

MOHAWK DIABETES AND FAT PATTERNING

MOHAWK DIABETES, OBESITY  
AND FAT PATTERNING

by

NAOMI ADELSON, R.N., B.A.

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AUTHOR: Naomi Adelson, R.N. (Vanier College)

B.A. (McGill University)

SUPERVISOR: Professor E.J.E. Szathmary

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## ABSTRACT

This research project examines the high rate of non-insulin dependent diabetes mellitus (NIDDM) among the Mohawk Indians of Kahnawake, Quebec in relation to current knowledge about diabetes among Amerindians and in the context of the association of NIDDM with a centripetal patterning of subcutaneous fat. The hypothesis tested is that NIDDM is positively correlated with a centripetal distribution of body fat in the adult Mohawk Indians of Kahnawake. In this cross-sectional case-control matched study, Mohawk Indians from the Kahnawake reserve near Montreal, Quebec were matched for age, sex and diabetic status. The results of skinfold ratios, waist to hip circumference ratios, and principal components analysis indicate that while nearly all of the participants fall above the current standards of obesity, there is no distinct trunkal distribution of subcutaneous body fat in those with NIDDM. Thus, contrary to the stated hypothesis diabetic Mohawk Indians do not show a propensity to trunkal obesity. Several possible explanations for these findings are suggested. Other findings of this study are: 1) a significantly lesser amount of fat on the lower extremities of the diabetic men, and to a lesser degree, of the diabetic women than their respective controls; 2) a strong intra-generational effect present in the sub-population of diabetics; 3) no difference in degree of non-Native ancestry can be discerned between those with or without NIDDM; 4) the pattern of obesity and disease occurrence (NIDDM and cholelithiasis) does not follow that of the New World Syndrome as is it currently defined; and, 5) high rates of macro-vascular disease were found in those with diabetes. Suggested future research involves longitudinal studies from time of diagnosis or earlier in order to discern the relative contribution of environmental factors in the occurrence of NIDDM as well as continued study of the relationship between NIDDM and body fat in this population.

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The Indians "...are never troubled with Scurvy, Dropsy, nor Stone. The Phthisick, Ashma (sic), and Diabetes they are wholly strangers to..." John Lawson, 18th century explorer (in Vogel 1977).

## Chapter One: Introduction

The aim of this research project is to examine the high prevalence of non-insulin dependent diabetes mellitus (NIDDM) among the Mohawk Indians of Kahnawake, Quebec in relation to current knowledge about diabetes among Amerindians and in the context of the association of NIDDM with a centripetal patterning of subcutaneous fat. Approval for the study was granted by the Kateri Memorial Hospital Centre (KMHC) Board of Directors. The Board of Directors acts on behalf of the Band Council on all matters related to the health of the people of Kahnawake (see note 1).

Presented in this thesis are the results, analysis and conclusions drawn from the research done in Kahnawake. A short term study of known diabetics and non-diabetic controls from the same population was conducted in an effort to understand the recently documented high prevalence rate of NIDDM on this Iroquoian reserve (Montour and Macaulay 1985).

The sudden rise in the rate of diabetes in the male and female adults of Kahnawake over the last two to three decades parallels the prevalence pattern seen among many other Amerindian populations (West 1974; Zimmet 1982). There is at present no clear explanation for the apparent epidemic-like

occurrence of NIDDM. The type and extent of obesity that tends to accompany NIDDM is thought to play a role in the triggering of this disease (Vague 1956; Albrink & Meigs 1964; Karam 1982). Thus, the hypothesis being tested is that non-insulin dependent diabetes mellitus (NIDDM, Type II) is positively correlated with a centripetal distribution of body fat in the adult Mohawk Indians of Kahnawake, Quebec.

Other objectives of this project include the determination of the degree to which duration and extent of obesity, parity, familial inheritance patterns and Caucasian admixture in the population affect the occurrence of NIDDM. A brief overview of the prevalence of clinical disorders that are related to NIDDM in this population is also presented.

#### 1. Diabetes Mellitus

Diabetes mellitus is now recognized as a genetically heterogeneous group of disorders characterized by the inability to metabolize glucose. This inability to breakdown and store glucose is conventionally thought to occur as a result of a deficiency or lack of insulin. In fact, this is only partially true, for some forms of diabetes are characterized by insulin excess, as described below. Although there are over thirty known types of this chronic disease the two most common are Insulin-dependent (IDDM, Type I) and non-insulin

Type I diabetes is distinguished by the lack of pancreatic insulin and a proneness to ketosis (that is, an abnormal accumulation of the end products of fat catabolism) and hence a dependence on injected insulin to sustain life. Onset of IDDM is abrupt and usually occurs in individuals before they are forty years of age. At the cellular level, there is an association between IDDM and an alteration of certain histocompatibility antigens (HLA) encoded on chromosome 6 as well as abnormal immune and auto-immune antibody responses. Type I diabetes is not associated with obesity and is uncommon in certain ethnic groups, even where the prevalence of NIDDM is extremely high (Bennett 1982; Stryer 1981; NDDG 1979).

The hyperglycemia of Type II diabetes is associated with a relative underutilization and not the lack of insulin that is characteristic of IDDM. The life-threatening biochemical imbalance of IDDM is not seen in Type II diabetes since the insulin reserve may be unhampered. The pancreatic exhaustion leading to the insulin deficiency found in advanced cases of NIDDM is believed to stem from a decreased cellular response to this hormone. As less glucose can be disposed of, the resulting hyperglycemia precipitates a higher blood insulin level. This chronic hyperinsulinemia may lead eventually to a relative insulin deficiency (Steinke 1974).

The onset of non-insulin dependent diabetes mellitus is usually slow and insidious. The syndrome usually occurs

after the fourth decade of life and is often only discovered during routine examination and not because the individual has symptoms or complaints. According to Bennett there are at least as many persons with undiagnosed NIDDM as there are those who have been made aware of the diagnosis (1982: 391). While some individuals with Type II diabetes may require insulin for the correction of persistent fasting hyperglycemia they are not prone to ketotic episodes and are thus not dependent on injected insulin. The hyperglycemia of Type II diabetes can usually be corrected by controlling dietary intake and with oral hypoglycemic agents.

Unlike Type I diabetes NIDDM has not yet been clearly associated with any specific HLA markers or with abnormal antibody activity. Recent research has revealed though that there may be a specific marker associated with NIDDM in certain populations. A relationship has been found between the antigen HLA-A2 and non-insulin dependent diabetes in Pima Indians and in South African Blacks (Knowler et al. 1981; Briggs et al. 1980)

The differences in etiological and genetic bases for the two distinct types of diabetes just described do not affect the rate of occurrence of the debilitating vascular sequelae associated with this disease. Micro- and macro-angiopathies involving the blood vessels and major organs ultimately affect diabetics with the same frequency despite

the origin or type of insulin deficiency (Bennett 1982; NDDG 1979; Karam 1982).

#### ii. The Prevalence of NIDDM

The overall prevalence rate of NIDDM in the United States was 2% in 1974 (no similar statistics are available for Canada) (National Diabetes Commission 1975 in Health & Welfare Canada 1985). This figure however does not reflect the actual occurrence of the disease. Differences attributable to age, sex or ethnic origin are not discerned nor are undiagnosed cases of NIDDM accounted for. Bennett estimates that the prevalence of Type II diabetes in the United States is actually closer to 6% in those between 40 and 59 years of age, as high as 13% in individuals over the age of 60 and is always higher in women (1982:392).

What is still not readily seen in any of the general prevalence figures is the enormous ethnic and geographic variability in the occurrence of the disease irrespective of age or sex. Prevalence and incidence studies have shown that despite the lack of uniformly controlled diagnostic criteria there is a striking diversity in the prevalence of NIDDM between different ethnic groups (Zimmet 1982; Bennett 1982). Compare, for example, rates as high as 50% in the Uto-Aztecan Pima Indians of Southcentral Arizona (Sievers and Fisher 1981; Knowler et al. 1981) with even the highest figure that

Bennett has proffered.

Nowhere is the diversity in the occurrence of NIDDM more apparent than across Amerindian linguistic groups. Rates of NIDDM range from up to fifty percent to less than one percent of the adult population in different Amerindian language families. High rates of Type II diabetes have been reported for the Choctaws, Chickasaws, Seminoles and Creeks; all sub-groups of the larger Southeastern Muskogean family (West 1974). Prevalence rates are as high as 10% and 12% of the adult population in the Paiute, Washoe and Yuma Indians. Each of these groups is linguistically related to the Pima, whose adult population exhibit the highest rate of NIDDM worldwide (Bartha et al. 1973; Cohen 1954; West 1978). Iroquoian Cherokee, Seneca and Mohawk Indians all share a similar high prevalence of NIDDM (Stein et al. 1965; Doebelin et al. 1969; Montour & Macaulay 1985, respectively). Epidemiological studies of the Cree and Ojibwa Indians of Central Canada reveal a moderately lower overall prevalence rate of NIDDM. Young et al. (1985) found the number of diagnosed cases of Type II diabetes to be about 3%. When broken down by age the prevalence of NIDDM in these two Algonkian groups increased to 10% in those over 65; almost identical to urban Caucasian rates of NIDDM (Bennett 1982).

Northern and Southern Athapaskans, on the other hand, exhibit a substantially lower frequency rate. NIDDM is in-

frequent or rare among the Navajo (Cohen 1954; Rimoin 1969), northern Alaskan (Mouratoff 1969) and Northwest Territorial Dogrib (Szathmary 1983) Indians. Similar low rates of Type II diabetes are reported for the Inuit of northern Canada (Schaefer 1968; Schaefer et al. 1972; Mouratoff et al. 1973).

The apparent variability in the rate of NIDDM among the Indians of North America may be attributed to variations in diet, habitat, or genetic predisposition. These causes are currently being debated. In assessing the differences in the distribution of the disease however, factors such as method of diagnosis and actual reporting may be confounding the data base. As West (1978) notes, the variability in the rates could be attributed to incomplete reporting, unavailability or insufficient use of the medical services provided. Szathmary (1986), in a recent paper, points to factors that may be contributing to a falsely high pattern of prevalence. In a thorough review of the Amerindian research to date, she found a consistent lack of standardized diagnostic criteria used in screening studies prior to the availability of the NDDG guidelines. The methods of determining diabetic status were so inconsistent that the results can not be assumed to be accurate. The high rate of NIDDM in some populations, suggests Szathmary (1986), may be a reflection of inaccurate baseline values or diagnoses based on single screenings rather than the actual distribution of the disease.

Despite the inconsistent reporting, there are similar-

ties in the prevalence of Type II diabetes among Amerindian populations. Three features in particular distinguish the pattern of occurrence seen in Amerindian populations. Most discernible and yet enigmatic is the universal relative increase (in the reporting) of NIDDM over the last four to five decades. Other consistent findings include the virtual non-existence of IDDM and the rarity of ketosis among Indian diabetics (West 1974; Zimmet 1982). Once thought to be an innocuous disease among Amerindians, the debilitating vascular sequelae of diabetes are increasingly becoming a health concern for the Aboriginal peoples of North America (West 1978; Zimmet 1982; Nutrition Newsletter 1983).

### iii. An Interaction of Genes and Environment

The great number of prevalence studies to date have prompted new theories about the etiology of NIDDM. The combination of different rates and yet a general rise in the number of diabetics over all linguistic groups suggests a multifactorial model of causation for this disease. There is thought to be an integration of a genetic susceptibility with certain environmental elements that triggers the onset of NIDDM (NDDG 1979); in Keen & Jarrett's (1976) terminology, a 'diabetogenic mix'.

Strong familial occurrence patterns suggests an in-

heritable predisposition to NIDDM. Studies among Oklahoma and Pima Indians indicate that those with siblings or parents with Type II diabetes were at a significantly greater risk for the disease independent of any other contributing factors (Lee et al. 1985; Beaty 1982; Knowler et al 1983; Bennett et al. 1976). Although there may be a "diabetic genotype", there is no evidence to date that a major gene which regulates glucose exists or is responsible for this metabolic disorder (Neel 1962; Zimmet 1982; Szathmary 1985).

The actual onset of NIDDM is seen only when the susceptible individual is exposed to certain environmental stimuli (NDDG 1979; Bennett 1982; Weiss 1985; Rimoin 1969; West et al. 1976). The exact stimulus is unknown although there are many factors that have been implicated. Most of the hypothesized elements suggested as being causally related to NIDDM tend to parallel factors related to acculturation.<sup>1</sup> Indeed, despite differing hypotheses to explain the higher rate of NIDDM and different Amerindian populations, studies by both Knowler et al. (1981) and Szathmary and Holt (1983) suggest that there is a positive association between degree of acculturation and onset of hyperglycemia. As well, migrant studies of Pacific Melanesian populations show the prevalence

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<sup>1</sup> Defined as major culture changes that occur as a result of prolonged contact between societies and in this case, the effects of prolonged contact between Amerindian and Western (Euroamerican) societies.

of NIDDM increases when members of the same ethnic group move from a rural to an urban environment (Zimmet et al. 1981; O'Dea et al. 1980; Tan et al 1981; Bennett 1982; NDDG 1979; Zimmet 1982).

Changes in type and quantity of diet, level of activity, socio-economic status, exposure to toxins and increased psychological and physiological stress associated with the transition to Western lifestyles have all been investigated as possible etiological agents (Zimmet 1982; Mouratoff et al. 1969 & 1973; Cohen 1954; Knowler et al. 1981; O'Dea et al. 1980; Eaton 1977). Age and parity have also been proposed as possible factors (Zimmet 1982; Mouratoff et al. 1969; Bennett et al. 1976; Lee et al 1985; Kalkhoff 1983 [studied age only]; Kissebah et al. 1982 [studied age only]).

#### iv. Obesity and Fat Patterning in NIDDM

Obesity in Amerindians, as in Caucasians, is highly correlated with NIDDM. In fact, obesity is often implicated as the sole or primary causal factor in the onset of NIDDM independent of the etiological basis for the increased fat mass (NDDG 1979; West 1976; Bennett et al. 1976; Zimmet 1982; Young et al. 1985; Beaty et al. 1982).

However, whether obesity alone is diabetogenic or whether it is an adequate explanation for the increase in the prevalence of diabetes in Amerindian as in Caucasian popula-

tions is currently under dispute (Weiss et al. 1984; Vague et al. 1979; Armellini et al. 1979; Keen et al. 1979; KÙbberling 1979; Kissebah et al. 1982; Stern et al. 1983; Bartha et al 1973). Duration, extent and especially location of adipose tissue may play a larger role in the onset of NIDDM than previously thought.

As early as 1956 Vague (1956) differentiated between android (centripetal, trunkal), or predominantly an upper body fat patterning and gynoid, or lower body fat patterning. His research indicated that diabetes occurred 80-90% of the time in association with a pronounced centripetal fat pattern in both men and women.

Trunkal fat patterning is not merely a random placement of labile adipose tissue. Fat located primarily on the upper body differs morphologically and metabolically from that distributed to the lower parts of the body. Centripetally located fat is the result of a hypertrophy or enlargement of adipocytes whereas lower body fat arises from an increase in the number, or hyperplasia of fat cells in a particular area. Differences in the biochemical and hormonal activity of the two types of of adipose cells are thought to be linked to insulin resistance and the consequent diabetes mellitus (Karam 1982; Vague 1956; Vague et al. 1979; Kissebah et al. 1979; Hartz et al 1984; Kissebah et al 1982).

Studies of age, sex and race matched Caucasian, Negro,

Mexican American and Athapaskan Indian individuals have independently revealed a significant correlation between centripetal distribution of body fat and glucose intolerance (Feldman et al. 1969; Kissebah et al. 1982; Kalkhoff et al. 1983; Mueller et al. 1985; Szathmary and Holt 1983). The implication of these findings for Amerindians is far-reaching. As Szathmary and Holt (1983) noted, even if trunkal fatness is a racial characteristic in Amerindians as some have proposed (West 1978), this does not mean that diabetics cannot be distinguished from their kinsmen on the basis of fat patterning.

Few studies have investigated the association between diabetes and trunkal fat patterning in Amerindians. Presented here are results of the research that was conducted at Kahnawake in 1985 which builds upon and adds to the current knowledge of body morphology and NIDDM in North American Indian populations.

## CHAPTER TWO: SUBJECTS AND METHODS

Data collection took place from mid-July to late September of 1985 in facilities provided by the Kateri Memorial Hospital Centre.

### 1. The Mohawk Indians of Kahnawake, Quebec

Mohawk Indians are members of the Six Nations Confederacy, a political alliance which is comprised of the six northeastern North American Iroquoian tribes (Five Nations originally: Mohawk, Oneida, Onandaga, Cayuga and Seneca, and the Tuscarora who joined the confederacy at a later date). The League of Six Nations was founded as a social and political group that in its actions reflects the Iroquoian conceptualization of the harmony of the universe. The Longhouse is the symbolic representation of that confederacy, both in spirit as well as in territorial delineations (Blanchard 1980; Hertzberg 1966; Prudeck 1981).

The Mohawks were responsible for guarding the eastern doorway of the Longhouse, that is, the eastern borders of Iroquoian territory. As sentrymen of what is today northeastern North America and the St. Lawrence River the Mohawks

appropriately became the first members of the Confederacy to make contact with European explorers and traders. Economic ties were established in the early 1600's as the Mohawks became brokers in the fur trade and then voyageurs for the British. At the end of the nineteenth century many of the men of Kahnawake found employment in the new structural steel industry as high steel workers, hence continuing a long established tradition of commercial association with the Euroamericans of North America. The structural steel industry remains to this day a viable professional choice for many of the men of Kahnawake. (Blanchard 1980; Jennings 1975; Mitchell 1978).

Kahnawake is located approximately 20 kilometres southwest of present day downtown Montreal on the shores of the Lachine rapids (kahnawake means "at the rapids"). The reserve was settled in 1676 by Mohawk converts to Catholicism as settlements further south became overpopulated. The site was chosen in an effort to increase trade with Europeans (Blanchard 1980).

Kahnawake is today a suburban and partly rural reserve with a total population of approximately 5,400 (Indian & Northern Affairs Canada 1984). Despite the early and pervasive contact with Euroamericans and the conversion by many to Catholicism, the Mohawk Indians of Kahnawake have always retained a cultural, economic and political sovereignty and identity (Blanchard 1980). There is evidence on the reserve

of a continued effort to maintain this identity in face of the pressures of assimilation through programs such as cultural and language education. The health status of the community is one of the many concerns that is dealt with at the local political level (see Note 2 for a description of the range of services available). Recognition by the medical staff of the Kateri Memorial Hospital Centre (KMHC) of the increase of diabetes in the adult population of Kahnawake prompted the acceptance of this research project by the Band Council through the KMHC Board of Directors (Note 2).

#### **ii. Study Population: Sampling and Recruitment**

In order to minimize sampling bias, selection criteria were established prior to the recruitment of the study population. The four basic criteria are outlined as follows: each participant had to 1- be at least thirty-five years of age, 2- use (or have used) the Kateri Memorial Hospital Centre for their health care needs, 3- live on the reserve, and 4- have two parents and/or one parent and three grandparents of Mohawk descent.

A minimum age limit of thirty-five years in both diabetics and controls was determined by the actual distribution of NIDDM in Kahnawake. All known diabetics except one are thirty-five years of age or older. Universal usage of KMHC

minimizes any disparities attributable to differential access to health care facilities between the diabetics and non-diabetics although three of the participants were no longer using the facilities for their current care (in those cases, and after written approval by the participant, their respective physicians were contacted by N. Adelson and the questionnaire was completed with their assistance). Controlling for place of residence reduces the effect of inter-reserve variability although it does not control for possible intra-Kahnawake differences in acculturation. The hypothesis being tested is population specific, thus Indian origin and homogeneity of the study population was both specified and assured.

A successful study was anticipated since previous research done through KMHC revealed that approximately 92% of the adult population utilize the health services provided by the Kateri Memorial Hospital Centre (Montour and Macaulay 1985) and that there has been a high rate of participation by community members in research done through the centre in the past (Macaulay 1981).

Participation in the study was voluntary. Sample size selection was based on the number of known diabetics as of June 1985 such that at least one third of the participants be one half of previously diagnosed diabetics and the other two thirds be non-diabetic controls (a diabetic to control ratio of 1:2). A list of known diabetics was provided by the home care nurse a month prior to the start of the study. From

-TABLE I-  
Number of Subjects in Each Age Range  
Versus Target Number#

Age Range:	Actual (Target)			
	Male N=80		Female N=124	
	Diabetic	Non-Diabetic	Diabetic	Non-Diabetic
35-45	3 (3)	8 (6)	5 (4)	11 (8)
46-55	9 (8)	15 (16)	11 (9)	16 (19)
56-65	14 (11)	11 (22)	17 (12)	21 (24)
66-75	6 (5)	7 (10)	10 (8)	19 (17)
76-90	2 (2)	5 (4)	5 (3)	9 (6)
<b>Column Total</b>	<b>34 (29)</b>	<b>46 (58)</b>	<b>48 (36)</b>	<b>76 (74)</b>

# Excludes the controls who on testing for Hb-A1 were found to be hyperglycemic

this list of 138, one was below the minimum age limit, one was deceased, two were non-Indian, and two were non-residents.

The remaining 132 known diabetics were stratified by sex and then further divided by year of birth into decade age ranges. Only 66 of the 132 diabetic individuals were required to meet the needs of the study although all diabetics were contacted in order to achieve the target figure. For the first mailing of letters requesting participation in the study every other member of each sub-group was selected and contacted. The second and subsequent mailings went to all of the remaining known diabetics who were eligible to participate. By the end of the data collection period 82 (124% of the target number) individuals participated. A breakdown by age range and a comparison to the target number is provided in Table I.

Non-diabetics were selected from the 1984 Kahnawake Band Date of Birth List (1985). The number of individuals in each age range was divided by the number of diabetics in that age range. The resultant percentage provided a means of selecting individuals from the already randomized list. For example, of those born between 1910 and 1919, 27 of the 324 (or 8%) have been diagnosed as diabetic. Using the figure randomly selected by this method (that is, 8) every eighth male and every eighth female was chosen for inclusion in the study. If that individual failed to meet the study criteria

(see note 3) or opted not to participate then the next person of the same sex in the particular age range was selected. At the end of the data collection a total of 261 non-diabetic individuals were selected for study. Of the 261, 94 (or 36%) agreed to participate.

Discussions of the study on the local radio station increased local awareness of both the study and of the problem of NIDDM in this community. Two interviews with the co-investigators (Adelson, Montour and Macaulay) and daily reminders on the afternoon talk show kept the community up to date on the progress of the study. Twenty-eight non-diabetic individuals who satisfied the study criteria were recruited by this method and participated in the project (see Chapter 3 for discussion of this inclusion process).

One hundred and thirty controls represents 98% of the target number of 132 (twice the projected target number of diabetic participants). Of the 130, one was subsequently excluded because she was below the minimum age limit and seven were excluded from the analysis because they were found to be hyperglycemic according to the glycosylated hemoglobin results taken at the time of the study. The final number of non-diabetic participants is 122 (or 92% of the target). A breakdown by age range with a comparison to the target number is provided in Table I.

All potential participants were sent a letter explaining the reason for the study, that it had been approved by

the Band Council, what their participation entailed, and that they were being asked to participate irrespective of their diabetic status (Appendix A:1). Since letters sent within the reserve are cancelled and distributed directly to the post office boxes (there is no delivery service) the mail could be picked up the day after it was sent out. A follow-up call made by the research assistant confirmed receipt of the letter. The assistant also answered any of the questions of the potential participants. If they agreed to participate, a day and time was scheduled for the data collection process. A second follow-up and reminder call was made to each participant the day prior to his or her appointment. A letter thanking each of the participants was mailed out at the end of the data collection phase of the project (Appendix A:2).

### **iii. Data Collection**

#### **a. Overview**

Data collection included anthropometric measurements and a single venipuncture as well as completing a family, personal and medical history questionnaire with information provided by both the participant and his or her respective medical chart.

The process began with a short discussion of the study

and of what was to be done in the following half hour. Participants were then asked to sign a consent form (Appendix A:3). Consent was obtained from every participant prior to inclusion in this study. Anonymity was ensured since questionnaires were coded, only the master list having the name and corresponding identification number of the participant.

#### b. Questionnaire

The questionnaire was designed in order to obtain information both from the participant and the medical charts. Data was collected from each individual about ethnic origin, parent, spouse or sibling diabetes, civil status, smoking habits, weight history, parity, body image, and for diabetics, their treatment regimen and year of onset of NIDDM. All questions were asked by and responses recorded by N. Adelson.

Appendix A:4 is a copy of the questionnaire used in this study. The majority of the questions are self-explanatory and only where the method differed from that stated or where a definition is necessitated will there be fuller explanations provided. In consultation with Band members certain revisions were made to the original questionnaire. Questions were rephrased or omitted based upon either the cultural parameters of the population or the feasibility of the question itself. For example, as would be appropriate in this matrilineal society, ethnic origin of first degree

relatives was asked first of maternal and then of paternal kin.

In questions 3 - 5 of the Family and Personal History section of the questionnaire the place of residence and the names of relatives of the participants was omitted. Instead only the number of children and siblings was collected.

In the Individual's Medical History section of the questionnaire, question 20 was phrased as 'what do you do for your diabetes?'. It was felt that in asking the question this way there might reveal any discrepancy in the treatment prescribed and actually used for NIDDM. Question 28 of the same section, 'what do you usually weigh?' was found to be difficult to answer because of the ambiguity of the word 'usually'. The question was rephrased to read as 'what has your average weight been in the last 2 - 5 years?'.

Question 32, '...what do you think your ideal weight should be?' could not be answered simply. Instead, 'how much would you want to lose?' was asked of those participants who answered affirmatively to question question 31 ('do you think that you are now overweight?').

The Medical History Questionnaire was collected from the medical charts at KMHC by N. Adelson. Methodology for testing diabetic status has not been standardized and thus both oral glucose tolerance test results and results of fasting and 2-hour post-prandial blood glucose values were

used to confirm diagnosis of disease status. Considered to be diabetic were all those whose plasma glucose levels conformed to the National Diabetes Data Group classification (1979) of non-insulin dependent diabetes mellitus and who had been previously diagnosed as diabetic. Those who did not meet the criteria or for whom no confirmatory lab values were available were considered to be diabetic based upon a positive diagnosis by a physician and if they were currently taking oral hypoglycemic agents or insulin.

All complications of diabetes and chronic diseases, except hypercholesterolemia were based upon a medical diagnosis. In the case of hypercholesterolemia, results of standard SMA-12 total serum cholesterol levels taken after 1982 were used. The criteria followed what is outlined on the questionnaire. Cancer, question 45e, was too broadly defined to be included in the analysis. For question 45f, only chronic diseases were recorded (see Note 1).

#### c. Description of Anthropometric Measurements

Anthropometric variables include stature, weight, chest, abdominal, hip (gluteal), upper arm and calf circumferences and skinfold measurements at the forearm, biceps, triceps, subscapular, midaxillary, suprailiac, abdominal and medial calf sites (exact site locations are given in Feldman 1969; Standardized Test of Fitness 1984). Measurements were taken with participants standing, wearing light under-

clothing and a hospital gown and with their shoes removed. All measurements were taken by N. Adelson. Those measurements not taken at the midline were taken at the right side of the body. Any of the known effects of inter-observer variability were minimized as there was only one investigator collecting the anthropometric data (Jamison et al 1976; Burkinshaw et al 1973; Roche et al. 1985). Retesting of measurement technique was performed at one week and one month to verify uniformity of the method and to assure that there was minimal intra-observer variability (concordance between 95 and 100%).

Stature was measured with an anthropometer (GPM) to the nearest millimetre while the individual was standing shoeless and erect. Weight was taken to the nearest tenth of a kilogram using a Seca scale that was placed on a flat surface and checked daily for accuracy.

Circumferences were measured with a flexible metal tape to the nearest millimetre. Chest girth was taken at the midline of the sternum with the tape horizontal and at the end of the participant's normal expiration. Abdominal circumference was taken at the level of minimum girth on the unclothed abdomen. Hip girth was measured posteriorly at the maximum protrusion of the gluteals and anteriorly at the level of the symphysis pubis. Arm circumference was taken on the right upper arm halfway between the acromion process of

the scapula and the tip of the elbow. Calf circumference was measured at the maximum circumference (by visual inspection).

The eight skinfold measurements of subcutaneous adipose tissue were taken to the nearest millimetre using a Lange skinfold caliper (model no. 1278). In order to increase accuracy in taking a stable reading each site was measured twice and the average of the two readings used. Measurements were performed using standard technique:

-Forearm: with the elbow bent ninety degrees, the forearm skinfold was measured at one third the distance from the olecranon process to the ulnar styloid along the posterior aspect of the ulna.

-Triceps: with the arm bent, the distance halfway between the distal ends of the acromion process and the proximal end of the olecranon was determined. The skinfold was lifted parallel to the long axis and along the posterior aspect of the arm. The triceps measurement was taken with the participant's arm lowered and hanging freely at his side.

-Biceps: the same midpoint as for the triceps was used to measure the biceps skinfold along the anterior aspect of the pendant arm, taken parallel to the long axis of the upper arm.

-Subscapular: this skinfold measurement was taken at approximately one centimetre below and along the line of the inferior aspect of the scapula.

-Midaxillary: one third the measured distance from the apex of the axilla to the iliac crest along the midaxillary line was used as the point of measurement.

-Abdominal: this skinfold measurement was taken at five centimetres to the right of the umbilicus.

-Suprailiac: this skinfold was measured at three centimetres above the iliac crest with the fold running parallel to the crest at the midline of the body.

-Medial Calf: measurement of this skinfold was taken along the medial aspect of the calf at its largest diameter (by visual inspection). (Source: Feldman 1969; Standardized

Test of Fitness 1984)

d. Blood Analysis

After all the measurements were taken and with the individual sitting, a single 10 ml. blood sample was drawn using a disposable B-D 20G needle and holder and a violet top (EDTA) B-D Vacutainer. The blood samples were kept refrigerated until received by the laboratory in Pittsburgh.

The blood analysis included testing for glycosylated hemoglobin concentrations (Hb-A1) and red cell antigen markers. The latter study will provide a genetic description of the Kahnawake Mohawk Indians. As well there will be a determination of the proportion of non-Indian admixture in the Mohawk population and an estimation of the extent of genetic differentiation within this population and between other Iroquoian nations (see note 1) (Szathmary 1986).

For the purposes of this study, the case-control design necessitates that diabetics and their controls have comparable degrees of Indian ancestry. To this end, information obtained from genetic markers would allow the calculation of average amounts of non-Indian ancestry in the diabetic subpopulation and their non-diabetic controls. The two group means should not be significantly different if the groups are, on average, properly matched. The laboratory methods used to determine the phenotypes at the five loci used in admixture estimation (ABO, Kell, Rh, ACP and Immunoglobulin

Gm) are given in Szathmary (1983) and Szathmary et al. (1983). Because this is not a thesis in population genetics, nor is my training in this disciplinary area, all calculations were carried out by Dr. E.J.E. Szathmary (Szathmary 1986: unpublished calculations).

Glycosylated hemoglobin concentrations were obtained to ensure that non-diabetic controls were in fact normoglycemic and not to ascertain diabetic status (refer to Ferrell et al. 1984 and Abraham et al. 1983 for the laboratory method in measuring the stable fraction of glycosylated hemoglobin). The concentration of Hb-A1 reflects the blood glucose concentration over the lifetime of the red blood cell. Since there is a high correlation between the Hb-A1 level and long-term blood glucose concentrations this test is an effective indicator of long term hyperglycemic status. The method is slightly less accurate than a fasting blood glucose level or a full 3-sample oral glucose challenge in diagnosing new diabetics but has been found to be highly effective in population screening surveys and as an indicator of diabetic control (Ferrell et al. 1984; Modan et al 1984). A cut-off point of 8% is both the most sensitive and specific (87%) indicator for separating normals and diabetics in a population screening survey (Ferrell et al. 1984) and is the value that was used in this study.

Testing for Hb-A1 concentrations revealed that seven

of those who were selected as controls had Hb-A1 results over 8% and were considered to be hyperglycemic. These seven were subsequently removed from the analysis phase of the study and have been notified of the results.

#### e. Statistical Methods

All analyses were done using the SPSS-X program package (SPSS Inc. 1986). Unless where specified, men and women were analyzed separately using standard statistical methods.

Skinfold measurements, circumference ratios and the body mass index (BMI) are the basic measurement units that have been used in the process of determining differences in body fat patterning between the diabetic and non-diabetic participants.

The body mass index is a highly reliable and non-invasive method of determining total body fat (NDDG 1979; Sims 1982). This calculation of weight/height<sup>2</sup> (kilograms divided by metres squared) generates an index that meets the criteria necessary to estimate overall body fatness in adults. The BMI is independent of height yet sensitive to changes in weight and has the highest correlation with both skinfold thickness and body density (Khosla and Lowe 1967; NDDG 1979; Cronk and Roche 1982; Bouchard 1985). The National Diabetes Data Group (1979) recommended definition of obesity (BMI >27 for men and >25 for women) has been used for this study.

The body mass index however is not sensitive to the location of adipose tissue. The direct measurement of skin-folds on the other hand is a non-invasive procedure that can be used to determine the pattern of body fat distribution with a high degree of accuracy and only a small degree of technical error (overall reproducibility coefficient of variation is about 15%, smallest for the triceps and largest for the medial calf) (Bouchard 1985). The measurements can be compared either directly with a comparison of triceps to subscapular measurements, or indirectly using higher levels of analysis (Durnin & Wormsley 1974; Cronk et al 1982; Damon and Goldman 1964; Leonhardt et al. 1978).

In this study three basic techniques (discussed below) have been used to test the stated hypothesis: NIDDM is positively correlated with a centripetal distribution of body fat in the Mohawks. All three methods, despite differences in technique, population size and ethnic origin have shown a significant and positive association between a centripetal fat distribution pattern and the prevalence of NIDDM. I have chosen to analyze these data by each of the following methods in an effort to best detect differences between the diabetic and non-diabetic groups and to compare my findings with those of the other researchers.

Univariate analyses include the comparison of (1) skin-fold ratios and then, (2) girth ratios based on work done by

Feldman et al. (1969) and Hartz et al. (1984), respectively. Feldman et al. (1969) used both direct skinfold measurements and a comparison of triceps and subscapular ratios to nine other skinfold sites to estimate the pattern of subcutaneous fat distribution. In the work done by Hartz et al. (1984) a simple waist to hip girth ratio provided an indicator of disease prevalence based on body fat location in a group of Caucasian women.

Prior to the actual data analysis the distribution of each skinfold in the population was examined and it was found that most departed significantly from a normal pattern of distribution (norm of test:  $p < 0.05$ ) and were positively skewed. Each measurement was then normalized with the log (base 10) transformation and analyses were performed on the transformed data.

The Mann-Whitney U test was used in all of the univariate comparative analyses of the diabetic and non-diabetic subgroups. The Mann-Whitney statistic is non-parametric and can be used on variables that are not normally distributed. The baseline significance level employed in all of the calculations was  $p < 0.05$ .

A multivariate model utilizing (3) principal components analysis of the skinfold data provides an estimation of relative fat patterning that, unlike ratio comparisons, is independent of overall fatness (Mueller and Reid 1979; Szathmary and Holt 1983). Principal components analysis is based

on a partial correlation model whereby a set of correlated original variables (in this case, the skinfold data) are replaced by a set of uncorrelated variables that are linear transformations of the original data. The extent to which a single principal component can explain most of the variance is dependent upon the degree to which the  $n$  original variables were correlated. Subsequent components are independent of the other and each component explains less of the variance than the one preceding it. Using an unrotated matrix preserves the independence of each component which is essential in the identification of patterns typical of the type of analysis done in fat patterning research (Rummel 1970; Mueller and Reid 1979; Szathmary and Holt 1983).

In skinfold analysis there is a highly positive loading on the first component indicating that it explains much of the variance. Mueller and Wohlleb suggest that this first component reflects overall fatness and have termed it the "index of obesity" (1980). Genetic and environmental factors that affect the all the sites uniformly are included in the first component (Mueller and Reid 1979). With an unrotated matrix the general effect of overall fat is then removed from the subsequent component scores. The degree and direction of the loading of the matrix on the subsequent components will explain differences attributable to the actual distribution of the subcutaneous adipose tissue (Mueller and Reid 1979).

Principal components analysis using the transformed skinfold data was carried out separately for each of the four male and female diabetic and non-diabetic sub-groups. Hierarchical analysis of variance (ANOVA) was then performed on the resultant factor scores of the entire sample population. Using sex and then diabetic status as dependent variables removes the effect of sex from the resultant analysis score. Only principal components 1 (fatness) and 2 (centripetality) were employed in this test. The results justifying the use of these two components are given in Chapter 3, section k.

### Chapter Three: Results

The results of the data analysis are presented in this chapter. Included is a description of the sample population which provides an overview (not representative) of the larger group from which the participants were drawn. Next is a review of the clinical characteristics of the diabetic subgroup and then a comparative examination of the diabetic and non-diabetic participants. Immediately following is the analysis of the anthropometric data in the context of the current knowledge about both fat patterning and non-insulin dependent diabetes mellitus (NIDDM) among Native North Americans. As well there is a presentation of the results of the genetic marker analysis illustrating the degree of Caucasian admixture in the diabetic versus the non-diabetic population.

#### **i. Demographic Characteristics of the Sample Population**

##### **a. Participation in the Study and an Estimation of NIDDM Prevalence**

As of December of 1984 the total population of Kahnawake, Quebec was 5,409. Of this number 1,036 were born prior 1949, constituting the adult population aged thirty-five and over. The combined total of 204 participants in the study (excluding the newly found hyperglycemics) represent approxi-

mately 20% of the adult population of Kahnawake.

An estimation of the prevalence of NIDDM among those in whom the disease has been diagnosed can be calculated from the census figures and the known number of diabetics in the community. That is, 132 of 1036 individuals have been diagnosed as having NIDDM as of July 1985 giving a prevalence of 12.7 per 100.

Table I (page 18) outlines the number of subjects in comparison to the estimated goal. As explained in the methods chapter the number of known diabetics provided the target number of participants in each of the sex and decade age range categories. In all of the diabetic sub-groups regardless of age or sex the target number was either met or exceeded (Table I).

Participation among the non-diabetic controls was generally high although there is a degree of self-selection bias inherent to the recruitment method used (94 of 261 recruited actually participated). That is, although participants were selected at random from the band list (see Chapter 2 for details), those who chose not to participate were replaced by the next individual on the band list who met the study criteria. It is unlikely though that the rationale for participation was based upon either diabetes or, more importantly, issues surrounding obesity since all participants were curious about the details of the study at the time of their interviews. The voluntary participation by radio recruitment

of 28 non-diabetics (23% of the control group) would not substantially alter the findings since there already was a degree of self-selection inherent to this study. It is only in the 56-65 year old age range for both men and women in which under-representation (based upon the targeted figures) is evident. Of a possible twenty-seven, only 21 women in this middle age range were successfully recruited. Among the men, just over half of the target number participated (Table I).

All participants in the study met the designated entrance criteria. All are reserve-dwelling adult Mohawk Indians who use (or have used) the KMHC clinic services. Specific data about level of income and education or socio-economic status was not collected.

#### b. Employment Status of the Sample Population

The types of employment held by the study participants reflect the urban nature of the reserve (Table II). Among the men, for example, jobs range from construction and maintenance to educator and architect. All of the men who were interviewed stated that they are currently or have been employed.

Structural steelwork remains a viable profession among those who are working (11.8% of the diabetic and 15.2% of the non-diabetic men). Indeed the actual number of individuals

-TABLE II-  
Occupation of Male and Female Mohawk Participants  
by Diabetic Status

Occupation:	Frequency (percent)	
	Diabetic	Non-Diabetic
<b>MALES:</b>		
	N=34	N=46
Never worked	00	00
On disability	6 (17.6)	4 (8.7)
Steelworker	4 (11.8)	7 (15.2)
Retired	13 (38.2)	14 (30.4)
Hospital worker	00	1 (2.2)
Management-Admin.	00	1 (2.2)
Construction	2 (5.9)	5 (10.9)
Maintenance-mover	5 (14.7)	8 (17.4)
Educator	1 (2.9)	00
Farmer	1 (2.9)	00
Gov't-postal	00	1 (2.2)
Social worker	1 (2.9)	1 (2.2)
Police	1 (2.9)	1 (2.2)
Store-restaurant	00	2 (4.3)
Engineer-architect	00	1 (2.2)
<b>FEMALES:</b>		
	N=48	N=76
Never worked	38 (79.2)	48 (63.2)
On disability	1 (2.1)	00
Retired	2 (4.2)	9 (11.8)
Secretarial	00	4 (5.3)
Hospital worker	2 (4.2)	3 (3.9)
Management-Admin.	00	1 (1.3)
Band councillor	00	1 (1.3)
Maintenance-mover	1 (2.1)	2 (2.6)
Educator	00	2 (2.6)
Gov't-postal	00	2 (2.6)
Social worker	00	3 (3.9)
Factory worker	2 (4.2)	00
Store-restaurant	2 (4.2)	1 (1.3)

who have worked in the high steel industry would be much higher if all of those who are disabled and/or retired were included in the figures. It is interesting to note that, with very few exceptions, all those who are not currently employed in the steel industry work locally on the reserve.

There are few employment differences between diabetic and non-diabetic men. The only real disparity between the groups is that there is a higher percentage of diabetic men who are on disability. This figure may reflect the generally poorer health status of the diabetic men (Section ii of this chapter).

Table II also reveals that the women of this study do not make up a large portion of the labour force. Although 65% of those who participated are over 55 years of age, 79.2% of the diabetic sub-group and 63.2% of the non-diabetic sub-group of women stated that they have never been employed. Most of these women described their role as that of a homemaker although they did not acknowledge this as a "job".

Except for two women who are employed as factory workers all of those who do work outside of the home are, like the men, employed on the reserve. Type of work for these women includes hospital employee as well as secretarial, management and administrative positions (Table II).

A comparison by diabetic status reveals little information relevant to this study. Of interest though is that the non-diabetic women are employed in a broader range of

jobs than those who are diabetic and only 1 (2.1%) of the diabetic women is currently receiving disability insurance. This latter finding does not necessarily reflect a "healthier" profile of the diabetic women of Kahnawake. It is more likely an indication that the men, because of their jobs, are able to collect compensatory wages for their illnesses and can thus be considered as being on disability.

#### c. Civil Status of the Sample Population

Most of the participants are or have at one time been married (Table III). Although it appears that a higher percentage of the men in this sample are married, the figures are more likely an indication of the high number of widowed women in this community (29.2% of the diabetic and 34.2% of the non-diabetic women).

Few of those seen as a part of this study are either single or divorced. Overall, only 14% of the entire sample population falls into either of these categories.

#### ii. An Overview of the Known Mohawk Diabetics

##### a. Duration of NIDDM and Mean Age of the Diabetic Population

Summarized in Table IV are the clinical characteristics of the known diabetics who participated in this study. The

-TABLE III-  
Civil Status of Mohawk  
Participants by Diabetic Status and Sex

Civil Status:	Frequency (percent)			
	Diabetic		Non-Diabetic	
	Male	Female	Male	Female
Married	25 (73.5)	28 (58.3)	39 (84.8)	39 (51.3)
Widowed	4 (11.8)	14 (29.2)	2 (4.3)	26 (34.2)
Divorced	1 (2.9)	3 (6.3)	3 (6.5)	6 (7.9)
Single	4 (11.8)	3 (6.3)	2 (4.3)	5 (6.6)

-TABLE IV-  
Clinical Characteristics of the Mohawk  
Male and Female Diabetic Participants

	Male N=34	Female N=48
Mean Age (years)	58.9	60.2
Mean Duration of Diabetes, years#	7.1	5.8
± Standard Deviation	4.15	4.71
Range	1 - 14	1 - 25
Mean Age at Diagnosis, years#	51.6	54.1
± Standard Deviation	10.74	10.67
Range	32 - 87	33 - 78
Symptoms at Diagnosis# (frequency and percent)		
Yes*	10 (41.7)	16 (45.7)
No	14 (58.3)	19 (54.3)
Current Therapy# (frequency and percent)		
Diet	8 (23.5)	15 (31.3)
Oral Hypoglycemics	5 (14.7)	12 (25.0)
Insulin	19 (55.9)	21 (43.8)
Vascular Complications# (frequency and percent)		
Retinopathy	8 (26.7)	3 (8.1)
Neuropathy	4 (11.8)	00
Nephropathy	2 (6.1)	1 (2.1)
Peripheral Vascular Disease	4 (11.8)	6 (12.5)

# Male : Duration Diabetes: 1 missing observation  
 Current therapy: 2 m.o.  
 Retinopathy: 4 m.o.  
 Nephropathy: 1 m.o.  
 Symptoms: 10 m.o.

Female: Duration of Diabetes: 3 m.o.  
 Age at Diagnosis: 3 m.o.  
 Retinopathy: 11 m.o.  
 Symptoms: 13 m.o.

\* Symptoms include any or all of the following: weight loss, polyuria, polydipsia and/or fatigue.

mean age of the diabetic men is 58.9 years and of the diabetic women, 60.2 years. Duration of disease from time of diagnosis to the time of the study ranges from 1 to 14 years in the men and from 1 to 25 years in the women. The average length of time with NIDDM is somewhat higher in men than in women (7.1 and 5.8 years, respectively). As with any duration figures though these may be more of a reflection of the time of diagnosis rather than the natural history of disease.

As would be expected, in all decades except one, the duration of NIDDM in this population increases with increasing age (Table V). The mean number of years from time of diagnosis to the study period ranges from 4.67 to 8.5 years in men and from 4.2 to 9.0 years in women. Only in the 56-65 year old women does the duration fall to 4.25 years and then rise up to 8.33 years in the next decade. One possible explanation for this drop and then sharp rise in the duration figure in the older women may be that fewer of those with diabetes survive past the age of fifty-five. Those who do live beyond the fifth decade may be individuals who experience fewer complications or illnesses associated with NIDDM and thus are able to live substantially longer lives.

Mean age at time of diagnosis of NIDDM is similar in the two groups (Table IV). In women the mean age at time of diagnosis is 54.1 years and in men it is only slightly lower at 51.6 years. The range of ages when diabetes was diagnosed

-TABLE V-  
Duration of Diabetes, in Years from Time of  
Diagnosis, of Male and Female Mohawk Participants

Age Range:	Years ( $\pm$ Standard Deviation)	
	Males N=33#	Females N=45
36-45	4.67 (4.04)	4.20 (1.92)
46-55	6.67 (3.87)	5.20 (4.32)
56-65	7.46 (4.75)	4.25 (2.98)
66-75	7.50 (2.88)	8.33 (4.06)
76-90	8.50 (7.78)	9.00 (9.57)
Total:	7.06 (4.15)	5.80 (4.71)

# 1 missing value

are from 32 to 87 in the men and from 33 to 78 in the women.

b. Symptoms at Diagnosis and Current Therapy

Just over one half of all the known diabetics were asymptomatic at the time of diagnosis (58.3% men and 54.3% of the women; Table IV). That is, of those whose records stated the individual's status at the time of diagnosis, the disease was only discovered following a blood or urine analysis that was either routinely ordered or requested because of a suspicion by the physician of NIDDM (for example, chronic yeast infection in women). The remaining 41.7% of the men and 45.7% of the women had one or more of the known recognized symptomatology (weight loss, polyuria and/or polydipsia).

One quarter of the diabetic women and 14.7% of the diabetic men are currently taking supplementary oral hypoglycemics as part of their therapeutic regimen (Table IV). Control of dietary intake alone is the form of therapy prescribed for 23.5% of the men and 31.3% of the women. Fifty-five percent of the men and 43.8% of the women are currently managing their diabetes with injected insulin. One reserve physician acknowledged that those on insulin have episodes of uncontrolled hyperglycemia (without ketosis) for which insulin is felt to be the only effective treatment (Macaulay 1986; personal communication).

### c. Vascular Complications

Mohawk diabetics, according to the findings of this study, are experiencing the full range of vascular complications associated with diabetes. Presented here is a synopsis of the complications diagnosed among those who participated in this study. A more thorough analysis of the vascular complications of NIDDM among the Mohawk diabetics is forthcoming (see Note 1).

Diabetic retinopathy has been diagnosed in 8 of the 34 males (26.7%) and in 3 of the 48 women (8.1%). Only 2 (6.1%) of the women and 1 (2.1%) of the men has been diagnosed positively for kidney disease (Table IV). Peripheral vascular disease is at present slightly more common in women than in men. Six or 12.5% of the women and 4 or 11.8% of the men have been positively diagnosed as having peripheral vascular complications of NIDDM. Neuropathologic complications have not been diagnosed in any of the women seen although they have been found in 4 (11.8%) of the men (Table IV).

Occurrence of ischemic heart disease, smoking and hypertension are included in Tables VI and VII for reference only and will also be included in the adjunct complications study.



-TABLE VII-  
A Comparison of Anthropometric Variables, Familial  
 Occurrence Patterns and Clinical Observations  
 of the Mohawk Diabetic and Non-Diabetic Women

	Diabetic N=48	Non-diabetic N=76
Mean BMI	33.40§	29.01
Mean BMI at 18 years#	22.96	21.37
Mean Weight, kg.	81.89§	70.91
Mean Weight at 18 years, kg. #	56.65	52.21
Mean Waist to Hip Ratio	0.86	0.85
Hb-A1		
Mean	7.23§	5.28
Range	3.69 - 12.91	3.10 - 7.50
Parity, mean ( $\pm$ SDM)	4.68 (3.29)	4.05 (3.25)
Frequency (percent) :		
Diabetic mother	12 (25.0)	21 (27.6)
Diabetic father	5 (10.4)	9 (11.8)
Siblings with diabetes	22 (45.8)§	13 (17.1)
Spouse with diabetes ##	8 (16.7)	14 (18.0)
Children with diabetes	6 (12.5)	5 (6.6)
Smokers	13 (27.1)	23 (30.3)
Hypertension	36 (75.0)§	37 (48.7)
Hypercholesterolemia	9 (18.8)	11 (14.5)
Ischemic Heart Disease	26 (54.2)	14 (18.4)
Cholelithiasis	22 (45.8)	24 (31.6)

# Mean weight: diabetic: 4 missing observations; non-diabetic: 2 m.o.

## Percentage is of those with spouses only

Mann-Whitney U test significant at: \* p < 0.05  
 § p < 0.005

### iii. Comparison of Diabetic and Non-Diabetic Mohawk Indians

#### a. NIDDM in First Degree Relatives and Parity

Among the Mohawk, the diabetic status of the parent has no significant effect in the occurrence of NIDDM in the offspring (Tables VI and VII). In both sexes and in all of the disease status sub-groups there is a comparable percentage of parents with NIDDM.

The number of diabetics who have siblings with the same diagnosis however is significantly greater than non-diabetics with diabetic sibs. Just over thirty-five percent of the male diabetics stated that they had at least one sibling with NIDDM while only 15.2% of the non-diabetic men have siblings with diabetes (Table VI).

The difference between diabetic and non-diabetic women who reported having siblings with NIDDM is even greater than that which was found between the men. Nearly forty-nine percent of the diabetic women seen have one or more siblings with diabetes in contrast to 17.1% of the non-diabetics who have sibs with the same diagnosis. This difference was significant at  $p < 0.005$  (Table VII).

Although there is a slightly lower percentage of women with diabetic husbands (16.7% and 18% in the diabetics and non-diabetics) there is twice the percentage of diabetic

women with children who have been diagnosed with NIDDM (12.5% of the diabetics and 6.6 % of the non-diabetics). None of the male participants reported a diagnosis of NIDDM in their children although both groups reported having spouses with NIDDM (8.8% and 10.4%, respectively). The findings of this study reveal that among the Mohawk Indians of Kahnawake there is a greater intra- rather than inter-generational effect on the development of NIDDM upon the manifestation of glucose intolerance.

There was no significant correlation found between parity (number of pregnancies) and occurrence of diabetes in the women who participated in this study. These findings are in concordance with other recent studies wherein parity is thought to have a minor, if any, effect as a causal agent in NIDDM (Zimmet 1982).

#### b. Gallbladder Disease

Cholelithiasis was found to be present in almost one half of the diabetic (45.8%) and in about one third of the non-diabetic (31.6%) women (Table VII). Although the difference between the women was not statistically significant, that nearly one half of all the diabetic women have gallstones is of clinical importance and in combination with the diabetes may indeed be indicative of the susceptibility of these women to this array of metabolic and digestive disorders mentioned above.

Fewer of the Mohawk men, as in other populations, experience diseases of the gallbladder. Only 17.6% of the diabetic and 10.9% of the non-diabetic men had diagnoses of cholelithiasis. The difference between these figures is not significant (Table VI).

These findings are in concordance with the recognized pattern of occurrence of gallbladder disease (GBD); that is, GBD is more prevalent among women than among men, among diabetics than among non-diabetics and occurs at a higher rate in Amerindians than in Caucasians (Snodgrass et al. 1974; Sievers and Fischer 1981).

#### iv. Anthropometry

The following data and analysis provide the basis for testing the hypothesis central to this thesis; that is, whether there is a correlation between a central distribution of body fat and NIDDM in the adult population of Kahnawake, Quebec. Three methods have been employed for this purpose: two are based upon the skinfold data and the third upon the circumference measurements.

##### a. Body Mass Index

Body mass index (BMI) broken down by sex, diabetic status and age range is presented in Table VIII. The body mass

index, a highly reliable determination of weight for stature (Bouchard 1985), is derived from the calculation of weight, in kilograms, divided by stature, in metres<sup>2</sup> (the weight and height data are included in Appendix B: Tables 1 & 2).

The National Diabetes Data Group (1979) suggested that the criterion for obesity be sex-specific. Thus a BMI greater than 25 kg/m<sup>2</sup> for women and greater than 27 kg/m<sup>2</sup> for men would be considered obese. According to these criteria, the group means of Mohawks in both sexes fall above the NDDG standards and would thus be considered obese. All age range sub-group BMI means except two are above the recommended upper limit criterion for obesity. The two exceptions are the non-diabetic men in the 66-75 year old age range and the eldest non-diabetic women with mean BMIs of 26.84 and 24.46, respectively (Table VIII). These values fall just short of the stated criteria.

Notwithstanding the overall tendency to higher BMIs, there are clear and statistically significant differences between those with and without diabetes. Both in the men and in the women at all ages, those with NIDDM have significantly greater BMIs than those without the disease (Tables VI and VII).

Calculating the percentage of individuals that fall within each of the BMI ranges by sex and diabetic status (Table VIII) provides an indication of the proportionate distribution of BMI values in this study population. The three

-TABLE VIII-  
Body Mass Index, in Kg/M<sup>2</sup>,  
by Age Range, Diabetic Status, and Sex

Age Range:	Mean ( $\pm$ Standard Deviation)			
	Male		Female	
	Diabetic N=33#	Non-Diabetic N=46	Diabetic N=48	Non-Diabetic N=76
35-45	30.38 (2.63)	27.53 (5.41)	34.94 (6.73)	28.73 (5.69)
46-55	31.56 (4.50)	29.59 (4.12)	40.80 (6.93)	28.74 (4.60)
56-65	29.96 (3.32)	29.03 (4.30)	32.99 (6.24)	29.12 (4.11)
66-75	29.50 (4.07)	26.84 (3.00)	29.35 (3.22)	31.43 (5.07)
76-90	30.48 (0.54)	29.75 (8.94)	25.01 (4.86)	24.46 (3.66)
Column	30.38	28.69	33.39	29.00
Total	(3.58)	(4.84)	(7.45)	(4.96)

# 1 missing observation

-TABLE IX-  
Proportion of Individuals Within Each Designated  
BMI Range By Sex and Diabetic Status

BMI Range:	Frequency (Percent)			
	Male		Female	
	Diabetic N=33#	Non-Diabetic N=45#	Diabetic N=48	Non-Diabetic N=76
</= 26.00	3 (9.1)	11 (24.4)	5 (10.5)	24 (31.5)
26.01 - 29	9 (27.3)	16 (35.6)	10 (20.8)	17 (22.4)
>/= 29.01	21 (63.6)	18 (40.0)	33 (68.7)	35 (46.1)

BMI categories are all within or above the NDDG stated categories of obesity for either sex but correspond to the given BMIs for this population. As indicated in Table IX, 63.6% of the male diabetics fall into the highest BMI range ( $\geq 29$  kg/m<sup>2</sup>), whereas just 40% of the non-diabetics are in this category. Only 9% of the diabetic men have BMIs under 26 kg/m<sup>2</sup> yet one quarter of the non-diabetic men (24.4%) fall into this BMI range. Among the women, as in the case of the men, there is an overall greater percentage of women in the highest BMI category. Sixty-nine percent of the diabetic women and 46 percent of the non-diabetic women (still a high percentage and more than the men in the same category) fall into the highest range of BMI values. Only 10.5% of the diabetic women have BMIs at or under 26 kg/m<sup>2</sup> compared to the 31.5% of the non-diabetic women in the same category.

There is what can only be considered a trend toward greater weight for height in the diabetic participants beginning in early adulthood if not sooner. The mean BMI at 18 years (calculated as: recall weight, in kilograms/current height, in metres<sup>2</sup>) is higher in the diabetic men and women (Tables VI and VII, respectively) than in their non-diabetic controls although not significantly so.

#### b. Skinfold Measurements

Comparison between untransformed skinfold means are

presented in Table X as an indication of the sex and age differences in the skinfold measurements between the diabetic and non-diabetic participants. Means of eight skinfold measurements are shown.

The raw means at each of the eight skinfold sites are similar irrespective of diabetic status among the male participants (Table X). In the women, on the other hand, the differences in the skinfold means are more apparent between those with and without NIDDM. The forearm and triceps sites are significantly larger in the diabetic women. The mean biceps and subscapular measurements also appear greater in the diabetic women, although the difference is not significant. The medial calf mean measurements are significantly smaller in the diabetic sample of women when compared to their non-diabetic controls. Comparatively smaller values were also found at the midaxillary and suprilliac sites although the differences are not significant. The mean abdominal skinfold measurement on the other hand is exactly the same in both diabetic and non-diabetic Mohawk women.

The triceps and subscapular skinfold measurements are reported to be useful indicators of both the proportionate location of subcutaneous body fat and overall proportion of body fat (Damon and Goldman 1964). These data are included in order to provide an indication of the difference in the distribution of subcutaneous fat over the age ranges within each of the sex and disease status categories (Appendix B:

-TABLE X-  
Skinfold Measurements, in Millimetres,  
by Diabetic Status and Sex

Skinfold:	Mean ( $\pm$ Standard Deviation)							
	Male				Female			
	Diabetic N=34		Non-Diabetic N=46		Diabetic N=48		Non-Diabetic N=76	
Forearm	6.9	(4.8)	7.8	(4.3)	16.9	(8.6)**	13.3	(6.4)
Triceps	16.7	(7.0)	16.2	(9.0)	36.2	(12.4)*	31.0	(9.3)
Biceps	10.9	(5.4)	10.8	(6.8)	27.2	(12.9)	23.1	(10.8)
Subscapular	30.0	(10.3)	26.5	(10.0)	39.8	(13.5)	36.4	(12.8)
Midaxillary	30.6	(10.7)	34.2	(13.8)	35.3	(11.7)	37.0	(11.2)
Suprailiac	34.7	(12.8)	38.9	(14.0)	41.4	(15.2)	44.6	(12.6)
Abdominal	43.1	(11.8)	42.3	(13.3)	50.6	(13.1)	50.6	(12.5)
Medial Calf	8.6	(5.1)	10.8	(7.1)	16.0	(11.3)**	19.7	(8.8)

Mann-Whitney U test significant at: \* p < 0.05  
 \*\* P < 0.02

Tables 3 and 4). Regression by age or duration of NIDDM on the values however were not performed nor were significance tests done between the diabetics and their controls in the indicated age ranges. The observations are described briefly for heuristic purposes only.

Differences that were not discernible in the mean skinfold values are more apparent when the results are broken down by age range. There is an overall increase, for example, in the triceps skinfold measurements with increasing age in the non-diabetic men but not in the diabetic men. Diabetic male triceps mean measurements are greater than the non-diabetic values in only the two youngest age groups (Appendix B: Table 3).

The breakdown by age range of the subscapular skinfold values reveals a somewhat different trend. The subscapular measurements tend to at first increase and then decrease in the older age groups in the diabetic men. In the non-diabetic men the mean subscapular measurement is lowest in the youngest age range and then increases yet varies very little in the next four age groups (Appendix B: Table 4).

Among the women, the triceps values are greater in the diabetic than in their non-diabetic controls in the first three age ranges (Appendix C: Table 3). The mean values then decrease with increasing age such that the smallest triceps skinfold mean is in the eldest of both diabetic and non-diabetic women.

Subscapular values broken down by age range reveals a higher mean measurement in all except one of the age ranges among the diabetic women (Appendix B: Table 4). As well there is a general overall decrease in the subscapular value with age in the diabetic women. There is a substantial decrease in the subscapular skinfold mean value only in the eldest age range of the non-diabetic female participants.

### c. Skinfold Ratios

All subsequent analyses and comparisons between the diabetic and non-diabetic participants are done using log transformed skinfold data (see Chapter 2:c).

In skinfold ratio data the assumption is that in those with a trunkal patterning of body fat an extremity skinfold will have a lesser value than that of a more proximal site (Feldman et al. 1969). Thus for the skinfold to triceps ratio, as the proximal skinfold increases the numerator gets larger and the resulting ratio will increase above one. The same assumption holds for the skinfold to subscapular ratio but in the opposite direction. If there is a tendency toward trunkal fat patterning the extremity measurements will be less than the constant denominator (that is, the subscapular skinfold measurement) and the ratio will decrease away from one.

The Mohawk data were analyzed by methods similar to

those of Feldman et al. (1969). The mean values of the log transformed data for each of the eight skinfold sites was first compared to the triceps and then to the subscapular measurements in the male and female subgroups.

#### d. Skinfold Ratios: Male Data

A comparison of the skinfold to triceps ratios of the male participants is presented in tabular (Table XI) and graphic form (Figure 1). The four central body skinfold measurements (subscapular, suprailliac, midaxillary and abdominal) to the triceps skinfold in both the diabetic and non-diabetic Mohawk men reflect a male pattern obesity consonant with the high BMIs but unexpected according to the stated hypothesis. In fact, contrary to the expected results, the ratios appear greater in the non-diabetic men although there are no significant differences between the groups.

Significant differences were found in the forearm and medial calf to triceps ratios (Table XI). In both cases the non-diabetic value is greater than that of the diabetic. These results suggest that there is a lower proportion of body fat at the more distal sites in the diabetic men.

Skinfold to subscapular ratios in the male participants decrease below one in the ratio calculation at each of the extremity sites (forearm, biceps, triceps and medial calf) (Table XII). The values differ significantly at the forearm and medial calf sites whereas the biceps and triceps to

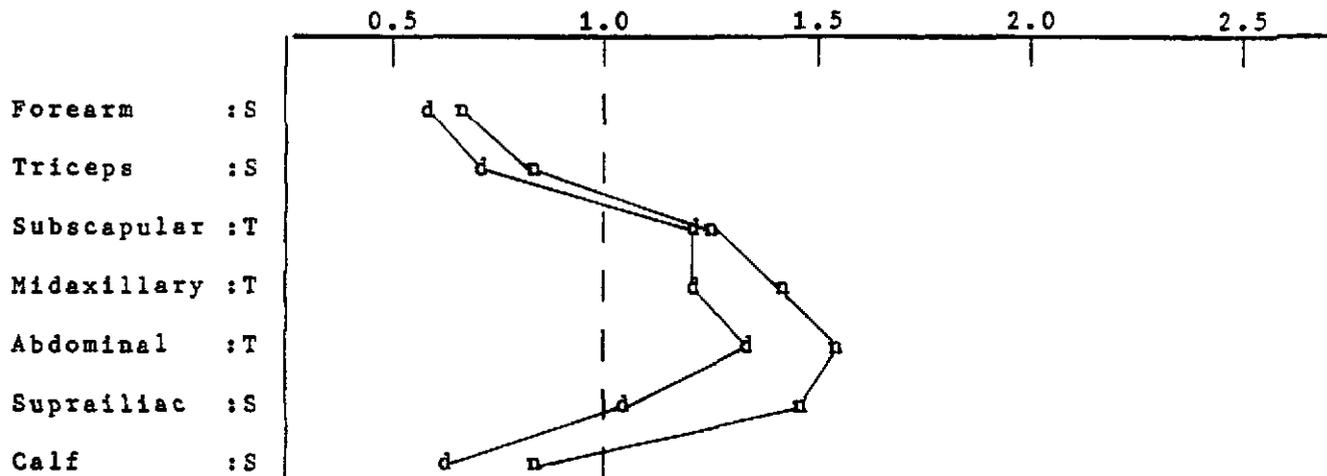
-TABLE XI-  
A Comparison of Mohawk Male Diabetic and Non-Diabetic  
Skinfold to Triceps Ratios

Mean ( $\pm$ Standard Deviation)		
Skinfold/Triceps#:	Diabetic	Non-Diabetic
Forearm	0.67 (0.14)**	0.76 (0.20)
Biceps	0.83 (0.22)	0.85 (0.15)
Subscapular	1.24 (0.14)	1.26 (0.30)
Suprailiac	1.28 (0.16)	1.40 (0.29)
Midaxillary	1.24 (0.17)	1.35 (0.30)
Abdominal	1.37 (0.16)	1.46 (0.33)
Medial Calf	0.74 (0.28)**	0.85 (0.16)

#Log transformed values

Mann-Whitney U test significant at: \*\*  $p < 0.02$

- FIGURE 1 -  
Comparison of Male Mohawk Diabetic and Non-Diabetic  
Fat Distribution by Selected Mean  
Triceps (T) and Subscapular (S) Ratios#



Key:

d = Diabetic

n = Non-Diabetic

-TABLE XII-  
A Comparison of Mohawk Male Diabetic and Non-Diabetic  
Skinfold to Subscapular Ratios

Mean ( $\pm$ Standard Deviation)		
Skinfold/Subscapular#:	Diabetic	Non-Diabetic
Forearm	0.55 (0.12)§	0.61 (0.10)
Biceps	0.68 (0.15)	0.70 (0.13)
Triceps	0.82 (0.08)	0.83 (0.16)
Suprailiac	1.04 (0.09)§	1.12 (0.09)
Midaxillary	1.01 (0.11)§	1.08 (0.09)
Abdominal	1.12 (0.10)	1.17 (0.12)
Medial Calf	0.59 (0.18)**	0.70 (0.17)

#Log transformed values

Mann-Whitney U test significant at: \* p < 0.05  
 \*\* p < 0.02  
 § p < 0.005

subscapular ratios are only slightly less in the diabetic men. The lower ratio values at the forearm and medial calf sites, as in the triceps ratios, follow the expected pattern. Significance was also reached in the suprailiac and mid-axillary to subscapular ratios. However here, as in the triceps figures, the diabetic ratios are less than those of the non-diabetic controls. One explanation for these findings might be that in those with NIDDM there is a greater deposition of fat in the subscapular region than in other trunkal sites although, as is indicated in Table X, there is no significant difference between diabetic and non-diabetic subscapular skinfold measurements.

Following the format of Feldman et al. (1969), I have plotted a selection of the skinfold to tricep and subscapular ratios which best provide a graphic representation of the difference between the diabetic and non-diabetic groups vis a vis trunkal fat patterning (Figure 1). Figure 1 clearly depicts what is described above. The non-diabetic men appear to have greater trunkal skinfold to triceps ratios than the diabetic men although the diabetic men have less subcutaneous fat at the distal extremity to subscapular sites.

The comparison of skinfold ratios in the diabetic versus the non-diabetic Mohawk men reveals two important findings. The diabetic men have significantly less subcutaneous fat in the more distal regions (medial calf and forearm) than the non-diabetic controls. However, contrary to the

suggested hypothesis the diabetic men do not have a greater degree of adipose tissue in the trunkal region than their non-diabetic controls.

e. Skinfold Ratios: Female Data

The comparison of skinfold to triceps ratios (Table XIII) indicates that among the women there is a tendency toward trunkal fat patterning independent of diabetic status. Values greater than or just under (0.99) one are reached at each of the four proximal sites (subscapular, suprailiac, midaxillary, abdominal) in both the diabetic and the non-diabetic women. Significant differences between the ratios were reached at the midaxillary and the abdominal sites although in the direction of the non-diabetic sub-group. As in the male data however, the medial calf to subscapular ratio in the diabetic subgroup is significantly less than that of the non-diabetic. This finding is indicative of proportionately less body fat in the distal and lower extremity of the diabetic Mohawk women.

Skinfold to subscapular ratios (Table XIV) confirm the significantly lesser amount of body fat at the medial calf site in the diabetic women. Other distal site ratio values provide little supportive evidence of a trunkal patterning in the diabetic women. For example, the triceps to subscapular ratio, according to Feldman and associates (1969), should



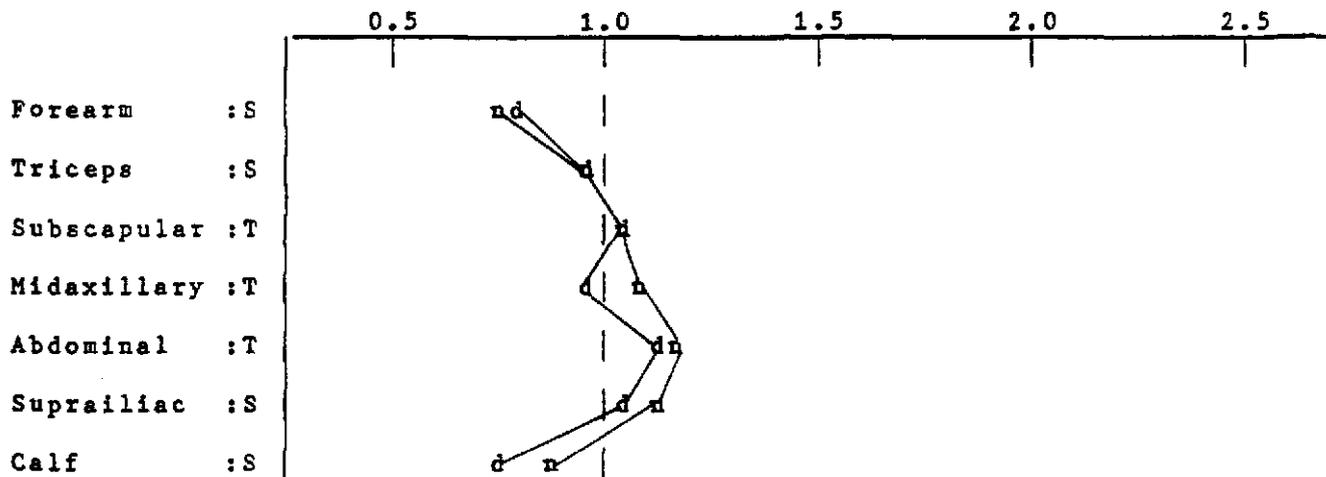


provide a clear indication of the difference between trunkal and distal body fat deposition. In this case however the results are very similar and close to one in both of the female sub-groups (diabetic: 0.98, non-diabetic: 0.97). Factors that may account for this are the following: it may be that the predominance of very high BMIs in the diabetic as well as the non-diabetic groups is confounding the analysis of the female data. In addition, the diabetic women are on a controlled dietary regimen and upper body fat is lost more readily than lower body fat (Bjorntorp 1982). Thus the diabetic women, who are at varying stages of disease management, may no longer be displaying an adipose tissue pattern that may have been discernible at the time of diagnosis.

As in the case of the men, selected skinfold to triceps and subscapular ratios have been plotted in order to compare the diabetic and non-diabetic female findings (Figure 2). That there is minimal difference between the diabetic and non-diabetic Mohawk women at any of the skinfold site ratios is clearly illustrated in this diagram.

The third graph compares the non-diabetic Mohawk male ratios to those of the diabetic women in order to discern any "male" pattern in the diabetic women (Figure 3). As illustrated in Figure 3, no such pattern is found in this sample of diabetic women. The trunkal patterning in the non-diabetic men is not replicated in the female Mohawk diabetics.

- FIGURE 2 -  
Comparison of Female Mohawk Diabetic and Non-Diabetic  
Fat Distribution by Selected Mean  
Triceps (T) and Subscapular (S) Ratios#

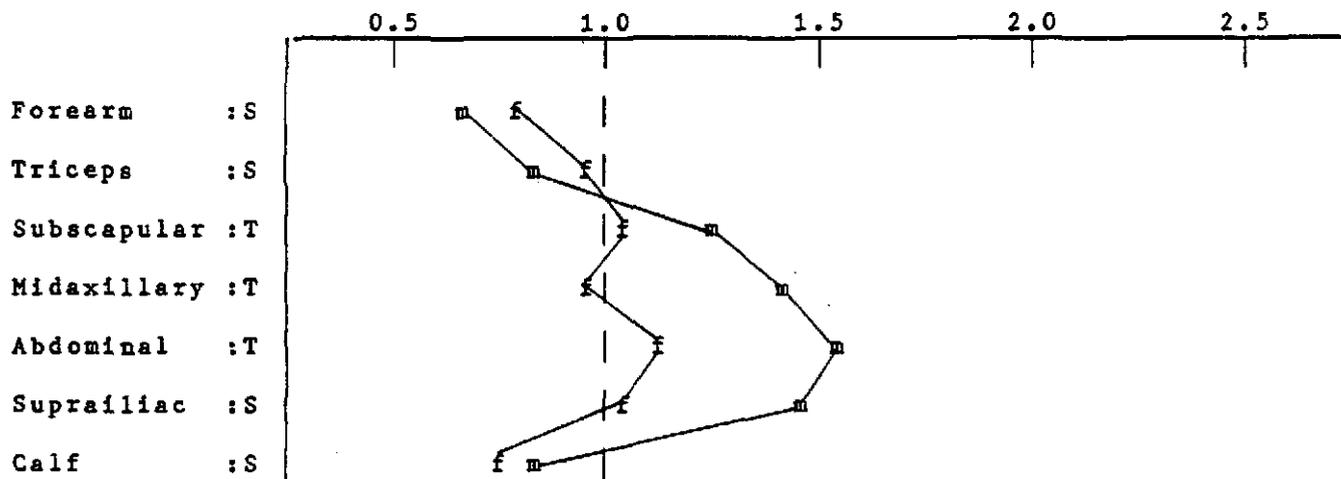


Key:

d = Diabetic

n = Non-Diabetic

- FIGURE 3 -  
Comparison of Mohawk Female Diabetic and Male Non-Diabetic  
Fat Distribution by Selected Mean  
Triceps (T) and Subscapular (S) Ratios#



## Key:

f = Female Diabetic  
m = Male Non-Diabetic

#### f. Circumference and Waist to Hip Ratio

Body girth ratios differ from skinfold ratios in the type of comparative analyses that can be made. Whereas skinfold ratios compare the relative distribution of trunk and extremity subcutaneous fat, body girth ratios contrast lower to upper torso dimensions. As the ratio increases towards or above one, the greater the inference that there is an increase in the proportion of upper to lower body fat distribution.

Hartz et al. (1983) found that the degree of trunkal fat patterning increased with an increase in the degree of obesity. Using a similar type of analysis in this study, waist to hip ratios are broken down within categories of body mass index (non-obese: <26, moderately obese: 26-29 and obese: >29 kg/m<sup>2</sup>; Table XVI and XVII).

#### g. Circumference and Waist to Hip Ratio: Male Data

Circumference measurements were taken of the chest, abdomen, hip, upper arm and medial calf (Table XV). The comparison of circumference means between the diabetic and non-diabetic Mohawk men indicates that the diabetic men have greater mean values at all sites although significance was reached only at the abdomen and hip sites.

The overall abdominal to hip ratio (abdominal girth/hip girth, in millimetres) mean value is included in Table VI



-TABLE XVI-  
A Comparison of Waist to Hip Ratios,  
(Abdominal Girth/Hip Girth, in Millimetres),  
by Age Range, Diabetic Status, and Sex

Age Range:	Mean ( $\pm$ Standard Deviation)			
	Male		Female	
	Diabetic N=34	Non-Diabetic N=46	Diabetic N=48	Non-Diabetic N=76
35-45	0.92 (0.11)	0.94 (0.06)	0.84 (0.05)	0.80 (0.07)
46-55	0.96 (0.18)	0.94 (0.06)	0.88 (0.06)	0.84 (0.07)
56-65	0.94 (0.24)	0.96 (0.06)	0.88 (0.07)	0.87 (0.07)
66-75	0.98 (0.04)	0.96 (0.05)	0.80 (0.13)	0.85 (0.05)
76-90	0.96 (0.05)	0.91 (0.07)	0.81 (0.05)	0.84 (0.05)
Column Total	0.95 (0.18)	0.94 (0.06)	0.86 (0.08)	0.85 (0.07)



(page 45). There is a significant difference between the diabetic and non-diabetic men such that the diabetic men appear to have the expected exaggerated male patterning although the actual ratio values differ only slightly. Breaking down the ratios by age range (Table XVI) reveals that the men generally have a greater waist to hip ratio (that is, all ratios are greater than 0.9) independent of diabetic status and age, although no significance testing was performed between the diabetic men and their controls in the indicated age ranges.

Distinguishing the patterning according to degree of obesity based upon a range of body mass indexes provides a somewhat clearer pattern of fat distribution in the comparison of the diabetic and non-diabetic Mohawk men. The values in Table XVII demonstrate that while there is an increase in the non-diabetic male pattern toward trunkal patterning with increasing obesity, there is a tendency toward that pattern in the diabetic men at all BMI ranges (and despite the paucity of men in the lowest BMI category). That is, although the non-diabetic male tends toward an upper body fat pattern this becomes apparent only at the higher BMI values. In the diabetic men, on the other hand, the tendency to trunkal fat patterning occurs independent of degree of obesity. Thus, there is evidence of exaggerated trunkal fat patterning seen in all of the diabetic men independent of degree of obesity.

I would suggest though that these data are not suffi-

cient evidence of an increased trunkal patterning in the diabetic men because at the higher levels of obesity the data are so similar as to preclude any assumptions about the association between trunkal patterning and diabetes. That is, these findings would suggest that the waist to hip ratio is not a sensitive enough measure to distinguish the obese diabetic from the obese non-diabetic men.

#### h. Circumference and Waist to Hip Ratio: Female Data

Circumference measurement means are significantly greater in the diabetic women at all sites, except the calf, when compared to their non-diabetic controls (Table XVIII). If circumference measurements can be taken as indicators of generalized obesity then these findings support the BMI data (Table VII) which indicate that the diabetic women are significantly more corpulent than the non-diabetic women.

It is expected that the waist to hip ratios will illustrate a clear distinction between the diabetic and non-diabetic women. As was found in the work done by Hartz and associates (1983), there should be a distinguishable difference between those women with and without NIDDM such that those with NIDDM will have a greater waist to hip ratio.

The overall waist to hip ratio mean value (Table VII) reveals that there is little difference between those women with NIDDM and those without the disease (0.86 and 0.85,

respectively). Contrasting the diabetic with the non-diabetic women by age range also provides no clear indication of any greater abdominal patterning in those with NIDDM (Table XVI).

The breakdown of waist to hip ratios by BMI range provides no indication of abdominal patterning in those who are diabetic (Table XVII). Those who fall into the lowest BMI range have waist to hip ratios that are lower than those in the other two categories but, unlike the men, the diabetic women exhibit a lower mean value than the non-diabetic women. All of the women in the other two categories exhibit a proportionately greater degree of upper to lower body fat. Unlike the comparison of the men and unlike the observations of Hartz and co-workers (1983), there is no discernible patterning effect independent of the degree of obesity in diabetic Mohawk women.

The difficulty in determining the effect of centrality at increasing levels of obesity must be considered however as a confounding factor in the analysis of these findings. That is, these data do not separate the effect of generalized obesity from that of central obesity and its association with the presence of NIDDM. Also, waist to hip circumference comparisons measure only centripetal fat distribution and do not take into account the relative contribution of distally located subcutaneous fat.

i. Principal Components Analysis of Skinfold Measurements and Analysis of Variance

Neither skinfold nor waist to hip ratios has shown a clear or significant relative patterning difference between the diabetic and non-diabetic in either of the sexes. Two possibilities must be considered at this point. First, that there is no real difference in the body composition of either male or female adult Mohawk Indians that can be correlated with diabetic status and, as previously thought (West 1978), this may be a benign morphological characteristic of Amer- indians. Second, that this general adiposity among the participants in this study is, as alluded to in the previous section, confounding the effect of fat patterning relative to the presence of NIDDM. It is the latter possibility that will be considered in this section. The statistical method that can best determine the relative proportion of subcutaneous body fat independent of overall obesity is principal components analysis.

As discussed in the statistical methods section (i) of Chapter 2, principal components analysis (PCA) can be used as an indicator of the effect of localized body fat on diabetic status. This multivariate analytic method will extract the necessary data so that the effect of centripetal fat upon NIDDM, the dependent variable, can be determined. Analysis of variance is then used to determine the statistical significance of these data. Researchers (Mueller et al. 1979 &

1981; Szathmary & Holt 1983) have found that using PCA is an effective method of determining the effect of body morphology on such factors as NIDDM since the obesity factor can be readily controlled.

#### j. Principal Components Analysis: Male Data

Component scores of the log transformed skinfold data were determined for each of the diabetic and non-diabetic sub-groups. Principal component scores, eigenvalues and percent variation explained for the diabetic and non-diabetic men are included in Table XIX. The first component, an "index of obesity" (Mueller and Wohlleb 1981), is highly and positively correlated with all skinfold sites in both sub-groups. This "index of obesity" explains 54% (eigenvalue = 4.31) of the variation in the diabetic men and 69.8% (eigenvalue = 5.59) in the non-diabetic.

The second principal component is a "pattern component" which contrasts extremity and trunk fat independent of overall fatness (Mueller and Reid 1979). This central versus distal factor explains 14.9% and 11.2% (eigenvalues = 1.19 and .90) of the variation in the Mohawk diabetic and non-diabetic men, respectively. Trunk sites (and the triceps in the diabetic men) contrast with the forearm, biceps and medial calf sites. These results indicate that there is a greater amount of variation explained by the pattern compo-

-TABLE XIX-  
Principal Components 1 (Fatness) and 2 (Centripetality)  
of Skinfold Thickness Measurements in  
Diabetic and Non-Diabetic Mohawk Males#

Skinfold and Eigenvector:	Principal Components			
	1	2	1	2
	Diabetic		Non-diabetic	
Forearm	.67	.48	.76	.20
Triceps	.81	-.19	.85	.27
Biceps	.68	.50	.87	.22
Subscapular	.83	-.17	.85	-.39
Midaxillary	.76	-.19	.87	-.39
Suprailiac	.82	-.19	.90	-.26
Abdominal	.77	-.42	.85	-.10
Medial Calf	.41	.63	.71	.59
Eigenvalue	4.31	1.19	5.59	.90
% Variation Explained	54.00	14.90	69.80	11.20
Total % of Variation	68.8		81.1	

#Log transformed values

nent in those men with NIDDM than in those without the disease.

k. Principal Components Analysis: Female Data

Principal component analysis of the female data reveals that the first component explains 71.4% and 63% (eigenvalue = 5.71 and 5.04) of the variance in the diabetic and non-diabetic Mohawk women (Table XX). This "fatness" component loads positively on all variables and heavily on all but the medial calf site in both subgroups of women.

Contrary to the expected results the second principal component explains less of the variance in the diabetic than in the non-diabetic women. In those women without NIDDM the second component explains 15.4% of the variance (eigenvalue = 1.23) whereas in the diabetic women this component of "centripetality" explains just 13.2% of the variance (eigenvalue = 1.06). The eigenvectors associated with the triceps, biceps, subscapular, suprailiac, and abdominal sites in the diabetic women contrast with those of the forearm, midaxillary, and medial calf sites.

In the non-diabetic women the proportionate loadings of the second component eigenvectors differ from the pattern seen in the diabetic women. In this case the eigenvectors associated with the medial calf, forearm, and biceps contrast with those of the triceps, subscapular, midaxillary, suprailiac, and abdominal.

-TABLE XX-  
Principal Components 1 (Fatness) and 2 (Centripetality)  
of Skinfold Thickness Measurements in  
Diabetic and Non-Diabetic Mohawk Females#

Skinfold and Eigenvector:	Principal Components			
	1	2	1	2
	Diabetic		Non-diabetic	
Forearm	.75	.48	.75	.36
Triceps	.89	-.12	.89	-.04
Biceps	.91	-.001	.80	.35
Subscapular	.94	-.20	.83	-.33
Midaxillary	.91	.18	.83	-.19
Suprailiac	.86	-.20	.86	-.26
Abdominal	.93	-.11	.89	-.21
Medial Calf	.45	.83	.38	.85
Eigenvalue	5.71	1.06	5.04	1.23
% Variation Explained	71.40	13.20	63.00	15.40
Total % of Variation	84.6		78.4	

#Log transformed values

According to these findings, factor 2 (centripetality) accounts for a greater proportion of the variance in these morphological data in women than in men and in non-diabetic than in diabetic women.

#### 1. Analysis of Variance of the Principal Components

Hierarchical analysis of variance, with sex and diabetic status used as the dependent variables, was performed on the means of the two principal components and the results of that analysis is presented in Table XXI. Analysis of the first principal component reveals that only sex has a significant effect on the degree of overall fatness (F ratio = 65.97; Significant at 0.0001). Similar results were obtained in the analysis of the second principal component; sex, not diabetic status, has a significant effect on the centripetality component (F ratio = 51.20; Significant at 0.0001). Women had higher average scores on each component than did men. Both the range of difference and the proportion of the variance explained are greater in the first (fatness) component.

These results suggest that after the removal of the effect of sex from the equation, in Mohawk men and women diabetic status does not have a significant effect on either overall fatness or a centripetal distribution of body fat. An unexpected finding that contradicts results presented earlier however is that obesity alone can not be correlated

-TABLE XXI-  
F-Ratios Derived from Hierarchical Analysis of  
Variance of the First and Second Principal Components  
in Sex and Diabetic Status Groups

Source of Variation:	Principal Components			
	1 (Fatness)		2 (Centripetality)	
	df	F-Ratio	df	F-Ratio
Main Effects	2	33.00*	2	25.67*
Sex	1	65.97*	1	51.20*
Diabetic Status	1	0.02		0.14

\* Significance of F = 0.0001

with diabetic status.

**v. Genetic Marker Analysis: Percent Mohawk  
of the Population Sample**

This section presents the results of the genetic marker analysis that was conducted as an adjunct to this study (Note 1). Five of the seven genetic systems useful in the detection of European admixture, including the two most powerful in the detection of admixture (Rh and Gm), were examined in order to discern whether there is a difference in the average amount of non-Indian ancestry between the diabetic and non-diabetic sub-groups (Appendix B: Table 6). The results of the analysis on the Mohawk genetic markers (ABO, Kell, and Rh blood group systems, immunoglobulin Gm, and the red cell enzyme Acid Phosphatase) strongly suggest that 1: there is no difference between the diabetic and non-diabetic in the average proportion of non-Indian ancestry, 2: on average, approximately 40% of the genes of the Mohawks in this study are non-Indian in origin, and 3: that gene flow into the Mohawk sample is from European origin only (Szathmary 1986, unpublished calculations).

## vi. Summary

The results presented in this chapter indicate that contrary to the stated hypothesis there is no clear correlation between NIDDM and a trunkal fat patterning in either the male or the female diabetic participants in comparison to their respective non-diabetic controls in this Mohawk population. These data do reveal though that there is proportionately less extremity fat (medial calf and forearm) in the male diabetics, and to a lesser degree, in the female diabetics. Whether obesity alone can be correlated with NIDDM in those with diabetes requires further investigation, since these data provide apparently contradictory findings. Number of pregnancies in women could not be correlated with diabetic status; nor could parental diabetic status. However, there is a significant intra-generational effect for both of the sexes confirming the recent occurrence pattern and suggesting either a strong additive environmental effect or, perhaps, an increase in the overall number of diagnoses in the recent past. Diabetes could not be correlated with degree of Amerindian ancestry.

Other findings include a high rate of vascular complications secondary to the diabetes and a high, although not statistically significant rate of gall bladder disease in the diabetic women.

## Chapter Four: Discussion

### 1. Introduction

The primary goal of this study has been to discern whether, in Mohawk Indians, there is a correlation between trunkal fat patterning and non-insulin dependent diabetes mellitus (NIDDM). As well, extent of obesity, familiarity of NIDDM and extent of vascular complications have been examined. The findings of this study which were presented in the previous chapter will now be discussed in the context of other studies on NIDDM and especially NIDDM in Amerindian populations.

Diabetes mellitus, as Keen (1974) notes, is a historical and descriptive term which encompasses many mechanisms that have in common the presence of an increased blood glucose level. A separation of disease entities sharing this one fundamental symptom was produced by the National Diabetes Data Group (1979) in an effort to break down this heterogeneous group of disorders according to etiological mechanisms and treatment regimes (e.g. Insulin-dependent [IDDM] versus non-insulin dependent [NIDDM] diabetes mellitus). Gaining increasing recognition is that there are some ethnic groups that apparently have a greater propensity for this

disease today than was previously known. North American Indian populations, despite their heterogeneity, are among those ethnic groups that are displaying recent and growing rates of Type II diabetes. Doubtful that this is an artifact of increased screening and health care practices, researchers are asking why and how NIDDM has become such a health problem in Native populations (for example, the Canadian Workshop on Diabetes in Native Populations, October 1986). Although few prevalence and no incidence studies have been conducted to date there is a clear indication that NIDDM is a new and increasingly pernicious disease among Canadian Native populations (Young 1985; Shah 1986). Speculation has arisen as to whether these populations are somehow more prone to NIDDM or whether this is somehow a different disease in these populations. Early 'natural history' studies of diabetes tended to substantiate the latter since North American Indians were asymptomatic at the time of diagnosis and apparently developed fewer of the vascular sequelae that are associated with diabetes. The findings of this study and its associated projects (see Note 1) lend support to the current belief that while the disease is relatively new in Canadian Aboriginal populations it is by no means a benign disorder.

The question remains then as to how and why Amerindian populations have become so susceptible to a disease once thought not to exist at all in these populations. This study

is one of an increasing number of research efforts attempting to gain greater insight into factors thought to be related to NIDDM in Amerindian populations.

#### ii. Diabetes in Amerindians: Neel's Hypothesis and Beyond

If North American Indians (as one example of what Neel [1962] referred to as less acculturated or primitive populations) have become more prone to diabetes in the recent past, asked Neel (1962), could it be that they are now experiencing the deleterious effects of an interaction of environmental and metabolic processes that was at one time essential for survival? And, if this is true, are they now revealing a "telescoped" version of how NIDDM developed in more "Westernized" populations? Neel argued that based on the models of early humans as hunters and gatherers there were times of low and high food availability. To survive these periods a biologic mechanism that increased caloric storage capacity would have been beneficial. Neel's term for this hypothetical survival mechanism is the "thrifty genotype". With the ready and abundant food supply available today the metabolic process that at one time would have been advantageous may now be deleterious (Neel 1962 & 1982).

Neel (1962) suggested that in those individuals who manifest a "thrifty genotype" there is a "quick insulin trigger" mechanism involved in what would now, with a con-

stant food supply, be a pathologic process. The effect of the relatively recent constant stimulation could lead to pancreatic beta-cell decompensation and eventually diabetes. Originally Neel (1962) thought that the mechanism leading to a net decrease in the efficiency of the available insulin involved insulin antagonists. This physiological process has since been found untenable (Neel 1982).

Despite the inability of anti-insulins to explain the exact process leading to diabetes, Neel's basic hypothesis remains as a fundamental organizing model under which many alternative mechanisms have been proposed and disputed. A "quick insulin trigger" in response to continual rather than sporadic alimentionation, for example, may induce an overstimulation of beta-cells and eventually lead to pancreatic decompensation and NIDDM (Neel 1982).

Other more recent hypotheses about the pathogenesis of NIDDM in Amerindians as well as Caucasians primarily involve the insulin receptor site on the cell, and especially the adipocyte, surface. There may be a down-regulation of normal insulin receptors in response to a repeated high level of circulating insulin in those predisposed to this type of reaction. The insulin receptors remain normal in function but there are relatively fewer functioning in response to the given carbohydrate load. Insulin secretion remains high and this persistent hyperinsulinemia would eventually result in

decreased pancreatic activity (Zimmet 1982; Neel 1982). Hyperinsulinemia has been documented in the Pima Indians as well as some Pacific Aboriginal populations with concomitant high rates of NIDDM (Bennett et al. 1976; Zimmet 1982).

Other theories that have arisen to explain the prevalence of NIDDM in Amerindians are based upon Neel's fundamental idea of a relatively recent metabolic insult that would have had past benefits. Schaefer et al. (1972) and Szathmary (1986), for example, both argue that the origin of the disorder lies in the initial digestive stage of the metabolic pathway. Each suggests that a diet high in protein and fat (more likely the traditional diet of the early northern hunting and gathering ancestors of the New World peoples) has led to an inability to metabolize the relatively recently added high carbohydrate foods. Schaefer (1968) (and Schaefer et al. 1972) found that among Canadian Inuit glucose metabolism was enhanced when preceded by a protein supplement and that intravenous glucose was more efficiently metabolized than that administered orally. The conclusion drawn from this work is that the focus of insult was in the gut and suggest that there may be a hormonal deficiency of some sort in those now prone to or experiencing hyperglycemia resulting in the inability to properly digest the oral carbohydrates.

Szathmary (1986) argues that a high rate of gluconeogenesis (that is, high blood glucagon concentration) would have been biologically advantageous in an environment without

any available carbohydrates. The result of the relatively recent addition of regularly available carbohydrates in addition to the already established high rate of glucagon secretion would substantially increase the blood glucose and hence insulin concentrations and eventually lead to pancreatic exhaustion.

Working from historical models however provides only a limited perspective on the etiology of NIDDM in Amerindian populations. Central to each of the models is the concept of relatively recent overnutrition and a new, constant fat mass. As will be discussed in the next section, adipocyte tissue, independent of overall body mass, may play a particularly important role in the etiology of NIDDM in North American Indians.

### **iii. The Relationship Between NIDDM and Adiposity in Amerindians**

#### **a. Obesity and NIDDM**

Obesity is often implicated in the etiology of NIDDM despite no known causal relationship between the two entities. Indeed, the question has arisen as to whether obesity is diabetogenic or diabetes causes obesity (Berger et al 1978) (for a review of adipose tissue physiology see Bjorn-  
torp 1982 and Stern & Johnson 1978). Of particular concern is that while "obesity is clearly a major concomitant of

NIDDM it is not necessarily an antecedent of, or sufficient explanation for the degree of, elevation of diabetes mellitus in New World Peoples" (Weiss et al. 1984:158).

There is no discernible associative pattern between NIDDM and obesity (Keen 1974; Armellini 1979; Berger 1978; K8bberling 1979; Baird 1982; Bierman et al. 1968). When they looked at differences in the prevalence of diabetes between populations with similar degrees of obesity and similarities in diabetes prevalence between populations with differing frequencies of obesity, Keen and others (1979) concluded that obesity per se cannot be the only determinant of diabetes.

The Mohawk data lend new and strong support to the thesis that overall fatness cannot by itself account for the higher rate of NIDDM. Mohawk Indians without NIDDM are, on the average, also obese. That there is a significant difference in BMI values between diabetics and their non-diabetic controls in both males and females is not sufficient evidence (especially since the principal components analysis contradicts the BMI finding) for an association of diabetes and obesity independent of other factors. Non-diabetics, and their diabetic counterparts, fall above the upper limits of obesity according to the NDDG (1979) guidelines.<sup>2</sup>

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<sup>2</sup> Although it can be argued that Amerindians exhibit a greater weight for height when compared to Caucasians (Johnson and Schell 1979), the range of BMI values for this population fall well above those reviewed in the Johnson and Schell (1979) study.

When obesity is implicated it is not the degree of fatness that is associated with disease but the familiarity of that fatness which in turn may "unmask" a discrete susceptibility in certain individuals and ethnic groups. That is, if obesity is a factor in the development of NIDDM then it may be a concomitant of but not causally related to the disorder (Keen 1974; K8bberling 1979; Keen et al 1979). In fact research among different ethnic groups reveals that NIDDM is more closely related to ethnic affiliation and familiarity of obesity than degree of obesity alone (West & Mako 1976; Knowler et al. 1983). For example, Stern and co-workers (1983 & 1986; see also Haffner et al. 1986) found that lean Mexican-Americans were at greater risk for Type II diabetes than their lean Caucasian controls and concluded that although obesity contributes to the development of NIDDM, fatness alone can not explain the high rate of NIDDM in the Mexican Americans. Similarly, in Oklahoma Indians, Lee found an increased risk of NIDDM in obese individuals but attributed it to inheritance patterns and degree of "Indian-ness" (albeit that this was a qualitative association) (1985). Knowler and co-workers (1983) (see also Bennett et al. 1976 and Nagulesparan et al. 1980) have found that in the Pima Indians although there is a strong relationship between NIDDM and fatness this alone could not account for the high occurrence of the disease. They also posit a possible but as

yet unknown primary inherited factor that would implicate both obesity and ethnic affiliation in the development of NIDDM.

b. Duration of Obesity

Currently under investigation is the extent to which duration of obesity affects future disease. The trend toward greater weight for height in diabetics appears to begin in early adulthood if not sooner although there is little conclusive evidence here of a causal association. There is speculation though that a complex of diseases peculiar to Amerindians involves early-onset obesity. Along with NIDDM, cholesterol gallstones, gallbladder cancer and a higher rate of cancer at all sites, early-onset obesity is an adjunct of this conglomeration of discrete diseases and symptomatology, termed New World Syndrome (NWS). NWS is believed to be a twentieth century affliction of Native populations regardless of geographic location or linguistic affiliation (Weiss et al. 1984 and 1986). The relationship of early obesity, NIDDM and cholelithiasis seen among the Pima Indians (Knowler et al. 1981) lends preliminary support to the NWS model (see also Section vi:a of this chapter).

In contrast to the NWS model of long term obesity and its association with NIDDM, Lanska and co-workers (1985) suggest that long-term hyperadiposity is of a different type than that of adult-onset obesity (predominantly hypertrophic,

see next section) and that NIDDM is currently believed to be related to the latter. As such, early weight gain should not, by current definition, play a key role in the later development of NIDDM. A long-term cohort study of Israeli men revealed that duration of obesity has no apparent relationship to the occurrence of NIDDM in that population (Modan et al. 1986).

The Mohawk data reveal no clear trend to early fatness in the male or the female diabetics when compared to their controls (Table VI & VII). However in this as in other studies the data is cross-sectional and based upon recall data. Thus no conclusions can be drawn about early fatness as a predisposing factor of NIDDM in this population without adequate longitudinal testing.

### c. Body Fat Distribution and NIDDM

Despite the general medical consensus that obesity is related to NIDDM the preceding discussion reveals that the nature and extent of the interassociation remains a subject of debate. Increasingly there is evidence<sup>3</sup> that the critical denominator in the development of metabolic disorders such as NIDDM is the proportion of body fat in relation to muscle

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<sup>3</sup> Trunkal adiposity has been specifically associated with other diseases that have long been accepted as being primarily "diseases of obesity." Independent longitudinal

mass and especially the location of that adipose tissue (Ruderman 1981; Leonhardt et al 1978; Bjorntorp 1982; Sims 1982; Vague et al. 1979). As Vague and co-workers have stated: "Predominance of fat in the upper part of the body is at least the clinical reflection of factors which lead obesity to progress towards diabetes mellitus"; that is, upper body fat is "diabetogenic" (1979: 145). Possible physiological and biochemical explanations for this "diabetogenic" effect are presented in the next two sections.

#### d. Hyperplastic and Hypertrophic Adiposity

Research conducted as early as 1955 ascertained that there are individual differences in body fat distribution (Garn 1955) and that a pronounced upper body fat accumulation is more likely to be associated with diabetes (Vague 1956; Vague distinguished between 'gynoid' and 'android' obesity, although these terms are no longer used). These observable topographic differences in subcutaneous body fat pattern are in turn associated with metabolic and morphologic differences in the adipose tissue (Kissebah et al. 1979). Lower body adipose tissue is predominantly hyperplastic whereas upper body fat is predominantly hypertrophic (Sims 1982).

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studies of European and American men found trunkal fat patterning a better predictor of cardiovascular disease, hypertension, and death than degree of obesity alone (Larsson et al. 1984; Damon et al. 1969 [Framingham men]; Weinsier et al. 1985).

The development of an excess number of adipocytes characterizes hyperplastic obesity. At a certain critical fat cell size new adipocytes develop from precursor cells. As a result of the continued increase in the number of new fat cells this type of obesity is often quite severe yet is related to few metabolic complications. Hyperplastic obesity is generally long-standing, often but not always present since childhood (Stern and Johnson 1978; Bjorntorp 1982).

Hypertrophic obesity is characterized by an increase in the size of the adipocyte. There is thought to be a triggering mechanism to stimulate new growth of fat cells here as well but, as the name suggests, the maximal capacity of each cell is reached before any new cells are formed. The hypertrophy of adipocytes is typical of adult-onset obesity; indeed, Bjorntorp et al. (1972) suggest that there is a direct correlation between maturity-onset obesity and NIDDM. And as mentioned above, hypertrophic fat has been clinically correlated with what are known as the "diseases of obesity" (NIDDM, hypertension and hyperlipidproteinemia) (Albrink & Meigs 1964; Krotkiewski et al 1974; Gibson et al. 1975). As Kissebah et al. state: "The distinct [hypertrophic] fat cell morphology and metabolic activity associated with the site of fat predominance may play an important role in the evolution of metabolic complications in obesity" (1982: 259).

#### e. Hypertrophic Fat and NIDDM

The role of insulin receptors as the primary site of disorder in NIDDM may be directly linked to the hypertrophic fat cell. Using a skinfold ratio comparative method, Feldman and co-workers (1969) investigated the correlation between NIDDM and trunkal fat patterning in Caucasian and Negroid individuals. Controlling for age, sex, race, height and weight, the relationship between nine skinfold sites and the triceps and then subscapular measurements were tested. The ratio measurements of the diabetic participants varied significantly from those of the non-diabetics. Feldman et al. (1969) found that both diabetic men and women were more apt to be obese and that the excess weight was located primarily in the trunkal region rather than on the extremities. Whereas the diabetic women gained in a pattern resembling that of the normal male, the physique of the diabetic men was described as an exaggeration of that centripetal pattern. Feldman and associates (1969) concluded that there is a positive association between an increased deposition of subcutaneous body fat in the trunkal region and non-insulin dependent diabetes mellitus.

In another study, Hartz et al. (1983) tested the relationship between trunkal obesity and NIDDM in 15,532 diabetic and non-diabetic Caucasian women who were known to be overweight. In a comparison of waist to hip ratios in combination with degree of obesity, a strong association between

obesity, trunkal fat patterning and the occurrence of NIDDM was found. Based on their findings Hartz et al. (1983) concluded that upper body fat patterning acts as an additive factor in the association of obesity and NIDDM.

The actual physiological mechanism responsible for the relationship between a distinct body morphology and NIDDM is currently under investigation. Measurement of insulin binding to receptors on the surface of adipose cells has demonstrated that the increased size and volume of the adipocyte is accompanied by a decrease in the concentration of the existing insulin receptors on the cell surface. Concomitant to the proportionate decrease of insulin receptors is a decrease in the cellular uptake of glucose and an increase in blood glucose concentration. The insulin insensitivity and hyperinsulinemia seen in NIDDM are believed to be associated with this change in the cell morphology and not with the number of fat cells per se (Karam 1982; Bjorntorp 1982).

Karam is (1982) quick to note however that this theory of the role of adipose tissue in glucose metabolism attributes a greater role than is now known to adipocytes in energy regulation and it assumes an associated although as yet unproved decrease in the uptake of insulin in muscle and liver tissue. Alternative explanations de-emphasize the role of the hypertrophied fat cell. Salans and Cushman (1978) propose that the site of receptor insult is located primarily

in the muscle and liver tissue and not in the adipocytes. They reason that the enlarged adipocyte cannot be directly responsible for the glucose intolerance and insulin resistance in the obese individual since caloric restriction is often associated with a significant reduction in plasma glucose and insulin concentrations long before there is a substantial change in body weight and adiposity.

Alternative explanations include an aberrant intrinsic regulatory mechanism (e.g. "down-regulation" of insulin receptors at all sites) and pre- and post-receptor anomalies. Despite the many theories advanced to date however none fully explain the complex interplay that results in NIDDM in those with a trunkal patterning of fat tissue (Neel 1982; Karam 1982).

#### f. Trunkal Fat Patterning and NIDDM in Amerindians

The association between NIDDM and trunkal fat patterning in Amerindians stems from the combination of observations of anthropometric variation in Amerindian adults, the prevalence pattern of NIDDM in Amerindian populations and speculation about the mechanisms underlying adipose tissue morphology. Specifically, in light of their findings Szathmary & Holt (1983) challenge West's assertion that the "peculiar distribution" of fat in North American Indians is "probably a racial characteristic" and hence is not meaningful in the context of diabetes onset (1978b:248 in Szathmary

and Holt 1983:494). West's assertion about the benign nature of this type of fat patterning in Amerindians is, according to Szathmary and Holt, precipitous and speculative in light of what is currently known about trunkal fat patterning and about the distribution of NIDDM in Amerindian populations. That is, although in some populations there is a tendency to a trunkal fat patterning it does not mean that within a group the diabetics may not have an even greater exaggeration of that pattern. If hypertrophic weight gain is a factor in the occurrence of NIDDM then this may be a significant factor in the etiology of NIDDM in populations now thought to be at greater risk for this disease. Szathmary and Holt (1983), for example, found that the Dogrib Indians who tested hyperglycemic had significantly greater proportion of trunkal fat than those who were normoglycemic.

The Mohawk data presented here is the first to test the relationship between NIDDM and trunkal fat patterning in an Amerindian population with a percentage of known diabetics. Comparative studies of diabetics and non-diabetics in Mexican-American and Caucasian groups, as mentioned earlier, reveal that in both sexes, and especially in women, upper body fat patterning is associated with NIDDM (Feldman et al. 1969; Hartz et al. 1983; Mueller et al. 1984; Kalkhoff et al. 1983; Sims 1979).

g. The Mohawk Study

In this study the findings did not support the initial hypothesis that there is more fat on the upper trunk of those who are diabetic. No clear patterning effect could be ascertained in either the male or the female diabetic Mohawk participants in comparison to their non-diabetic controls and there were differences in patterns between the sexes. Although there is a tendency toward trunkal patterning at higher BMI values this association is not born out in the principal components analysis. The results of three separate analytic techniques indicate that neither the male nor the female diabetics in this Mohawk population exhibit a significantly greater proportion of trunkal fat when compared to their age and sex matched controls.

What must be emphasized however is that the patterning effect may be hidden or lost as a result of one or many confounding factors. There is, for example, evidence of patterning in the extremity to subscapular skinfold ratios. These data revealed proportionately less lower limb fat in diabetic male and female participants than in their respective control groups. Mueller and Wohlleb (1981) have stressed the importance of leg fat in studies of NIDDM, since it is the obverse phenomenon to that which is being studied. When examined, diabetics do seem to have significantly less lower limb fat than non-diabetics (Vague 1956; Feldman 1969; Vague et al 1971). The importance of lower limb fat has yet

to be fully understood but in the case of the Mohawks it may be indicative of a strong relative fat patterning effect that is otherwise hidden.

Haffner and co-workers (1986), in their research among Mexican-Americans, explain how a patterning effect could be obfuscated. They suggest that the effect of centrality decreases at higher levels of centrality; that there is a "plateau effect" of centrality in NIDDM above which the effect is no longer seen. That the waist to hip ratios do not increase at increasing BMI values combined with the fact that more than 90% of the population fall above standard levels of obesity strongly suggests this type of "plateau" effect may be confounding the Mohawk data.

Haffner and co-workers (1986) suggest another factor that may also be confounding the Mohawk data. They found that, contrary to their initial speculation, there was a sex difference in the occurrence of central obesity (greater in men than in women) among the Mexican-American diabetics. They point out however that their work as well as this and most other studies are based upon prevalence and not incidence data. "If, for example", explain Haffner et al., "increased centrality were associated with shorter duration of disease due to higher mortality in men, then a prevalence study might fail to detect an association in men due to a selective removal of centrally obese diabetic men" (1986:

158). Thus the recruitment of a cross-section of living known diabetics may in and of itself influence the type and quality of the data collected such that those diabetics with central fat patterning may never be accounted for.

Another critical confounder in this study is the length of disease and hence treatment program. Diagnosed diabetics enter into strict dietary programs regulating their caloric intake and as a result usually tend to gradually lose weight. Since hypertrophic weight is more easily reduced (Kissebah et al. 1979) than those with diabetes, independent of sex or age, may have proportionately less upper body fat than those individuals without the disease. Concomitantly, the high proportion of obese non-diabetic participants is probably an additive confounder.

Furthermore, the number of individuals in each of the sub-divided (by sex and diabetic status) data sets may be responsible for the results attained. In the analysis of principal components, for example, the small numbers of individuals within each of the sub-groups may account for the fact that the differences between centrally located fat (PC2) and overall fatness (PC1) did not reach statistical significance in the analysis of the principal components. The male and female data could not be combined however since there is a difference between the sexes (Table XXI).

Finally, the discrepancy between the results of the BMI and principal components analysis must be addressed. Ac-

According to the comparison of BMIs there is a significant difference between those with and those without diabetes. With the principal components analysis, which is based on factors derived from the original skinfold measurements, this difference was not replicated. The discrepancy may be the result of differences in efficacy of techniques employed or differences in the statistical manipulation of the data. Measurements taken to determine overall fatness include skinfolds (principal component 1) and weight and stature (BMI). The BMI might indeed adjust for differences in stature but there were no within-sex differences in stature between the diabetic and non-diabetic participants. Thus the BMI values can be assumed to be accurate reflections of differences in body mass between the groups.

At higher levels of obesity there may be a decrease in the accuracy of caliper measurements although it is doubtful that this is a significant factor in this study. The raw data indicate that of all the skinfold sites measured (8 sites x 208 participants = 1,664) only 20, or 1.2%, were greater than 65 millimetres and primarily at the abdominal site, which proved the most difficult to attain in the excessively obese. As well, all sites were measured twice, the average of the two readings was recorded, and there was minimal intra-observer variation based upon mid-collection reassessments of technique (see Chapter 2, iii:d). It is

thus felt that the caliper readings also accurately reflect the degree and relative location of adiposity in this population.

The difference between the BMI and the principal components scores (PC) may be an artifact of the analytic techniques employed. As in the case of centrality, the number of participants within each of the data sets may be a factor in the stated discrepancy.

The discrepancy between the BMI and PC, on the other hand, does in no way deter from original finding that there is a high degree of obesity within all subsets of this sample population whereas only certain individuals have NIDDM. That is to say, if obesity was in and of itself a predisposing factor then an even greater proportion of Mohawk adults should have NIDDM. A factor that must also be considered is the relatively new but high rate of NIDDM in this population as it is being diagnosed by the medical staff of the KMHC.

#### iv. Familiality of NIDDM

Factors that may be influencing the above-mentioned high rate of occurrence NIDDM were examined in this study. Primarily, the distribution of diabetes among first degree relatives was investigated since familial inheritance patterns do affect an individual's predisposition to NIDDM. Having one or more diabetic parents has been shown to in-

crease one's risk for Type II diabetes (Zimmet 1983). There is no clear evidence to date though that explains the extent to which a hereditary factor can precipitate the onset of diabetes alone or in combination with external triggers or with the type and degree of obesity.

Studies to date in Amerindians as in Caucasians show that while there is evidence of a hereditary component, this effect is seen only concomitantly with others factors such as obesity. Lee et al. (1985) found that among Oklahoma Indians there were significantly more diabetics than non-diabetics who were both obese and had diabetic parents. But these researchers found that there was no difference in the occurrence of NIDDM based on the number of parents with diabetes. Knowler et al. (1983 and 1981) reached similar conclusions in their studies among the Pima Indians. They found a higher incidence of NIDDM among those who were obese and who had at least one diabetic parent than those without any parent with NIDDM. Knowler at al. (1981) also found that the incidence of NIDDM increased in those with two diabetic parents.

Although parental diabetes plays a role in the occurrence of NIDDM this inter-generational effect is not always an accurate indicator of future disease. More specific is the intra-generational effect between siblings and their age cohorts. This within-generation effect however may be more indicative of newly antagonistic environmental influences

acting upon a previously "dormant" inherited predisposition. Beaty et al. (1982), working with Caucasians, found that the number of affected sibs was a better predictor than the number of parents with NIDDM in the risk for diabetes among their probands. They suggest that this within-generational effect may be genetic in origin but that a "diabetic" genotype may have a higher probability of being expressed in the offspring generation who are being exposed to an increased number of possibly detrimental environmental stimuli. That is, changes in lifestyle patterns and especially diet and exercise habits may precipitate an increase in the rate of NIDDM among a cohort of sibs.

Szathmary (1983), working among the Athapaskan Dogrib Indians, found that there was a strong intra-generational effect in the occurrence of impaired glucose tolerance. She attributes these findings to the influences of an increased rate of acculturation in those who underwent this process during childhood or in their youth.

Among the Mohawk, the stated<sup>\*</sup> diabetic status of the parent has no significant effect in the occurrence of NIDDM in the offspring (Tables VI and VII). In both sexes and in all of the disease status sub-groups there is a comparable percentage of parents with NIDDM. While the proportion of

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\* There may be a certain degree of recall bias inherent in this data set since parental diabetic status data was collected only by offspring recall.

women with diabetic offspring is almost twice as great in diabetic women, that difference is not statistically significant (Table VII). The number of siblings with diabetes stands out as the only first degree relationship in which there is a significantly greater percentage of diabetics with brothers or sisters who also have NIDDM. Although this clearly reflects an inherited susceptibility, it still remains unclear as to what factors are now precipitating the occurrence of NIDDM in the last quarter century among those who are at greater risk.

#### v. Environmental Factors in NIDDM

Which and to what extent environmental factors influence the onset of NIDDM in this Iroquoian population is a subject that is in need of further extensive investigation. An association has been made between urbanization and westernization and diabetes in Pacific aboriginal populations (O'Dea et al. 1980; Zimmet et al. 1981; Stanton et al. 1985). The implication of this change is that those undergoing or who have undergone change now consume a higher proportion of carbohydrates and hence calories and pursue a less active lifestyle. The combination of these factors is a physiological proneness to and development of obesity. This marked change in lifestyle patterns is accompanied by the develop-

ment of diseases that are common to the "westernized" Euro-american living. A highly significant correlation in the occurrence of NIDDM among siblings in the absence of any other evidence reflects both an environmental and a genetic component at play without implicating one more than the other.

The Mohawk findings are similar to those of Beaty et al. (1982) and Szathmary (1985) wherein there is a significantly higher percentage of hyperglycemic siblings than parents or children of diabetics or non-diabetics. Whether there have been major shifts in lifestyle patterns must be addressed but this is the subject of another study. Extrinsic factors that impinge upon normal glucose regulation such as quality and quantity of foodstuffs, activity patterns and change, socio-economic factors, environmental toxins and stress must all be evaluated in relation to the occurrence of NIDDM in the Mohawk population of Kahnawake.

The sedentary, suburban lifestyle and the range of employment in the labour market (Tables IIa and b) is not new to this reserve community. As discussed in Chapter 2:ii structural steelwork, the primary livelihood of many Mohawk men, has existed since the end of the last century. On the other hand, the gardens that people keep are smaller than in the past and indoor water and plumbing have, over the last ten to fifteen years, been incorporated into all of the homes (Artesian wells are still in evidence in front of many). Also, there is much less physical work done as people walk

less, have more food to eat and take advantage of many more mechanized appliances and equipment (Montour 1986, personal communication).

Parity has been considered a causal agent in the development of NIDDM in some societies; an increase in the number of pregnancies and births proportionately increasing the risk of NIDDM (for a brief review see Keen and Jarrett 1976). In this study, the Mohawk women show no correlation between parity (number of pregnancies) and occurrence of diabetes. These findings are in concordance with other recent studies wherein parity is thought to have a minor, if any, effect in the etiology of NIDDM (Zimmet 1982).

#### v. Disorders Associated with NIDDM in Amerindian Populations

##### a. Gallbladder Disease and New World Syndrome

There are two groups of disorders that are related to NIDDM and require discussion since they are of particular importance in the Amerindian context: they are New World Syndrome and the vascular sequelae of NIDDM.

Weiss and associates (1984a and 1984b) suggest that there is a relationship between NIDDM and GBD and that the two are components of a greater syndrome to which Native People of the New World have become particularly susceptible. They propose that the combination of a tendency toward early

onset obesity, adult onset diabetes mellitus, the formation of gallstones and gallbladder cancer is a complex syndrome peculiar to Amerindian populations and especially Amerindian women. This New World Syndrome is thought to be the result of a similar type of culture-gene interaction that is responsible for the increased prevalence of NIDDM (Weiss et al. 1984a).

The findings of this study reveal that although the difference between diabetic and non-diabetic women is not statistically significant, nearly one half of all the diabetic women have gallstones. That is, cholelithiasis is extremely prevalent in Mohawk women regardless of diabetic status. Also, as discussed in Section iii:b of this chapter there is no evidence that diabetic Mohawk women begin to gain weight earlier than non-diabetics and even if they did, this may not be related to either diabetes or to NWS. Fewer incidences of cholelithiasis were discovered among the diabetic men and even fewer among the non-diabetics. Thus, although this Iroquoian population is experiencing a high rate of NIDDM and of gallbladder disease, it does not appear to be part of the complex of disorders making up the New World Syndrome as it is currently defined.

#### b. Vascular Complications of NIDDM

Diabetes is not, as was once thought (Saiki and Rimoin 1968), a benign disorder in Amerindians; nor is it an iso-

lated and complication-free disease among these peoples. With an increase in the duration of the disorder the full range of sequelae are afflicting Amerindian populations. Whether there is truly a variation in the extent and severity of complications between Native American groups will only be known after many more years of study.

Studies such as those done by Rate and co-workers (1983) and Gillum and associates (1984) confirm the findings of this study: Amerindians are not exempt from either the micro- or the macro-vascular sequelae of NIDDM. In Rate and associates' (1983) study, for example, the Hopi and Navajo Indians of the southwestern United States with NIDDM of at least ten years duration, experienced high rates of retinopathy (57%), nephropathy (40%), peripheral neuropathy (21%), and peripheral vascular disease (28%). In an overview of the literature, West (1974), finds that there are inter-tribal differences in the occurrence of complications; nephropathy is common in all groups that have been studied, whereas retinopathy varies from one group to another (and may be more closely linked to the rate of hypertension). Weiss and others (1984a) suggest that despite an apparent higher prevalence of the disease itself, Amerindians "do not differ significantly from other populations with respect to the natural history of the disease and the long term effects of chronic hyperglycemia" (1984a: 157); that is, the sequelae of

NIDDM will occur to the same extent in Amerindian populations as in others. Thus, although there is little agreement about the inter-group rate of occurrence, there is clear evidence that NIDDM is not a benign disorder in the Mohawk as in many other Amerindian populations (see Note 1).

#### vii. Summary and Conclusions

Contrary to the original hypothesis, diabetic Mohawk Indians do not show a propensity to trunkal obesity. In this cross-sectional case-control matched study, Mohawk Indians from the Kahnawake reserve were matched for age, sex, and diabetic status. The results of the investigation indicate that while nearly all of the participants fall above the standards of obesity, there is no distinct trunkal distribution pattern in those with diabetes. There is a significantly lesser amount fat on the extremities of the diabetic men and, to a lesser degree, of the diabetic women than their respective controls. Several possible explanations for the findings have been suggested since a proportionately greater distribution of fat in the trunkal region may not have been manifested because of factors other than those controlled for in this study.

A strong intra-generational effect is present in this sub-population of diabetics suggesting a recent environmental

"triggering" factor impinging upon those who are already "at risk" for the disease. According to the preliminary red cell marker analysis, degree of Indian ancestry does not appear to be a predisposing factor although the familial occurrence pattern does suggest a genetic basis.

The limitations of this study as presented in previous sections of this thesis include: a self-selection bias on the part of the control population, duration of disease and hence treatment in those with NIDDM, and the limitations of analysis at upper levels of obesity. Despite these limitations however the results provide substantial data where none previously existed and will form the baseline for further research in this population.

Future research should, first and foremost, be longitudinal. Since most of the residents of Kahnawake use the health services provided on the reserve and with the already present concern within the community for increased health status and awareness, enlistment from an early age into a long-term project is both plausible and of minimal inconvenience to those who participate. Whether those with newly discovered NIDDM do in fact tend toward truncal obesity could be addressed directly as could the question of early weight gain as a predisposing factor in NIDDM. In light of the strong within-generational effect found in the Mohawk population much more research needs to be conducted in order to discern the relative contribution of such environmental fac-

tors as level of activity, dietary factors (quality and quantity), and socio-economic level.

Clinical investigations must occur in unison with studies of the cultural factors that will elucidate the meaning and the understanding of diabetes specific to this Mohawk community. Such factors include: the concept of weight and obesity in terms of either status or prosperity in this population (see Rittenbaugh 1982; West 1974 re: obesity as a culture-bound syndrome), culturally specific attitudes towards, meanings inherent within and intrinsic methods of treating NIDDM (see Judkins & Judkins 1984; Hagey 1984); in other words, what it means to be sick with diabetes from a uniquely Mohawk perspective.

On a day to day basis, individuals on the reserve with diabetes learn how to take control of their own treatment and care. Each newly diagnosed diabetic becomes a member of an educational group at KMHC and begins to learn about realistic long-term modifications in his diet and activity patterns. This program provides all of its participants the opportunity to take control of their disease rather than the disease overtaking them. Individual involvement and responsibility of care is stressed at every stage in the clinical encounter whether it be by the dietician, the home care nurse or the physician. If obesity, in any form, plays a role in the development of NIDDM, then the results of this study indicate

that nutritional and physical education must begin at an early age in order to decrease the risk of adulthood diseases such as NIDDM and its sequelae.

## NOTES

1. The two adjunct studies will 1) look at the rate of complications among the diabetic population and 2) provide a genetic profile of the entire study population. As a follow up to the original study by Montour and Macaulay (1985), Dr. L.T. Montour, Dr. A.C. Macaulay and N. Adelson are conducting a retrospective review of the prevalence of micro- and macro-vascular complications of NIDDM, hypertension, ischemic heart disease and hypercholesterolemia and comparing these rates to those found in a control Mohawk population. Please refer to this work for the definitions of the complications of NIDDM.

Dr. E.J.E. Szathmary and N. Adelson are determining the average proportion of non-Indian admixture in this population as it relates to this study. As well, comparisons are being made with two other Iroquoian populations and the extent of genetic differentiation within Iroquians is being compared to that in subarctic hunting and gathering peoples. Also the genetic relationship of the Mohawk to non-Iroquoian peoples will be determined and interpreted in the context of the current ideas about the peopling of the Americas.

2. There is an extensive range of health services available to and utilized by this community. The extent of these facilities typifies the degree to which this Native population is politically organized and thus able to support its members so comprehensively.

The Board of Directors of the Kateri Memorial Hospital Centre includes the hospital administrator (Ms. J. Delisle), the medical director (at present: Dr. A.C. Macaulay), 2 members of the Mohawk Council, representatives from the profession staff (medicine, nursing, social services), a representative of the non-professional staff, and 5 community representatives.

The hospital has the facilities to support 33 long term and 10 short term beds. The medical out-patient clinic is open six days a week during daytime hours. There are five physicians who provide a rotating full coverage to the hospital and clinic. Also available through the clinic are a nutritionist, home care nurse, physiotherapist, psychologist, speech therapist, dental clinic, eye clinic, and a full range of social services. Whatever can not be handled by the medical services at Kahnawake is sent to hospitals in Montreal. Transportation to Montreal is provided through KMHC.

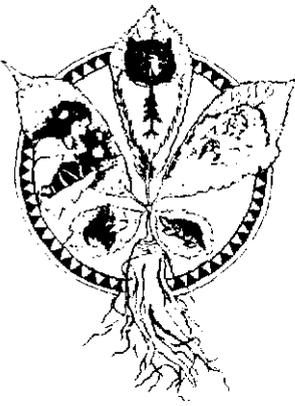
Community Health Services is staffed by three registered nurses and provides pre- and post-natal classes and check-ups and a well-baby clinic. It also serves the local

check-ups and a well-baby clinic. It also serves the local pre-school, primary school and high school (Survival school) providing general health services and education as well as immunization programs and visual and hearing tests.

Community Health Representatives (who on remote reserves often dispense medications) disseminate information about health and community development issues, investigate health and/or environmental complaints and serve as a type of liason between the community and the health facilities.

3. Celina Montour was the research assistant hired for this project. She is a reserve-dwelling Mohawk Indian who is currently working part time as the secretary/receptionist in the eye clinic. She is very familiar with the functioning of the medical clinic as well as with the community members of the reserve. The criteria of this research project were well known to her and she was able to determine at the stratification stage whether or not an individual lived on the reserve, used the hospital facilities or had at least one Indian parent.

- APPENDIX A -



**Kateri Memorial Hospital Centre  
Tehsakotitsén : tha**

**P.O. Box 10, Kahnawake, Que., J0L 1B0  
Tel.: (514) 632-7620**

July 22, 1985.

Dear

As you may have recently heard on the radio, there will be a study on diabetes and the relationship between diabetes and fat patterning conducted here in Kahnawake throughout the months of August and September, 1985. This study has been approved by the Board of Directors of the Kateri Memorial Hospital Centre. We are studying non-diabetic as well as diabetic individuals.

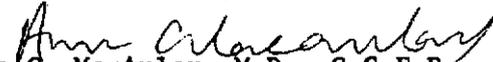
As a chosen participant in this study, you will be able to help us better understand why many people here in Kahnawake have diabetes. Whether you are diabetic or not, your voluntary participation would be greatly appreciated.

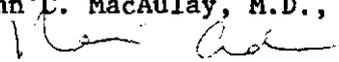
Should you decide to help us, you will be asked to come to Kateri Memorial Hospital Centre at a scheduled day and time (transportation available, if needed). The study, which should take no more than one hour, will include taking your height, weight and skinfold measurements of your legs, arms and chest. You will be asked questions regarding your health status and family background. There will also be one tube of blood drawn for chemical analysis.

We, the members of the research team, appreciate your participation and look forward to seeing you in the very near future. Mrs. Celina Montour will contact you to schedule an appointment time.

Sincerely,

  
Louis T. Montour, M.D., C.M., C.C.F.P.

  
Ann C. MacAulay, M.D., C.C.F.P.

  
Naomi Adelson, R.N., B.A.

Mohawk Diabetes, Obesity and Fat Patterning

- CONSENT FORM -

I, \_\_\_\_\_, consent to participate in the study of  
please print  
diabetes and fat patterning. I understand that there is no risk to  
me and that all information gathered in this study is COMPLETELY CONFIDENTIAL.  
I also understand that I will be asked questions regarding my medical and  
family background, that I will have my height, weight and skinfold  
measurements taken and that Ms. Adelson will take one sample of blood.  
I permit access to my medical charts to the research team. I sign this  
consent form of my own free will and understand completely the nature  
of this study and my participation in it.

---

Signature of participant

-Mohawk diabetes, obesity and fat patterning-

DIABETIC - Yes / No

-QUESTIONNAIRE-

M: \_\_\_\_\_

F: \_\_\_\_\_

I.D. \_\_\_\_\_

Family and Personal History Questionnaire

1. What is your date of birth

1.    /   /     
    d    m    y

2. Civil Status

- married (incl. living with someone) = 1
- widowed = 2
- divorced = 3
- single = 4
- other = 5

2. \_\_\_\_\_

a) Is, or was, your spouse Indian (Mohawk)?

2a) \_\_\_\_\_

Yes = 1, no = 2, don't know = 9

3. Do you have any children?

3. \_\_\_\_\_

Yes = 1, no = 2

If yes, give name, age and place of residence of offspring (if deceased, note with a 'd'.)

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

4. Do you have any brothers?

4. \_\_\_\_\_

Yes = 1, no = 2.

If yes, what are their names?

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

I.D. \_\_\_\_\_

Family and personal History, cont'd.

5. Do you have any sisters?

5. \_\_\_\_\_

Yes = 1, no = 2

If yes, what are their names?

\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

6. Was your mother originally Indian (Mohawk)?

6. \_\_\_\_\_

Yes = 1, no = 2, don't know = 9

a) If no, do you know what nationality she was/is?

6a. \_\_\_\_\_

7. Was your father Indian (Mohawk)?

7. \_\_\_\_\_

Yes = 1, no = 2, don't know = 9

a) If no, do you know what nationality he was/is?

7a. \_\_\_\_\_

8. Was your mother's mother Indian (Mohawk)?

8. \_\_\_\_\_

Yes = 1, no = 2, don't know = 9

a) If no, nationality?

8a. \_\_\_\_\_

9. Was your mother's father Indian (Mohawk)?

9. \_\_\_\_\_

Yes = 1, no = 2, don't know = 9

a) If no, nationality?

9a. \_\_\_\_\_

10. Was your father's mother Indian (Mohawk)?

10. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

a) if no, nationality?

10a. \_\_\_\_\_

I.D. \_\_\_\_\_

Family and personal History, cont'd.

11. Was your father's father Indian (Mohawk)? 11. \_\_\_\_\_  
Yes = 1, no = 2, don't know = 9
- a) If no, nationality? 11a. \_\_\_\_\_
12. What type of work do you do?  
(include if work is seasonal or  
if retired)  
12. \_\_\_\_\_
13. Do you now smoke cigarettes? 13. \_\_\_\_\_  
yes = 1, no = 2  
If NO, go on to Question 16.
14. How many cigarettes do you usually smoke 14. \_\_\_\_\_  
in one day?
15. How old were you when you began 15. \_\_\_\_\_  
smoking regularly?
16. Do you chew tobacco? 16. \_\_\_\_\_  
Yes = 1, no = 2
17. Do you smoke a pipe? 17. \_\_\_\_\_  
Yes = 1, no = 2.  
If NO, go on to Question 20.
18. How often do you smoke your pipe 18. \_\_\_\_\_  
each day?
19. How old were you when you began to smoke 19. \_\_\_\_\_  
a pipe regularly?.

I.D. \_\_\_\_\_

Individual's Medical History

IF DIABETIC (If not, go on to Question 22).

20. What type of treatment has the doctor prescribed for your diabetes? 20. \_\_\_/\_\_\_/\_\_\_  
 [What do you do for your DM?]  
 diet = 1, oral hypoglycemics = 2,  
 insulin = 3, other = 4
21. When was your diabetes diagnosed? 21. \_\_\_\_\_  
 (years ago)
22. Did your mother have diabetes? 22. \_\_\_\_\_  
 yes = 1, no = 2, don't know = 9
23. Did your father have diabetes? 23. \_\_\_\_\_  
 yes = 1, no = 2, don't know = 9
24. Do any of your brothers or sisters have diabetes? 24. \_\_\_\_\_  
 yes = 1, no = 2, don't know = 9  
 a) if yes, how many? 24a. \_\_\_\_\_
25. Do any of your children have diabetes? 25. \_\_\_\_\_  
 yes = 1, no = 2, don't know = 9  
 a) if yes, how many? 25a. \_\_\_\_\_
26. If married, does your spouse have diabetes? 26. \_\_\_\_\_  
 yes = 1, no = 2, don't know = 9
27. Ask women only:  
 a) how many pregnancies have you had 27a) \_\_\_\_\_  
 b) how many liveborn children do you have? 27b) \_\_\_\_\_

I.D. \_\_\_\_\_

Individual's Medical History Cont'd.

28. What do you usually weigh? (kg.) 28. \_\_\_\_\_  
(av. last 7-5 yrs)
29. What was your weight at 18 years of age? 29. \_\_\_\_\_
30. What is the most that you have ever weighed? 30. \_\_\_\_\_
- a) How long ago did you weigh this amount? 30a. \_\_\_\_\_
31. Do you think that you are now overweight? 31. \_\_\_\_\_
- yes = 1, no = 2, don't know = 3
32. If different from present weight, what do you think your ideal weight should be? 32. \_\_\_\_\_

I.D. \_\_\_\_\_

Medical History Questionnaire  
(Collected from medical charts)

DIABETES HISTORY: (if not diabetic, go on to Question 45).

Diagnosis

3. Oral Glucose Tolerance Test 33. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

if yes, results of fasting : \_\_\_\_\_ mg/dl  
2h: \_\_\_\_\_ mg/dl

n.b. plasma ( )  
whole blood ( )

34. Presence of (yes = 1, no = 2,  
don't know = 9)

- a) polyuria 34a. \_\_\_\_\_
- b) polydipsia 34b. \_\_\_\_\_
- c) rapid weight loss 34c. \_\_\_\_\_
- d) Ketonuria 34d. \_\_\_\_\_
- e) other 34e. \_\_\_\_\_

if other, please specify \_\_\_\_\_

35. Age at onset of symptoms (years) 35. \_\_\_\_\_

36. Age at diagnosis (years) 36. \_\_\_\_\_

37. Duration of diabetes (years) 37. \_\_\_\_\_

38. Prescribed therapy (yes = 1, no = 2,  
don't know = 9)

- a) diet 38a. ( ) \_\_\_\_\_  
note: calories/exchange/free
- b) oral hypoglycemics 38b. ( ) \_\_\_\_\_  
note: type, dosage, duration
- c) Insulin 38c. ( ) \_\_\_\_\_  
note: type, dosage, duration
- d) urine testing 38d. ( ) \_\_\_\_\_  
note: qd, qwk, never
- e) other, specify \_\_\_\_\_

I.D. \_\_\_\_\_

Medical History Questionnaire (cont'd.)

COMPLICATIONS - NATURE AND EXTENT

39. Ischemic heart disease

- a) angina 39a. \_\_\_\_\_
- b) myocardial infarction 39b. \_\_\_\_\_
- c) coronary bypass surgery 39c. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

40. Cerebrovascular

- a) transient ischemic attack 40a. \_\_\_\_\_
- b) amaurosis fugax 40b. \_\_\_\_\_
- c) stroke 40c. \_\_\_\_\_  
(not cerebral aneurysm)

yes = 1, no = 2, don't know = 9

41. Peripheral vascular disease

- a) claudication 41a. \_\_\_\_\_
- b) ischemic foot 41b. \_\_\_\_\_
- c) amputation 41c. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

42. Retinopathy

42. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

43. Diabetic nephropathy

43. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

- a) if yes, proteinuria = 1 + = 1 43a. \_\_\_\_\_
- 2 + = 2
- 3 + = 3
- 4 + = 4

b) 24h. urine protein = 43b. \_\_\_\_\_gms

I.D. \_\_\_\_\_

Medical History Questionnaire (con'td).

44. Diabetic neuropathy 44. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

n.b.:

- diabetic neuropathy

SUBJ: decreased sensation  
decreased vibration sense  
tingling or pain  
cotton ball sensation

OBJ: decreased light touch  
decreased/absent vibration sense

- hypertension

present if: male < 45 B/P > 140/90  
                  male > 45 B/P > 140/95  
                  female all ages B/P > 150/95

45. Other diseases

a) Hypertension 45a. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

b) if yes, and diabetic was BP present when diabetes diagnosed? 45b. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

c) hypercholesterolemia 45c. \_\_\_\_\_

cholesterol > 280 = 1  
cholesterol < 280 or less = 2  
no = 3  
don't know = 9

d) gall bladder disease 45d. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

if yes, specify nature of disease (especially cholelithiasis) and type of surgery, if any:

\_\_\_\_\_

\_\_\_\_\_

I.D. \_\_\_\_\_

Medical History Questionnaire (con'td.)

45. e) cancer

45e. \_\_\_\_\_

yes = 1, no = 2, don't know = 9

If yes, specify location and type: \_\_\_\_\_

\_\_\_\_\_

f) other diagnosed diseases

specify: \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

46. Present medications (other than insulin or oral hypoglycemics) - list type, dosage, duration:

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

I.D. \_ \_ \_ \_

ANTHROPOMETRIC MEASUREMENTS

47. Weight (to nearest 10th kg) 47. \_ \_ \_ . \_

48. Stature (to nearest mm) 48. \_ \_ \_ . \_

ALL OF THE FOLLOWING TO THE NEAREST mm. (no exam = 999)

49. Forearm skinfold 49. \_ \_ \_ . \_

50. Triceps skinfold 50. \_ \_ \_ . \_

51. Biceps skinfold 51. \_ \_ \_ . \_

52. Subscapular skinfold 52. \_ \_ \_ . \_

53. Midaxillary skinfold 53. \_ \_ \_ . \_

54. Suprailiac skinfold 54. \_ \_ \_ . \_

55. Abdominal skinfold 55. \_ \_ \_ . \_

56. Medial calf skinfold 56. \_ \_ \_ . \_

57. Chest girth 57. \_ \_ \_ . \_

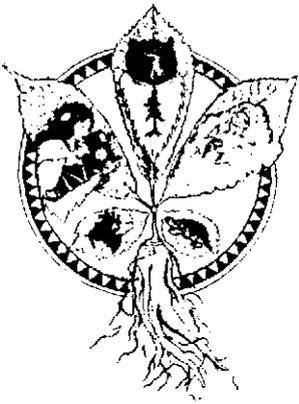
58. Abdominal girth 58. \_ \_ \_ . \_

59. Hip girth 59. \_ \_ \_ . \_

60. Arm circumference 60. \_ \_ \_ . \_

61. Calf circumference 61. \_ \_ \_ . \_

62. Blood sample taken \_\_\_\_\_



**Kateri Memorial Hospital Centre**  
**Tehsakotitsén : tha**  
P.O. Box 10, Kahnawake, Que., J0L 1B0  
Tel.: (514) 632-7620

October 9th, 1985

Dear

We would like to thank you for your participation in our study on diabetes conducted here at Kateri this summer. Your participation in the study helped to make it a wonderful success and has provided us with much needed information about diabetes here in Kahnawake.

The final results of the research will be available for publication in just under one year's time from now and we plan to be on the radio with that information.

Again, please accept this letter as our personal thanks for your generous cooperation.

Sincerely yours,

Louis T. Montour, M.D., C.M., C.C.F.P.

Ann C. Macaulay, M.D., C.C.F.P.

Naomi Adelson, R.N., B.A.

- APPENDIX B -

APPENDIX B - TABLE 1

Weight, in Kilograms,  
by Age Range, Diabetic Status and Sex

Age Range:	Mean ( $\pm$ Standard Deviation)			
	Male		Diabetic	Non-Diabetic
	Diabetic N=33#	Non-Diabetic N=48	N=48	N=76
35-45	88.83 (7.69)	82.25 (20.89)	91.20 (16.83)	72.32 (17.22)
46-55	92.22 (14.91)	85.87 (12.35)	99.59 (16.54)	72.27 (11.00)
56-65	86.39 (10.08)	82.96 (9.84)	80.35 (15.06)	71.38 (11.80)
66-75	81.83 (9.04)	79.79 (9.60)	71.15 (9.87)	74.08 (11.41)
76-90	80.50 (2.12)	86.70 (23.31)	60.30 (12.08)	57.67 (8.91)
Column Total	87.02 (11.23)	83.71 (14.25)	81.89 (18.66)	70.91 (12.85)

# 1 missing observation

APPENDIX B - TABLE 2

Stature, in Metres,  
by Age Range, Diabetic Status, and Sex

Age Range:	Mean ( $\pm$ Standard Deviation)			
	Male		Female	
	Diabetic N=33#	Non-Diabetic N=46	Diabetic N=48	Non-Diabetic N=76
35-45	1.71 (0.00)	1.72 (0.08)	1.61 (0.05)	1.58 (0.08)
46-55	1.71 (0.45)	1.70 (0.07)	1.56 (0.06)	1.60 (0.04)
56-65	1.70 (0.08)	1.69 (0.04)	1.56 (0.05)	1.56 (0.06)
66-75	1.67 (0.05)	1.72 (0.06)	1.56 (0.04)	1.54 (0.04)
76-90	1.62 (0.007)	1.71 (0.05)	1.55 (0.05)	1.54 (0.08)
Column Total	1.69 (0.06)	1.71 (0.06)	1.57 (0.05)	1.56 (0.06)

# 1 missing observation

APPENDIX B - TABLE 3

Triceps Skinfold, in Millimetres,  
by Age Range, Diabetic Status, and Sex

Age Range:	Mean ( $\pm$ Standard Deviation)			
	Male		Female	
	Diabetic N=34	Non-Diabetic N=46	Diabetic N=48	Non-Diabetic N=76
35-45	17.7 (9.9)	11.8 (7.3)	41.0 (12.2)	31.6 (14.4)
46-55	17.4 (6.0)	15.3 (6.3)	47.1 (10.0)	31.6 (8.8)
56-65	17.0 (7.4)	17.6 (7.0)	35.4 (11.0)	30.5 (7.3)
66-75	12.8 (5.9)	13.7 (7.8)	30.6 (8.8)	35.3 (7.2)
76-90	23.0 (9.9)	26.6 (17.1)	21.4 (6.9)	22.1 (3.6)
Column Total	16.8 (7.0)	16.5 (9.1)	35.6 (11.8)	31.1 (9.3)

APPENDIX B - TABLE 4

Subscapular Skinfold, in Millimetres,  
by Age Range, Diabetic Status, and Sex

Age Range:	Mean ( $\pm$ Standard Deviation)			
	Male		Female	
	Diabetic N=34	Non-Diabetic N=46	Diabetic N=48	Non-Diabetic N=76
35-45	28.3 (8.1)	23.88 (12.9)	46.4 (15.8)	39.6 (16.9)
46-55	33.8 (9.6)	27.1 (9.4)	52.0 (9.0)	35.3 (9.6)
56-65	30.5 (11.2)	27.6 (10.2)	39.1 (9.0)	38.2 (11.0)
66-75	25.8 (11.0)	30.0 (10.2)	32.0 (10.7)	40.0 (13.3)
76-90	24.0 (7.1)	27.0 (14.3)	24.2 (12.9)	23.0 (6.8)
Column Total	30.0 (10.3)	27.0 (10.6)	39.8 (13.5)	36.4 (12.8)

APPENDIX B - TABLE 5

Percent Glycosylated Hemoglobin  
by Age Range Diabetic Status and Sex

Age Range:	Mean ( $\pm$ Standard Deviation)#			
	Male		Female	
	Diabetic N=33	Non-Diabetic N=45	Diabetic N=48	Non-Diabetic N=75
35-45	5.92 (1.90)	4.36 (0.58)	8.04 (3.03)	4.92 (1.00)
46-55	7.72 (2.82)	4.85 (0.84)	7.80 (2.61)	5.38 (1.04)
56-65	7.13 (1.64)	5.06 (1.36)	6.77 (2.12)	5.11 (0.68)
66-75	6.70 (4.46)	5.42 (0.79)	6.90 (3.22)	5.46 (1.06)
76-90	5.21 (0.00)	4.96 (0.74)	7.36 (1.05)	5.52 (1.03)
Column Total	7.04 (2.61)	4.91 (0.95)	7.23 (2.47)	5.28 (0.95)

# 3 missing observations

APPENDIX B - TABLE 6

Mean Admixture Values at the ABO, Kell, Rh, Gm, and ACP Loci  
in Diabetic and Non-Diabetic Mohawk Indians\*

Locus:	Mean ( $\pm$ Standard Error of Mean)	
	Diabetic N=81#	Non-Diabetic N=119#
ABO	.2710 (.1194)	.2191 (.0884)
Kell	.2715 (.1912)	0§
Rh	.2804 (.1094)	.3258 (.0655)
Gm	.5339 (.0589)	.4759 (.0457)
ACP	0§	0§
Weighted Mean	.4342 (.0462)	.3951 (.0345)

\* From: Szathmary 1986, unpublished data

# 4 missing values in total

§ No Caucasian allele found

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