, peripheral neuropathy, or peripheral vascular disease) should be established and maintained. This group should receive frequent foot examination, foot-care, and education. The education program should be based on "Instructions for Patients at risk for Diabetic Foot Ulcers" contained in Grunfeld's review article and used by Health Sciences Center, Winnipeg.[99] **See ATTACHMENT D.**

- Professional podiatry services should be available to diabetics with insensate feet.

- Fitting and supply of appropriate footwear should be available. The present sources (visiting physiotherapist

and podiatrist) have been inadequate.

- When staff (both physicians and nurses) are unfamiliar with the management of foot ulcers, or ulcers are slow to heal, patients should be managed in conjunction with professionals specializing in that area of medicine. Patients with non-healing ulcers should be referred to multidisciplinary clinics (such as at Health Sciences Center, Winnipeg) for assessment.

6.3 EYE CARE

In North America, diabetic retinopathy is the leading cause of blindness among adults, however it has been conclusively shown that treatment of diabetic retinopathy and macular edema significantly reduces the incidence of subsequent blindness. The recommendations of the expert Committee of the Canadian Diabetic Association for the ophthalmological care of diabetics are included in section 2.6.1. These guidelines state that the examination should be performed by an ophthalmologist and this is consistent with the widely held view that examinations by non-ophthalmologists miss as many as 50% of serious lesions. It remains a logistic challenge to provide this high, but necessary, level of care to the large numbers of people with diabetes, especially those living in remote locations. It would appear to be more cost-effective for an ophthalmologist to visit aboriginal communities on a

regular basis to screen for diabetic retinopathy rather than send all diabetics to Winnipeg by plane for screening.

The communities of Garden Hill and Red Sucker Lake have been fortunate to have had regular ophthalmologist visits and this, combined with some examinations carried out in Winnipeg, has allowed over 70% of the diabetics in these communities to have had ophthalmological examination in the last two year period. Experience has shown however, that it is difficult to recruit ophthalmologists to visit other communities. DQ personally contacted all ophthalmologists in Winnipeg; two offered to visit aboriginal communities for one week per year, all others declined. The fact that some patients are screened by a visiting ophthalmologist, others are screened by ophthalmologists in Winnipeg, and others are travelling to Winnipeg regularly for treatment makes it a complex task for nursing station staff to ensure that diabetics all receive the necessary screening, treatment, and follow-up.

Suggested policy

All diabetics should receive ophthalmological care consistent with the Guidelines of the Expert Committee of the Canadian Diabetic Association.

Recommendations for implementation of policy

- A register of the results of diabetic eye examinations should be established and maintained; this should include results of examinations performed both at the community and in Winnipeg. This register would be used to ensure compliance with the appropriate screening protocols and attendance at treatment follow-up appointments in Winnipeg.

- A concerted effort should be made to recruit ophthalmologists to visit isolated aboriginal communities to screen for diabetic retinopathy. If necessary, the ophthalmological professional associations should be approached for help in managing the screening program.

- The utility of alternative methods of screening should be studied:

  (a) A detailed study should be undertaken to determine the utility of dilated fundal photography to screen for diabetic retinopathy. This technique allows photographs of the fundus to be taken by staff with relatively low levels of training; these photographs are then checked for retinopathy by a centrally located ophthalmologist. This system is used in some areas of the United States Indian Health Service.

(b) As most isolated aboriginal communities are regularly serviced by optometrists, a detailed study should be undertaken to determine the utility of using optometrists to screen for diabetic retinopathy.

6.4 DIABETES CARE COORDINATOR

Sections 6.1.1 to 6.1.3 each recommend the establishment of a register of diabetics to ensure correct medical care. Practical experience has shown that, given the pressures of day-to-day primary care on both the nursing staff and physicians, such registers, when established, tend to quickly become outdated and ineffective.

Recommendation

A permanent position should be created with the mandate to establish and maintain a computerized register of diabetics, their complications, and their medical follow-up schedule. The chronic disease register maintained by Medical Services Branch is unsuitable for this purpose. The incumbent would be responsible to attempt to ensure that diabetics receive all appropriate follow-up and to coordinate this follow-up (e.g., trips to Winnipeg) in a cost-effective manner.

6.5 HYPOGLYCEMIC MEDICATIONS

At present, there is no evidence that use of medication in populations such as that studied here significantly improves metabolic control.

Recommendation

- In these populations it appears that there is little overall benefit from the use of hypoglycemic medications; the onset of **symptomatic** NIDDM may be the main indication for the use of these drugs.

- When hypoglycemic therapy is to be instituted, it appears that drugs of the sulphonylurea class offer better metabolic control than the other hypoglycemic medications, however they are associated with less weight loss (or actual weight gain) than the use of metformin. In cases of symptomatic NIDDM, sulphonylureas should be used for non-obese patients (BMI < 27 kg/m^2) and metformin should be used for obese patients (BMI $\geq$ 27 kg/m^2). If the use of a sulphonylurea fails to relieve the symptoms of a non-obese diabetic, metformin should be added to the regimen. If metformin fails to relieve the symptoms in an obese diabetic, a sulphonylurea drug should be added to the regimen.

- When oral hypoglycemic medications are used, they should

be used in doses which accord with those used in the UKPDS. In that study doses were increased at 1- or 2-weekly intervals until the fasting plasma glucose became less than 6 mmol/l. For glyburide the maximum dose used was 10 mg twice a day, for metformin the maximum dose was 1700 mg at breakfast and 850 mg at the evening meal.

- Because of the relative complexity of administration, difficulties of storage, associated hypoglycemic episodes, and lack of proven superior efficacy, insulin should not be used except, as recommended in the Canadian Diabetic Association guidelines[1], for the short-term management of acute infections in diabetics and the management of pregnancy.

- Clinical trials should be undertaken on communities such as those studied here to determine the effectiveness of combination hypoglycemic therapy, especially the combination of metformin and sulphonylureas. It is possible that a correctly selected diabetes care coordinator would also be able organize these studies.

6.6 LIFESTYLE MODIFICATION PROGRAMS

Ideal interventions would help to prevent diabetes in the broader community as well as providing information and counseling for those who have already developed the disease.

The question remains: "What forms of behaviour modification programs would effectively lead to healthy changes in lifestyle in communities such as the ones studied here?" The Sioux Valley Diabetes Primary Prevention Project[61], which concludes in August 1995, is assessing the impact of a community based health promotion program on diet, physical fitness, and the prevalence of obesity and hyperglycemia. Interventions target various levels of complexity (individuals, groups, organizations, the community at large) and will be introduced into appropriate existing community health related programs, including school, child care, and personal home activities. We await the results of this intervention so that lessons learned can be shared with the communities studied here.

Recommendations

- A thorough review of the literature involving lifestyle modification in diabetes prevention/treatment programs should be undertaken.

- Further investigation of community perceptions of diabetes, obesity, and the need for physical exercise are required.

- The results of the Sioux Valley Study, the literature

review, and a summary of this paper should be presented to the communities concerned.

- Sufficient resources should be made available to allow the communities, with expert advice, to develop behaviour modification programs based on cultural traditions. Such programs may require multimedia (using print, radio, and television) format.

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Diabetic Retinopathy Classification

Nonproliferative diabetic retinopathy
Microaneurysms only
Mild/moderate nonproliferative retinopathy
  Milder degree of venous loops, retinal hemorrhages, hard exudates, soft exudates or IRMA, but less than severe retinopathy
Severe nonproliferative retinopathy—one of the following:
  Hemorrhages or microaneurysms in four retinal quadrants
  Venous beading in two quadrants
  IRMA in one quadrant
Very severe nonproliferative retinopathy
  Two or more severe nonproliferative characteristics

Proliferative diabetic retinopathy
Early proliferative retinopathy
  Proliferative retinopathy, but not high risk (one or two risk factors)
High-risk proliferative retinopathy*—three or four of the following risk factors:
  New vessels in the eye
  New vessels on or near the optic disk
  Moderate or severe new vessels (greater than one-quarter the disk area)
  Vitreous hemorrhage
Advanced proliferative retinopathy
Extensive neovascularization, vitreous hemorrhage or fibrovascular proliferation with or without retinal detachment

IRMA = intraretinal microvascular abnormalities.

**—Unequivocal disk neovascularization or any neovascularization with hemorrhage (the two most common patterns of high-risk retinopathy).*

Derived from Fundus photographic risk factors for progression of diabetic retinopathy. ETDRS report number 12. Early Treatment Diabetic Retinopathy Study Research Group. Ophthalmology 1991;98(5 Suppl):823-33.

Schedule of Ophthalmologic Follow-up Examinations for Patients with Diabetes

Status of retinopathy	*Follow-up (months)*
No retinopathy or microaneurysms only	12
Mild/moderate nonproliferative retinopathy without macular edema	6 to 12
Mild/moderate nonproliferative retinopathy with edema not threatening the macular center	4 to 6
Mild/moderate nonproliferative retinopathy with edema involving the macular center	3 to 4
Severe/very severe nonproliferative retinopathy	3 to 4
Early (mild/moderate) proliferative retinopathy but not high risk	2 to 3
High-risk proliferative retinopathy: immediate scatter laser treatment, then monitor	3 to 4

Adapted from American Academy of Ophthalmology, Quality of Care Committee, Retina Panel. Diabetic retinopathy: preferred practice pattern. San Francisco, Calif.: American Academy of Ophthalmology, 1989.

ATTACHMENT B

DIABETES INPUT FORM DATE OF ENTRY _____

DRUG USED _____

IDNO	1.	_ _ _ _ _	ENTNO 2. _ _ OF _ _
LSTNME	3.	_____	LAST NAME
FSTNME	4.	_____	FIRST NAME
MIDNME	5.	_____	MIDDLE NAME
TREATY	6.	_ _ _ _ / _ _ _ _	TREATY NUMBER/BAND
LOCN	7.	_ _ _ _	LOCATION
MHSC	8.	_ _ _ _ _ _	M.H.S.C.
DOB	9.	_ _ / _ _ / _ _	D.O.B.
AGE	10.	_ _ _	
SEX	11.	_	SEX (M = 0, F = 1)
DXYR	12.	_ _ _ _	YEAR OF DIAGNOSIS
DXAGE	13.	_ _	AGE AT DIAGNOSIS
DXWT	14.	_ _ _ . _	WEIGHT AT DIAGNOSIS
DXCRIT	15.	_	CRITERION OF DIAGNOSIS FBS = 0, RBS = 1, GTT = 2
DIABYRS	16.	_ _	YEARS SINCE DIAGNOSIS
WT	17.	_ _ _ . _	CURRENT WEIGHT (KG)
HT	18.	_ _ _	HEIGHT (CM)
BMI	19.	_ _ . _	(KG/M^2)
WHR	20.	_ _ _ / _ _ _	WHR (CM/CM)
WHRNO	21.	_ . _ _	WHR (FRACTION)
SMOKNOW	22.	_	NOW SMOKING? NO = 0
SMOKPAST	23.	_	PAST SMOKER? YES = 1
IHD	24.	_	NIL = 0 ANGINA = 1 MI = 2
CVD	25.	_	NIL = 0 TIA = 1 CVA = 2

STN1	26.	_	ATTENDED NURS.STN. > 1 LAST YR?
			EYES
OP2YR	27.	_	OPHTHALMOLOGIST EXAM IN PAST 2 YEAR?
OPDATE	28.	_ _ _ _	YEAR OF LAST OPTHAL CHECK
OPPLCE	29.	_	COMMUNITY = 0, WINNIPEG = 1.
OPDOC	30.	_	GOULD = 1, BELLEN = 2, MATHEN= 3 OTHER = 4.
RET	31.	_	RETINOPATHY NO = 0, YES = 1
TYPE	32.	_	NIL = 0 MACULAR OEDEMA = 1 BACKGROUND = 2 PROLIFERATIVE = 3 HAEMORRHAGE = 4 DETACHMENT = 5 BLIND = 6
CATS	33.	_	NO = 0, YES = 1.
LAS	34.	_	NO = 0, YES = 1.
		RENAL	
DIP2YR	35.	_	URINE DIPPED IN LAST 2 YRS? NO = 0, YES = 1
PRT	36.	_	PROTEIN IN LAST 2 DIPS NO = 0, YES = 1
AMT	37.	_	NIL = 0 (USE LESSER TRACE = 1 AMOUNT) 0.3 g/l = 2 1.0 g/l = 3 3.0 g/l = 4 20 g/l = 5
CR2YR	38.	_	CREAT LAST 2 YEARS? NO = 0, YES = 1
CRNORM	39.	_	<110 = O, >110 = 1
LSTCR	40.	_ _ _	LAST CREAT IF < 2 YEARS
DIAL	41.	_	NO = 0, YES = 1
CRF	42.	_	0 = REQUIRES NO TREATMENT 1 = PERITONEAL DIALYSIS 2 = HEMODIALYSIS 3 = TRANSPLANT

NEUROPTHY/PVD

FTCHK	43.	_	NO = 0, YES = 1(IN LAST 2 YR)
METH	44.	_	METHOD 10g = 0 PIN = 1 VIBRAT = 2 OTHER = 3
NEUROPL	45.	_	NO = 0, YES = 1
NEUROPR	46.	_	NO = 0, YES = 1
PVDL	47.	_	NO = 0, YES = 1
PVDR	48.	_	NO = 0, YES = 1
ULCRL	49.	_	NO = 0, YES = 1
ULCRR	50.	_	NO = 0, YES = 1
AMPL	51.	_	NO = 0, YES = 1
AMPLEVL	52.	_	NIL = 0 TOE = 1 MIDTARSAL = 2 BEL KNEE = 3 ABOVE KNEE = 4
AMPR	53.	_	NO = 0, YES = 1
AMPLEVLR	54.	_	NIL = 0 TOE = 1 MIDTARSAL = 2 BELKNEE = 3 ABOVE KNEE = 4

----------BLOOD PRESSURE----------

BP2YR 55. _ BP MEASURED IN LAST 2 YEARS?
NO = 0, YES = 1

SYST 56. _ _ _

DIAS 57. _ _ _

HT 58. _ HYPERTENSION? NO = 0, YES = 1
(ON TREATMENT OR DIASTOLIC > 90 OR SYSTOLIC > 160)

BPTRT 59. _ INCLUDES: NIL = 0
ACE = 1
CALCIUM = 2
ACE + CA = 3
OTHER = 4

BPCONT 60. _ DIASTOLIC <90 = 0
90 - 104 = 1
105 - 114 = 2
>115 = 3
IF DIAST < 90 BUT SYST 140 - 150 = 4
> 160 = 5

THIS INTERVAL OF THERAPY

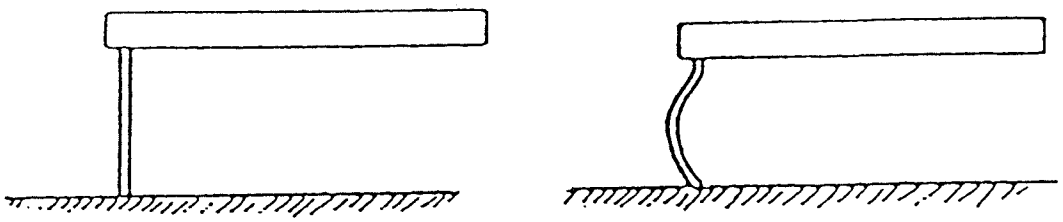
TRT	61.	_	NO DRUG = 0 SULPH = 1 MET = 2 INSULIN = 3 SULPH + MET = 4
		_ _ /_ _ /_ _	START DATE (DD/MM/YY)
		_ _ /_ _ /_ _	STOP DATE (DD/MM/YY)
MTHSTRT	62.	_ _ _	NO OF MONTHS ON TREATMENT
INWT	63.	_ _ _ . _	START WEIGHT (KG).
FNWT	64.	_ _ _ . _	STOP WEIGHT (KG).
WTCH	65.	_ _ _ . _	WT CHANGE (KG) (REMEMBER SIGN)
HBAIC	66.	_ _ . _	AVERAGE (%)
FBS	67.	_ _ . _	AVERAGE FBS
RBS	68.	_ _ . _	AVERAGE RBS
2HRPC	69.	_ _ . _	AVERAGE 2HRPC
CAT	70.	_	CATEGORY OF CONTROL (0-2)
PRVTRT	71.	_	THERAPY USED BEFORE PRESENT
NEXTTRT	72.	_	THERAPY USED AFTER PRESENT NO DRUG = 0 SULPH = 1 MET = 2 INSULIN = 3 SULPH + MET = 4 NO SUBS OR PREV THERAPY = 5 (USE CAT 5 UNLESS PREV OR SUBS TREATMENT IS CLASSIFIED AS A FULL INTERVAL OF THERAPY)

Filament Application Instructions

Note: The sensory testing device used with the FOOT SCREEN is a nylon filament mounted on a holder that has been standardized to deliver a 10 gram force when properly applied. Our research has shown that a patient who can feel the 10 gram filament in the selected sites will not develop ulcers.

Instructions for sensory testing on the foot:

1. Use the 10 gram filament provided to test sensation.
2. The sites to be tested are indicated on the Diabetic Foot Screen Form.
3. Apply the filament perpendicular to the skin's surface. (see diag. A)
4. The approach, skin contact and departure of the filament should be approximately 1 1/2 seconds duration.
5. Apply sufficient force to cause the filament to bend. (see diag. B)



A B

6. Do not allow the filament to slide across the skin or make repetitive contact at the test site.
7. Randomize the selection of test sites and time between successive tests to reduce the potential for patient guessing.
8. Ask the patient to respond "yes" when the filament is felt and record response on the Diabetic Foot Screen Form.
9. Apply the filament along the perimeter of and NOT on an ulcer site, callous, scar or necrotic tissue.

Developed by
Rehabilitation Branch
Gillis W. Long Hansen's Disease
Carville, Louisiana 70721

INSTRUCTIONS FOR PATIENTS AT RISK FOR DIABETIC FOOT ULCERS

Foot Care

1. Inspect the inside of your shoes for foreign objects before putting the shoes on.
2. Inspect your feet daily for blisters, cuts, scratches, redness, dryness, hot spots or infection.
3. Wash feet daily and dry carefully, especially between the toes.
4. Avoid hot temperatures; test water with elbow before bathing.
5. If your feet are cold, wear socks. **DO NOT** use hot water bottles or heating pads.
6. **DO NOT** cut corns or callouses or use chemical removal agents.
7. **DO NOT** use adhesive tape on your feet unless instructed by your physician.
8. Cut nails in contour with the toes; do not cut deep down the sides or corners.
9. Consult your physician before soaking feet or using lubricating creams or oils.
10. If your vision is impaired, a family member or friend should be trained in these items.

Foot Wear

1. Shoes should be measured properly at the time of purchase; do not depend on them to stretch out.
2. Break shoes in slowly, wearing them for no more than a few hours at a time in the beginning.
3. Wear shoes of a material that breathes.
4. Avoid pointed toes or high heels.
5. **DO NOT** walk barefoot; particularly avoid hot surfaces in the sun, on beaches or around swimming pools. At night, turn on the lights and **DO NOT** walk barefoot to the bathroom.
6. Wear shoes appropriate to the weather: avoid wearing wet shoes; use thick socks or lined boots in the winter.
7. Avoid sandals or thongs.
8. Avoid garters, tight elastic bands on socks, or rolling hose. Always wear socks or stockings with shoes, but avoid thick seams or mended areas.

Professional Care

1. See your physician regularly and be sure that your feet are examined at each visit.
2. Notify your physician at once if you develop a blister, sore or crack in the skin of your feet.