

HUMAN OBESITY AND ARCTIC ADAPTATION:

**Epidemiological Patterns, Metabolic Effects,
and Evolutionary Implications**

Theron Kue Hing YOUNG

BSc, MD, CM (McGill) MSc (Toronto) FRCP(Canada)

Linacre College

and

Institute of Biological Anthropology

Dissertation Submitted to the University of Oxford for the Degree of

Doctor of Philosophy

Trinity Term 1994



ABSTRACT

Title: Human Obesity and Arctic Adaptation: Epidemiological Patterns, Metabolic Effects, and Evolutionary Implications

Name: T.K.H. YOUNG

College: Linacre

Degree: D.Phil.

Term: Trinity 1994

The objective of this dissertation is to investigate the occurrence, determinants and consequences of obesity among the Inuit people in the central Canadian Arctic, based on the Keewatin Health Assessment Study (KHAS), conducted during 1990/91 in 8 Inuit communities in the Northwest Territories ($n=434$ adults aged 18yr+). Data from three other surveys are included for comparison: (1) the 1990 Manitoba Heart Health Survey among 2,200 predominantly Caucasian residents of the province of Manitoba; (2) the 1986-87 Northern Indian Chronic Disease Study among 704 Cree-Ojibwa Indians from northern Ontario and Manitoba; and (3) the 1990-91 Chukotka Chronic Disease Survey among 362 Chukchi and Inuit in coastal Chukotka in the Russian Far North.

Judged by both body mass index and two skinfold thicknesses, obesity among the Inuit in the Keewatin region is as prevalent as it is in the general North American population. This is a new development over the past two or three decades, the result of rapidly changing physical activity, diet and other lifestyles. Obesity is more prevalent among women, among whom there is also a higher prevalence of central fat patterning.

Age, education and non-smoking status (females only) are consistently identified as independent predictors of various obesity indices on multivariate analysis. While better educated men are more likely to be obese, the relationship is reversed in women, possibly due to the different sex roles and their associated stress levels in a rapidly acculturating and modernizing society.

When different categories of obesity indices are compared, there is a consistent pattern of an increasing trend in blood pressure and one or more of the lipids but no significant change in glucose or insulin level. This observation distinguishes the Inuit and Chukchi from Caucasians and Amerindians. Even where a relationship exists, as with triglycerides and HDL-cholesterol, the magnitude of response is also lower among the Inuit.

The differential effect of obesity on glucose, blood pressure and lipids in Inuit compared to non-Inuit suggests a type of selective insulin resistance, the underlying mechanism of obesity and several chronic diseases. The Inuit metabolism reflects their almost exclusive diet of fat and proteins. Apart from its public health importance, the study of Inuit obesity can shed some light into issues related to the peopling of the Americas: are the Inuit "exempt" from the "New World syndrome", and can the "thrifty genotype" explain the differential occurrence of diabetes among Arctic and Subarctic hunter-gatherers? It provides an opportunity to elucidate fundamental questions relating to the interaction of genetic and environmental factors in disease causation and distribution.

TABLE OF CONTENTS		i
ACKNOWLEDGMENTS		iii
LIST OF TABLES		iv
LIST OF FIGURES		vi
PART ONE	INTRODUCTION AND BACKGROUND	1
Chapter One	Introduction	1
Chapter Two	Obesity and its Health Effects	7
	2.1 Definition and Measurement	7
	2.2 Aetiology and Pathogenesis	12
	2.3 Obesity-Associated Diseases	15
	2.4 Sociocultural Aspects	26
Chapter Three	Inuit in the Canadian Arctic	29
	3.1 Physical and Social Environment	29
	3.2 Human Biology and Demography	38
	3.3 Pattern of Mortality and Morbidity	81
PART TWO	DESIGN AND METHODS	97
Chapter Four	The Keewatin Health Assessment Study	97
	4.1 Study Setting and Objectives	97
	4.2 Sample Selection and Participation Rate	100
	4.3 Survey Procedures and Data Collection	101
	4.4 Data Management and Analysis	115
Chapter Five	Comparison Populations	133
	5.1 Siberian Eskimos and Chukchi	133
	5.2 Subarctic Cree and Ojibwa Indians	141
	5.3 General Population of Manitoba	145

PART THREE	RESULTS AND DISCUSSION	152
Chapter Six	Obesity and Regional Fat Patterning	152
	6.1 Prevalence and Distribution	152
	6.2 Sociocultural Determinants	164
	6.3 Metabolic Consequences	170
	6.4 Between-Population Comparisons	179
Chapter Seven	Conclusions and Synthesis	191
	7.1 Epidemiological Patterns	191
	7.2 Metabolic Pathways	199
	7.3 Evolutionary Perspectives	205
BIBLIOGRAPHY		216

ACKNOWLEDGMENTS

I wish to thank Professor Geoffrey Harrison for his guidance during all phases in the preparation of this dissertation. I particularly benefitted from his insightful comments on the data analysis and presentation. The staff and students at the Institute of Biological Anthropology provided me a congenial, collegial and intellectually stimulating environment in which to pursue my D.Phil.

The research described in this thesis could not have taken place without my co-investigators, particularly Drs. Michael Moffatt and John O'Neil, at the University of Manitoba. The Department of National Health and Welfare of Canada provided the research funds. The people, local government, and health board in the Keewatin Region, Northwest Territories, offered the research team their hospitality and support.

Professor Roy Shephard of the University of Toronto, and Dr. Otto Schaefer, former Director of the Northern Medical Research Unit, Department of National Health and Welfare, read the manuscript and corrected various errors of fact and interpretation. They have pioneered much of the research into human biology of the Inuit in the Canadian Arctic, which I hope to continue.

LIST OF TABLES

- 2.1 Metabolic changes during evolution of obesity to diabetes **p.20**
- 3.1 Sources of income in two Inuit communities, NWT, 1950s and 1960s **p.36**
- 3.2 Selected socioeconomic indicators, NWT Inuit and all Canadians, 1986 Census (percent of population aged 15 and above) **p.37**
- 3.3 Historical data series (pre-1960s) on mean height and weight of male, adult Eskimo/Inuit populations in the circumpolar zone **p.47**
- 3.4 Data from anthropometric surveys among Eskimo/Inuit populations in the circumpolar zone, post-1960s **p.48**
- 3.5 Body composition of Inuit in Igloolik, 1970-71 **p.54**
- 3.6 Standardized mortality ratio for selected causes, NWT Inuit compared to all Canadians **p.84**
- 4.1 The Keewatin Health Assessment Study: Sample selection and response rate **p.101**
- 4.2 Contents of the Individual Survey (IS) **p.103**
- 4.3 Distribution of anthropometric variables **p.119**
- 4.4 Correlation matrices for anthropometric variables **p.120**
- 4.5 Distribution of metabolic and physiologic variables **p.121**
- 4.6 Distribution of log-transformed continuous variables **p.122**
- 4.7 Frequency distribution of selected sociodemographic variables, all ethnic groups combined **p.124**
- 4.8 Frequency distribution of selected sociodemographic variables among Inuit **p.125**
- 4.9 Frequency distribution of cigarette smoking status **p.127**
- 4.10 Cross-tabulation of two variables measuring physical activity **p.129**

- 4.11 . Cross-tabulation of time spent on the land during spring/summer and autumn/winter **p.130**
- 4.12 Frequency distribution of past medical history **p.131**
- 4.13 Family history of selected chronic diseases **p.132**

- 5.1 Distribution of total number of individuals (A) eligible (B) randomly selected and (C) participated in survey by age and sex **p.138**
- 5.2 Components of the Chukotka Chronic Diseases Study **p.140**
- 5.3 Contents of the Northern Indians Chronic Disease Study **p.144**

- 6.1 Means (and standard errors) of anthropometric measures by age and sex among Keewatin Inuit **p.152**
- 6.2 Comparison of sex-specific and age-adjusted mean body mass index by selected sociocultural variables: Keewatin Inuit **p.165**
- 6.3 Comparison of sex-specific and age-adjusted mean skinfolds by selected sociocultural variables: Keewatin Inuit **p.166**
- 6.4 Comparison of sex-specific and age-adjusted mean circumferences by selected sociocultural variables: Keewatin Inuit **p.167**
- 6.5 Significant independent predictors of obesity/central fat patterning indices established by stepwise multiple regression analyses: Keewatin Inuit **p.169**
- 6.6 Comparison of sex-specific and age-adjusted means of selected metabolic variables according to risk categories of body mass index (BMI) and waist-hip ratio (WHR): Keewatin Inuit **p.171**
- 6.7 Comparison of sex-specific and age-adjusted means of selected metabolic variables according to quartiles of obesity indices: Keewatin Inuit **p.173**
- 6.8 Significant independent predictors of selected metabolic variables established by stepwise multiple regression analyses: Keewatin Inuit **p.176**
- 6.9 Summary of comparison of sex-specific, age-adjusted means of selected metabolic variables according to risk categorie of BMI and WHR: Comparison populations **p.180**

- 6.10 Comparison of sex-specific and age-adjusted means of selected metabolic variables according to quartiles of obesity indices: Comparison populations **p.181**
- 6.11 Summary of significant independent predictors of selected metabolic variables established by stepwise multiple regression analyses: Comparison populations **p.183**
- 6.12 Magnitude of standardized coefficients of regression of BMI on metabolic variables: Comparison populations **p.189**

LIST OF FIGURES

- 1.1 Map of Northwest Territories, Canada, showing communities included in the Keewatin Health Assessment Study **p.6**
- 2.1 Alternative classifications of body composition compartments **p.8**
- 2.2 Components and determinants of daily energy expenditure **p.13**
- 2.3 Model of causal relationship between insulin resistance and obesity, diabetes, hypertension, atherosclerosis and dyslipiæmia **p.17**
- 2.4 Metabolic events involved in insulin action and resistance **p.18**
- 2.5 Model of hyperinsulinæmia, insulin resistance and abdominal obesity **p.24**
- 2.6 An integrative view of obesity, insulin resistance and associated conditions **p.25**
- 2.7 Interrelationships between socioeconomic status and obesity **p.28**
- 3.1 Map of the Arctic showing traditional territories of the Eskimo/Inuit **p.32**
- 3.2 Dendrogram showing genetic affinities among 11 Asiatic and North American populations **p.42**
- 3.3 Dendrogram showing genetic affinities between 42 aboriginal populations around the world **p.45**
- 3.4 Comparison of mean weight-for-age between Inuit, Indian and Canadian adults, Nutrition Canada Survey, 1972 **p.51**

- 3.5 Comparison of age-sex-specific mean weight and height between Inuit and Canadian adults, Nutrition Canada Survey, 1972 **p.52**
- 3.6 Prevalence of obesity risk categories based on ponderal index, Inuit and Canadian adults, Nutrition Canada Survey, 1972 **p.55**
- 3.7 Change in mean body mass index and sum of three skinfolds in Igloolik between 1970, 1980, and 1990 **p.57**
- 3.8 Median intake of calories and proteins, Inuit and Canadians, Nutrition Canada Survey, 1972 **p.65**
- 3.9 Distribution of serum cholesterol level: Inuit and Canadians, Nutrition Canada Survey, 1972 **p.69**
- 3.10 Growth of Inuit population in Greenland, Alaska and the Northwest Territories, 1950-80 **p.72**
- 3.11 Crude birth rate among NWT Inuit and all Canadians, 1955-87 **p.73**
- 3.12 Age-sex composition of NWT Inuit and Canadian populations, 1941, 1961, 1981 **p.76**
- 3.13 Infant mortality rate among NWT Inuit and all Canadians, 1955-87 **p.79**
- 3.14 Neonatal and postneonatal mortality rate among NWT Inuit and all Canadians, 1961-85 **p.80**
- 3.15 Crude mortality rate for selected causes, NWT Inuit, 1964-88, and percent distribution by cause, 1979-88 **p.83**
- 3.16 Trend in tuberculosis incidence: NWT Inuit and all Canadians, 1960-86 **p.86**
- 3.17 Prevalence of diagnosed diabetes in the circumpolar indigenous populations of Chukotka (Russia), Alaska (USA), Yukon and NWT (Canada) **p.90**
- 4.1 The Keewatin Region, Northwest Territories, Canada **p.99**
- 4.2 Anthropometric measurement techniques **p.105**
- 4.3 Pamphlet in Inuktitut syllabics promoting the Keewatin Health Assessment Study **p.113**
- 4.4 Analysis plan: dependent and independent variables **p.117**

- 5.1 Map of Chukotka showing communities participating in the Chukotka Chronic Diseases Study **p.137**
- 5.2 Map of northwestern Ontario and northeastern Manitoba showing communities participating in the Northern Indians Chronic Disease Study **p.143**
- 5.3 Map of Manitoba showing communities included in the Manitoba Heart Health Survey **p.148**
- 6.1 Frequency distribution of body mass index (BMI) by sex **p.156**
- 6.2 Frequency distribution of waist-hip ratio (WHR) by sex **p.157**
- 6.3 Frequency distribution of triceps skinfolds by sex **p.158**
- 6.4 Frequency distribution of subscapular skinfolds by sex **p.159**
- 6.5 Prevalence of obesity (BMI 26-30 and BMI >30) by age and sex **p.161**
- 6.6 Mean body mass index (BMI) and waist-hip ratio (WHR) by age and sex **p.162**
- 6.7 Mean subscapular and triceps skinfolds by age and sex **p.163**
- 6.8 Mean systolic and diastolic blood pressure levels by categories of BMI **p.187**
- 6.9 Mean triglycerides and HDL-cholesterol levels by categories of BMI **p.188**
- 7.1 Relative ranking of body mass index of Keewatin Inuit among populations in INTERSALT STUDY **p.193**
- 7.2 Relative ranking of body mass index of Keewatin Inuit among populations in MONICA STUDY **p.194**

PART ONE INTRODUCTION AND BACKGROUND

CHAPTER ONE INTRODUCTION

The deposition of subcutaneous fat as insulation is a major adaptive mechanism to the cold environment of the Arctic among warm-blooded animals. Human beings appear to be an exception. Among the indigenous Inuit (or Eskimo) population, who have occupied the Arctic regions of North America for at least 5,000 years, studies in the past have generally concluded that obesity had not been prevalent. It has been suggested that cultural innovations such as efficient clothing and shelter, vigorous physical activity and a high protein and fat diet obviate the need for subcutaneous fat to conserve body heat. Traditionally, Inuit have also been "spared" the deleterious health effects of obesity such as diabetes, coronary heart disease, and hypertension. Over the past two decades, substantial social and cultural changes have occurred among the Inuit - sedenterization in towns and villages, reduction in traditional hunting activities, and declining dependence on the land and marine mammal-based diet. Given such a background, three questions can be posed:

- (1) Is obesity prevalent among Inuit today and has it significantly increased?
- (2) Is such obesity as occurs metabolically "harmful"?
- (3) What are the evolutionary implications of the emergence of obesity and associated diseases in this Arctic population?

The objective of this dissertation is to address these three questions, based on the results of a population health survey among the Inuit in the Central Arctic, supplemented by a thorough review of the relevant literature. It should be noted that "obesity" is used as an operational term defined in accordance with several existing criteria. Its appropriateness to the Inuit is the subject of this dissertation.

The Keewatin Health Assessment Study (KHAS) was a comprehensive community health survey conducted during 1990/91 in 8 Inuit communities in the Keewatin Region of the Northwest Territories (NWT), Canada (Fig.1.1). While the KHAS covered a wide variety of areas in all age groups, only adults aged 18 and above (n=434) and those variables relevant to the three research questions above are analyzed and presented in this dissertation. Details on the design and methods of this study are provided in Part Two. In addition, data from three recently completed population surveys are also included for comparison:

- (1) the Manitoba Heart Health Survey (MHHS), conducted during 1990 on a sample of the total population of the province of Manitoba as part of a national survey of cardiovascular risk factors (n=2,200);
- (2) the Northern Indian Chronic Disease Study (NICDS), conducted during 1986-87 among the Algonkian speaking Cree and Ojibwa Indians inhabiting 6 communities in the subarctic boreal forest of northern Ontario and Manitoba (n=704);
- (3) the Chukotka Chronic Disease Survey (CCDS), conducted during 1990-91 in 4 Chukchi and Inuit villages in the Chukotka peninsula of the Russian Far North by the Siberian Branch, Academy of Medical Sciences in Novosibirsk (n=362).

I was one of three co-principal investigators of the KHAS and MHHS, jointly responsible for the design of the studies and their direction in the field. I was the sole principal investigator of the NICDS. I had close liaison with the Russian investigators of the CCDS through joint planning meetings and site visits. The rationale for using these comparison groups is that the MHHS provides data on a predominantly Caucasian, southern Canadian population, the NICDS a linguistically, culturally and ecologically distinct Canadian aboriginal group, and the CCDS

a second tundra-living aboriginal group with close genetic affinities with the Inuit. All four studies are comparable in their data collection protocol for the key variables of interest.

Organization of Dissertation

This dissertation is divided into three Parts: **Introduction and Background; Design and Methods; and Results and Discussion.** In Part One, Chapter Two reviews the definition, measurement, etiology and pathogenesis of obesity in human beings and its sociocultural and health impact. Chapter Three provides a summary of existing knowledge about the human biology and pattern of health and disease among the Inuit and the specific physical and social environment in which they live. Part Two describes in detail the methodology used in the Keewatin Health Assessment Study (Chapter Four) and the three other studies from which comparative data have been derived (Chapter Five). In Part Three, results relating to the prevalence, determinants and consequences of obesity among the Inuit are presented and the extent to which the three questions posed earlier in this chapter have been answered is discussed. The statistical data are placed within a theoretical context of adaptation to the Arctic environment and the role of diseases in past and future microevolution of this population.

A Note on Terminology

The indigenous population which inhabits the Canadian Arctic has traditionally been referred to by anthropologists and the general public as *Eskimos*. Over the past two decades, this term has been replaced by *Inuit*, meaning "the people". [*Inuit* is the plural form, not Inuits; the singular is *Inuk*]. It is widely believed that *Eskimo* is a derogatory term, originally used by Algonkian Indians for "eaters of raw meat", an inaccurate interpretation not supported by linguists. In Alaska, *Eskimo* is still an acceptable term for self-identification, although

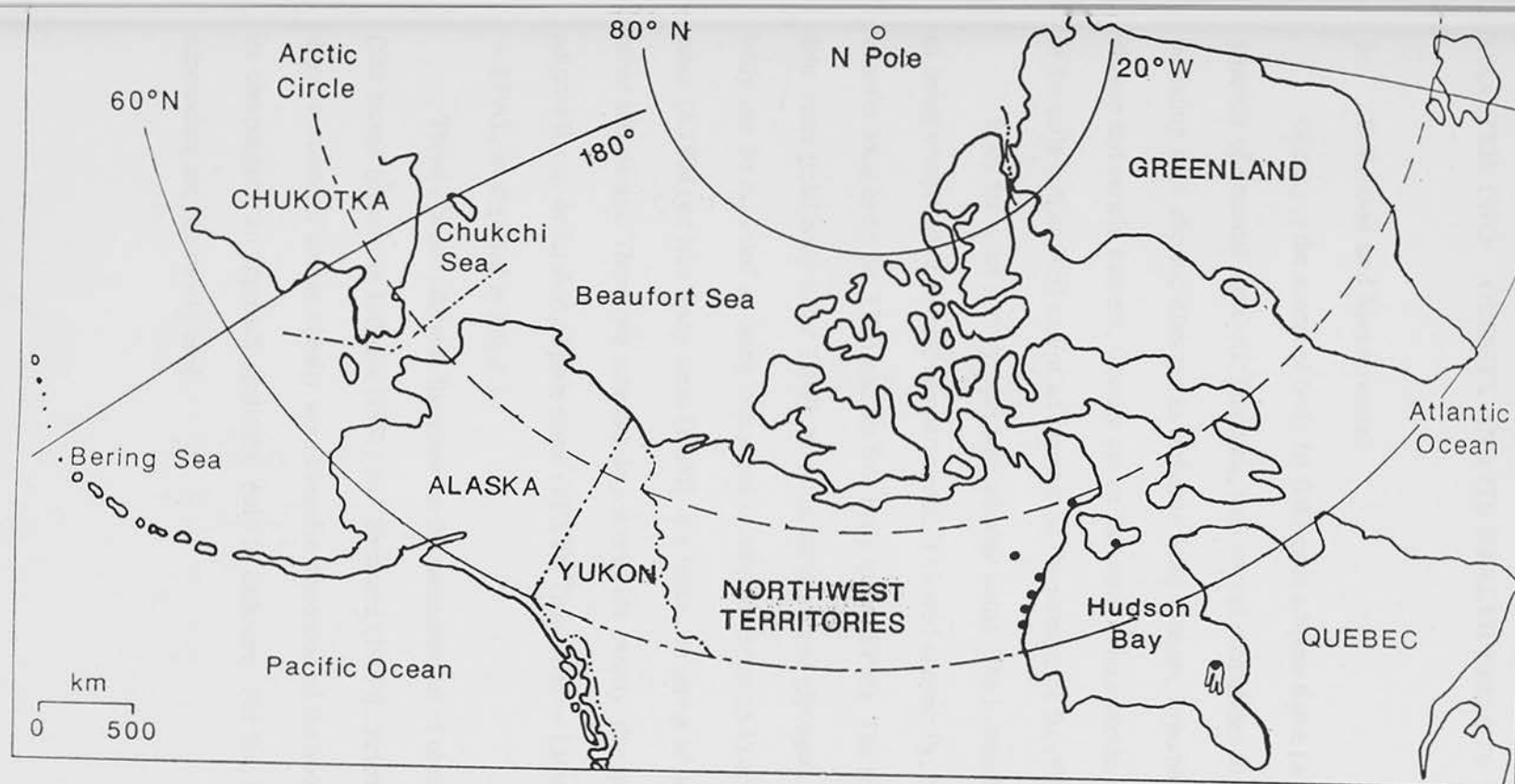
increasingly the terms *Yupik* and *Iñupiat* are used for specific regional/linguistic groups. In addition, the term *Alaska Native* is used collectively when referring to all Eskimos, Indians and Aleuts living in that state. In Greenland, the only geographical entity where the Eskimos have achieved nationhood, the term *Greenlander* (or *Kalaallit*) is preferred. In this dissertation I have used *Inuit* and *Eskimo* interchangeably, favouring *Inuit* when referring to present-day Canadians but retaining *Eskimos* when referring to historical populations or quoting the contemporary literature. I have also used the term *Eskimo/Inuit* when describing the group "continentally".

Ethnologists have generally included Eskimo/Inuit as one of the North American Indian groups. The Eskimos, for example, are included in the Smithsonian Institution's *Handbook* series on North American Indians (Damas 1984). However, general usage tends to make a distinction between North American Indians or Amerindians, and Eskimos. To avoid confusion, I have used the term *Native American* when referring to all the indigenous populations in North America, and restrict *North American Indian*, *Amerindian*, and *Indian* to all indigenous groups except the Eskimos. As discussed in more detail in Section 3.2, the Eskimo/Indian distinction is important in the debate regarding the origins and affinities of Native Americans.

An explanatory note is also required for the use of the term *adaptation*. The concept of adaptation and adaptability is central to human biology (Harrison et al 1988). Many definitions are proffered in textbooks of anthropology, physiology, and ecology, and in works which deal with their interfaces (eg. Prosser 1964, Folk 1966, Damon 1975, Moran 1982). In the broadest sense, adaptation can be applied to any property of an organism which favours survival in a specific environment (Prosser 1964). This is the usage common in the biological literature. Some physical anthropologists, however, tend to restrict the term to the product of evolutionary

changes generated by natural selection on genetic variability. Thus for them *adaptation* is to be distinguished from *adjustment*, which occurs during an individual's lifetime. Such adjustment may be acclimatory - reversible physiological adjustments to an environmental stress; developmental - nonreversible physiological and morphological changes during early growth and development; and regulatory - relatively rapid behavioral, social or cultural responses by the physical organism (Moran 1982). The laboratory-based physiologist further distinguishes *acclimation* from *acclimatization*: the former referring to compensatory alteration in an animal under laboratory conditions altered for a single stressor, while the latter refers to changes under natural conditions where multiple factors vary (Prosser 1964). It should be recognized that *cultural adaptation*, while obviously non-genetic, is nevertheless a long-range, transgenerational process (Alland 1970).

Thus a rather confusing array of meanings have become attached to the term adaptation, depending on whether it involves genetic or non-genetic mechanisms, at the level of the individual or the population, the reversibility and speed of the changes, the number of environmental variables involved, and the stage of development when the changes occur. In this dissertation I have opted to use the term "adaptation" in its broader context. The definition offered by Baker (1984) in his Huxley Memorial Lecture to the Royal Anthropological Institute is thus particularly helpful: Adaptation is *any biological or cultural trait which aids the biological functioning of a population in a given environment*. It includes genetic adaptation and physiological acclimatization but excludes those sociocultural adjustments which have no demonstrable effects on human biology.



— international boundary
 - - - provincial/territorial boundary

• Communities included in the Keewatin Health Assessment Study

Fig.1.1 Map of Northwest Territories, Canada, showing communities included in the Keewatin Health Assessment Study

CHAPTER TWO OBESITY AND ITS HEALTH EFFECTS

2.1 Definition and Measurement

Obesity is the excess of body fat (**BF**) or of adipose tissue (**AT**). While BF and AT serve a variety of essential metabolic functions, their excess predisposes to significant health problems, including such chronic diseases as ischemic heart disease, hypertension, diabetes, gallbladder disease and certain cancers. Obesity can be the result of an increase in the size (ie. hypertrophy) of fat cells (adipocytes) and/or an increase in the number of fat cells (ie. hyperplasia).

Body fat is not synonymous with adipose tissue. The human body can be conceptualized as being composed of several compartments. If viewed chemically, these compartments are: fat mass(or total body fat **TBF**) and fat-free body mass (**FFM**). The latter can be further divided into water (total body water **TBW**), proteins, minerals and glycogen. If viewed anatomically the body can be regarded as being composed of adipose tissue (**AT**) and adipose tissue-free body mass (**ATFM**) or lean body mass (**LBM**), the latter consisting of bones, skeletal muscles and other soft tissues. These two schemes do not coincide exactly, due to the presence of non-fat in adipose tissue, or fat-free adipose tissue (**FFAT**). Thus $FFM = LBM + FFAT$ and $AT = TBF + FFAT$, as depicted in Fig.2.1.

There is a considerable literature on the measurement of obesity and body composition. [See recent reviews in Lukaski (1987) and Shephard (1991)]. Strictly speaking, the only truly direct measure of human obesity would involve dissection of the body and chemical analysis of its components, an approach applicable only to cadavers. For live human beings, three basic approaches are commonly used:

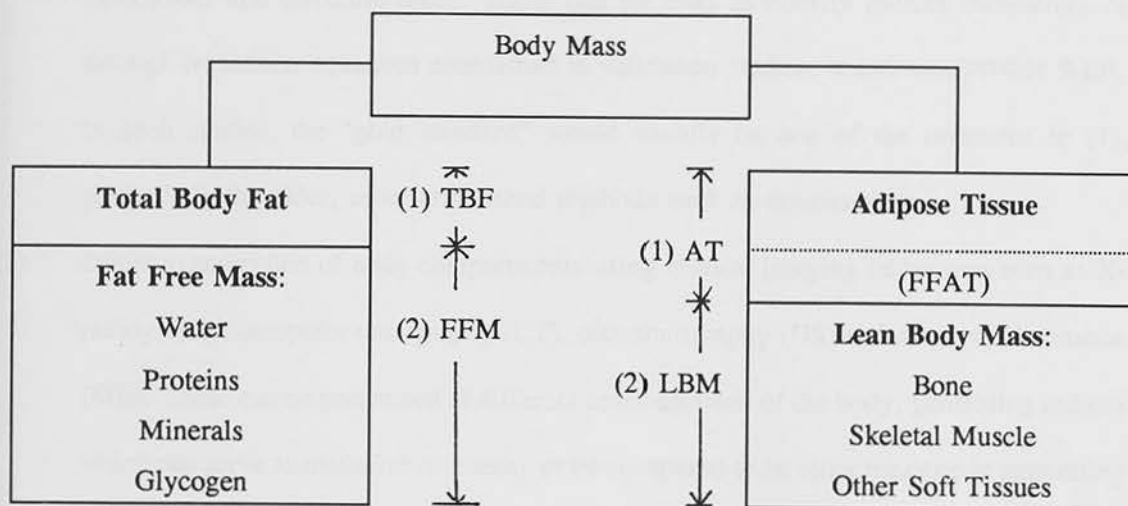


Fig.2.1 Alternative classification of body composition compartments

- (1) Measures which provide estimates of percent body fat (%BF) by estimating the various compartments. Models with different degrees of complexity may consist of two, three or more compartments. A whole array of techniques have been developed, including hydrodensitometry, isotope dilution, neutron activation, dual photon absorptiometry, total body electrical conductivity, bioelectrical impedance and infrared interactance. It should be noted that these methods require the acceptance of various assumptions, such as the absence of variation within a compartment, that TBW is a fixed proportion (73.2%) of FFM, that proteins consist of 16% nitrogen and all body nitrogen exists in the form of proteins, that the density of fat is 0.9 gm/ml and that of FFM 1.10 gm/ml, and so on.

- (2) Measures which are based on anthropometry such as height and weight, skinfold thicknesses and circumferences. These can be used as obesity indices themselves or through regression equations established in validation studies, to estimate/predict %BF. In such studies, the "gold standard" would usually be one of the measures in (1), particularly the older, more established methods such as densitometry.
- (3) Direct visualization of body compartments using clinical imaging techniques such as X-radiography, computer tomography (CT), ultrasonography (US) and magnetic resonance (MR). These can be performed at different cross-sections of the body, generating indices which can serve as measures of obesity or be compared to an older measure in generating prediction equations for %BF.

Most of the methods listed in (1) and (3) are cumbersome and not suitable for large population surveys. Indices based on height and weight are attractive because of their ease of measurement and reproducibility. The **body mass index** (BMI) or Quetelet's index is widely used in epidemiology. In most populations it correlates only minimally with height and thus is not affected by the amount of muscle and bone. On the other hand it correlates well with weight, skinfolds and body composition measures (Revicki and Israel 1986, Micozzi et al 1986). However, Garn and others (1986) cautioned that BMI was not completely independent of height, particularly in younger ages, and correlated with lean body mass as much as body fat. Of particular relevance to the Eskimo/Inuit is that BMI correlates with relative sitting height and tends to be higher in short-legged people. That the Eskimo/Inuit are generally "short-legged" has been recognized in the older physical anthropology literature [reviewed in Szathmary 1984)]. As recently as the Nutrition Canada Survey during the early 1970s, Canadian Inuit adults still

showed higher relative sitting height than Canadians nationally in all age-sex groups (Canada, Department of National Health and Welfare 1980:108).

While **skinfold thicknesses** provide more direct measures of subcutaneous fat than height-and-weight-based indices, they are technically more demanding and more liable to interobserver variation. There is also considerable variation in fat tissue deposits in different body sites. Moreover, skinfolds have been shown to be highly correlated with densitometric indices of body fat (Revicki and Israel 1986, Mueller and Stallones 1981).

Data on body composition among Native Americans are limited. The correlation of BMI with such indices as %BF, TBF and FFM was demonstrated in the Pima Indians in the southwestern United States (Knowler et al 1991). Among the Inuit, body composition studies in Igloolik were reported by Shephard and his team, using deuterium oxide dilution method in 1970 (Shephard, Hatcher and Rode 1973) and hydrodensitometry in 1990 (Rode and Shephard 1992). They also provided regression equations to predict body density based on the logarithm of the sum of three skinfolds (subscapular, triceps and suprailiac).

Few epidemiological studies linking obesity with adverse health outcomes used %BF or TBF as the predictor variable. Most used BMI or "percent overweight" based on comparisons with actuarial standards. There are no universally accepted criteria for obesity, although various investigators have proposed guidelines based on the risks of complications. For BMI, values of 25-30 have been defined as "overweight" and those over 30 as "obese" (Bray 1989). Such cut-off points are widely used in population studies, especially in international comparisons (eg. Millar and Stephens 1987, Gurney and Gorstein 1988). Garrow introduced a system of "grades"

for obesity: grade 0 for BMI from 20-24.9, grade I from 25 to 29.9, grade II from 30-40 and grade III, greater than 40 (Garrow 1988).

Increasingly it is recognized that it is the regional distribution of body fat and not simply overall obesity which has important health implications (Björntorp et al 1988). There are two main types of fat patterning: (1) *central*, also called *abdominal*, *android*, *truncal*, or *upper-body*; and (2) *peripheral*, *gluteo-femoral*, *gynaecoid*, *centripetal*, or *lower-body*. Commonly used measures of regional fat patterning are the **ratio of waist to hip circumference** (WHR) and the **ratio of subscapular to triceps skinfolds** (STR). Studies have shown that these two indices are not highly correlated and are independent predictors of various measures of glucose and lipid metabolism (Haffner et al 1987, Young and Sevenhuysen 1989). As in BMI, cut-off points are problematic, although for WHR the values of 0.9 for men and 0.8 for women have been suggested (Bray 1989). Alternatively, individual skinfolds such as subscapular have been used to represent "central" and triceps "peripheral" fat. Different skinfolds from a variety of sites in the body can also be aggregated by principal component analysis to represent the different fat patterns (Mueller and Stallones 1981).

A central fat patterning is in fact composed of a subcutaneous and an intra-abdominal or visceral component. Advances in computed tomography (CT) offer a non-invasive method to visualize and measure intra-abdominal fat. While such a method cannot be used in large-scale population screening, it has been shown that the ratio of the CT-determined area of intra-abdominal fat to the area of subcutaneous fat at the umbilicus level correlated well with the WHR, after adjusting for age and BMI (Ashwell et al 1985). Other studies have also shown that

a high CT-based intra-abdominal/subcutaneous ratio was associated with the usual abnormalities of glucose and lipid metabolism (Fujioka et al 1987).

A useful classification system has been proposed by Bouchard (1991) in recognition of the distinction between general and central fat, and between subcutaneous and visceral fat. Type I refers to excess body mass or percentage of fat; type II, excess subcutaneous truncal-abdominal fat; type III, excess abdominal visceral fat; and type IV, excess gluteofemoral fat.

2.2 Aetiology and Pathogenesis

Obesity can be regarded simplistically as a chronic dynamic imbalance between energy intake and energy expenditure (Ravussin and Swinburn 1992). An excess of the former (as in overnutrition) and/or a reduction in the latter (such as through physical inactivity) could lead to obesity. The precise relationship is difficult to establish. The pathophysiological mechanisms of obesity are complex, involving a host of regulatory hormones and enzymes and the actions of the autonomic nervous system. At the level of the organism one is concerned, at the energy intake end of the equation, with total caloric intake, the macro and micronutrient composition of food, and neurobehavioural aspects of taste, appetite, and satiety. On the expenditure side, important factors include basal and resting metabolic rate, thermogenesis due to food, stress or cold, and the effects of exercise and habitual level of physical activity (Fig.2.2). At the cellular and molecular level - within the individual adipocyte -the storage and release of energy is expressed in terms of the interplay between the uptake and disposal of glucose on the one hand and fat metabolism on the other, with insulin playing a major role. Within adipose tissue, fat cells around the waist and in the abdomen are more active metabolically than those in the thighs

and buttocks. [For a state-of-the-art review of the immense literature, one is referred to the 800-page volume edited by Björntorp and Brodoff (1992)].

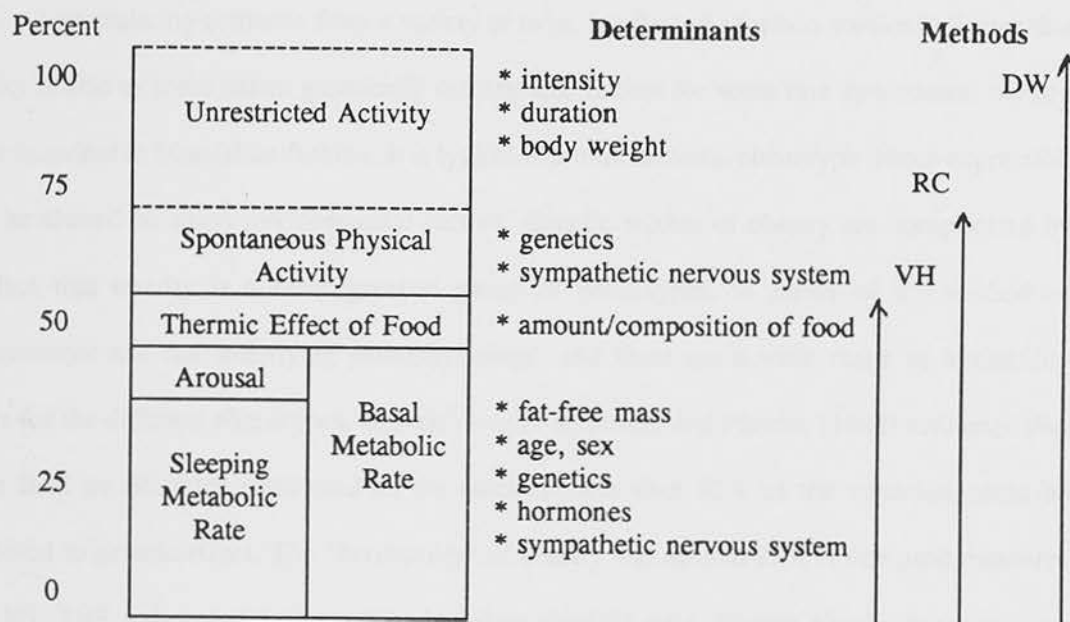


Fig.2.2 Components and determinants of daily energy expenditure

(Based on Ravussin and Swinburn 1992)

Note: VH ventilatory hood, RC respiratory chamber, DW Double-labelled water
BMR: 50-70% of daily energy expenditure; thermic effect of food 10%; physical activity 20-40%

Most of the metabolic studies were conducted in laboratory settings with the primary objective of discerning basic "universal" biological mechanisms, with little attention to potential ethnic or racial differences. Much of the accumulated knowledge, however, has been derived from studies among the Pima Indians, a population with an excessive prevalence of obesity

which had undergone an explosive epidemic of diabetes over the past four decades. The extensive Pima data from *in vitro* fat cell studies, metabolic ward investigations, and energy balance analyses have been summarized by Howard and others (1991).

Accumulating evidence from a variety of twin, family and adoption studies indicates that obesity is also to some extent genetically determined. Except for some rare syndromes, obesity is not inherited in Mendelian fashion. It is typical of a multifactorial phenotype whose expression may be altered by many environmental factors. Genetic studies of obesity are complicated by the fact that obesity is a heterogeneous group of phenotypes, in terms of the method of measurement and the underlying pathophysiology, and there are a wide range of heritability levels for the different phenotypes. In their review, Bouchard and Pérusse (1988) estimated that when BMI or skinfolds were used as the measure, less than 10% of the variation could be attributed to genetic effect. The "heritability" of obesity was around 25% if one used measures of %BF, TBF and central fat patterning based on skinfold ratio. Human obesity genotypes are likely complex multigenic systems with networks of gene-gene and gene-environment interactions (Bouchard and Pérusse 1993).

A study of weight gain among the Pima showed that low 24-hour energy expenditure (which incorporates the metabolic rate at rest, the thermogenic effect of food and the caloric cost of physical activity) increased fourfold the risk of gaining at least 7.5 kg over 2 years (Ravussin et al 1988). There was also familial aggregation of low energy expenditure, which may contribute to the observed familial aggregation of obesity.

Maternal diabetes is an important determinant of obesity in the offspring. The offspring of women who were diabetic during pregnancy, regardless of the degree of maternal obesity,

were more likely to be heavier at birth and develop obesity during childhood and young adulthood compared to those born to non-diabetic or "pre-diabetic" women. Even if the birth weight was normal for gestational age, offspring of diabetic women still became heavier eventually (Pettitt, Knowler et al 1987).

The data supporting the association between childhood obesity and subsequent obesity in adulthood are inconsistent. The relationship depends on the age of onset of obesity and the type of indicator used (Johnston 1985).

2.3 Obesity-Associated Diseases

Extensive data on the serious health effects of obesity, which include premature mortality, coronary heart disease, stroke, diabetes and various cancers, have been accumulated and reviewed in several recent symposia (NIH Consensus Development Panel 1985, Björntorp et al 1988). Several prospective cohort studies, such as the Framingham, Gothenburg and Mexican-American studies and life insurance actuarial data, have provided strong and consistent support for the independent aetiologic role of obesity, particularly central obesity, in these conditions. While the pathogenetic pathways remain to be elucidated, there is consistent evidence that obesity is directly related to the development of a disadvantageous coronary risk profile of plasma lipids and blood pressure [reviewed in Hubert (1986), Donahue et al (1990)].

Obesity is considered one of the strongest risk factors of diabetes mellitus. In a review of diabetes and obesity, Barrett-Connor (1989) pointed out the complexity of the relationship and listed several issues that still remained to be clarified: the existence of a threshold effect; the relative importance of current vs previous obesity, childhood vs adult-onset obesity, and degree

vs duration of obesity; the independent contributions of overall obesity and regional fat distribution; and the role of weight loss in reducing the risk of diabetes and its complications.

There is increasing evidence to suggest that **insulin resistance** may be the key primary metabolic defect that leads to such related disorders as obesity, hypertension, diabetes, dyslipidaemia and atherosclerosis, which tend to co-occur or cluster in the same individuals. The inter-relationships between insulin resistance and the pentad of metabolic disorders can be represented by a "metabolic cascade", an example of which was presented by DeFronzo and Ferrannini (1991). Fig.2.3 is a modification and simplification of this model.

Insulin resistance represents a metabolic state whereby body tissues have become less sensitive to the action of insulin such that increasingly more insulin is required to do the same job. Insulin resistance is demonstrated *in vivo* by an increased insulin response during the oral glucose tolerance test (OGTT) above the normal range. In recent years, the more elaborate "hyperinsulinaemic euglycaemic clamp" has become the standard tool, which actually measures how much glucose disposal has been reduced. [It works as follows: the test subject is continuously infused with insulin until endogenous glucose production is suppressed. The amount of glucose which must then be infused to maintain euglycaemia thus represents the amount of glucose disposal that has taken place. The use of isotope tracer can also allow the estimation of endogenous glucose production].

Insulin resistance (IR) is primarily a disorder of carbohydrate and fat metabolism. [The basic science and clinical literature in this field is extensive. The following discussion is heavily dependent on several recent reviews: Dinneen et al (1992), DeFronzo et al (1992), Felber et al (1993), V.R.Young and Marchini (1990)]. In order to understand IR, one must first understand

the different tasks performed by insulin. These inter-linked processes are summarized in Fig.2.4. IR may occur because of defects in the binding of insulin with its receptors on the plasma membrane and/or one or more components of various regulatory intracellular enzyme systems.

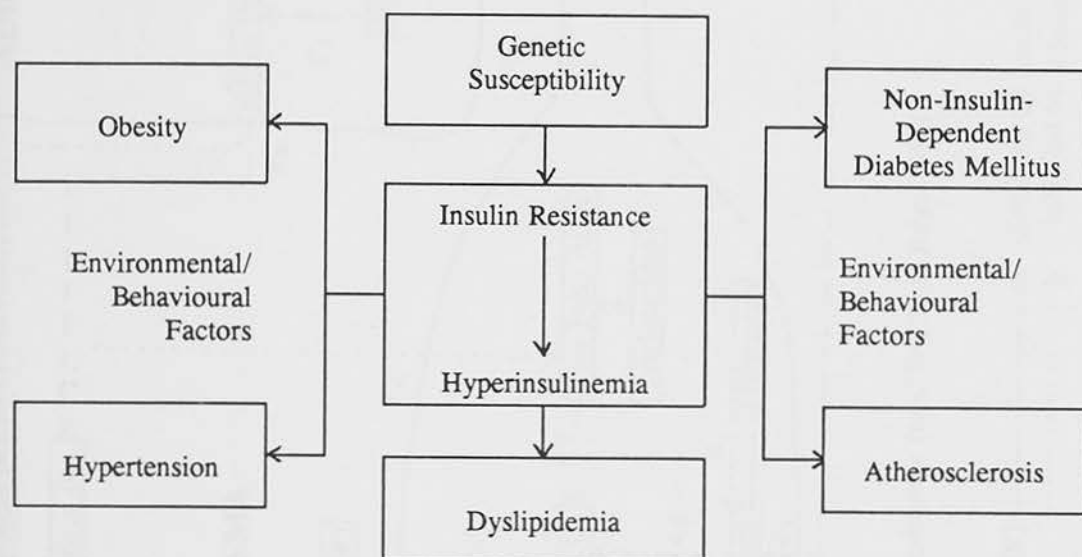


Fig.2.3 Model of causal relationships between insulin resistance and obesity, diabetes, hypertension, atherosclerosis and dyslipidaemia (Adapted from DeFronzo and Ferrannini 1991)

In the body, glucose is "disposed of" by being removed from the plasma and through a series of steps become oxidized to provide energy or stored as glycogen. Glucose is also produced from glycogenolysis and gluconeogenesis. These processes are regulated by insulin. In IR, glucose *disposal* is depressed while *endogenous glucose production* is increased.

CARBOHYDRATE		FAT	PROTEIN
C1	↓ glycogenolysis ↑ glycogen storage [This occurs mostly in the liver and, to a limited extent, muscles]	F1	↑ production of VLDL rich TG in liver and release in plasma P1 ↑ uptake of AA by muscles ↓ release of AA to plasma ↓ plasma level of AA
C2	↓ gluconeogenesis from AA and lactate [C1+C2 = ↓ hepatic glucose production]	F2	LPL in endothelial cells break down TG to FFA: In AT: LPL ↑ In muscles: LPL ↓ P2 ↑ protein synthesis ↓ proteolysis
C3	glucose uptake by liver [20-25% of glucose load] independent of insulin ↓ release of glucose from liver to plasma	F3	In AT: FFA resynthesized to TG for storage (↑ lipogenesis) ↓ lipolysis [regulated by hormone-sensitive lipase, suppressed by insulin, increased by glucagon, catecholamines, cortisol and HGH] [TG directed to AT for storage in postprandial state and to muscles for oxidation in postabsorptive state]
C4	↑ glucose uptake by muscles [75-80%] and other tissues; no exit of glucose from muscles C1+C2+C3+C4 = ↓ plasma glucose concentration		
C5	↑ glucose uptake by AT [insignificant in man, ~ 1% glucose load]	F4	↓ FFA oxidation via acetyl CoA and citric acid cycle [FFA not immediately oxidized, but first stored as TG in muscles taken up from plasma TG]
C6	↑ glycolysis and glucose oxidation in muscles, AT, and other tissues C3+C4+C5+C6 = ↑ glucose disposal	F5	<i>de novo</i> synthesis of fat from CHO insignificant in man; TG stores in AT reflect dietary fat intake F4,C6 FFA and glucose compete as substrates for citric acid cycle and aerobic energy production

Notes: AA amino acids, AT adipose tissue, CHO carbohydrate, FFA free fatty acid, HGH human growth hormone, LPL lipoprotein lipase, TG triglycerides, VLDL very-low-density lipoprotein

The presence of IR triggers off a series of events. Because of the reduced glucose disposal, more insulin is required to clear the excess plasma glucose. This is achieved by the hypersecretion of insulin by the β cells in the pancreas [in central obesity there is in addition decreased clearance of insulin in the liver], thus resulting in hyperinsulinaemia. The secretion of insulin is a complex process, depending not only on the level of glucose available, but also on the action of the parasympathetic nervous system and gut hormones stimulated by feeding. As IR worsens, the obese subject with normal glucose tolerance goes through the stages of impaired glucose tolerance (IGT) and non-insulin-dependent diabetes (NIDDM). Finally there is total decompensation of the regulatory mechanism and a stage of NIDDM accompanied by hypoinsulinaemia ensues (Table 2.1).

Table 2.1 Metabolic changes during evolution of obesity to diabetes

Metabolic processes/markers	Increasing duration of obesity ---->			
	NGT	IGT	Early NIDDM	Late NIDDM
Lipid oxidation (indirect calorimetry)	+	+	+	+
Glucose disposal (clamp)	-	-	-	-
Insulin response	Fasting insulin	+	+	+
	PC insulin	+	+	-
Oral glucose tolerance	Fasting glucose	N	+	+
	PC glucose	N	+	+
Hepatic glucose production	N	N	N	+

Notes: PC post-challenge NGT normal glucose tolerance
 IGT impaired glucose tolerance NIDDM non-insulin-dependent diabetes
 +/- increased/decreased N no change

Source: Modified from Felber et al (1993)

Unlike glucose, which after ingestion is oxidized quickly, fat is first stored as triglycerides in muscles and adipose tissue and later mobilized as fuel for energy. Insulin resistance is characterized by increased lipid oxidation (measurable by indirect calorimetry), which remains elevated regardless of the state of glucose tolerance. The breakdown products of triglycerides¹ - glycerol and free fatty acids (FFA) - link up with glucose metabolism at two points. Increased FFA leads to increased concentration of acetyl CoA, which depresses glycolysis and thus reduces glucose disposal.

Fat metabolism reflects entirely dietary fat intake and is independent of carbohydrate intake. Very little fat is synthesized from excessive carbohydrate intake. The spectrum of metabolic changes seen in IR can be accounted for by the fact that fatty acids produced by increased lipolysis in AT and increased fatty acid oxidation in muscles (due to a decrease in insulin's suppressive action] have displaced glucose as the main substrate for energy production. The restriction of glucose oxidation leads to compensatory responses to maintain glucose homeostasis - ie. hyperglycaemia and hyperinsulinaemia. With time, compensation fails and diabetes occurs. IR can be conceived of having two separate arms, one each in glucose and fat metabolism. While they may be interrelated, there is evidence to suggest that they are controlled separately genetically and in some populations (eg. the Pima and Australian Aborigines) only one may be operative - the phenomenon of "selective insulin resistance". The implications for Inuit obesity will be further explored in Chapter VII.

1 Triacylglycerol is the preferred term used by lipid biochemists. However, the term "triglyceride" still persists in the clinical literature.

In obesity, IR reduces the effective action of insulin (coupled with an increase in free androgen and glucocorticoids), resulting in increased lipolysis in AT, increase in free fatty acids delivered to the liver, and increase in the production of VLDL. In the peripheral circulation, IR is associated with higher plasma levels of triglycerides (TG), total cholesterol (TC), and low-density-lipoprotein (LDL), and lower levels of high-density-lipoprotein (HDL)². It is believed that the activity of lipoprotein lipase, the enzyme located on capillary endothelial cells responsible for hydrolysis of TG carried in plasma VLDL and chylomicrons, is lowered, leading to a reduced clearance of TG from the circulation. In the liver, the increased influx of free fatty acids leads to overproduction of TG-rich VLDL, measurable as increased plasma level of TG. Note that in "central" obesity, the increased fatty acids have direct access to the liver via the portal circulation. The increase in production of VLDL in the liver also results in more LDL being released, and a measurable increase in plasma LDL level. Another reason for the increased plasma level of LDL is the decreased clearance of LDL from plasma, due to the suppression of LDL-receptor activity in the liver. The increased production of VLDL uses up cholesterol esters from HDL, leading to a decrease in plasma level of HDL.

Ordinarily the amount of protein ingested in excess of requirements for nitrogen metabolism is oxidized to produce energy. In starvation, protein stores have to be mobilized. Liver and skeletal muscles are the major sites where proteolysis occurs. In the liver, amino

2 Cholesterol and triglycerides are transported in plasma as lipoproteins, of which there are several kinds, consisting of varying proportion of proteins (apolipoproteins), phospholipids, triglycerides, and cholesterol esters. Lipoproteins are classified according to their density and electrophoretic mobility into: Chylomicrons, VLDL, LDL and HDL. Chylomicrons and VLDL contain predominantly triglycerides whereas LDL and HDL contain mostly cholesterol esters. The apolipoprotein components also differ in terms of the A, B, D, and E subtypes.

acids are deaminated, producing urea, while the carbon skeletons are catabolized to such intermediary metabolites as oxaloacetate and pyruvate, thus joining the glucose metabolic pathways either towards oxidation or gluconeogenesis. Protein intake can therefore add to the carbohydrate load. The conversion of amino acids to glucose is regulated by insulin, glucagon and the availability of substrates (ie. amino acids).

Protein metabolism in the obese differs little from the non-obese but non-diabetic person. The suppressive action of insulin on proteolysis is not altered and plasma amino acid levels are similar in the two groups. In the obese NIDDM patient with poor metabolic control, however, proteolysis is increased, reflecting the deficient action of insulin in liver and muscle cells.

The Primary Defect?

The above review presents one scenario of interlocking metabolic pathways in insulin resistance and the progression of obesity through impaired glucose tolerance to NIDDM, which is largely based on the work of Felber et al (1993) and DeFronzo et al (1992). In reality it is very difficult to determine which is the initial 'lesion'. While there is basic agreement on the types of metabolic events taking place, it is much more difficult to decide the correct time sequence. As many metabolic events are interconnected, many through feedback loops with one metabolite serving as counter-regulator of another, it is exceedingly difficult to decide which is the primary event, which is the compensatory response, and which is the final state when this compensatory response can no longer cope (or "decompensated"). A case in point is obesity, insulin resistance and hyperinsulinaemia. While it is clear that the three tend to occur together, it is not at all clear which comes first. An alternative conception, which considers insulin resistance as the compensatory response to hyperinsulinaemia and central obesity (Kissebah and

Peiris 1989), is equally compatible with the corpus of metabolic findings (Fig.2.5). An interesting but unorthodox view even suggests that obesity is an advantageous adaptation to modern foods, especially refined carbohydrates, and should not even be viewed as a metabolic disorder (Bradley 1982). In response to hyperinsulinaemia brought on by increased glucose intake, insulin resistance serves to conserve endogenous glucose for the glucose-sensitive tissues such as the brain.

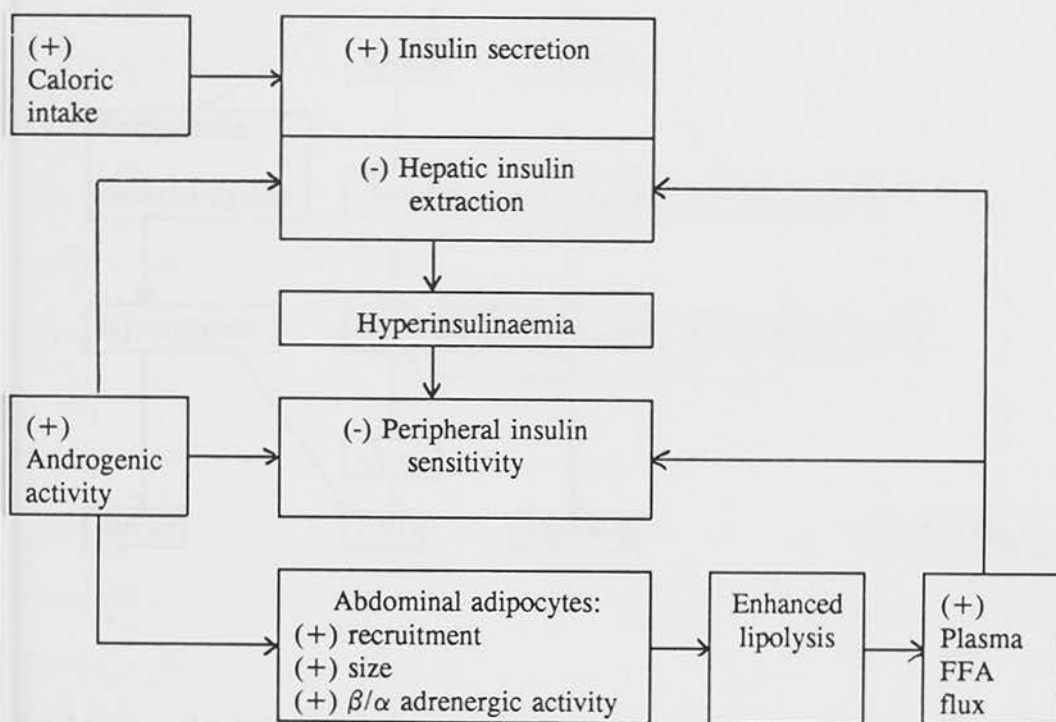


Fig.2.5 Model of hyperinsulinaemia, insulin resistance and abdominal obesity (After Kissebah and Peiris 1989)

Björntorp (1988), in an attempt to encompass all known environmental and metabolic events associated with obesity, insulin resistance and related diseases, hypothesised that the primary pathogenetic factor lies in neuroendocrine dysregulation at the hypothalamic level, perhaps chronic stress-induced arousal brought on by life events, triggering off responses in the sympathetic nervous system and adrenal cortex (Fig.2.6).

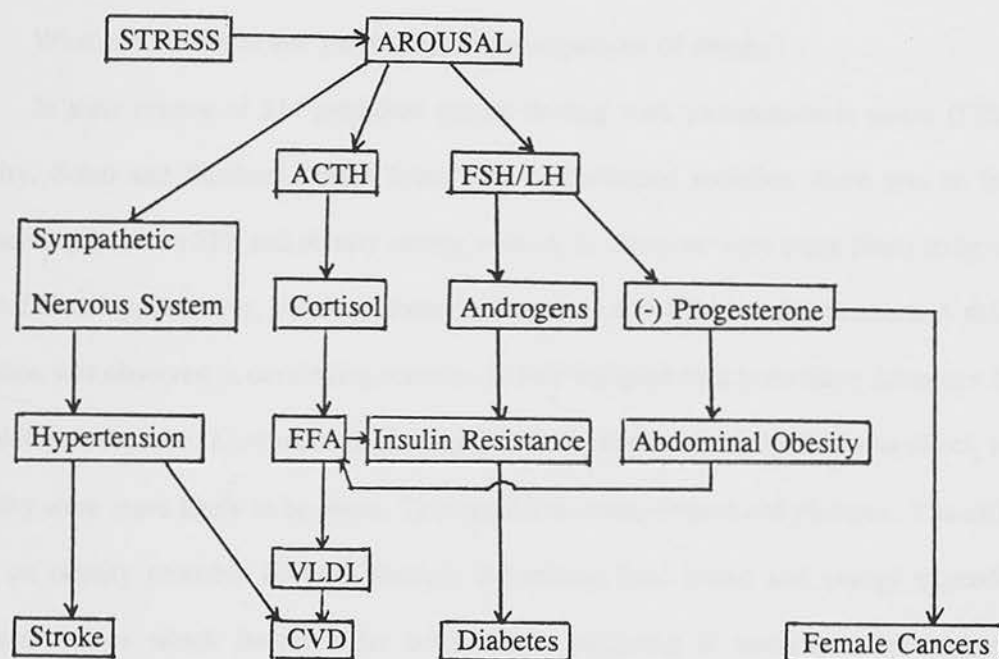


Fig.2.6 An integrative view of obesity, insulin resistance and associated conditions

(After Björntorp 1988)

Notes: ACTH Adrenocorticotrophic Hormone LH Luteinising Hormone
FSH Follicle-Stimulating Hormone FFA Free Fatty Acids
VLDL Very-Low-Density-Lipoprotein CVD Cardiovascular Disease

2.4 Sociocultural Aspects

The relationship between sociocultural factors and obesity can be considered in the following perspectives:

- (1) What social, cultural and economic factors, if any, are determinants or predictors of obesity?
- (2) What are the prevailing attitudes towards obesity in different cultures?
- (3) What are the social and psychological consequences of obesity?

In their review of 144 published papers dealing with socioeconomic status (SES) and obesity, Sobal and Stunkard (1989) found that in developed societies, there was an inverse relationship between SES and obesity among women, ie. the poor were more likely to be obese. The relationship, however, was inconsistent among men and children of both sexes. A different situation was observed in developing societies [which included data from three American Indian populations - Apache, Kiowa/Comanche, and Navajo], where the relationship was direct, ie. the wealthy were more likely to be obese. This applied to men, women and children. The effect of SES on obesity probably operates through differential food intake and energy expenditure. Cultural values which favour a fat body shape, assigning it social prestige and sexual attractiveness, promote greater food intake among the well-off, who possess the necessary means.

Different cultures view obesity differently, and not all would regard it as a "health problem". Concepts of obesity are intertwined with concepts of self-worth, sexuality, ideal body type and female beauty. In fact, many pre-industrial societies probably have no ethnomedical definition of obesity (Brown and Konner 1987). Little data can be found in the ethnographic

literature on what the Inuit thought about obesity. Ritenbaugh (1982) went further and called obesity a *culture-bound syndrome*, at least in modern western society. Culture shapes the definition, diagnosis and treatment of the "disease" and defines its etiology. Whereas biological data on height and weight are *etic*, biomedical categories such as disease and obesity are culture-specific (ie. *emic*). Western biomedicine is but one specific conceptual framework for interpreting biological data. In support of her arguments, Ritenbaugh documented a downward shift in the standards since 1940s of what American biomedicine considered "obesity". It is however, also probable that this reflects changing scientific knowledge.

As a "disease", obesity carries with it unfavourable social stigmas, a situation which likely gives rise to the increasing prevalence of so-called eating disorders, particularly among young females in western society. Subtle discrimination against the obese as physically "deviant" also exists. Some of these attitudes may conceivably be transmitted from the dominant society to indigenous populations in North America because of modern communications and the influence of mass media. To what extent have such attitudes been accepted by Inuit and other Native American populations is not known and would be worthwhile subject of further ethnographic research.

A useful framework for understanding the mechanisms linking socioeconomic status and obesity has been proposed by Sobal (1991). SES leads to variation in behaviour which influences energy intake, expenditure and intermediate metabolism, resulting in obesity. Operating in the other direction, the social perception of obesity can be interpreted through prejudicial beliefs with subsequent stigmatization and discrimination, with the result that access to higher SES roles becomes limited (Fig.2.7).

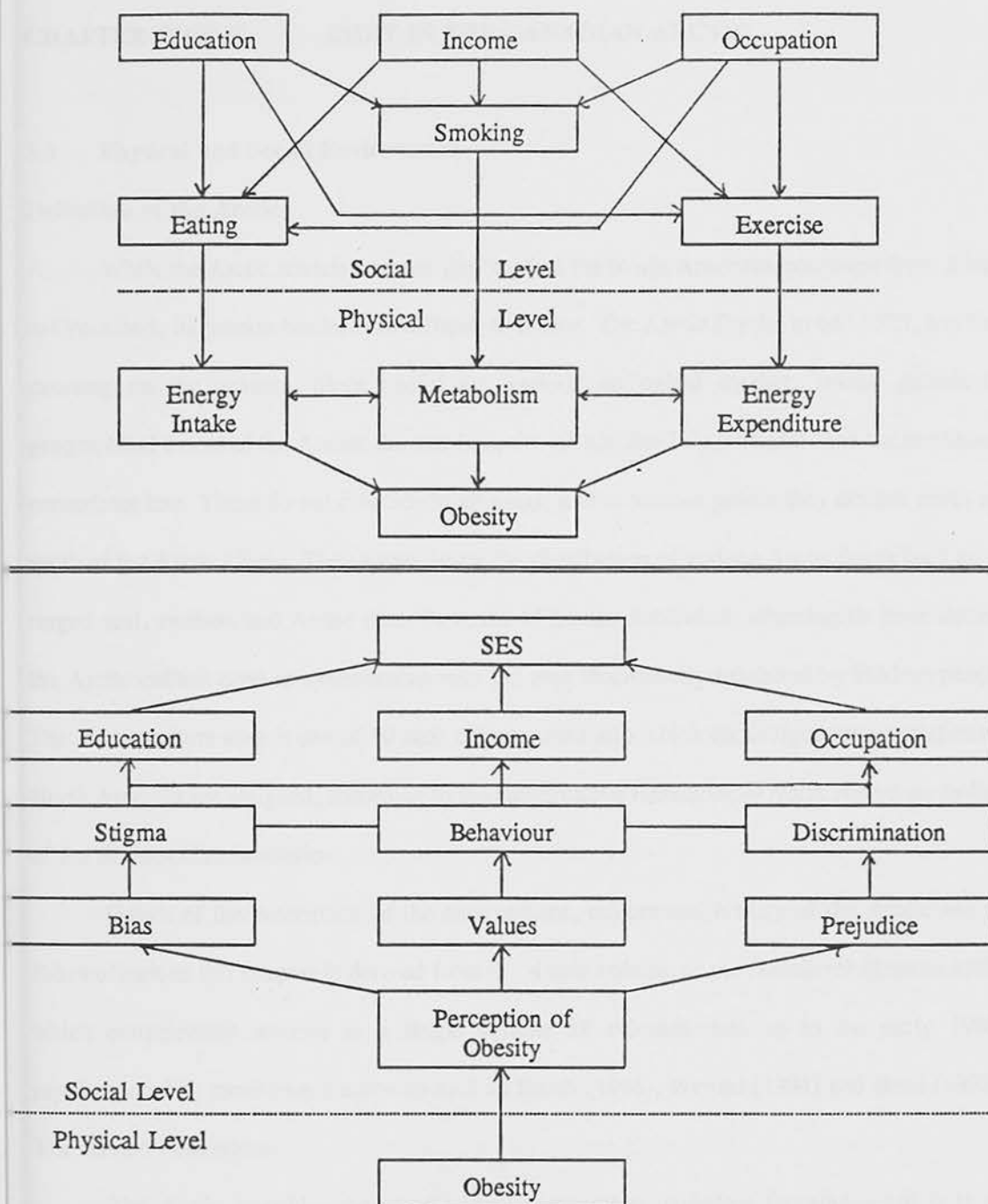


Fig.2.7 Interrelationships between socioeconomic status and obesity (After Sobal 1991)

3.1 Physical and Social Environment

Definition of the Arctic

While the Arctic stretches across the "top" of the North American continent from Alaska to Greenland, its precise borders are difficult to define. The Arctic Circle, at 66° 30'N, has little meaning on the ground. More useful are various ecological markers which delimit the geographical extent of the Arctic: the tree-line, the 10° Celsius July isotherm, and the continuous permafrost line. These do not coincide in all areas, and at various points they deviate north and south of the Arctic Circle. They approximate the distribution of various Arctic fauna such as the ringed seal, caribou and Arctic char. In terms of human habitation, ethnologists have defined the *Arctic culture area* as synonymous with the area traditionally inhabited by Eskimo people. The Arctic culture area is one of 10 such culture areas into which the indigenous populations of North America are assigned, according to the authoritative *Handbook of North American Indians* of the Smithsonian Institution.

[Much of the description of the environment, culture and history of the Arctic and the Eskimo/Inuit in this chapter is derived from the *Arctic* volume of the *Handbook* (Damas 1984), which conveniently reviews in a single volume all relevant data up to the early 1980s, supplemented by more recent accounts such as Burch (1986), Wenzel (1991) and Bone (1992)].

The Arctic Ecosystem

The Arctic is cold - the mean annual temperature is below freezing - but it is not uniformly so. Climatic variation exists on account of the location of mountain barriers, proximity

to open waters, extent of surface snow and ice cover, and the regime of daylight and darkness. Due to the high latitudes, the sun is very low on the horizon much of the year, with its rays hitting the earth at an oblique angle, thus conferring much less heat per unit area than at the lower latitudes. The polar ice and snow also reflects much of the heat that reaches the surface. There is net radiation loss, such that heat needs to be "imported" from the southern latitudes via atmospheric circulation and the ocean currents.

Despite the image of a white expanse, precipitation is generally low, and strong blowing winds redistribute the snow deposits unevenly. While pack sea ice is found year round between the high Arctic islands, elsewhere the Arctic coast is ice free for several months of the year (Stager and McSkimming 1984, Bone 1992).

There is relatively low biodiversity in the Arctic. The major marine and terrestrial mammals include seals (ringed and bearded), beluga whales, polar bears, muskoxen, barren ground caribou, walruses, and small furbearers such as Arctic foxes, hares, and wolves. Avian species include ptarmigan, snowy owl, ducks, geese and raven. Fish such as lake trout and whitefish can be found in rivers and lakes, and species such as arctic char move seasonally from the sea into freshwater areas. While the number of species is small, individual species are present in large numbers, and there are seasonal bursts of high productivity. Within one locality, there is also considerable year-to-year fluctuation (Freeman 1984).

Much of the Canadian Arctic lies within the geologically ancient Canadian Shield, scoured by glaciation of relatively recent vintage. In the Central Arctic, the physical terrain is characterized by an undulating plain of low elevation. The Arctic soil is dry, thin and poor, and permafrost can be found everywhere at varying depths. Except in the high Arctic where there

is only bare rock and gravel, some vegetation, mostly lichens, moss, grass and sedges, covers large areas, much of it under snow cover during most of the year (Bone 1992). In the short summer growing season, blooming flowers enrich the landscape with a variety of colours. Plant life is an important food source for the herbivores which form a critical link in the Arctic's food chain. The lack of trees means that firewood, except for driftwood, is generally unavailable.

Subdivisions of Eskimo/Inuit

The term "tribe" is not really applicable to hunting-band societies such as the Eskimo/Inuit. Damas (1984:3) cited Helm in defining a tribe as "the greatest extension of population throughout which there is sufficient intermarriage to maintain many-sided communication". The Smithsonian's *Handbook* recognized some 20 "tribe-like" groupings of Eskimos, primarily as a means of organizing data (Fig.3.1):

- Greenland: (1) East Greenland; (2) West Greenland; (3) Polar Eskimos
- Canada: (4) Labrador Coast; (5) Northern Quebec; (6) Baffinland; (7) Iglulik Eskimos; (8) Netsilik; (9) Caribou; (10) Copper; (11) MacKenzie Delta Inuit
- Alaska: (12) North Alaska Coast; (13) Interior North Alaska; (14) Kotzebue Sound; (15) Bering Strait; (16) St. Lawrence Island; (17) Nunivak; (18) Mainland Southwest Alaska; (19) Pacific Eskimos
- Russia: (20) Siberian Eskimos

Although *Inuit* is the collective term preferred by Canadian Eskimos (singular: *Inuk*), various other terms are used in self-designation elsewhere in the Arctic, such as *Inuvialuit* (MacKenzie Delta), *Inupiat* (Northern Alaska), and *Yupik* (Central Alaska). The Inuit which today inhabit the study area, the Keewatin Region in the Northwest Territories, are derived primarily from the Caribou Eskimos, with some representatives of the Netsilik, Iglulik, and Eskimos of northern Quebec.

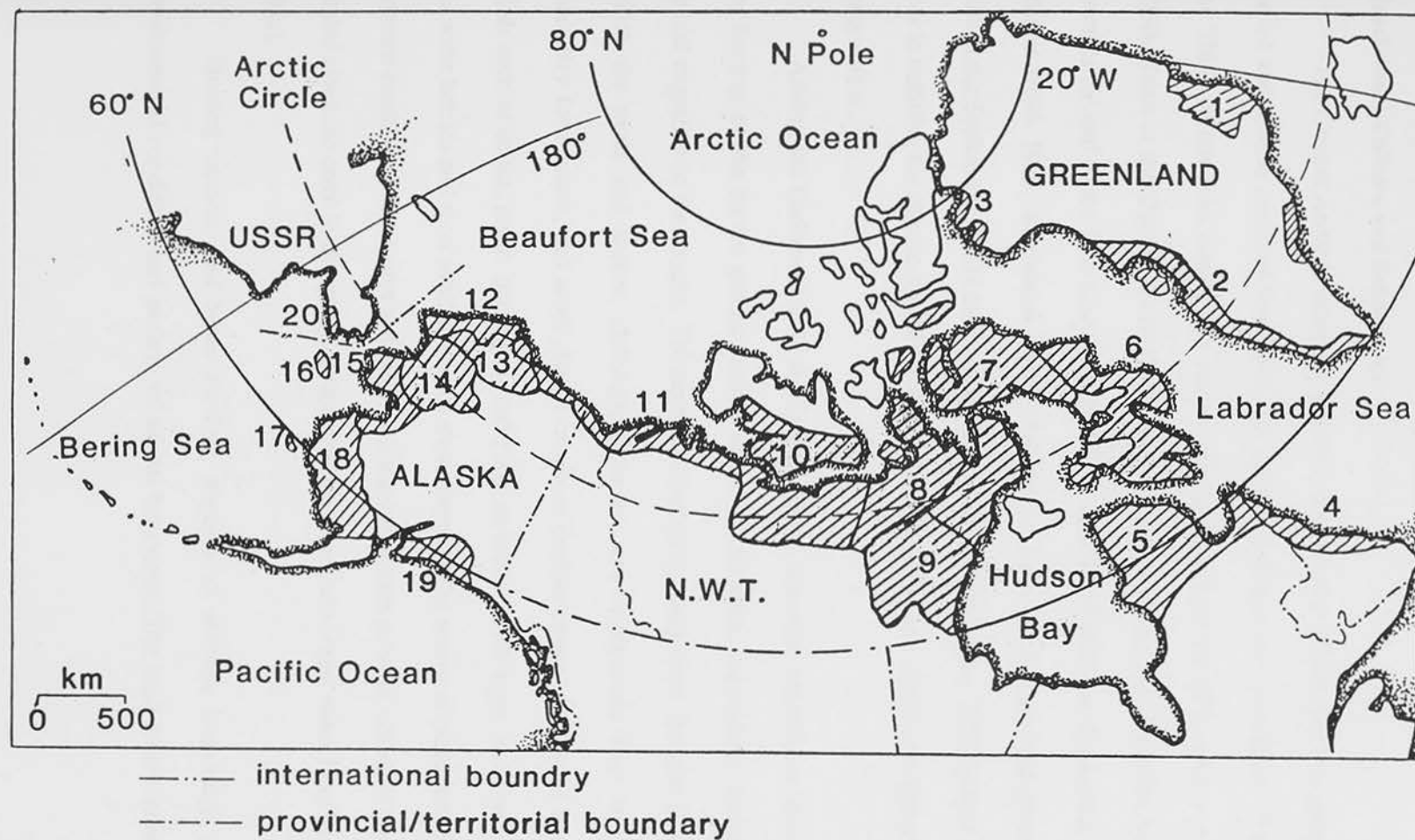


Fig.3.1 Map of the Arctic showing traditional territories of the Eskimo/Inuit

Traditional Culture and Subsistence

The Central Arctic is relatively desolate, compared to Alaska and Greenland, with their varied and diverse maritime food base. The Central Eskimos are descendants of the people of the Thule culture who moved into the central Arctic around the 12th century AD. Until the 1950s, Inuit in the Central Arctic lived much the same way as that described by Franz Boas towards the end of the 19th century (Boas 1888), and the Fifth Thule Expedition of the 1920s (Birket-Smith 1929, Rasmussen 1930). Popular accounts by explorers-adventurers such as Vilhjálmur Stefánsson (1921) and novelists such as Farley Mowat (eg. 1951) played an important role in instilling the romanticised Eskimo with his dog-sledge and snowhouse (*iglu*) in the public imagination.

Among the Caribou Inuit in the Central Arctic, the main subsistence activity has been the hunting of the barren ground caribou in the inland areas, particularly during the herd's annual migration in the autumn. The caribou furnishes not only meat, but also hides and bones which are made into shelter, clothings, footwear, and implements. Fish is an important secondary food source, and arctic char is caught in shallow streams during its spawning runs. Birds such as ducks, geese, ptarmigan, and gulls, as well as their eggs, are also eaten. Except for some berries and roots available in the short summer, the source of plant food is mainly the stomach contents of herbivores and birds. Sea mammals such as seals, walruses and whales are hunted along the coast in the spring, both at the floe edge and in open water from kayaks (Arima 1984).

Hunting success, and hence survival, requires an intimate knowledge of changing environmental conditions and patterns of animal behaviour. The Inuit hunter must be able to

predict the location of game and possess the necessary vehicles and weapons to track, pursue, kill and retrieve large mammals on land and in the sea. Hunting is an integral part of the annual cycle. The method of hunting and animal species hunted change with the seasons, along with changes in the type of housing, clothings, mode of transportation, and population clustering.

Contact History and Social Change

The contact history and subsequent sociopolitical developments differ between different parts of the Arctic. In Alaska, it was Russians and Americans who acted successively as agents of change. Greenland was colonized by the Danes. In the Central Arctic, the external influence was primarily British, and later, Canadian. While early explorers such as Henry Hudson in the 17th century and traders such as Henry Kelsey early in the 18th century visited the region, the Central Eskimo came into more sustained contact with Europeans during the 18th century, when the Prince of Wales Fort was established by the Hudson's Bay Company (HBC) in what was to become the modern town of Churchill, Manitoba. During 1770-2 the HBC trader Samuel Hearne made the epic journey from Churchill across the barrenlands to the shores of the Arctic Ocean at the mouth of Coppermine River in the northwest. While the HBC's main trade was directed towards Indians in the subarctic boreal forest to the south, the company's sloops traded with the Eskimos up the Bay coast. After 1790 such practice ceased and the Eskimos had to trek down to Churchill during the summer to trade. Initially the Caribou Eskimos acted as middlemen in the fur trade between the Europeans and other Eskimos further north - the Iglulik, Netsilik and Copper Eskimos. In the 19th century the more northern groups were frequented by whaling fleets and scores of naval expeditions in a renewed search for the Northwest Passage and later, for those (such as Captain John Franklin) who were lost in its search. The Eskimos assisted

whalers and sailor wintering on the ice, and benefitted from the infusion of metals and wood from abandoned ships.

Despite the introduction of items of European material culture, such as firearms, twine, and metal utensils and implements, the Inuit traditional way of life remained largely intact until the 20th century. Such contacts, however, did introduce new diseases which were responsible for the depopulation and even extinction of some tribes, such as the Sallirmiut on Southampton Island (Keenleyside 1990). By the beginning of the 20th century, whaling had declined while the fur trade, particularly in Arctic fox, prospered. The Royal Canadian Mounted Police, the Hudson's Bay Company, and various Catholic and Anglican missions established their presence and extended their activities into the Central Arctic.

During the decade 1915-24 the Caribou Eskimos suffered severe privations due to the failure of the caribou migration to materialize in consecutive winters. This led to a reduced dependence on the caribou and a shift to the trapping of small furbearers as the main source of livelihood. However, the fur trade collapsed during the 1930s. In the 1940s the fortunes of the Caribou Eskimos continued to plummet, as epidemics of tuberculosis, influenza and poliomyelitis struck on the heel of famine. The public outcry spurred the Canadian government into action and provided the impetus for the post-War increase in government services.

Since the 1950s, the pace of change has intensified. New townsites were developed, and the Canadian government actively encouraged sedentary settlement where health, education, and welfare services could be conveniently administered and provided. The new population centres also resulted in the mixing of different traditionally distinct groups of Eskimos: the Caribou, Iglulik and Netsilik, particularly in larger towns such as Rankin Inlet and Baker Lake. The

beginning of mining activities - nickel in the Rankin Inlet area in the 1950s and uranium near Baker Lake in the 1970s - further consolidated the local population as well as introduced large number of migrant workers from the south. Elsewhere in the Arctic, the Cold War (against the Soviet Union, not the climate) hastened the modernization of the northern frontiers with the construction of radar stations and airstrips. The transition of Inuit economy is evident from Table 3.1, based on data from Freeman (1971).

Table 3.1 Sources of income in two Inuit communities, NWT, 1950s and 1960s

Community	Years	Sources of Income (%)				Per capita cash income
		Furs/skins	Wages	Subsidies	Crafts	
Baker Lake	1951-2	56	6	38	0	\$56
	1961-2	3	46	48	3	\$329
Lake Harbour	1963-4	69	12	14	6	\$422
	1966-7	28	38	18	17	\$525

The visible changes include prefabricated houses; manufactured clothes (often with poorer insulative properties); store-bought food; decline in hunting and its replacement by wage labour, the cottage industry in handicrafts (soapstone carving, silkscreen prints) and social assistance payments; and motorized transports (snowmobiles and boats) in place of dog sledges and kayaks. While health status has generally improved, with the infectious diseases being brought under control, there has been a corresponding increase in alcohol and substance abuse, social problems, accidents and violence. Some observers have characterized the situation in the Arctic as an "internal colony" (eg. Brody 1975).

The unfavourable socioeconomic situation of the Inuit can be seen in Table 3.2, which provides a comparison of several indicators between Inuit in the NWT and all Canadians according to the 1986 Census (Statistics Canada 1989). It should be mentioned that comparison of income between the Inuit and other Canadians is not straight forward. Some expenses are heavily subsidized in the Arctic, for example, public housing. While the cost of food and supplies in the stores are often prohibitively expensive, food is also available from the land, though not without cost in terms of capital investment in snowmachines and rifles and operating expenses such as gasoline and ammunition. On the whole, income level does reflect the discrepancy in economic prosperity between the two populations.

Table 3.2 Selected socioeconomic indicators, NWT Inuit and all Canadians, 1986 Census (percent of population aged 15 and above)

Indicator	NWT Inuit		Canada	
	M	F	M	F
Highest level of schooling				
< grade 9	63.6	66.4	16.9	17.6
Grade 9 +	35.6	32.5	62.8	65.8
Some university	0.8	1.2	20.3	16.6
Labour force activity				
employed	41.8	32.9	70.0	49.6
unemployed	19.1	12.3	7.5	6.3
not in labour force	39.1	43.8	22.5	44.1
Per capita annual income				
< \$10,000	63.1	81.2	32.5	60.8
\$ 10,000-19,999	17.1	10.4	21.2	22.5
\$ 20,000+	19.7	8.4	46.3	16.7

3.2 Human Biology and Demography

Origins and Affinities

It is generally accepted that the aboriginal population of the Americas ultimately originated in the Asian continent. The details of the migration - its date, the number of "waves" of migration, the point(s) of entry, the routes of dispersion, and the time required to populate the length and breadth of the hemisphere, among others - have been the subject of much debate. Reviews of the archaeological, geological, linguistic, genetic, cultural and dental evidence are available (eg. Laughlin and Harper 1979, Kirk and Szathmary 1985, Fagan 1987). The geological evidence points to the Beringia landbridge, exposed during various times during the last (the Wisconsin) glaciation of the Pleistocene Ice Age, as the most likely route over which the migration occurred. In one scheme, Harper (1980) estimated that the organization of the gene pool which spawned the entire Native American population began around 19,000 BP (Before Present) in northeast Asia. Around 15,000 BP the Indians split off from the "Bering Sea Mongoloids", each taking a different route across the landbridge. Around 10,000 BP the Aleuts separated from the Eskimos. A further division into the present-day Yupik and Iñupiat/Inuit occurred around 5,000, with the former settling in the central and southwestern Alaskan coasts and the latter dispersing across the Arctic coast from Alaska to Greenland. Greenberg, Turner and Zegura (1986), based on archaeological, linguistic and dental data, suggested that three biologically differentiated groups of Asiatic peoples entered North America at different times: the Amerind (or Paleo-Indian), the Na-Dene (primarily Athapaskans), and the Aleut-Eskimo (or Eskaleut). A recent review of the geophysical and archaeological data concluded that the settlement of Beringia began around 12,000 BP, at the beginning of a major interstadial in

response to environmental changes (such as the appearance of trees), followed by rapid expansion to other regions of the New World, free from the constraint of the continental ice masses, which ceased being a physical barrier as early as 14,000 BP. After the onset of the final Pleistocene cold oscillation 11,000-10,500 BP there was an abrupt change in technology ("microblade") suggestive of a new population which never spread beyond the northern regions (Hoffecker et al 1993).

Much of the older physical anthropology literature on Eskimos [for example, the classic works of Jenness on the Copper Eskimos (1923), Hrdlička on Alaskan Eskimos (1930), Stefánsson on the MacKenzie Delta and Copper Eskimo (Seltzer 1933)] was concerned with detailed descriptions and measurements of such "racial" characteristics as hair colour, texture and distribution, cranial and facial dimensions, anthropometric indices, epicanthic folds and skin pigmentation. A review of these and more recent data collected in the early 1970s as part of the IBP was provided by Laughlin (1966), Dahlberg (1980), Eriksson et al (1980), and Szathmary (1984). Such data have been used to discern the relationship among Native Americans, and between them and other populations in the world. Turner's studies on dental morphology grouped Native Americans with the peoples of Siberia, northern China and Japan into a *Sinodont* group. Another group, the *Sundadont*, could be traced from southeast Asia into Polynesia (Turner 1989). Dermatoglyphics, for example digital patterns, have also been used. Recent analyses found intercorrelations among dermatoglyphic, geographic, genetic and linguistic distances in 18 Siberian, Amerindian and Eskimo populations (Crawford and Duggirala 1992).

The development of population genetics and the identification of human gene polymorphisms advanced the measurement of genetic distances between various North American

Indian populations, as well as between North American Indians and other races (Szathmary 1984, Roychoudhury 1978). In their world survey of polymorphic genes, Roychoudhury and Nei (1988) enumerated the following types of gene loci:

1. Enzymes (eg. acid phosphatase, cholinesterase, glucose-6-phosphate dehydrogenase)
2. Proteins (eg. group-specific component, haptoglobin, transferrin)
3. Blood groups and platelet antigens (eg. ABO, Rh, Duffy, Diego, and MNS systems)
4. HLA (histocompatibility) markers
5. Immunoglobulins (eg. Gm1, Km systems)
6. DNA polymorphisms
7. Miscellaneous (eg. isoniazid inactivation, phenylthiocarbamide tasting)

A large body of data has been accumulated from studies among the Inuit in the circumpolar zone. Data on Canadian Inuit have mostly been collected as part of the IBP studies in Igloodik (McAlpine et al 1974, Dossetor et al 1973), although there are earlier data from studies in other parts of the Arctic (Chown and Lewis 1959).

Earlier studies (eg. Laughlin 1963) tend to compare Eskimos with other groups based on single traits. Thus the blood group gene B is present in Eskimos and Aleuts but not among Indians, whereas Eskimos and Aleuts lack the Di^a gene of the Diego system, which is present among Indians. Szathmary and Ossenberg (1978) tackled the issue of the biological distinctiveness between Eskimos and Indians by analyzing two independent data sets from 18 aboriginal populations in Siberia and northern North America: (1) 24 discrete cranial traits; and (2) 35 alleles at 11 loci for blood groups and serum proteins. There was significant concordance between the two dendrograms. Eskimos in North America formed a distinct cluster, and they

were closer to Athapaskan Indians than to Aleut or Asiatic Eskimos. Further studies by Szathmary (1981) using information from 42 genes on 14 loci showed three basic divisions. One extended across the Bering Sea and included Reindeer Chukchi, all Eskimos and Athapaskan Indians. Within this aggregate there was no difference between the weighted mean Eskimo-Chukchi and Eskimo-Athapaskan distances. A second cluster included only Asiatics. More distant from this and the Eskimoan group were the Algonkian Indians. Szathmary suggested an Athapaskan-Eskimo-Chukchi continuum which could be explained either by gene flow between different populations that inhabited Beringia or a common origin of what are today linguistically disparate populations. Fig.3.2 reproduces one of Szathmary's dendrograms. More recent data based on 13 loci from Chukotka, Russia, seem to support the genetic affinity between Eskimos and Chukchi of Chukotka, and North American Eskimos, although the St. Lawrence Island Eskimos are in the anomalous position of being genetically more distant from their geographic neighbours the Chukotka Eskimos than Alaskan and Canadian Eskimos (Nazarova 1989).

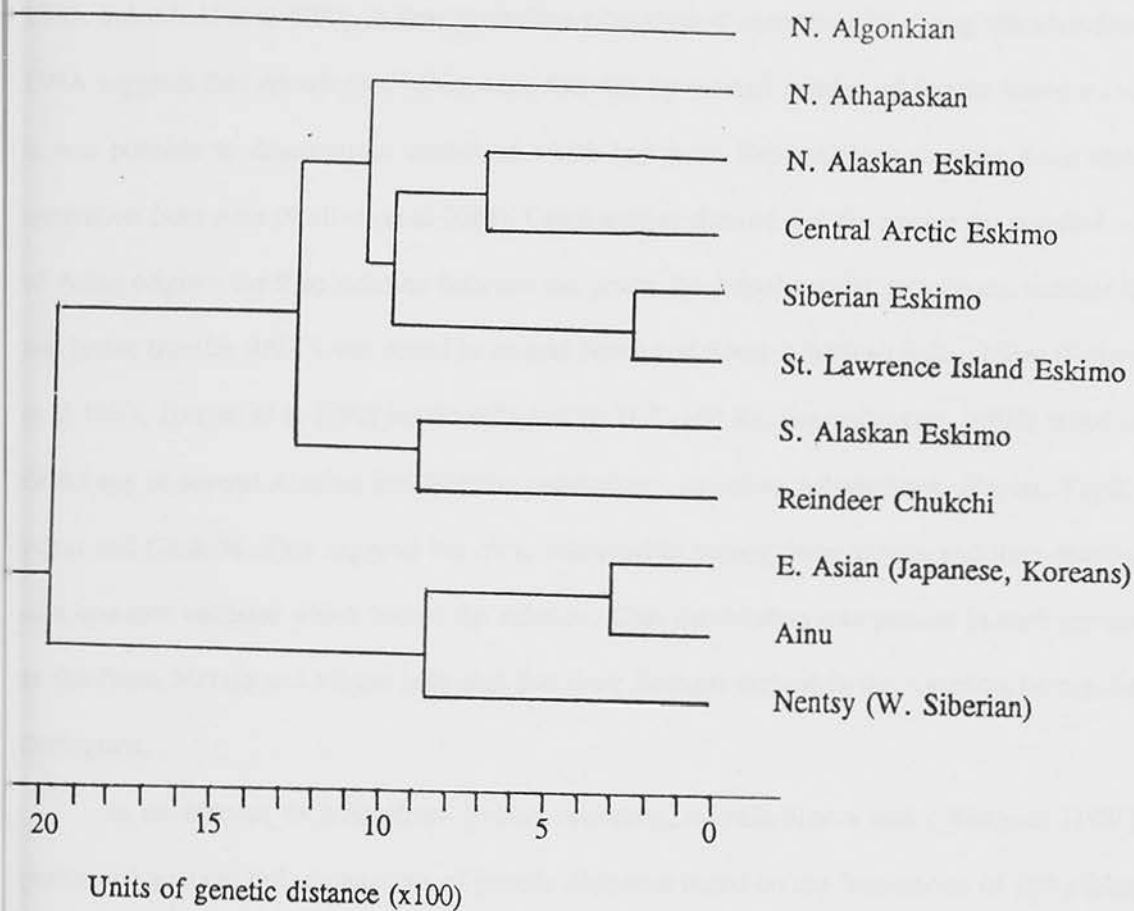


Fig.3.2 Dendrogram showing genetic affinities among 11 Asiatic and North American populations

Note: modified from Szathmary (1981); based on Nei's standard genetic distances between 14 loci (ABO, Rh, MNS, P, Kell, Duffy, Diego, Kidd, Gc, Hp, PGM, AcP, AK and G6PD)

The Asiatic origin and separate migrations of Eskimos, Na-Dene, and Paleo-Indians received additional support from more recent studies of Gm and Km allotypes (Williams et al 1985, Schanfield et al 1990). A new generation of analytical techniques involving mitochondrial DNA suggests that Amerindian tribes were founded by a small number of female lineages and it was possible to demonstrate mutations which had been fixed in these lineages since their separation from Asia (Wallace et al 1985). Other studies showed that the marker for populations of Asian origin - the 9-bp deletion between the genes for mitochondrial cytochrome oxidase II and lysine transfer RNA - was found in several North and South American Indian tribes (Schurr et al 1990, Torroni et al 1992) but Shields and his U.S. and Russian colleagues (1992) failed to detect any in several Alaskan and Siberian populations, including Athapaskans, Iñupiat, Yupik, Aleut and Chukchi. This suggests the close relationship among these groups and their sharing of a common ancestor which lacked the deletion. That the deletion was present in such groups as the Pima, Navajo and Mayan indicated that these lineages arrived in the Americas before the Beringians.

In an attempt to reconstruct human evolution, Cavalli-Sforza and colleagues (1988) performed average linkage analysis of genetic distances based on the frequencies of 120 alleles in 42 aboriginal populations around the world. Populations were pooled together geographically (and also linguistically in some groups, for example, the Na-Dene and the "northern Turkic" of northeastern Asia) and the gene frequencies obtained from the literature averaged. Their dendrogram is shown in Fig.3.3. In this scheme, the Eskimos and Chukchi are closer together and "belong" to a larger grouping of Arctic, northeastern Asiatic peoples and separate from the Amerindians of the western hemisphere.

Another approach to the affinities of North American Indian populations is to examine linguistic differentiation. The correlation between linguistics, archaeology and genetics has been demonstrated in many populations around the world (Renfrew 1992, Cavalli-Sforza et al 1988), including aboriginal North America (Spuhler 1979), where linguistic replacement such as that imposed by military conquest was not believed to be significant in aboriginal times. Considerable disagreement exists on how the hundreds of mutually unintelligible North American Indian languages should be classified and grouped together. Particularly controversial is the larger groupings of families and language isolates into higher-order "phyla" and "super-phyla" [analogous to Linnæan taxonomy]. At the most radical, Greenberg hypothesized that all Amerindian languages were ultimately derived from three stocks: Amerind, Na-Dene and Aleut-Eskimo, corresponding to the three migrations (Greenberg et al 1986). Attempts have been made to link North American languages with Asiatic ones, for example through a phylum called American Arctic-Paleosiberian, which grouped Eskimo-Aleut with Chukchi-Kamchatkan in northeast Asia (Voegelin and Voegelin 1966, cited in Driver 1969). In Ruhlen's survey of world languages, in which he "lumped" some 5,000 languages into 17 phyla, a "Eurasian" superphylum was envisioned which included Eskimo-Aleut and Chukchi-Kamchatka with all the other Indo-European and Asiatic languages. This was comparable to the even broader "Nostratic" superphylum proposed by some Soviet linguists. Amerind was also considered to be more distantly related to Eurasian. Na-Dene, on the other hand, was taken as the easternmost member of a "Dene-Caucasian" superphylum which included, among others, the Sino-Tibetan family of languages (Greenberg and Ruhlen 1992). Not unexpectedly, there is little agreement among linguists on such sweeping, global schemes.

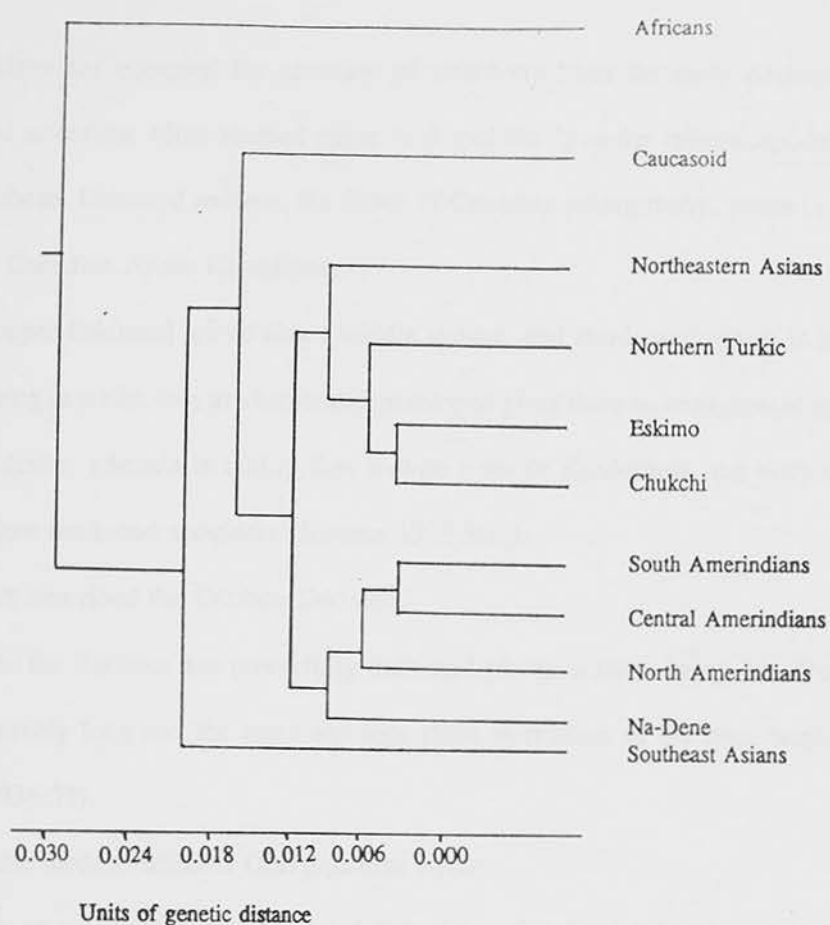


Fig.3.3 Dendrogram showing genetic affinities between 42 aboriginal populations around the world (modified from Cavalli-Sforza et al, 1988)

Body Build and Composition

Considerable evidence is available to indicate that obesity is now prevalent among many Native American populations (Broussard et al 1991, Harrison and Ritenbaugh 1992). The Inuit, and perhaps also Athapaskan Indians, appear to be less affected, which may be the result of genetic predisposition or a later and less intense exposure to acculturation. The issue of obesity

among the Eskimo has occupied the attention of observers from the early ethnographers to modern medical scientists. Most seemed eager to dispel the "popular misconception" that the Eskimos were obese. Diamond Jenness, the father of Canadian ethnography, wrote in his report of the 1913-18 Canadian Arctic Expedition:

The [Copper Eskimos] are of about middle stature, and sturdy and robust in build. The fur clothing in which they are habitually enveloped gives them an exaggerated appearance of corpulence, whereas in reality they incline towards slenderness, the body tapering a little below the broad shoulders (Jenness 1923:B38).

Kaj Birket-Smith described the Caribou Eskimo:

As a rule the Eskimos are powerfully built and plump without being fat. The trunk is comparatively long and the arms and legs short in relation to the total height (Birket-Smith 1936:32).

Decades later, the medical scientist Otto Schaefer wrote:

[There is a] popular misconception of Eskimos eating lots of blubber and being jolly rotund fellows whose presumably ample subcutaneous fat padding helps them to withstand the Arctic cold (Schaefer 1977).

Newman (1960) reviewed the available data series on adult male Eskimo/Inuit heights and weights, including some of the early ethnographic literature dating back to the late 19th century (Table 3.3). It is possible to compute body mass index for each of the series from the overall mean height and mean weight. It should be recognized that such data are illustrative only, as the sample sizes were small, the sampling method unclear (likely non-random), and the age-range unstated. It is noteworthy that the mean BMI, even from early times, was close to the

cut-off point for "overweight" (*vide supra* Section 2.1). Assuming a near "normal" distribution of BMI, this implies that around half of the population would fall into the "overweight" category. Other types of data, namely skinfold thicknesses and body composition, however, conflict with the impression of widespread obesity in historical times.

Table 3.3 **Historical data series (pre-1960s) on mean height and weight of male, adult Eskimo/Inuit populations in the circumpolar zone**

Year	Population	Sample Size	Mean Ht(cm)	Mean Wt(kg)	BMI
1901,1923	Copper Eskimo, NWT	20	163.5	74.0	28
no date	SW Greenland	43	157.7	64.3	26
1951	Aleut	28	161.2	65.3	25
1954	E Greenland	162	162.0	64.1	24
1949	SW Greenland	121	159.3	61.1	24
1885	Pt Barrow, Alaska	51	161.3	66.0	25
1958	E Greenland	133	162.4	63.6	24
1939	Chesterfield Inlet, NWT	30	162.7	65.3	25
1952	Alaska, various sites	17	165.7	63.3	23
1950	St.Lawrence Island	27	165.0	63.2	23

Source: Newman (1960)

More recent survey data on relative weight and skinfolds from Alaska, Canada, and Greenland are summarized in Table 3.4. Data from the Nutrition Canada Inuit survey (Canada DNHW 1980), which was based on a sample size of 366 (200 aged 20 and above) from 4 communities in the NWT, are shown graphically in Fig.3.4 and Fig.3.5. Compared to Canadians

nationally, the Inuit were less heavy and shorter in all age-sex groups. In Igloolik, Hildes and Schaefer (1973) separated their weight and skinfold data according to occupation - hunters vs employees. They found that, among males aged 17-39, hunters weighed on the average 4.5 kg less than employees, and 2mm lower in both their triceps and subscapular skinfolds. In a later survey, Schaefer and others (1980) found that adult Inuit aged 20-49 in Inuvik town in the western Arctic had two to three times the sum of skinfolds than those in the small isolated village of Arctic Bay at the northern tip of Baffin Island.

The prevalence of overweight or obesity among the Inuit depends on the indicator used. In Alaska, the Interdepartmental Committee on Nutrition for National Defence (ICNND) surveyed two groups of Eskimos: a southern group of residents from 5 villages and a northern group from 3 villages, including men from these villages serving in the National Guard. Using the criteria of ≥ 120 percent of U.S. life insurance actuarial standards, the ICNND classified 5 percent of adult Eskimo men as overweight, compared to 19% among women (Mann et al 1962).

Table 3.4 Data from anthropometric surveys among Eskimo/Inuit populations in the circumpolar zone, post-1960s

Authors	Years	Region	Sample size	Ht	Wt	BMI	Tri	Sub	SUM (3 SFs)*
Mann et al (1962)	1958	Southern Alaska	20-34 M:	53	160	64	25		
			35-54 M:	37	160	65	25		
			55+ M:	17	158	64	26		
			20-34 F:	28	147	53	25		
			35-54 F:	35	150	57	25		
			55+ F:	13	148	55	25		

		Northern Alaska	20-34 M:	48	168	64	23			
			35-54 M:	29	165	65	24			
			55+ M:	5	163	64	24			
			20-34 F:	26	155	62	26			
			35-54 F:	23	155	68	28			
			55+ F:	4	155	67	28			
Schaefer (1977)	1964- 1970	NWT, various sites	18-39 M:	396	164	67	25	5.8	7.6	20.5
			40-54 M:	263	163	68	26	6.5	8.2	21.5
			55+ M:	185	160	64	25	7.0	8.7	22.2
			18-39 F:	321	153	54	23	11.4	10.2	31.6
			40-54 F:	124	152	56	24	14.4	13.0	39.1
			55+ F:	89	149	59	27	17.3	16.5	45.9
Auger et al (1980)	1960s	NW Alaska	20-40 M:	55	166	70	26	8.0		
			41-60 M:	36	166	75	27	8.6		
			20-40 F:	75	156	63	26	17.8		
			41-60 F:	59	154	69	29	21.1		
		Igloodik, Hall Beach, NWT	20-40 M:	103	164	66	25	4.9		
			41-60 M:	31	161	65	25	4.8		
			20-40 F:	84	153	56	24	8.7		
			41-60 F:	30	150	56	25	12.0		
Hildes and Schaefer (1973)	1970	Igloodik, NWT	17-39 M:	44		67		5.3	7.0	
			40-54 M:	14		67		4.6	6.6	
			55+ M:	5		61		7.9	8.3	
			17-39 F:	10		56		12.7	9.0	
			40-54 F:	26		57		14.0	9.3	
			55+ F:	12		58		22.2	18.7	
Rode and Shephard (1984, 1992)	1970	Igloodik, NWT	20-39 M:	58	166	68	25			17.5
			40-59 M:	15	164	69	26			19.7
			20-39 F:	33	156	57	23			26.3
			40-59 F:	10	152	60	26			41.0
	1980	Igloodik, NWT	20-39 M:	69	165	67	25			23.2

			40-59 M:	28	164	71	27	28.6
			20-39 F:	47	154	57	24	40.0
			40-59 F:	23	152	62	27	45.2
1990	Igloolik, NWT	20-39 M:	94	164	65	24	31.5	
		40-59 M:	26	163	74	28	47.0	
		20-39 F:	62	153	56	24	50.1	
		40-59 F:	30	152	69	30	92.8	
Nutrition Canada Survey (Canada 1980)	1972	Eskimo Point, Pelly Bay, Frobisher Bay, Coppermine, NWT	20-29 M:	16	167	71	26	
			30-39 M:	17	164	67	25	
			40-49 M:	26	162	67	26	
			50-59 M:	21	160	65	26	
			60+ M:	22	158	61	25	
			20-29 F:	17	152	53	23	
			30-39 F:	24	152	59	26	
			40-49 F:	27	152	56	24	
			50-59 F:	17	151	62	27	
			60+ F:	11	151	50	22	
Ekoe et al (1990)	1982- 1984	N. Quebec	15+ M:	354			26	8.5
			15+ F:	424			26	15.2
Murphy et al (1992)	1987	Central Alaska	40+ M:	230			27	8.5
			40+ F:	279			29	15.2

Note: Ht Height
Wt Weight
BMI Body mass index
SUM (3 SF)* Sum of 3 (triceps, subscapular and suprailiac) skinfolds

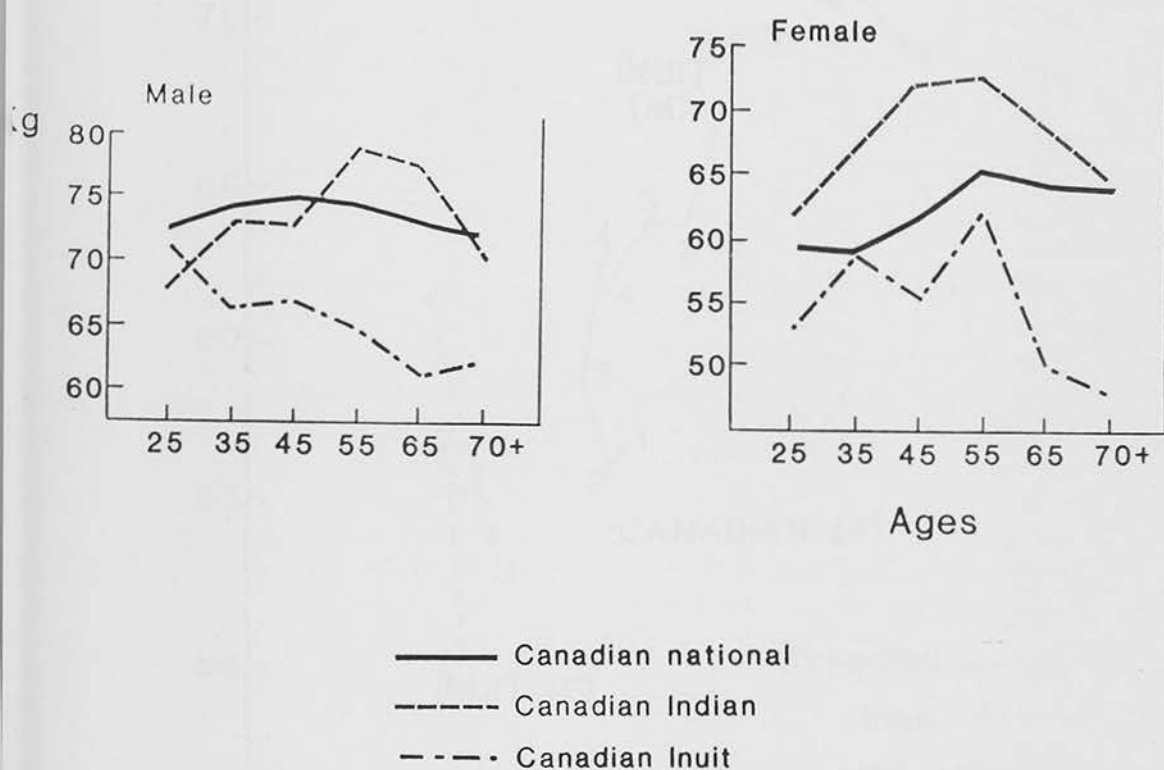


Fig.3.4 Comparison of mean weight-for-age between Inuit, Indian and Canadian adults, Nutrition Canada Survey, 1972

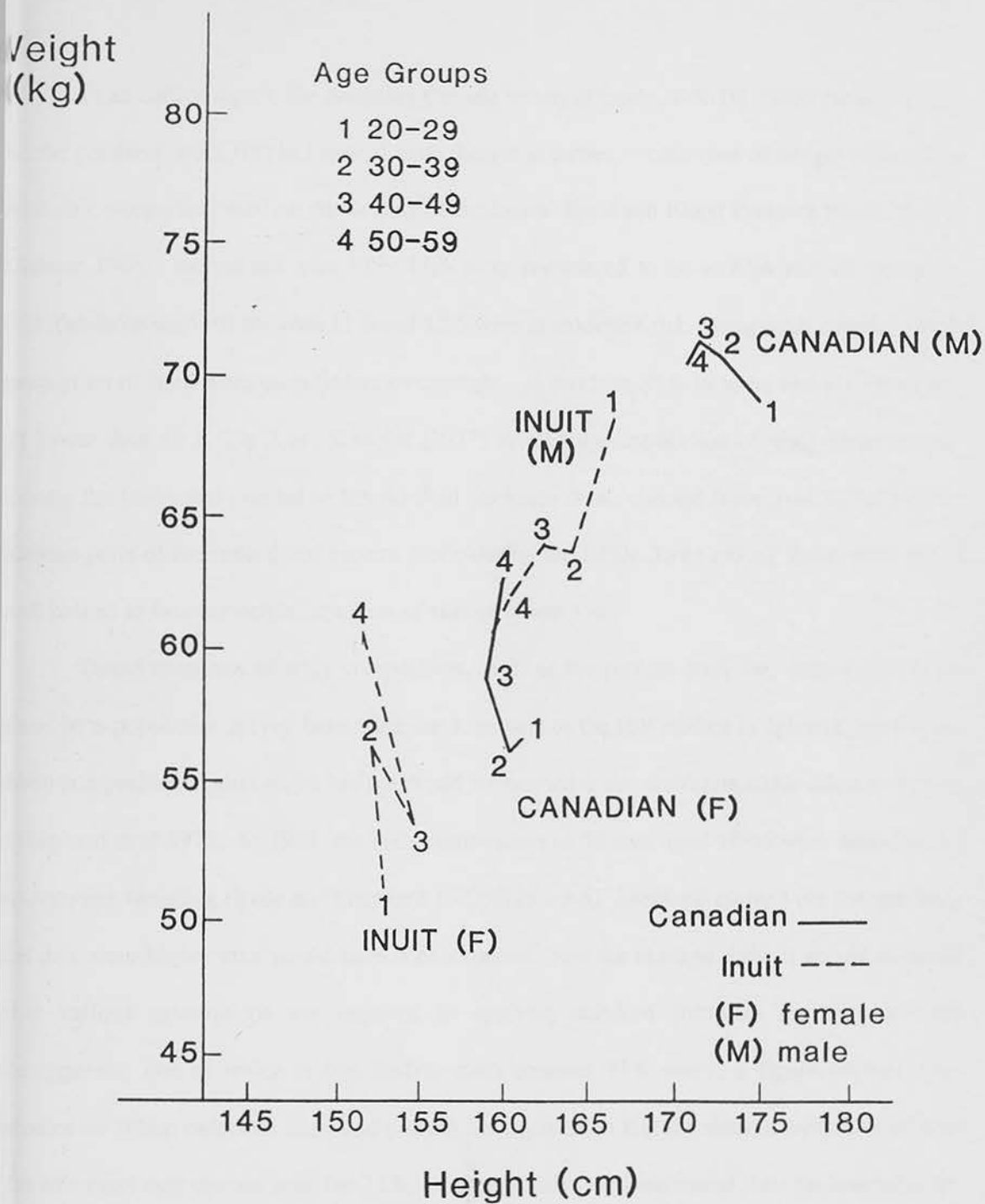


Fig.3.5 Comparison of age-sex-specific mean weight and height between Inuit and Canadian adults, Nutrition Canada Survey, 1972

In an earlier report, the Nutrition Canada survey (Canada, DNHW 1975) presented data on the ponderal index (PI) in imperial units (height in inches $\div$ cube root of weight in pounds), with risk categories based on the Society of Actuaries' Build and Blood Pressure Study of 1959 (Seltzer 1966). Individuals with PI < 11.6 were considered to be at *high risk* of mortality, whereas those with PIs between 11.6 and 12.5 were at *moderate risk*. Using such criteria, a high proportion of Inuit were classified as overweight - as much as 85% in some age-sex group had PI lower than 12.5 (Fig.3.6). Schaefer (1977) refuted the implication of widespread obesity among the Inuit, and pointed to his skinfold thickness data collected from over 1,000 Inuit in various parts of the central and eastern arctic during the 1960s. Even among those with low PI and judged to be overweight, the sum of skinfolds was low.

Direct measures of body composition, such as the percent body fat, cannot readily be done on a population survey basis. Shephard, as part of the IBP studies in Igloolik, performed body composition studies on 74 Inuit men and women using the deuterium oxide dilution method (Shephard et al 1973). In 1990, the body composition of 51 men aged 18-36 were measured by underwater weighing (Rode and Shephard 1992)(Table 3.5). Shephard pointed out that the body fat data were higher than would have been expected from the skinfold data. It should be noted that various assumptions are required in applying standard formulae for various body components, one of which is that fat-free mass contains 73% water, a figure derived from studies on White cadavers. Shephard posited the explanation that the state of hydration of Inuit fat-free mass may deviate from the 73%, or alternatively, the Inuit stored their fat internally (ie. in the omentum, mesentery, and retroperitoneum) rather than in the subcutaneous tissues (Shephard 1980). Direct evidence in support of the latter premise, however, is lacking. Recent

technological advances in computed tomography and B-mode ultrasonography and their application in the measurement of visceral and subcutaneous fat at various cross-sections of the body (Ashwell et al 1985, Ramirez 1992) would provide the means to collect the needed data.

Table 3.5 Body composition of Inuit in Igloolik, 1970-71

Age-Sex Group	Sample Size	Total Body Water (%)	Body Fat (%)
15-19 M	11	63.9	12.7
15-19 F	9	59.0	19.4
20-29 M	12	62.0	15.3
20-29 F	12	58.7	19.8
30-39 M	10	64.5	11.8
30-39 F	10	54.2	26.0
40-59 F	10	54.6	25.5

Source: Shephard, Hatcher and Rode (1973)

Prevalence of obesity risk categories

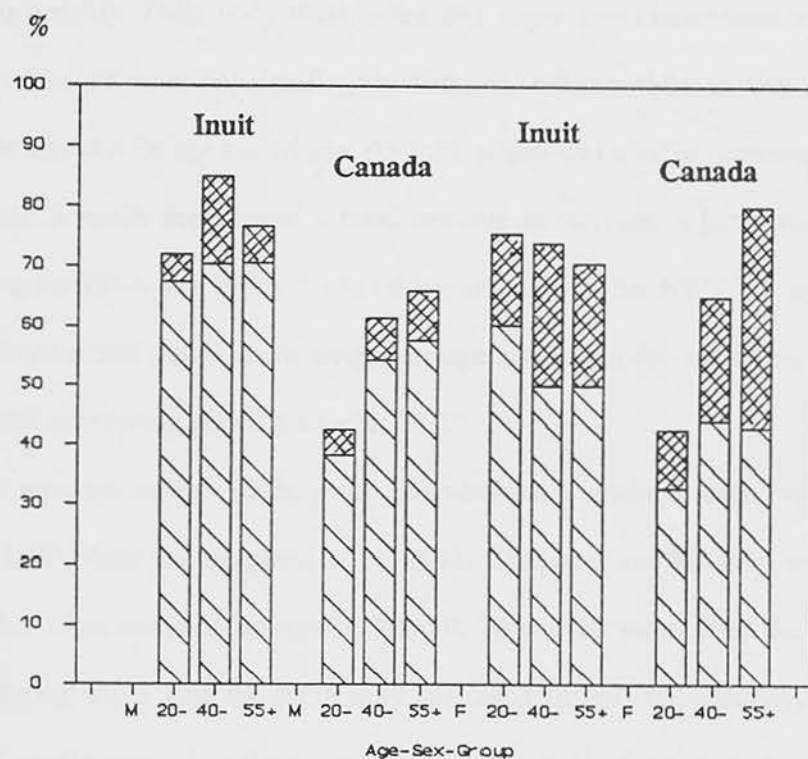


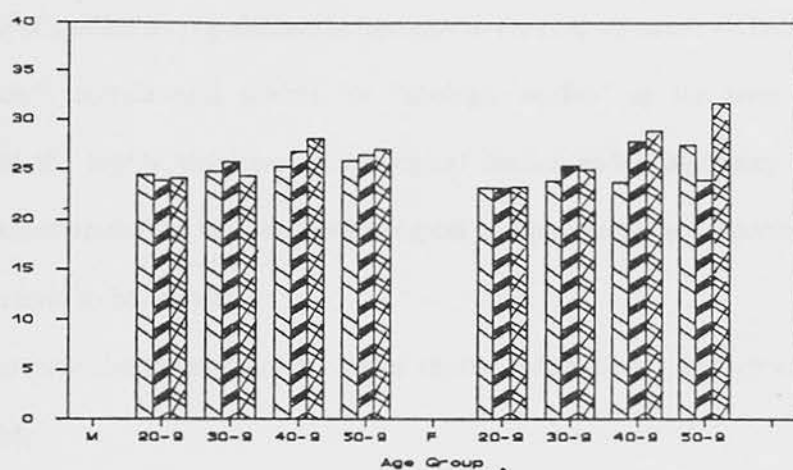
Fig.3.6 Prevalence of obesity risk categories based on ponderal index, Inuit and Canadian adults, Nutrition Canada Survey, 1972

moderate risk  high risk 

The Eskimo physique is evident in childhood, when compared to White and Black American children. In a study of the St. Lawrence Island Eskimo children and young adults up to age 20, Johnston and others (1982) computed various age-adjusted anthropometric indicators. Thus Eskimo children were generally shorter in stature (due to a shorter leg length, but comparable sitting height). Their body mass index and upper arm circumference were also higher but triceps skinfold were not significantly different. Eskimo children also had greater mean body weight adjusted for age and triceps skinfold, which was used to represent indirectly the lean body mass. Schaefer documented a trend towards an increase in birth weight among Inuit infants during the 1950s and 1960s in several communities of the NWT. Citing 1968 and 1938 data from Cumberland Sound, both weight-for-age and height-for-age in childhood had increased during the intervening period (Schaefer 1970).

Data from repeated surveys in the same community are available only from Igloolik, NWT (Shephard 1980, Rode and Shephard 1984, 1992). Shephard and his team were able to conduct two further cross-sectional surveys in Igloolik 10 and 20 years after the initial IBP survey in 1970, during which time the community has "modernized" considerably, as gauged by the number of people engaged in the wage economy, the decline of hunting as a means of livelihood, and increasing ownership of such labour-saving devices as snowmobiles. During this interval, there was a substantial increase in the sum of three skinfolds and, to a lesser extent, body mass index (Fig.3.7). Among Alaskan Yupiks three diabetes surveys in 1962, 1972 and 1987 showed that the prevalence of obesity had also increased substantially (Mouratoff and Scott 1973, Murphy et al 1992).

body mass index



sum of three skinfolds
(mm)

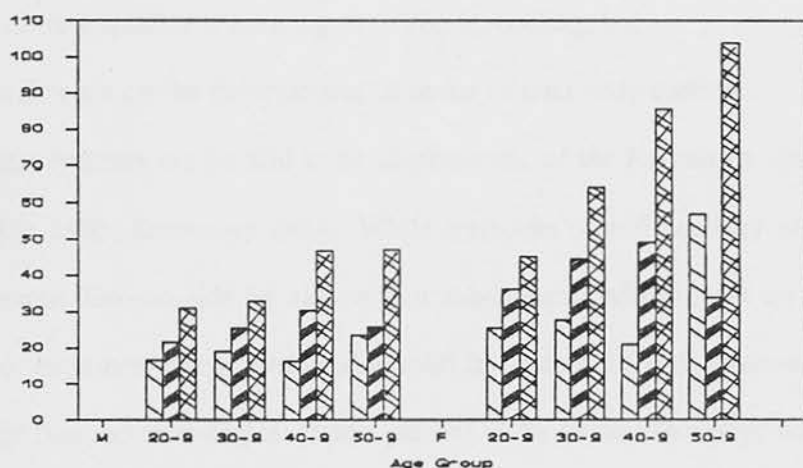


Fig.3.7 Change in mean body mass index and sum of three skinfolds in Igloolik between 1970, 1980, and 1990 (Source: Rode and Shephard 1992)

1970 1980 1990

Cold Adaptation

In a review of studies on populations indigenous to the cold climates, including both the "between-population", correlational studies (or "ecologic studies" as the term is used in epidemiology), and the highly elaborate, physiological studies under laboratory conditions, Steegman (1975) summarized the various morphological and physiological responses:

- (1) The people tend to be heavier;
- (2) they tend to have shorter extremities, larger chests, reduced body surface area per unit body weight;
- (3) they tend to have a narrower and higher nose;
- (4) they tend towards round headedness;
- (5) they tend to have quicker rewarming to extremity cooling; but
- (6) no clear difference can be demonstrated in terms of total body cooling.

Not all these features can be said to be characteristic of the Eskimo, or shared among Arctic dwellers (So 1980, Szathmary 1984). While textbooks of anthropology often showed photographs of Arctic Eskimo side by side with a sub-Saharan African, not all differences between these two "extremes" can be attributed to cold! It is indeed difficult to sort out the result of low temperature from other ecological factors present in the Arctic. That large body size and short extremities (with the effect of reduced surface area and thus reduced heat loss) are associated with cold climate has been demonstrated in warm-blooded animals, which have been codified into the so-called Bergmann's and Allen's rule since the latter half of the 19th century. The correlation of mean body height and weight and mean temperature was examined by Roberts (1953), who assembled data from 116 samples of men globally. He found that mean weight, but

not mean height, correlated with mean annual temperature. The weight/temperature correlation remained even when height was entered into the regression equation (and thus "adjusted for"). Roberts' series included 16 populations indigenous to the Americas, including Eskimos, and the correlation coefficient for this subgroup was -0.5. This type of study was replicated by Newman (1960) who restricted his analysis to 60 Native American populations in the western hemisphere, including 10 Eskimo groups in various parts of Alaska, Canada, and Greenland. With height being held constant, the partial correlation of weight and temperature was - 0.67, whereas that between height and temperature (controlling for weight) was +0.29. Newman further found that the correlation improved if the mean temperature of the coldest month was used instead of the mean annual temperature. As indicated in an earlier section in this chapter, there is little evidence that Eskimos in the past were characterized by thick subcutaneous tissues. It would therefore appear that the main strategy against cold has not been insulative, but rather metabolic.

Many studies (reviewed in Itoh 1980) reported that the basal metabolic rate (BMR) among Eskimos, particularly among those living in a more traditional lifestyle, was higher than standards established in Whites, although this observation had been attributed to a high protein diet and test anxiety rather than cold adaptation (Rodahl 1952). Roberts (1952) correlated BMRs with mean annual temperature in a global survey. In the western hemisphere, the indigenous populations generally had higher values than Euro-Americans. Among Native Americans, the Arctic Eskimos and subarctic Chipewyan Indians in the extreme north and the Mapuches in Chile in the extreme south reported the highest values, with the Mayans in central America occupying intermediate position. Among Native Americans, the correlation coefficient was -0.95 between

basal kcal/day and mean annual temperature; the r was -0.36 when kcal/kg body weight was used (Roberts 1952).

Physiological studies involving extremity cooling showed that, compared with non-Eskimos, the Eskimo had higher mean finger or hand temperature after cooling, higher manual dexterity, lowered skin sensitivity and higher pain tolerance, and earlier onset of cold-induced vasodilatation (Hildes 1966, Steegman 1975, Itoh 1980).

Perhaps more important in cold adaptation are behavioural strategies, including shelter, clothing, work/activity pattern, diet and eating habits, and risk avoidance/safety precaution practices (Moran 1981, Steegman et al 1983). That such adaptations have been remarkably successful is illustrated by the relatively low frequency of cold injuries among Native Americans inhabiting cold climate regions, provided that there is no impairment by alcohol. It has been pointed out that the main problem with Eskimos is one of heat dissipation when fully clothed and during vigorous physical activity, rather than one of heat conservation (Rennie et al 1962). It is interesting that Schaefer found a greater number of actively functioning sweat glands in the face of Eskimos than on the body and extremities, parts which were usually covered (Schaefer et al 1974).

Physical Activity and Fitness

As part of the IBP human adaptability studies in a variety of populations around the world, physiological studies were conducted in the field which provided measures of physical fitness and work capacity, including maximal oxygen intake (VO_{2max}) and muscular strength (force of hand grip and leg extension). The maximal oxygen uptake provides an indirect measure of the oxygen utilized in metabolic oxidation of foodstuffs and thus the energy output by aerobic

processes or aerobic power. It can be predicted from the heart rate and oxygen consumption measured from expired gas according to the Astrand nomogram during the ascent of a double step. It may be expressed in litres per minute (absolute) or ml per kg body weight per minute (relative). Shephard (1985) concluded that physiological differences between colonists of various extreme environments were very small. Differences that existed could be obscured by other factors such as malnutrition, disease, and socioeconomic status.

Among the Inuit in Igloolik in 1970, Shephard and his coworkers noted that they showed an exceptionally high level of aerobic power, little subcutaneous fat, and a superior leg strength. A high degree of physical fitness was required in order to wear bulky clothings and footwear and walk long distances over rough terrain of ice and snow. A hunting lifestyle was associated with a high level of energy expenditure: an average of 15-16 MJ/day, compared to 8-10 MJ/day of an office worker, with periods of even more intense exertion. By 1980 the maximum oxygen intake had decreased by an average of 10 and 12% in men and women respectively. Another repeat survey in 1990 showed further decline: compared to 1970 average values for hand grip force, leg extension force, absolute aerobic power, and relative aerobic power were lower by 23%, 41%, 17% and 22% respectively (Rode and Shephard, 1984, 1992).

Diet, Nutrition and Metabolism

The major food species exploited by the Inuit were mentioned earlier in Section 5.1. The earlier literature on Inuit foodways as described by ethnographers and analyzed by itinerant physiologists was reviewed by Sinclair (1953). The generalization that the "traditional" Eskimo diet was very high in protein and fat, in cholesterol and in retinol, and low in carbohydrates, fibre, ascorbic acid and tocopherols (Editorial, *Lancet* 1983), should be qualified by a

recognition of the considerable regional and seasonal variability in food sources. Cultural and ecological factors contribute to a widely different dietary pattern. Depending on the locality, fish, sea mammals or caribou may constitute the main source of food. In the past several decades, no area has been spared the introduction and increasing consumption of imported "modern" or "western" foods.

Among the Inuit, changes in the relative contribution of proteins, fats and carbohydrates to total caloric intake, the proportions of "country" and imported foods, the adequacy of specific nutrients, and peculiarities in carbohydrate and lipid metabolism all play a role in the emerging pattern of disease. It has been estimated that in the precontact Eskimo diet 66% of caloric intake were derived from fats, 32% from proteins, and only 2% from carbohydrates (Draper 1977). In the 1960s and 1970s, these proportions ranged from 20-40% for fats, 20-50% for proteins and 30-50% for carbohydrates (Schaefer and Steckle 1980:4).

Few large-scale population dietary surveys have been conducted in the Arctic. In Alaska the Dietary Survey of 1956-61 represented the first systematic effort in collecting dietary information from the Native population. This survey involved some 5,000 dietary records collected in 9 Eskimo and 2 Indian villages (Heller and Scott 1967). Dietary data were also collected in northwestern Alaska as part of the IBP human adaptability studies (Bell and Heller 1978). During 1987-88, a statewide survey covered many of the previously surveyed Native communities and provided a useful longitudinal perspective. Alaska Natives still derived 37% of their energy from fats, essentially unchanged from the earlier 1956-61 survey. The consumption of fish, however, continued to be high (Nobmann et al 1992).

In Canada, various small-scale community studies in the 1960s and 1970s were summarized by Schaefer and Steckle (1980). The Nutrition Canada Survey of 1972 included a sample of 366 Inuit from four communities in the NWT (as well as 1,800 Indians from 29 reserves across the country), using the 24-hr recall method (Canada DNHW 1975). Compared to Canadians nationally, Inuit reported a higher intake of meat (especially fish, wild game), but also of sugar, while consumption of all other foods was lower. Median intake of total calories was also lower, while that of proteins was higher (Fig.3.8). The intake of some minerals and vitamins, notably calcium, vitamin A, C, D and folate, were actually below the recommended daily allowance, while that of the B vitamins and iron was high. Schaefer had monitored the change in per capita sugar consumption during the 1960s in various communities and reported a four-fold increase, corresponding with a decline in protein (Schaefer 1970).

Small-scale studies involving weighing and chemical analyses of food sources and prepared meals have been conducted during the 1970s in Umanak, Greenland (Bang et al 1976, 1980) and Arctic Bay, NWT (Hoppner et al 1978), and more recently in Broughton Island (Innis and Kuhnlein 1987). Of particular interest is the fatty acid composition of the Inuit diet. In northwestern Greenland, about 39% of total caloric intake were from fats, comparable to the 42% reported from Danes in Denmark. Inuit also consumed twice as much dietary cholesterol than Danes. However, the proportion of saturated, monounsaturated, and polyunsaturated fats was 23%, 58% and 19% respectively among Inuit and 53%, 34% and 13% among Danes. The ratio of polyunsaturated (PUFA) to saturated fatty acids, or P/S ratio, was 0.84 among Inuit, much higher than the 0.24 among Danes. Of the PUFAs, those found in much higher proportions in the Inuit diet belonged to the n-3 (or omega-3) family of marine origin such as

eicosapentaenoic acid or EPA (20:5n-3) and docosahexaenoic acid or DHA (22:6n-3). By contrast, fatty acids of the n-6 family, such as linoleic acid or LA (18:2n-6) was found in greater proportions in the Danish diet (Bang, Dyerberg and Sinclair 1980). Dietary EPA acts on various enzyme systems involved in fat metabolism. It has been shown to increase the proportion of EPA and DHA, and decrease the proportion of LA in plasma, platelets and red blood cells. Dietary EPA depresses plasma triglycerides and elevates high-density-lipoprotein, with a variable effect on low-density-lipoprotein and total cholesterol (Goto et al 1993).

In Broughton Island, Innis and Kuhnlein (1987) analyzed the fatty acid composition of the adipose tissues of Arctic mammals and found high levels of long chain n-3 fatty acids in marine mammals such as narwhal, ringed seal, bearded seal and walrus and the amphibious polar bears but relatively low in the terrestrial caribou. However, the content of PUFA in caribou was still higher than that found in the beef of modern farm animals (Draper 1977, Hoppner et al 1978). In addition to being EPA-rich, the traditional Eskimo diet may be beneficial to health in lacking the unnatural isomers of unsaturated fatty acids found in such products as margarine produced by industrial processes (Booyens et al 1986).

It is difficult to assign a dollar value to traditional or country foods from estimates of the volume harvested and the proportion actually used by humans as food. Usher (1976) suggested that the best indicator to evaluate the economic importance of such foods was their *substitution cost*. The high cost of imported foods and inadequate income from employment and social assistance meant that the nutritional value of the land foods could not be substituted and compensated in full.

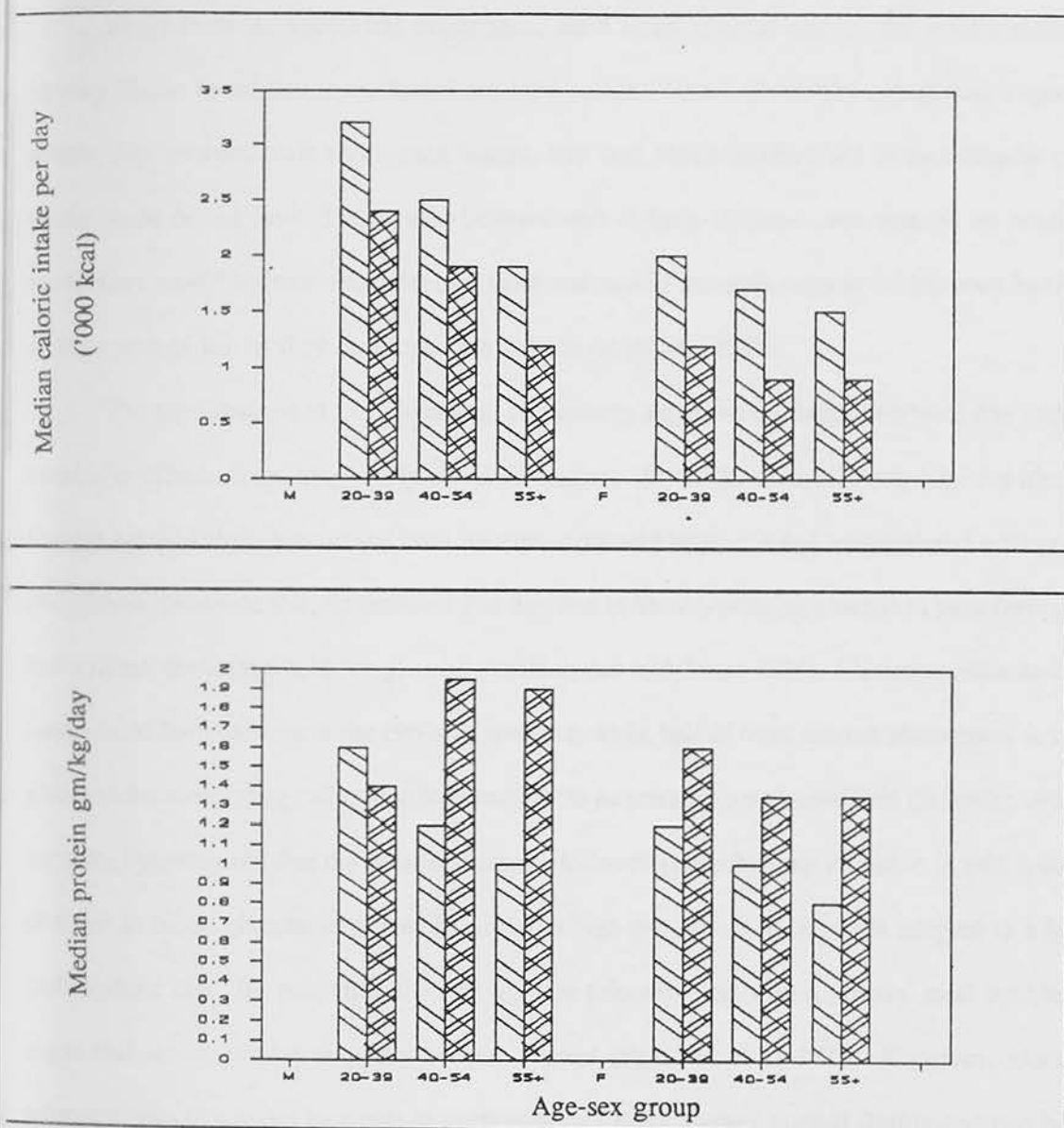


Fig. 3.8 Median intake of calories and proteins, Inuit and Canadians, Nutrition Canada Survey, 1972

Inuit  Canadians 

Apart from its nutritional importance, food is an integral part of the Inuit's cultural identity. Borre found that in the traditional Inuit cultural model of nutrition, there was a special relationship between seals and human beings, that seal blood fortified and in fact became part of the blood of the Inuit. Elders also believed that if Inuit children were weaned on country foods, they would become dependent on them and would maintain support subsistence hunting as their way of life as they developed into adulthood (Borre 1991).

The near absence of carbohydrates, particularly sugar, in the traditional Inuit diet and its metabolic effects have long intrigued physiologists. In 1935, scientists associated with the Eastern Arctic Patrol, who tested Inuit for glycosuria and ketonuria and administered a 50-gram oral glucose tolerance test, commented that they had difficulty utilizing glucose in pure form and had a raised renal threshold for glucose (Rabinowitch and Smith 1936). Clinical studies among hospitalized Inuit patients in the 1960s showed that while half of them reacted abnormally to oral glucose tolerance testing, all responded normally to an intravenous glucose load (Schaefer 1968). Schaefer hypothesized that the release of insulin following carbohydrate ingestion in anticipation of a rise in blood glucose level was lacking in a high proportion of Inuit not adapted to a high carbohydrate diet. By preceding the oral glucose tolerance test with a protein meal the blood sugar and serum insulin response became normal (Schaefer et al 1972). However, glucose tolerance tests in various localities in northwestern Alaska showed normal fasting and two-hour glucose and insulin levels (Feldman et al 1975, Bell et al 1973). Among youngsters aged 15-20, those living in a boarding school consumed twice as much carbohydrates and four times as much sugar than their peers in the remote village of Point Hope. Among the latter, who were less

"exposed" to sugar, the 1- and 2-hour glucose level was higher, while still within normal range (Feldman et al 1975).

The prevalence of diabetes and impaired glucose tolerance among Inuit continues to be low, compared to other Native American populations, although a trend towards its increase has also been observed. Diabetes is discussed in greater detail in Section 3.3.

A more important effect of a low carbohydrate diet than the issue of intolerance to ingested glucose, is the need to maintain adequate glucose level in the body to provide for essential functions. Since glucose cannot be synthesized from fatty acids, it must be derived from amino acids produced by the breakdown of proteins, a high intake of which must be maintained to achieve glucose homeostasis. The interaction of protein, lipid and carbohydrate metabolism and its role in the pathogenesis of obesity and diabetes is discussed in more detail in Chapter Seven.

The high fat (and cholesterol) intake in the Eskimo diet has not generally resulted in high plasma cholesterol and triglyceride levels. There has been a long tradition of studies on lipid metabolism among the Inuit dating back to pre-World War II days, in an attempt to discover the causes of the low incidence of ischemic heart disease in this population (Corcoran et al 1937, Wilber and Levine 1950, Scott et al 1958). The Nutrition Canada Survey in the early 1970s (Canada DNHW 1975) still showed generally low levels of total cholesterol among Canadian Inuit, particularly males (Fig.3.9). Studies in northwestern Alaska (Feldman et al 1975) and northwestern Greenland (Bang and Dyerberg 1972) further revealed low levels of triglycerides and very-low-density lipoprotein cholesterol among the Eskimos compared to whites. Greenland Inuit have also been found to have higher apolipoprotein A-I and lower apolipoprotein B than

Danes, but comparable levels of lipoprotein (a) (Gerdes et al 1992). In northern Quebec, low levels of cholesterol were also detected among the Inuit, where only 1-3% of men and 0-7% of women were considered hypercholesterolaemic (Thouez et al 1989). Studies among the Chukchi and Eskimos in Chukotka, Russia, similarly showed lower total cholesterol, triglycerides, LDL and apolipoprotein B and higher HDL than Muscovites (Gerasimova et al 1991).

The different extent of dietary acculturation has been suggested to account for an observed geographical gradient in serum cholesterol levels in Alaska (Draper 1977). In the NWT, health surveys in two Arctic communities also revealed higher serum cholesterol levels in the more "acculturated" town dwellers of Inuvik than in remote Arctic Bay (Schaefer et al 1980). In Arctic Bay itself, three successive surveys during 1976, 1978 and 1980 indicated an increase in median serum cholesterol values as well as the proportion of people considered hypercholesterolaemic (Verdier et al 1987).

Through a series of dietary, metabolic and haemostatic studies conducted during five medical expeditions to the Umanak District of Greenland in the 1970s and early 1980s, Bang, Dyerberg and colleagues proposed a hypothesis linking dietary fatty acids, serum lipids, prostaglandin synthesis, and platelet aggregability in the development of ischemic heart disease. Eicosapentaenoic acid or EPA found in fish and marine mammal common in the Eskimo diet and other polyunsaturated fatty acids (PUFA) inhibit platelet aggregation and reduce plasma triglycerides and VLDL cholesterol (Bang and Dyerberg 1972, Bang et al 1976, 1980, Dyerberg and Bang 1979, Jorgensen et al 1986).

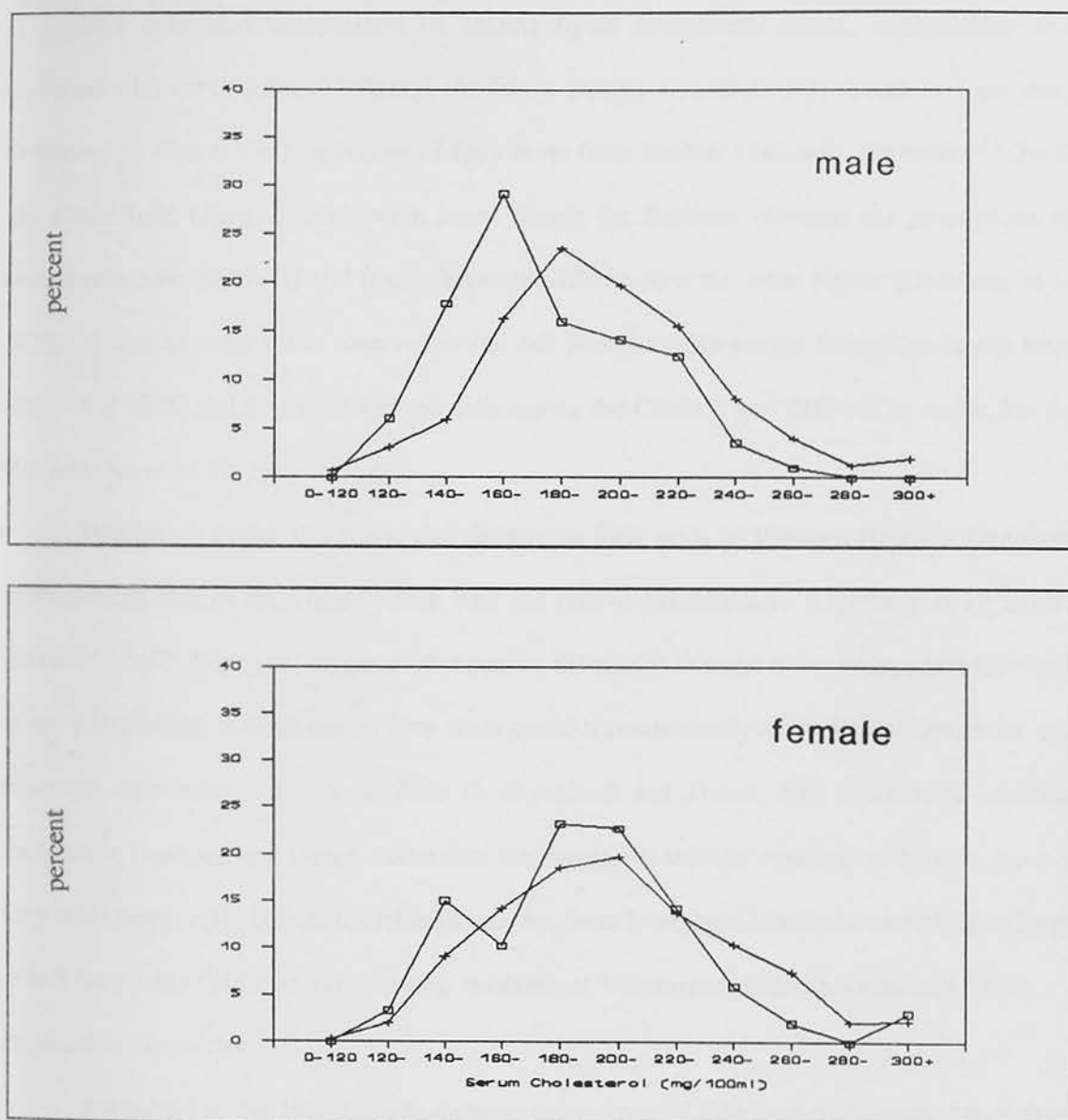


Fig.3.9 Distribution of serum cholesterol level: Inuit and Canadians, Nutrition Canada Survey, 1972

Inuit □ Canadians +

The fatty acid composition of plasma lipids (cholesterol esters, triglycerides and phospholipids) in Greenland Eskimos showed a pattern similar to that found in their diet. Compared to Danes, the proportions of fatty acids from linoleic (18:2n-6), linolenic (18:3n-3) and arachidonic (20:4n-6) acids were lower among the Eskimos, whereas the proportions of eicosapentaenoic (20:5n-3) and docosahexaenoic (22:6n-3) acids were higher (Dyerberg et al 1975). A similar pattern was observed in red cell phospholipids among Broughton Island Inuit (Innis et al 1988) and in plasma and red cells among the Chukchi and Eskimos in Arctic Russia (Gerasimova et al 1991).

Dyerberg's group also found that the plasma fatty acids of Eskimos living in Denmark approximated that of the Danes rather than the Native Greenlanders (Dyerberg et al 1975). Horrobin (1987), however, suggested that genetic deficiency in some of the desaturases involved in the intermediate metabolism of fatty acids could account jointly with dietary factors for the observed differences between Eskimos in Greenland and Danes, and similarities between Eskimos in Denmark and Danes. Some data also suggested that the exposure of Inuit to the n-3 fatty acids began early in life. Breast milk samples from Broughton Island showed a higher level of n-3 fatty acids than that found among residents of Vancouver (Innis and Kuhnlein 1988).

Population Dynamics

According to the 1986 Canada Census, there were 34,000 Inuit in Canada. More than half (55%) of the Inuit lived in the NWT (18,700), with substantial numbers in northern Quebec (21%) and Labrador (12%), and the remainder scattered across the country (Statistics Canada 1989, Norris 1990). In the circumpolar zone, Greenland has the largest Inuit population. In 1988 its total population was 55,000, 82% of whom were considered indigenous Inuit (cited in

Bjerregaard 1991). According to the 1980 U.S. Census, there were 42,000 Eskimos and 14,000 Aleuts in the U.S., of whom 80% and 60% respectively live in the state of Alaska (US Congress 1986). Across the Bering Straits, there are still 1,000-odd Siberian Yupik Eskimos remaining in the Chukotka Region. The growth of the Inuit population in the Greenland, Alaska and the NWT since 1950 can be seen in Fig.3.10. Canadian Inuit numbered around 6,000 in 1931 but has increased to 25,390 in 1981, a greater than four-fold increase (Robitaille and Choinière 1985).

One should be cautious in tracking population growth through the Census. Ethnic origin in the Canadian Census is based on self-ascription. It is therefore subject to changing perception of group identity, ethnic pride and also entitlement to special rights and benefits such as those resulting from land claims settlements. Enumeration practices also changed from Census to Census, for example in the phrasing of the question, the tracing of ancestry patrilineally, and the allowance for multiple origins. In the 1986 Census, 18% of self-identified Inuit reported mixed Inuit and non-Native origins (Statistics Canada 1989).

Fig.3.11 shows the trend in crude birth rate (CBR) for Canadian Inuit from 1955 to the late 1980s. It can be seen that the Inuit birth rate consistently exceeded that of the national population. There was a steady increase until the mid 1960s, followed by a precipitous decline over the next decade, which was halted in the mid 1970s, but the trend began to reverse during the 1980s. In the mid 1980s, the Inuit birth rate was 35/1,000, compared to a Canadian one of 15/1,000.

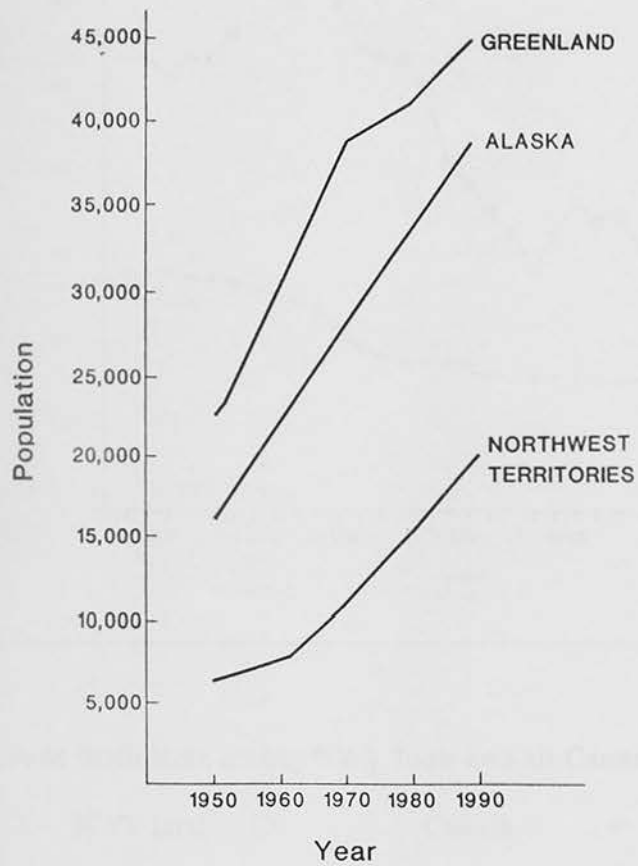


Fig.3.10 Growth of Inuit population in Greenland, Alaska and the Northwest Territories, 1950-80

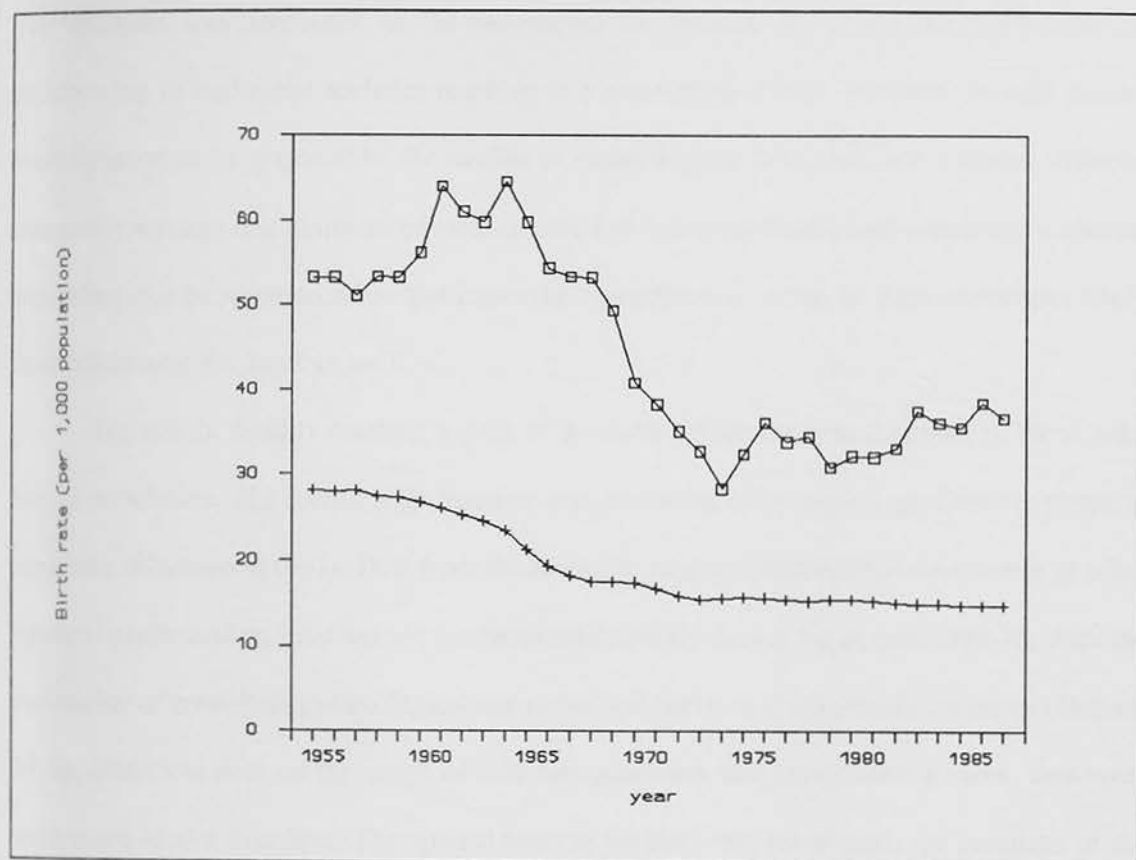


Fig.3.11 Crude Birth Rate among NWT Inuit and all Canadians, 1955-87

NWT Inuit □ Canada +

Source: Medical Services Branch, Department of National Health and Welfare and Statistics Canada

In examining the trend in crude birth rate among the James Bay Cree in northern Quebec, Romaniuk (1974) noted an increase from about 40/1000 in the pre-WWII years to just under 50/1000 in the early 1960s, corresponding to the early modernization period of this population.

The increase was attributed to the weakening or removal of biocultural inhibitions of childbearing in traditional societies resulting in a shortening of birth intervals. Several factors were believed to be responsible: the decline in breast-feeding frequency and duration, reduced pregnancy wastage due to improved medical care and living conditions, and reduction in spousal separation due to sedentarization and improved transportation. Some of these conditions likely operated among the Inuit as well.

The rise in fertility reaching a peak in the early 1960s was also observed in the Alaska Native population. The decline after that time was attributed to the beginning of family planning programs (Blackwood 1981). Data from the Keewatin Region indicated that the number of tubal ligations performed on Inuit women increased substantially during the decade 1966-76, with the availability of consultant gynaecological and obstetrical services in Churchill (Cohen and Baskett 1978). Statistical data on the usage of oral contraceptives and intrauterine devices, however, were much harder to collect. The upward trend in the birth rate (or at least, the cessation of the decline) observed during the 1980s can perhaps be explained by the fact that the large number of women born during the "boom" in the 1960s were entering the reproductive age themselves.

A more accurate indicator of fertility than the crude birth rate is the total fertility rate (TFR), which represents the sum of all age-specific fertility rates for women in the child-bearing ages. It can be interpreted as the number of children that would be born to a woman who experiences each of the age-specific fertility rates as she ages. In the late 1960s, the TFR for all Canadians was 2.5 children per woman, compare to 9.2 for Inuit. By the early 1980s, The TFR in both groups had declined, to 1.7 for all Canadian women, and more drastically, to 4.1 among Inuit respectively (Norris 1990).

Inuit fertility seems to fluctuate seasonally. Condon (1982) conducted interviews with women and reviewed medical records in Holman Island and found that prior to 1950, more births occurred during the first half of the year, peaking around March. During the period 1950-1969, characterized as a period of the greatest social change with the population being concentrated into settlements and adopting new form of economic activities, the distribution of births became bimodal, with a second peak during August. In the 1970s, with the establishment of the health clinic and the availability of birth control, the seasonality reverted to the earlier pattern with a single and more pronounced peak. This seasonality was observed among both planned and unplanned pregnancies. From a biological perspective, infants born during the first half of the year benefit from the warmer and brighter summer months during early infancy. Behaviours which promote this outcome include the absence of hunters out on the land during the autumn; when they return during the months of winter darkness, the major social activity is visiting other families, limiting the opportunity for sex. With approaching daylight, families leave for seal hunting camps and have more privacy.

The age-sex distribution of Canadian Inuit reveals a much younger population than the national population. The wide-based pyramid of the Inuit is comparable to that of many developing countries today or that of the general North American population of a century ago. In 1986 41% of Inuit were under the age of 15, while 3% were 65 and above. The corresponding Canadian proportions were 22% and 10% respectively. The changes of these proportions over successive censuses are shown in Fig.3.12.

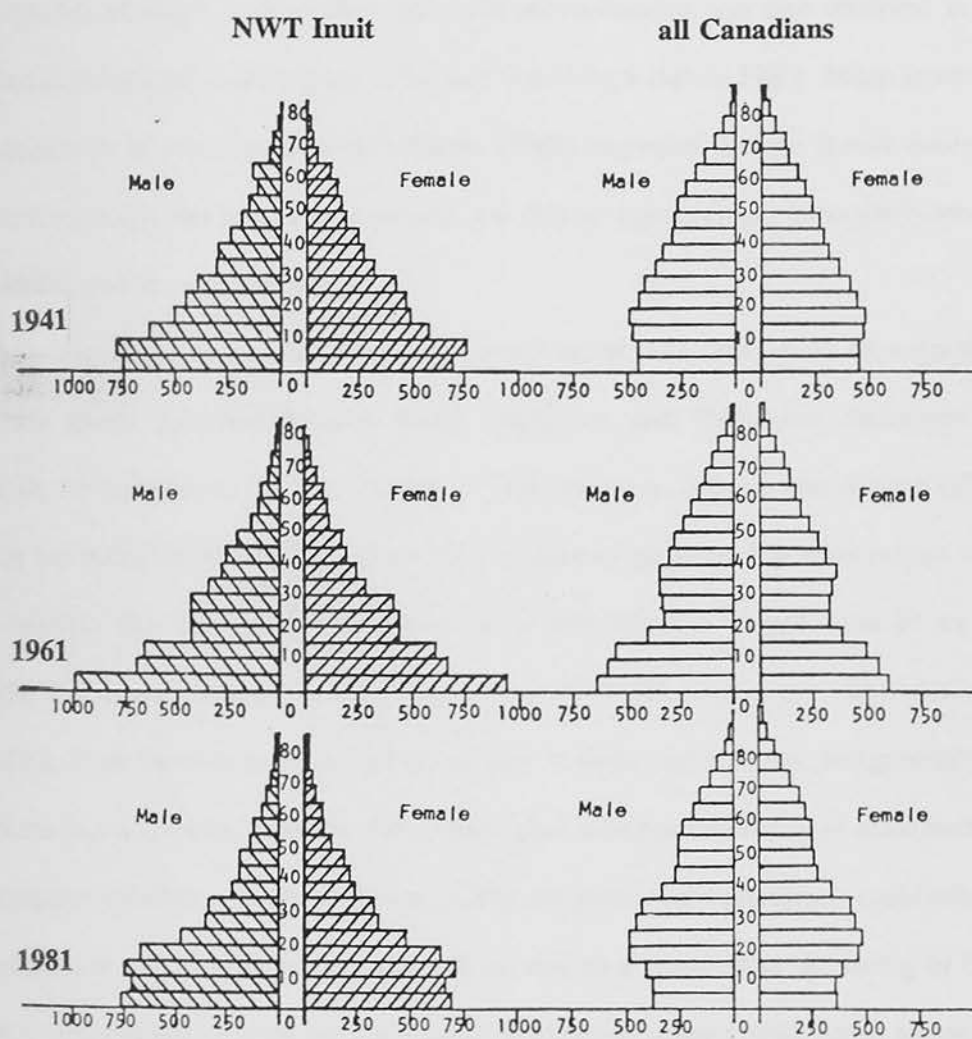


Fig.3.12 Age-sex composition of NWT Inuit and Canadian populations, 1941, 1961, 1981 (Reproduced from Robitaille and Choinière 1985)

Note: 5-year age groups per 10,000 population

The sex ratio among Inuit, with an overall male preponderance when all ages are combined, is the reverse of that observed in most populations. In 1986 the ratio was 1029 M to 1000 F. In studies of single communities, the male preponderance was also observed in the 1970s in Igloolik (McAlpine and Simpson 1976) and Wainwright (Milan 1980). Milan attributed this to out-migration of young men while Schaefer (1982) suggested that the female excess in mortality from such diseases as tuberculosis and post-partum hæmorrhage, particularly among the 1926-31 birth cohort, was responsible.

Female infanticide was at one time an important factor, and many early ethnographers such as Franz Boas, Diamond Jenness, Knud Rasmussen and Vilhjálmur Stefánsson all commented on its occurrence. In their review of this literature, Schrire and Steiger (1974) compiled the sex ratios of 12 Eskimo groups. The number of girls per 100 boys ranged from 37 to 104 (median 62). The number of adult women per 100 men ranged from 91 to 131 (median 105). The overall, all-age sex ratio ranged from 68 to 105 females per 100 males, with a median of 93. Thus there were severe deficits of girls in some communities, but generally by adulthood there was a reversal, probably due to the higher accidental mortality of male hunters. Based on computer simulation studies Chapman (1980) concluded that a population could tolerate a female infanticide rate as high as 30% and still survive as a group. The shortening of birth interval due to the cessation of lactation would result in relatively prompt replacement by another infant. In fact, there was some evidence that the Eskimo recognized this relationship and counted on the next child to be male. Cultural anthropologists pointed out that the Eskimos' decision to kill an female infant was made within the family in an *ad hoc* fashion in response to seasonal difficulties, rather than as a rigid social rule (Balikci 1967). Freeman (1971) cautioned that while

such a practice may well be adaptive (eg. improving group survival, counteracting adult male mortality, reducing the dependent population), Eskimos themselves were unlikely to be even aware of the demographic significance. Rather, its observance was a result of the social dynamics within the household.

In her demographic review of North American Indians, Johansson (1982) cited a skeletal-based estimate for life expectancy of about 23 years prior to contact, a figure not much different from most hunter-gatherer groups. It was actually quite close to the recorded Canadian Inuit life expectancy of only 29 years during the decade 1941-50. The life expectancy of the Inuit more than doubled between 1941-50 and 1978-82, when it was 66 years (Robitaille and Choinière 1985). While still about 10 years shorter than Canadians nationally, the substantial increase over a span of only four decades was a remarkable achievement, since it took many modern populations centuries to attain such a doubling in life expectancy.

The improvement in life expectancy in recent years can be attributed to the much reduced risk of death in childhood. Inuit who survive into adulthood have similar life expectancy as their national counterparts. The decline in infant mortality (IMR) among Canadian Inuit has been impressive (Fig.3.13). From a high of 145/1,000 livebirths during 1961-66 the IMR was reduced to a fifth, or 28/1,000, by the mid 1980s. This rate, however, was still three times that of the Canadian national rate. The IMR can be broken down into neonatal (under 28 days) and post-neonatal (from the 28th day up to the end of the first year of life) mortality rates. The former is generally considered to be more sensitive to medical care measures while the latter is reflective of socioeconomic conditions. The Inuit neonatal and post-neonatal mortality rates were still two and five times higher than the Canadian rate in the mid 1980s (Fig.3.14).

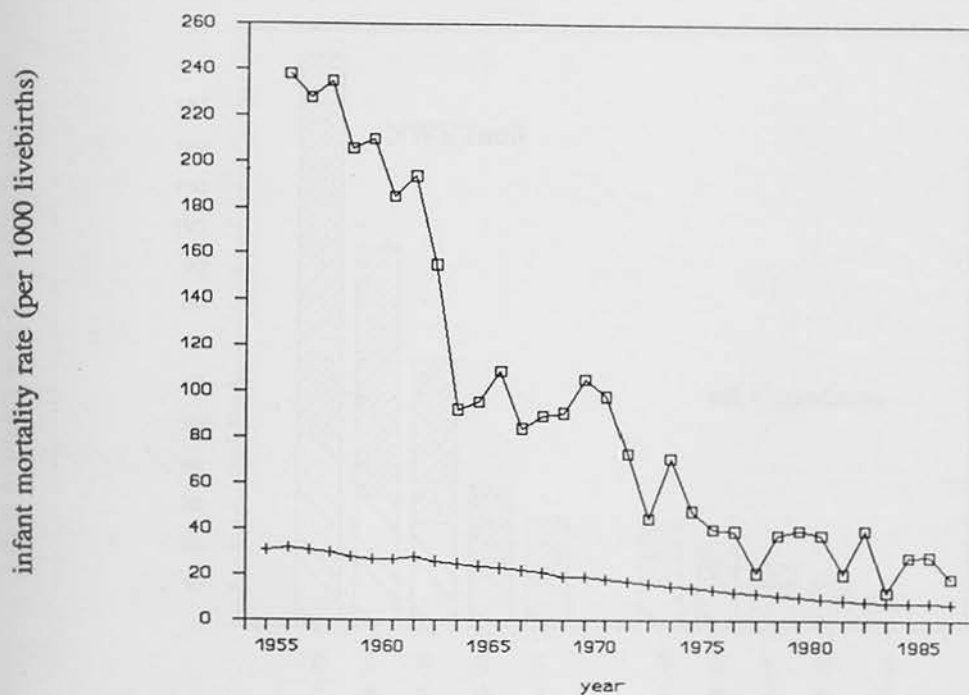


Fig.3.13 Infant mortality rate among NWT Inuit and all Canadians, 1955-87

NWT Inuit □ Canada +

Source: Medical Services Branch, Department of National Health and Welfare and Statistics Canada

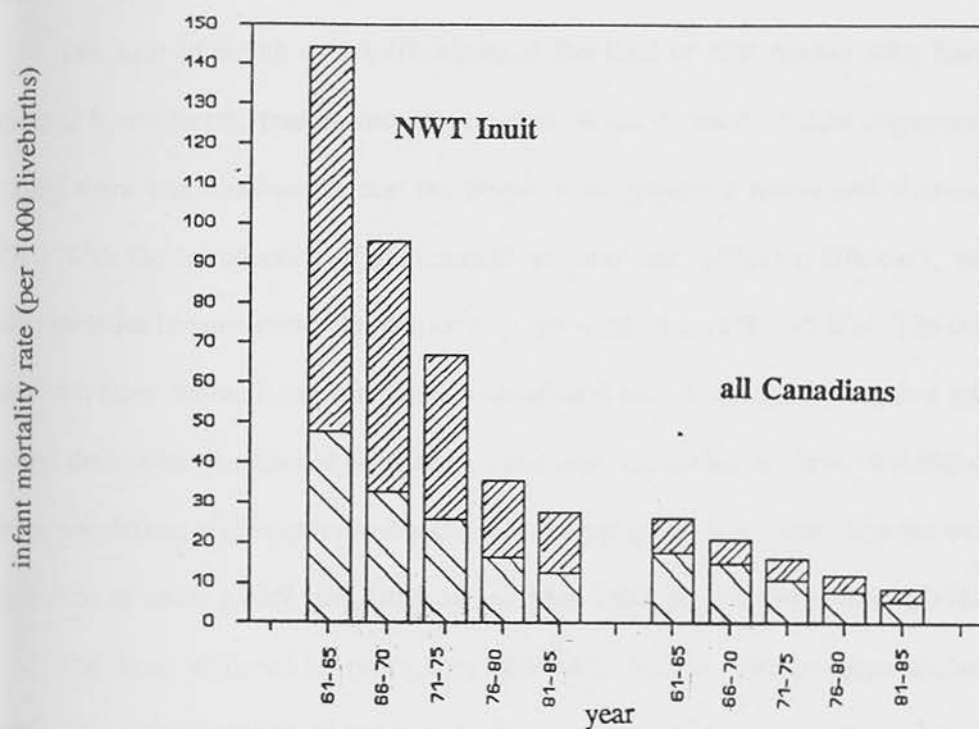


Fig.3.14 Neonatal and postneonatal mortality rate among NWT Inuit and all Canadians, 1961-85

Neonatal  Postneonatal 

Source: Medical Services Branch, Department of National Health and Welfare and Statistics Canada

3.3 Pattern of Mortality and Morbidity

Epidemiologic Transition

The state of health of Inuit/Eskimos at the time of first contact with Europeans was described by explorers, traders and missionaries. While detailed medical diagnoses were often lacking, there was a consensus that the people were generally robust and vigorous (Fortune 1971). With the introduction of government services and modern health care, more reliable health statistics became available, particularly since the Second World War. The Inuit/Eskimos share with other Native American populations several key features in their recent epidemiologic history: decline but persistence of infectious diseases, stabilizing at a level still higher than non-Native population; rise in chronic diseases but not yet quite as rampant; and the overwhelming importance of social pathologies (Broudy and May 1983, Kunitz 1983, Young 1988).

The Inuit achieved a precipitous mortality decline perhaps unparalleled by other populations. [Data on infant mortality and life expectancy at birth were presented in the previous section]. In terms of overall, all-cause mortality, the crude rate among NWT Inuit fell from a high of about 35/1000 towards the end of the 1940s to around 6/1000 four decades later (Robitaille and Choinière 1985). Fig.3.15 shows the mean crude mortality rates for several groups of causes since 1964 and their percent distribution during the period 1979-1988. Substantial reductions in mortality due to infectious/parasitic diseases, perinatal conditions and diseases of the respiratory system can be observed. [Note: these correspond to Chapters I, VIII and XV of the International Classification of Diseases, 9th revision.] Included under infectious diseases are tuberculosis, meningitis and gastroenteritis; the most important causes under the respiratory rubric are also infectious in origin - pneumonia and other respiratory infections]. In

the 1980s chronic diseases such as circulatory diseases and neoplasms were far more important than infectious diseases as causes of mortality. Injuries and poisoning [which supplanted the designation "accidents and violence" used in previous ICD editions] became the highest ranking cause in the 1980s, accounting for 30% of all deaths. There has been, however, a decline in mortality rate compared to the early 1970s.

The risk of death from various causes among NWT relative to the Canadian national population can be determined from the standardized mortality ratios (SMR). A SMR greater than 1 indicates that Canadian Inuit had a higher risk of death from that cause compared to all Canadians, having adjusted statistically for the different age distribution of the two populations. It can be seen that, when all causes of death were considered, Inuit had about twice the risk compared to Canadians (Table 3.6). Elevated risks of 3 to 6 times were associated with infectious, respiratory, perinatal conditions and injuries, whereas for neoplasms the risks were slightly elevated, and those of circulatory diseases, slightly reduced.

The extent and burden of selected diseases in the three categories of infectious, chronic, and injuries among NWT Inuit are discussed in further detail below.

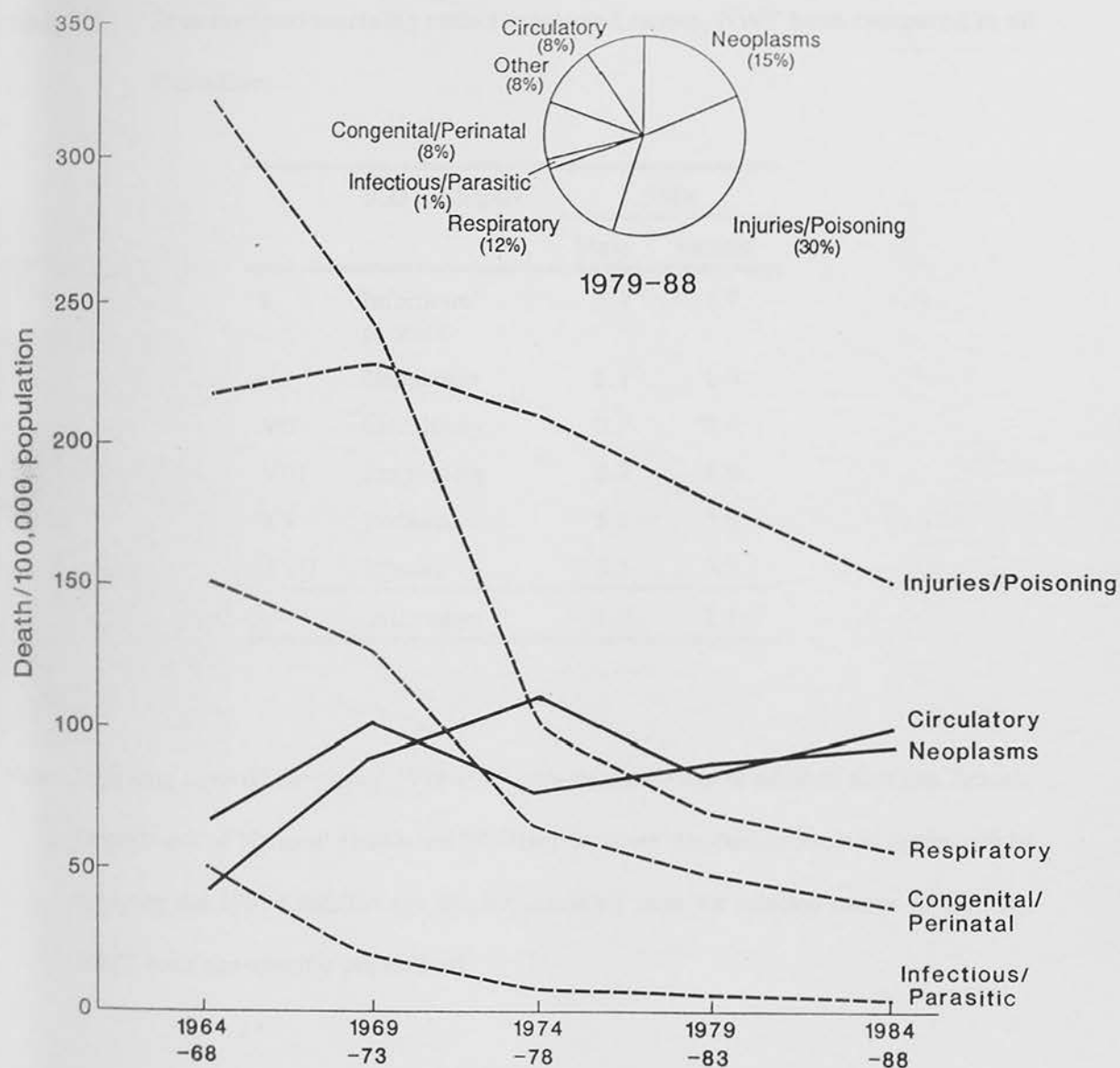


Fig.3.15 Crude mortality rate for selected causes, NWT Inuit, 1964-88, and percent distribution by cause, 1979-88

Table 3.6 **Standardized mortality ratio for selected causes, NWT Inuit compared to all Canadians**

ICD-9 Chapter		SMR	
		Male	Female
I	Infectious/ parasitic	3.4	5.7
II	Neoplasms	1.1	1.3
VII	Circulatory	0.7	0.9
VIII	Respiratory	2.7	5.9
XV	Perinatal	3.1	3.6
XVII	Injuries	3.5	3.9
All causes		1.7	2.1

Note: Inuit data covered the period 1978-1986 [raw data courtesy of Medical Services Branch, Department of National Health and Welfare]. Indirect standardization was performed by applying the 1981 Canadian age-specific mortality rates for selected causes to the 1981 NWT Inuit age-specific population].

Infectious Diseases

The success of immunizations has reduced the epidemiological significance of diseases such as measles, rubella, mumps, poliomyelitis, tetanus and diphtheria in Native American communities. It should be remembered that, as late as the 1950s, "virgin-soil" epidemics of these diseases still occurred in some areas in the Arctic. The building of the Alaska Highway

during World War II by the US Military resulted in a series of epidemics among Natives in the Yukon Territory (Marchand 1943). In Chesterfield Inlet, NWT, eight percent of the Inuit population contracted polio from white workers stationed in Churchill, Manitoba to the south, and 2% of the population died (Adamson et al 1949). In 1952, a measles epidemic swept through Baffin Island, NWT and the Ungava peninsula in northern Quebec, with an attack rate of 99% and mortality rate between 2% and 7%. This epidemic was traced to Inuit visitors to the Armed Forces base at Goose Bay, Labrador (Peart et al 1954).

Of all the infectious diseases, **tuberculosis** was the most important cause of mortality, morbidity and long-term disability among the Inuit during the first half of the 20th century, not only in Canada, but also in other circumpolar countries (Grzybowski et al 1976). The steep decline in incidence since the 1960s, with the availability of effective chemotherapy and other public health control measures, can be seen from Fig.3.16 (logarithmic scale).

The high incidence of **meningitis** has been demonstrated in many Native communities in Canada and the U.S. (Ward et al 1986, Wotton et al 1981). The most prevalent causative organism is *Haemophilus influenzae* type b (Hib), among other bacterial and viral agents. For Inuit in the Keewatin region of the NWT, the rate in excess of the province of Manitoba was 20 times among the under-5s and 33 times among infants (Hammond et al 1988).

Outbreaks of diarrhoeal disease or **gastroenteritis** still occur periodically in the Arctic (eg. Fournelle et al 1966, Bender et al 1972). While its persistence can be attributed to the overcrowding and inadequate sanitation that still exists in many communities, the frequency and severity is far less than that experienced in developing countries.

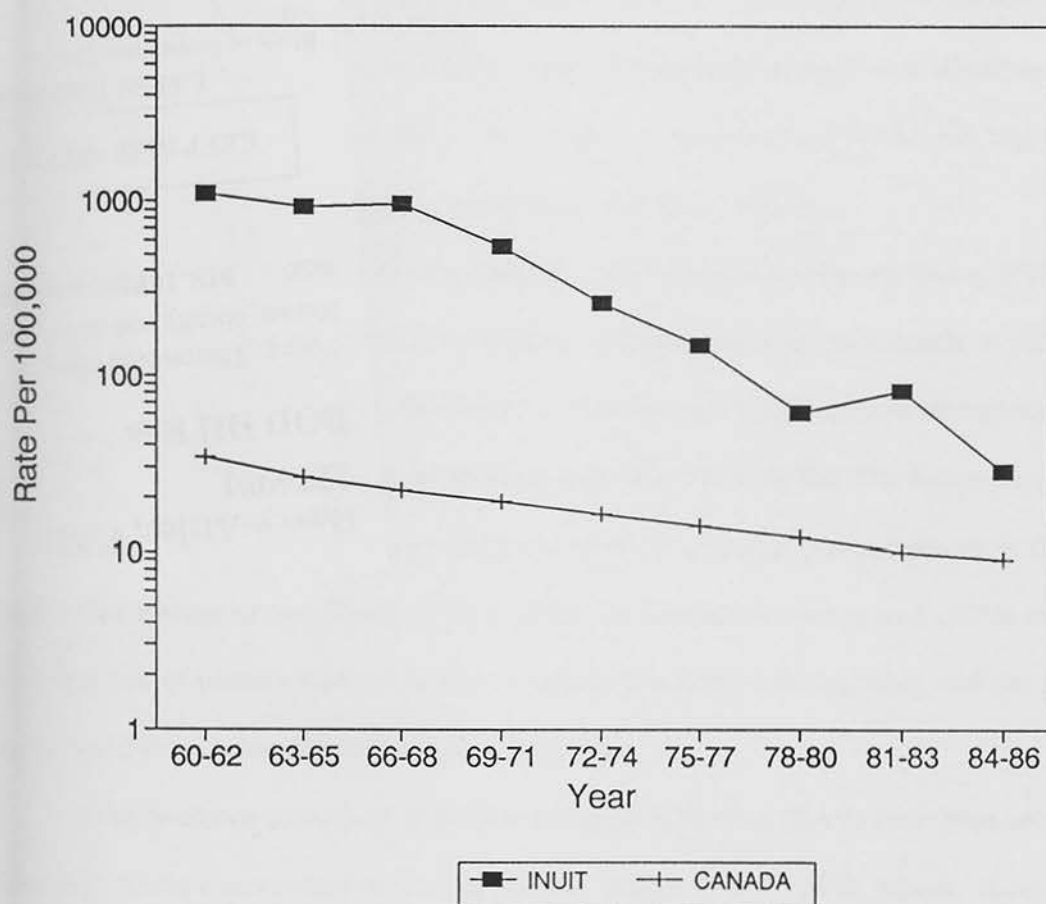


Fig.3.16 Trend in tuberculosis incidence: NWT Inuit and all Canadians, 1960-1986

A disease which can almost be considered a "culture-bound" disease among Inuit is botulism, a form of poisoning caused by the toxin produced by the bacteria *Clostridium botulinum* found in contaminated food. In Canada, between 1971 and 1984, 61 outbreaks involving 122 cases and 21 deaths were reported, almost exclusively among Inuit from northern Quebec and the NWT, and Indians from British Columbia (Hauschild and Gauvreau 1985). Among the Inuit, raw, parboiled or fermented meat, particularly the traditional delicacies *urraq* - uncooked seal flippers soaked in seal oil, and *muktuk* - fermented whale meat with blubber and skin, are particularly susceptible to contamination with *C. botulinum*.

While Fig.3.15 indicates that the mortality rate of respiratory diseases among NWT Inuit has declined substantially, the risk of respiratory infections such as **pneumonia** is still much elevated in the Arctic. In Alaska, the risk of pneumonia caused by *Hæmophilus influenzae* was 12 times greater than non-Natives in the same state (Ward et al 1986). The incidence of other forms of pneumonia such as pneumococcal pneumonia was also high among Eskimos, particularly those under the age of two (Davidson et al 1989). In Greenland, Bjerregaard (1983) found an increased rate of primary care contacts for respiratory infections among those with the poorest housing and those belonging to the lowest social class.

Seroprevalence surveys of antibodies to **hepatitis A virus (HAV)** have been conducted in several Arctic communities in Canada (Minuk, Waggoner et al 1982, Minuk, Nicolle et al 1982) and Greenland (Skinhøj et al 1977). These consistently showed extremely high prevalence (in the order of 70% and above), comparable to the situation in many developing countries. Seroconversion occurs at an early age, and by middle age, most if not all members of the community would be HAV-positive.

Because of the causal association between **hepatitis B (HBV)** and hepatocellular cancer, the prevalence of HBV has been extensively studied in various Inuit communities in the circumpolar region. Although the prevalence was generally high, there were important regional differences. The Greenland rate was the highest, suggestive of hyperendemicity (Skinhøj 1977). The Canadian Inuit rates were lowest (Minuk, Nicolle et al 1982, Minuk et al 1985, Larke et al 1987, Baikie et al 1989). HBV infection was uncommon before age 20 but increased in prevalence with age, reaching 80-90% in those over age 50.

While only a very small number of Native Americans have been afflicted, the human immunodeficiency virus (HIV) has definitely entered the Arctic. The potential of devastating epidemics exists, if the experience with other **sexually transmitted diseases (STD)** is any indication. While data on STDs in general are inadequate, notification rates of gonorrhoea and seroprevalence surveys of chlamydia infection suggest that the indigenous populations of the Arctic are at high risk for such diseases, attributable to the prevalent behavioral patterns. Surveys among Greenlanders found a higher number of lifetime sexual partners and earlier age of first intercourse compared to Denmark (Kjaer et al 1989).

Among Native Americans, parasitic infections are one group of diseases which are very sensitive to both ecological factors and cultural practices. The occurrence of various helminths and protozoa and their animal hosts and reservoirs are limited by such factors as climate, terrain, and the flora and fauna prevalent in an area. Exposure to such parasites, however, is determined by cultural traditions in food gathering and preparation methods and the relationship to domesticated animals. While the physical environment does not change radically (except, for example, when large tracts of lands are affected by natural resource development projects),

cultural changes as a result of modernization can reduce or eliminate the risk of parasitic diseases such as **trichinosis**, **diphyllobothriasis**, and **echinococcosis** (Tanner et al 1987, Curtis and Byland 1991, Desrochers and Curtis 1987).

Chronic Non-Communicable Diseases

The chronic, non-communicable diseases are characterized by insidious onset, slow progression, long duration and a non-infectious etiology. Within this diverse and disparate group, several diseases, including various cancers, diabetes, ischemic heart disease and stroke, have been called "diseases of modernization" or "Western diseases" (Trowell and Burkitt 1981) because of their increasing frequency in tandem with sociocultural changes experienced by so-called traditional societies. These diseases are also interrelated in their pathogenesis and share similar risk factors.

Studies during the 1960s and 1970s suggested that the Eskimo/Inuit appeared to have been "spared" the diabetes "epidemic" experienced by many Native American groups (Scott and Griffith 1957, Mouratoff et al 1967, Mouratoff and Scott 1973, Sagild et al 1966). Surveys during the 1980s indicated that, while the prevalence was still low relative to many Indian tribes, it had nonetheless increased when compared to two decades ago (Schraer et al 1988, Young et al 1990, Murphy et al 1992). A circumpolar survey of diagnosed diabetes among Chukchi, Aleuts, Eskimos, and Athapaskans from both sides of the Bering Straits indicated that considerable variation existed, although all were below the U.S. all-race prevalence (Fig.3.17), and substantially below the rate in such high-prevalence groups as Pima Indians in Arizona and Nauruans in the Pacific (Young et al 1992).

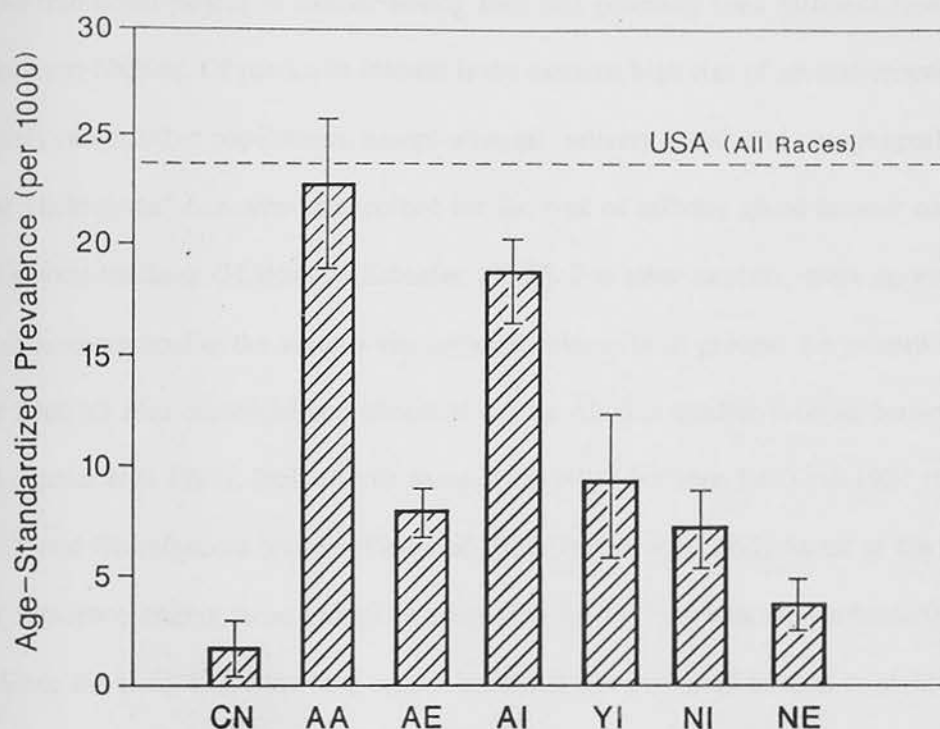


Fig.3.17 Prevalence of diagnosed diabetes in the circumpolar indigenous populations of Chukotka (Russia), Alaska (USA), Yukon and NWT (Canada)

CN	Chukotka Chukchi and Eskimos	AA	Alaska Aleuts
AE	Alaska Eskimos	AI	Alaska Indians
YI	Yukon Indians	NI	NWT Indians
NE	NWT Inuit		

(Source: Young et al *International Journal of Epidemiology* 1992)

The traditional pattern of cancer among Inuit has generally been different from that of Indians and non-Natives. Of particular interest is the extreme high risk of several cancers which are relatively rare in other populations: nasopharyngeal, salivary gland, and oesophageal cancer. [The term "Eskimoma" has even been coined for the type of salivary gland tumour commonly observed among the Inuit (Hildes and Schaefer 1984)]. For other cancers, there appears to be regional differences among the various circumpolar Eskimo/Inuit groups. An overall increase in cancer from all sites combined was observed among Alaskan Eskimo women between 1969 and 1983 (Lanier et al 1989), Inuit of both sexes in the NWT between 1970 and 1984 (Gaudette et al 1991), and Greenlanders between 1953 and 1985 (Prener et al 1991). Much of the increase in cancer incidence among these groups is accounted for by lung cancer, particularly among women. Since the early 1970s the lung cancer incidence has increased at least twofold in these populations. In Greenland, the rate has increased at least 10-fold compared to the 1950s. Schaefer noted a change in the relative proportions of the "traditional" (salivary, nasopharyngeal, oesophageal) and "modern" (lung, cervix, colon, and breast) cancers between 1950-66 and 1974-80 in the central and western Canadian Arctic (Schaefer et al 1975, Hildes and Schaefer 1984).

"Ethnospecific" dietary factors have been sought in some cancers prevalent among Inuit. The Inuit diet contains large amount of nitrosable compounds, suspected carcinogens in such cancers as nasopharyngeal cancer. Whale meat is rich in secondary amines, and fish and meat kept fermenting in the ground and eaten as a delicacy may also produce N-nitroso compounds. An analysis of salivary nitrite and urinary N-nitrosoproline levels in four Inuit settlements in the Northwest Territories revealed low levels (Stich and Hornby 1985).

Although smoking is by far the most important cause of lung cancer, some Native Americans are exposed to other carcinogens because of unique cultural conditions. Among the Inuit, prior to the 1960s lung cancer had been primarily a disease of elderly women. This observation led to the hypothesis that exposure to the fumes and smudge containing hydrocarbons and other volatile substances while tending open-flame lamps burning seal or fish oil - a virtually round-the-clock task undertaken by women - could have been responsible (Hildes and Schaefer 1984). There is some paleopathological evidence to support this as black pigmentation (anthracosis) has been found in the lungs of female Eskimo mummies from several centuries ago in Alaska and Greenland, long before the advent of tobacco and pollution (Zimmerman and Aufderheiden 1984, Hansen et al 1985).

Atmospheric nuclear testing during the 1950s and 1960s has attracted the attention of Arctic scientists because of the accumulation of such long half-life radionuclides as cesium 137 and strontium 90 in the food chain: lichens - caribou - Eskimo/Inuit. A review of exposure records and body burdens of Alaskan Eskimos revealed low maximal annual doses comparable to the natural "background" levels in many parts of the continent. Moreover, there has been no evidence of an increased risk in leukaemia, breast cancer and bone sarcoma - tumours which can potentially be induced by radiation - among Alaskan Eskimos (Stutzman et al 1986).

The low incidence of atherosclerotic or **ischemic heart disease (IHD)** among the Inuit has been recognized for some time, according to autopsy series during the 1950s and 1960s (Arthaud 1970, Lederman et al 1962). An analysis of death certificates in Alaska covering the period 1980-86 showed that Alaska Natives (which include both Eskimos and Indians) still had significant reduced risk of mortality from IHD, with a relative risk of 0.63 compared to non-

Natives in the state (Middaugh 1990). Similarly low rates have also been reported from the Northwest Territories (Young et al 1993) and Greenland (Bjerregaard and Dyerberg 1988).

It should be noted that IHD represents the endpoint of atherosclerosis, which begins early in life. The ability of portable ultrasonography to detect atherosclerotic plaques in peripheral arteries offers a non-invasive means to ascertain the prevalence of atherosclerosis in a free-living population. A study of 61 Greenland Eskimos from the remote northwest, compared with age-sex-matched Danes in Copenhagen, showed that Greenlanders had almost the same degree and extent of atherosclerosis in the carotid and femoral arteries, and by extrapolation, in other arteries of the body as well (Hansen et al 1990).

Whereas the risk of IHD mortality is reduced, the risk of **stroke** mortality among Alaska Natives was not significantly different from non-Natives in that state (Middaugh 1990). Greenlandic Eskimos are at an elevated risk compared to Danes (Kroman and Green 1980, Bjerregaard and Dyerberg 1988), particularly bleeding intracranial aneurysms.

In Alaska, a survey of Eskimo National Guardsmen in the 1950s (Mann et al 1962), studies under the International Biological Program in the 1970s (Colbert et al 1978), as well as a large cardiovascular risk factor survey among 2356 male adult Alaskan Eskimos in the mid 1970s (Maynard 1976), found that blood pressure (BP) levels were still generally low, and the prevalence of hypertension lower than the US population. A pocket of high hypertension prevalence, however, existed among the Aleuts (Torrey 1979).

Much interest has been shown in whether BP rises with age among Eskimos, a phenomenon not demonstrated by some preindustrial populations around the world. In Igloolik, NWT, a survey of 400 subjects in 1969-70 showed a low mean BP but no rise with age (Hildes

and Schaefer 1973). Later surveys showed an age-dependent increase in BP in both sexes in Inuvik, a large town, but not among males in remote Arctic Bay (Schaefer, Timmermans et al 1980). No present Native American populations can be considered "primitive" or "preindustrial". The multinational INTERSALT study, however, included two remote, relatively unacculturated Amazonian Indian populations from Brazil. These populations not only had the lowest mean BPs among the 52 sites, but there was no rise with age (Carvalho et al 1989).

Other risk factors for cardiovascular diseases such as obesity and plasma lipids have been discussed earlier in Section 3.2.

Accidents and Violence

Among the most serious health problems affecting Native Americans in the decades since the end of the Second World War are injuries due to accidents and violence. In the younger age groups injuries are by far the most important causes of mortality and morbidity, and overall, they may account for as much as 25%-40% of all deaths. The higher risks of injury mortality have been demonstrated in Alaska (Kraus and Buffler 1979), and Greenland (Bjerregaard 1990) and the NWT (Young et al 1993). Accidents, violence and alcohol and substance abuse have been collectively referred to as the "social pathologies". The excess mortality among aboriginal peoples have often been attributed, on the "macro" level, to the prevailing economic conditions and social stress that aboriginal people experience. Alcohol abuse is believed to play a major role in most violent and accidental deaths.

Data from the NWT indicate that among the injuries, mortality from motor vehicle accidents, suicide and homicide have increased whereas "other accidents" have declined. Included in the last mentioned are fires and burns, drownings, falls, etc (Young et al 1993). That

the pattern of injuries can be used as indicators of social change and modernization is illustrated by data from Greenland. Citing the historical landmark studies of Bertelsen, Bjerregaard noted that traditional Eskimo lifestyle was associated with high rates of drowning and boat accidents. With modernization, the mortality rate from such injuries declined as the people increasingly took on less hazardous, "modern" jobs. There was also geographical variation - the more remote (and thus traditional) the communities the higher the rate of drowning deaths. Suicides, on the other hand, had the reverse geographical distribution and historical trend. During 1979-1982 when there was alcohol prohibition in Greenland, many people reverted to traditional activities such as hunting and fishing, with the result that the rate of non-alcohol related accidents increased while that of alcohol-related accidents declined (Bjerregaard 1990).

Suicides are not a new phenomenon among Native Americans. Descriptions of the "traditional" pattern of suicide and the cultural beliefs regarding suicide can be found in the ethnographic literature. Among the Eskimo/Inuit, the image of the noble, but no longer productive, hunter walking out of a snow house into the blizzard to relieve his family's hardship has been a mainstay of Arctic lore. Such suicides, anthropologists have pointed out, occurred mainly among the elderly and infirm. They were carefully considered but non-ritualized acts, often carried out after considerable family consultations in response to sickness, suffering and a feeling of uselessness (Leighton and Hughes 1955). This is in sharp contrast with the typical pattern today, when they are committed primarily by adolescents and young adults.

It was possible in Greenland to reconstruct reasonably reliably the suicide rate back to the early twentieth century. Alfred Bertelsen, perhaps Greenland's first epidemiologist, culled church registers and estimated the average annual suicide rate between 1901 and 1930 to be as

low as 4/100,000. This compares with the steadily increasing rates of 9 during 1962-66, 17 during 1967-71, 54 during 1972-76, 69 during 1977-81, and 114/100,000 during 1982-86 (Lyng 1985, Thorslund 1990).

Opinions abound as to why circumpolar aboriginal people are at such high risk for suicide, homicide, and violence. Depending on the disciplinary orientation of the theorist, explanations have been sought in terms of individual psychopathology, family dynamics, social structure, economic relations, and cultural transition. Analytical epidemiological studies on risk factors have not been conducted in the NWT, although some small case-control studies have been reported from Alaska (Kettl and Bixler 1991) and Greenland (Grove and Lyng 1979).

The importance of alcohol in Native suicide was borne out by post-mortem blood alcohol levels in Alaska, which were available from 87% of all suicides. Just under 80% of Native suicides, compared to 48% among whites, had detectable blood alcohol. The proportion with levels greater than 100 mg/dl was 54% among Natives and 20% among whites (Hlady and Middaugh 1988).

PART TWO DESIGN AND METHODS

CHAPTER FOUR THE KEEWATIN HEALTH ASSESSMENT STUDY

4.1 Study Setting and Objectives

The Keewatin Health Assessment Study (KHAS) was conducted in 8 communities in the Keewatin Region of the Northwest Territories (NWT), Canada, during 1990-91. This survey was a collaborative effort between the Keewatin Regional Health Board and the Northern Health Research Unit of the University of Manitoba. It was also part of the collaborative research project between the University of Manitoba and the Institute of Internal Medicine of the Siberian Branch of the Russian Academy of Medical Sciences, which conducted a survey in the Chukotka Region using a compatible protocol. Funding was provided by the Department of National Health and Welfare of Canada.

The study was designed initially to provide health status and health care utilization data to the regional health board, which in 1987 took over the administration of health services from the federal health department. The scope was considerably expanded to take advantage of the unique opportunity offered by a population survey to address a variety of scientific issues in human biology, epidemiology and public health from both the basic and applied perspectives.

The Keewatin Region (Fig.4.1) is located in the central part of the Northwest Territories, between 60° and 69° N and 80° and 102° W. It comprises seven, predominantly Inuit, communities on the west coast of Hudson Bay. In addition, the community of Sanikiluaq, situated on the Belcher Islands near the eastern (Québec) shore of Hudson Bay, receives its

health services from the Keewatin Regional Health Board, although administratively it is part of the Baffin Region. It has therefore been included in the Keewatin Health Assessment Study.

The primary objective of the KHAS was to provide a comprehensive assessment of the current health status and health service needs of the population in the Keewatin Region of the NWT. Apart from the collection of scientific data, the KHAS was also intended to achieve the following secondary objectives:

- (1) to involve the regional health board and the eight communities in the design and implementation of the research process;
- (2) to raise the awareness of health issues in the region;
- (3) to serve as a benchmark against which success of specific health programs can be measured in the future;
- (4) to serve as a model of community health status and needs assessment for other aboriginal populations in the circumpolar zone.

Although the KHAS covered residents of all ages, only data pertaining to adults aged 18 and above were analyzed and presented in this dissertation. The design and methods of the entire study, however, are described in this chapter.

It should be emphasized that the focus of the analysis is in the Keewatin Region and its Inuit population as a whole, and not in inter-community differences within the region. The small sample size of each of the 8 communities greatly reduces statistical power. Inter-community differences in terms of genetic background, ecological conditions and level of acculturation can be expected to be small relative to the difference between the region as a whole and other non-Inuit populations which are used for comparison.

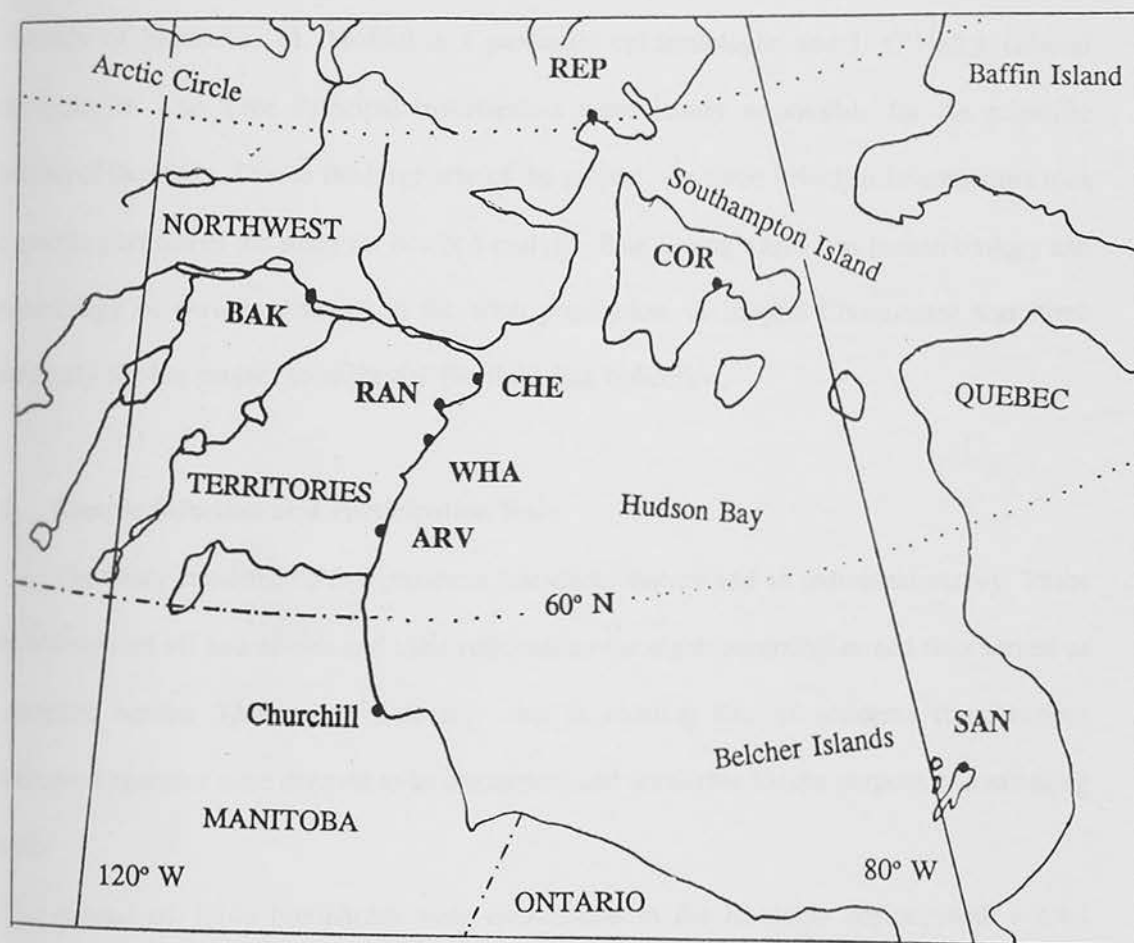


Fig. 4.1 The Keewatin Region, Northwest Territories, Canada

REP	Repulse Bay	COR	Coral Harbour
BAK	Baker Lake	CHE	Chesterfield Inlet
RAN	Rankin Inlet	WHA	Whale Cove
ARV	Arviat	SAN	Sanikiluaq

The KHAS was designed by a team of three Principal Investigators - Michael Moffatt, John O'Neil, and Kue Young, all members of the Department of Community Health Sciences, University of Manitoba. M. Moffatt is a paediatric epidemiologist and J. O'Neil a cultural anthropologist. The three Principal Investigators were jointly responsible for the scientific direction of the study. Due to the large size of the project, the three Principal Investigators took on specific portions of the study for detailed analysis. Kue Young's area was human biology and epidemiology of chronic diseases in the adult population. A Project Coordinator was hired specifically for the project to supervise the field data collection.

4.2 Sample Selection and Participation Rate

The study consisted of two phases: a household survey and an individual survey. Phase One enumerated all households and their residents in the eight communities and thus served as a complete census. This was a necessary step as existing lists of residents from various government agencies were deemed to be incomplete and unreliable for the purpose of a sampling frame.

A total of 1,146 households were enumerated in the Keewatin region, with a total population of 5,645 individuals. From the census list of names, a 20% random sample was selected from each of the 8 age-sex strata (1-17, 18-34, 35-54, 55+) in each of the enumerated communities. [This stratified sampling procedure would have resulted in the selection of essentially the same sample as would from a simple random sample of the entire region, since it was proportional to the actual age-sex distribution of the total population]. The random sample consisted of 1,135 individuals who were then invited to participate in Phase Two, the individual

survey. Among those invited to participate, 77% accepted and responded to all or part of the individual survey. The age-sex distribution of the number of people enumerated, invited and participated is shown in Table 4.1.

Table 4.1 The Keewatin Health Assessment Study: Sample selection and response rate

Age-sex group	Enumerated (A)	Invited (B)	Participated (C)	Sampling Fraction % (B)/(A)	Response Rate % (C)/(B)
(1) Male					
1 - 17	1357	272	207	20	76
18 - 34	902	181	114	20	63
35 - 54	444	87	54	20	62
55 +	182	38	31	21	82
Total	2885	578	406	20	70
(2) Female					
1 - 17	1313	2634	233	20	88
18 - 34	869	175	122	20	70
35 - 54	420	84	80	20	95
55 +	158	34	33	21	97
Total	2760	557	468	20	84
Grand Total	5645	1135	874	20	77

4.3 Survey Procedures and Data Collection

Household Survey (HS)

In addition to serving as a population census, the HS also included a 22-item questionnaire administered by trained, locally-hired research assistants to an adult member of

each household in his or her home. Data on the following sociodemographic variables were collected:

- (a) household size
- (b) age/sex composition
- (c) housing type
- (d) degree of crowding
- (e) sanitation facilities
- (f) household possessions and equipment
- (g) types of employment
- (h) household income
- (i) basic household expenses

One to three research assistants per community conducted the **HS**, which took from one to seven weeks to complete. To ensure that the data remained current, the **HS** in each community was administered no earlier than one to two months before the research team returned to conduct the individual survey.

Individual Survey (IS)

There were three different versions of the **IS**, directed at adults (ages 18 and above), adolescents (ages 13-17), and children (ages 1-12). Table 4.2 provides a list of the variables studied and the age group from which data were collected.

Questionnaire

The questionnaire was adapted primarily from existing health survey protocols such as the Canada Health Survey (Canada DNHW 1981), the WHO-MONICA study of cardiovascular

diseases (World Health Organization 1989), and the questions used in the standardized series of provincial Heart Health Surveys (MacLean et al 1992). If survey instruments were not previously established, we developed our own based on clinical experience, ethnographic observations and the Health Board's knowledge of local circumstances.

Table 4.2 Contents of the Individual Survey (IS)

Questionnaire (adults and adolescents)

- Sociodemographic Data
- Health/Safety Habits and Practices
- Nutrition
- Health Knowledge
- Traditional Activities
- Past Medical History
- Family History of Chronic Diseases
- Mental Health/Substance Abuse/Suicide Attempt
- Opinions of the Health Care Delivery System

Physical Examination

- Height, weight (all age groups)
- Subscapular and triceps skinfolds (adults only)
- Waist and hips circumference (adults only)
- Blood Pressure (adults only)
- Dental Examination (all age groups)
- Audiologic Assessment (all age groups)

Laboratory Tests

- Plasma Lipids (adults only)
 - Plasma Glucose and Insulin (adults only)
 - Electrocardiography (adults only)
 - Haematologic Indices (all age groups)
-

The interviewer-administered questionnaire included an abbreviated food-frequency questionnaire. This was devised primarily to determine the amount of traditional foods consumed, rather than a detailed quantitative analysis of caloric and nutrient intake. Physical activity levels and other health behaviours were assessed with questions designed to reflect the particular cultural practices of Inuit in the region.

A brief, self-administered, confidential questionnaire was included at the end of the personal interview, to be completed by adolescents and adults, dealing with mental health, sexual abuse and suicide attempts. Participants were instructed on how to complete the form, after which they were to remove the perforated pages from the survey booklet and seal the pages within the envelope which was provided. Mental health was assessed using a modified version of Goldberg's 12-item, abbreviated General Health Questionnaire (Goldberg 1972). This questionnaire was designed as a self-administered, screening instrument to detect potential psychiatric illness in general population surveys. The GHQ has been extensively validated and tested in different populations and national contexts (McDowell and Newell 1987:148-9), although not in an aboriginal population.

An extensive process of consultation, translation, pre-testing and back translation was undertaken to ensure that questionnaire items investigating health behaviour, knowledge and attitudes were culturally meaningful to the target population. All questionnaires were printed in both English and Inuktituk.

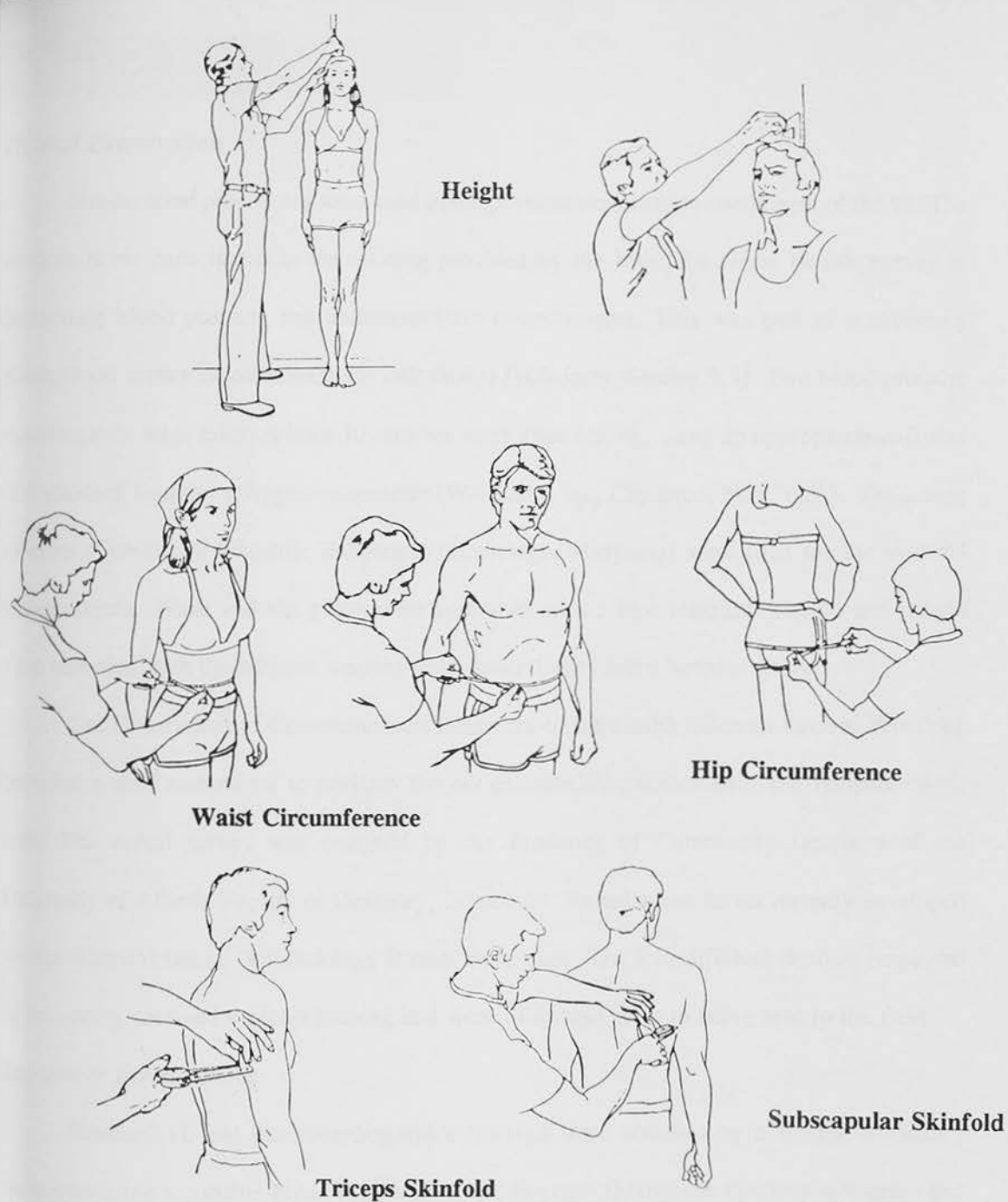


Fig.4.2 Anthropometric measurement techniques (Reproduced from Canada, Department of Health and Welfare. *Guide for Anthropometric Classification of Canadian Adults for Use in Nutritional Assessment*. Ottawa, 1983)

Physical Examination

Standardized procedures were used in the physical examination component of the IS. The research nurse participated in the training provided by the Manitoba Heart Health Survey in performing blood pressure and anthropometric measurements. This was part of a nationally standardized survey on cardiovascular risk factors [*vide infra*, Section 5.3]. Two blood pressure measurements were taken at least 10 minutes apart after resting, using an appropriate cuff size of a standard mercury sphygmomanometer (WA Baum Inc, Copiaque, New York). The Lange callipers (Cambridge Scientific Industries, Cambridge, Maryland) were used for the skinfold measurements. Waist and hip girths were measured with a tape measure. Height and weight were recorded with the subjects wearing only underclothes and a hospital gown.

The Department of Communication Disorders of the Health Sciences Centre, Winnipeg provided a staff audiologist to perform the ear examination, audiometric and tympanometric tests. The dental survey was designed by the Professor of Community Dentistry of the University of Alberta Faculty of Dentistry, Edmonton. Examination forms recently developed for the National Dental Epidemiology Project were used. The four different dentists employed in the survey received uniform training and were calibrated prior to being sent to the field.

Laboratory Investigations

Standard 12-lead electrocardiographic tracings were obtained by a trained laboratory technician using a portable MAC/PC Personal Cardiograph (Marquette Electronics/Ingram and Bell Medical, Don Mills, Ontario). The data were digitally transmitted by telephone modem to the Heart Disease Research Centre of Dalhousie University in Halifax for computer analysis. The tracings were coded according to the Minnesota Code (Prineas et al 1982).

Venipuncture was performed by the laboratory technician on adults after overnight fasting. All lipid analyses were performed by the national reference laboratory at the University of Toronto, and the protocols prescribed by this laboratory for centrifuging, storage, packaging and transportation were followed. The lipids were measured using a Technicon RA1000 and Technicon reagents for cholesterol and triglyceride (TG) determination. High-density-lipoprotein-cholesterol (HDL) was measured on the supernatant after precipitation of non-HDL cholesterol with heparin and 0.046M $MnCl_2$. Since Mn^{++} interferes with the cholesterol measurement, it was removed by precipitation with $NaHCO_3$, according to the method of Bachorik (Bachorik et al 1984). Low-density-lipoprotein-cholesterol (LDL) was derived from total cholesterol (TC), HDL and TG using Friedewald's formula (Friedewald et al 1972). The technical details of these procedures have been described elsewhere (Connelly et al 1992). The accuracy and precision of the lipid determinations were maintained in accordance with the Lipid Standardization Program of the US National Heart, Lung and Blood Institute and the Centers for Disease Control.

Testing for fasting and 2-hour glucose levels after challenge with a 75 gm glucose load (Glucodex by Rougier Inc, Chambly, Quebec) were performed on capillary blood obtained by fingerprick using an Ames Glucometer II (Miles Canada, Etobicoke, Ontario). In a subsample of subjects, fasting venous samples were analyzed for plasma glucose and insulin levels by the Clinical Chemistry and Endocrinology/Metabolism Laboratories respectively, at the Health Sciences Centre in Winnipeg. Glucose was determined by the kinetic hexokinase/G6P-DH method, using a Boehringer Mannheim BM/Hitachi 717 analyzer and the Gluco-quant glucose reagent. The Insulin RIA 100 radioimmunoassay kit from Pharmacia Diagnostics AB (Uppsala,

Sweden) was used. Plasma and red blood cell fatty acids were analyzed at the haematology research laboratory at the Manitoba Institute of Cell Biology.

The Ames Minilab system (Miles Canada, Etobicoke, ON) was used for the determination of haemoglobin (Hgb), haematocrit (Hct) and red blood cell count (RBC) in capillary blood obtained from finger-prick. From these the mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) were calculated. The Minilab was standardized by the Clinical Haematology Laboratory of the Health Sciences Centre prior to the field study, using a Coulter counter as comparison.

Staff Selection and Training

For the individual survey, the field research team consisted of the Project Coordinator, a nurse, a laboratory technician, an audiologist, a dentist and a dental recorder. A number of locally recruited Inuit research assistants were hired in each community to serve as interpreters, interviewers, receptionists and clerks.

The recruitment of local Inuit research assistants was accomplished after an extensive process of seeking input from local health board trustees and community leaders, a formal interview procedure, and intensive training and orientation. The assistants were brought together in Rankin Inlet, the largest settlement of the region, at the beginning of each fieldwork period for a week-long training session run by the Project Coordinator and one or more of the Principal Investigators. The training encompassed survey procedures and techniques, ethical concerns, as well as issues in cross-cultural interviewing and team research.

Ongoing supervision and quality control was provided by the Project Coordinator throughout the duration of the field work, and close and regular communication was maintained with the Inuit research assistants in the communities as well as with the Principal Investigators at the University.

Field Data Collection

The field work began early in 1990 and covered three communities during the spring; after a summer break the remaining five communities were covered between September and December 1991. The field work was preceded by an initial promotional tour of the targeted communities. During this tour the Project Coordinator initiated the HS, which was then continued independently by the assistants after the Coordinator's departure. The Project Coordinator maintained close telephone contact every 3-4 days. Large, self-addressed envelopes and postage monies were left with each assistant, with instructions to send off promptly every 30 completed survey forms to the University.

The professional field team made separate visits to each community for the IS. Some 600kg of equipment were assembled for each visit, part of which was shipped ahead of time to each community. A clinic-like configuration was employed to allow for a continuous flow of participants between different 'stations' where specific procedures were performed. Participants awaiting their turn were offered coffee and snacks.

In each community the initial stages of inviting and scheduling prospective participants were usually begun prior to the field team's arrival. One assistant concentrated exclusively on the task of contacting prospective participants, explaining the goals of the study and the different

parts of the survey to them, and then arranging an appointment date/time. Other assistants acted in various capacities as receptionist, driver and interpreter.

At each location, regular staff meetings were held by the Project Coordinator to inform the field team of the ongoing status of that particular community's data collection. This included the percentage of the desired sample already seen; revisions to scheduled clinic times (e.g. the need to hold an evening clinic); recent developments which were helping or hindering the survey's success; and general discussions regarding any interpersonal or intercultural concerns which might arise between field team members or their dealings with respondents.

For an adult, the entire survey lasted 2 to 2½ hours. Adolescents were exempt from fasting blood work and the ECG, and completed a shorter version of the questionnaire, resulting in a total survey time of approximately 1 to 1½ hours. Children under age 12 were only requested to complete the basic elements of the physical examination section and usually completed the survey within one hour. Adults scheduled for an early morning appointment were advised to arrive still fasting. For others seen for the first time later on in the day, a separate morning fasting visit was required. To provide maximum convenience and improve cooperation, participants had the option of having their venipuncture at home.

Community Consultation and Communication

As a collaborative, community-based research project, the Keewatin Health Assessment Study depended heavily upon input and assistance from the Keewatin Regional Health Board and local community members. All survey forms were developed in close consultation with and approved by the Board. All study literature and forms were professionally translated into Inuktitut. The health board also assigned a staff member as liaison to the project. All possible

attempts were made to avoid conflict with scheduled visits by health services staff. The health board made available field clinic sites and in some communities, also accommodation for the travelling field teams.

The study was extensively promoted utilizing community meetings, community radio phone-in shows, posters and pamphlets. All public presentations were attended by representatives from the University, the health board and local authorities. A copy of the pamphlet printed in Inuktitut syllabics is reproduced in Fig.4.3.

Each research assistant carried a letter of introduction which verified their employment with the study and indicated her awareness of the need for confidentiality. The concept of randomness was carefully explained to the respondents so that they understood why only certain individuals were invited to participate in the IS. People not on the random list who nevertheless wished to be interviewed and examined ("volunteers") were accepted. However, these data were kept separate from the main survey datafiles and were not included in the analysis.

The preliminary results of the KHAS were presented to the regional health board in the form of a two-day workshop held in Winnipeg in October, 1991. All graphs and charts contained captions in both English and Inuktitut syllabics. A summary report, amply illustrated and written in non-technical language, was also completed and submitted to the board.

Ethical and Cross-cultural Issues

The survey protocol was approved by the "Faculty Committee on the Use of Human Subjects in Research" of the Faculty of Medicine, University of Manitoba. Informed consent was obtained prior to the household survey interview and repeated again prior to the individual survey. A research license (No. 10016) was issued by the Science Institute of the Northwest

Territories, upon receipt of signed authorizations by the Hamlet Councils of each of the communities participating in the study.

All team members signed an oath of confidentiality at the start of their employment. Because of the small community size, the need for confidentiality was reiterated to local staff members who usually had personal knowledge of, and social interaction with, the participants prior to the study.

Signed consent forms for the HS and the IS were detached from the body of the questionnaire so as to avoid an actual name being associated with a particular form. These forms were stored and shipped separately from the completed survey forms. Each participant in the individual survey was assigned an identification number. Only the individual participant's identification number appeared on the survey forms.

Care was taken to ensure that all Inuit research assistants responsible for the interviews felt comfortable with the contents of the questions. Occasionally, a local assistant expressed hesitancy to interview a particular individual because of family and kinship associations. When this occurred, the interview was assigned either to another assistant or assumed by the Project Coordinator. The professional field team took every opportunity during non-working hours to participate in community social life such as dances, weddings, special festivals and excursions onto the land.

Although the KHAS was not a diagnostic exercise, the possibility existed for the discovery of potentially dangerous risk factor values. If these values were noted at the field clinic site, they were immediately discussed in a non-alarming manner with the participant. Protocols for referrals were established for specific laboratory/clinical values relating to plasma glucose and blood pressure. The ECG machine was capable of producing a preliminary narrative evaluation of the participant's cardiac status (eg. "normal", "old infarct", etc). Participants with abnormalities or deviations were advised to seek medical advice at the local health centre. The audiologist and dentist also referred participants for any necessary follow-up by future audiology/dentistry consultant visits.

On a daily basis, the Project Coordinator reviewed all the confidential, self-administered questionnaires in order to check for any suicidal tendency. If this assessment determined that the participant required any assistance or intervention, the nurse-in-charge of the local health centre would be notified.

Following the completed survey and laboratory analysis, all individual survey participants received a letter containing their personalized blood pressure, lipid and glucose values. Participants with normal laboratory and clinical values received a brief letter stating this fact. For those participants with deviations from established clinical norms, they were advised to seek medical follow-up, further testing, counselling or treatment as appropriate. All nurses-in-charge of the local health centres received an information package outlining guidelines for "abnormal" levels, a copy of the letter sent to each participant, and an information kit to be used as a reference and education tool if the participant sought the advice of the health centre staff.

4.4 Data Management and Analysis

Data Entry and Editing

All data from the questionnaires, clinical examination and laboratory investigations were initially edited in the field by the Project Coordinator prior to shipping to Winnipeg for data entry. The Biostatistical Consulting Unit of the Faculty of Medicine was contracted to keypunch the data into a computer file. The software package Statpac-Gold (Walonick Associates, Minneapolis, Minnesota) and SAS (SAS Institute, Cary, North Carolina) were used in the data management and analyses.

Preliminary frequency counts and descriptive statistics of the complete datafile revealed various inconsistencies and inaccuracies (eg. decimal point errors, imperial measures instead of metric, physiological impossibilities, etc). The original questionnaires were retrieved and checked. In cases where such errors could not be reconciled, the particular values were deleted from the datafile.

Analysis Plan

The analysis plan was designed with the following objectives:

- (1) determine the prevalence of overall obesity and central fat patterning in the study population;
- (2) investigate factors (demographic, socioeconomic, cultural, familial, behavioural and metabolic) associated with the presence of obesity and central fat patterning;
- (3) compare findings from this study with three other populations.

The following analytic approaches were taken (Fig.4.4):

- (1) Obesity/central fat patterning as *dependent variables* (as body mass index, waist-hip ratio, sum of skinfolds, skinfold ratios):
 - a. using analysis of variance and covariance, significant differences in the mean of the dependent variable (eg. BMI) between two or more categories of an independent variable (eg. education) could be determined, adjusting for covariates (eg. age);
 - b. using multiple linear regression techniques, the relationship between various independent variables (eg. education, marital status) and changes in the dependent variable (eg. BMI) could be determined;
- (2) Obesity/central fat patterning as *independent variables*:
 - a. using analysis of variance and covariance, significant differences in the mean of various continuous dependent variables (eg. blood pressure, lipids, glucose and insulin) between different categories of obesity and central fat patterning could be determined, adjusting for covariates;
 - b. using multiple linear regression techniques, the relationship between obesity and fat patterning indices and changes in the dependent variables (eg. blood pressure) could be determined.

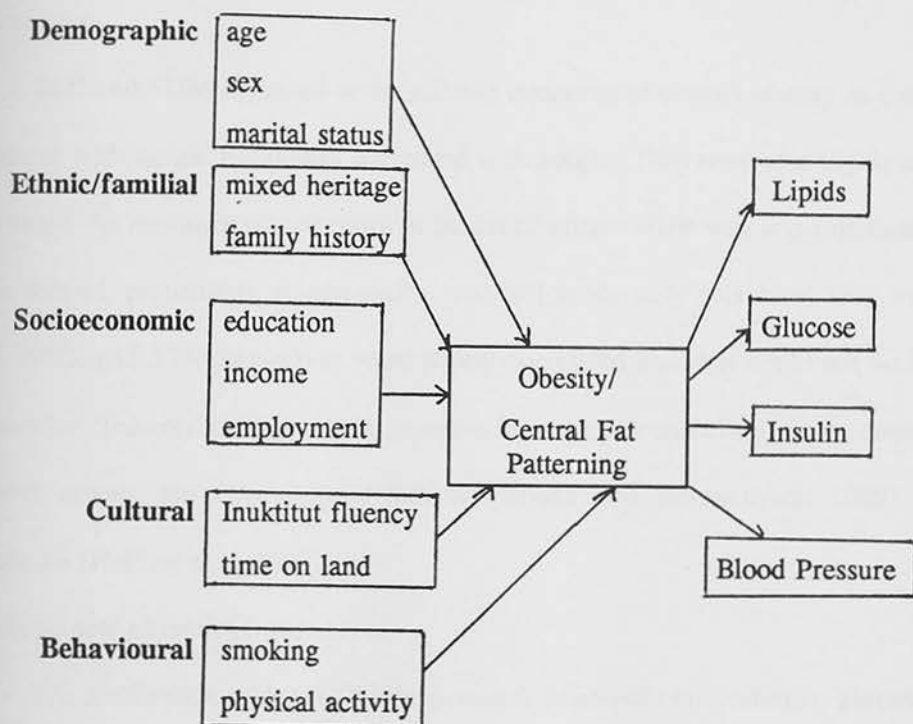


Fig.4.4 Analysis plan: dependent and independent variables

Distribution, Construction and Transformation of Variables

Obesity and fat patterning

Various indices were constructed from the raw data on measured heights, weights, triceps and subscapular skinfold thicknesses, and waist and hip circumferences. These included Quetelet's or **body mass index (BMI)**, defined as weight in kg divided by height in meters squared, **sum of skinfolds (SUM)**, **ratio of subscapular to triceps skinfolds (STR)**, and **ratio of waist to hip circumference (WHR)**. The distribution of these variables is shown in Table

4.3. The intercorrelations among the various anthropometric measures are shown in matrix form in Table 4.4.

BMI and SUM appeared to be suitable measures of overall obesity as they were highly correlated with weight but poorly correlated with height. They were also highly correlated with each other. As measures of centrality of fat distribution, WHR was less satisfactory than STR, as the former, particularly among males, was still moderately correlated with weight and with BMI. WHR and STR themselves were poorly correlated and thus could not be substituted for one another. The correlation matrices reported from the Keewatin Inuit were comparable to those reported among the Cree-Ojibwa Indians (Young and Sevenhuysen 1989) and Mexican Americans (Haffner et al 1987).

Metabolic and physiologic measures

The distribution of data on **blood pressure** (mean of two readings), **glucose** and **insulin**, and **plasma lipids** is shown in Table 4.5.

It is evident from Tables 4.3 and 4.5 that most of the anthropometric, physiologic and biochemical variables were not normally distributed. A normal (ie. Gaussian) distribution would have skewness near zero and kurtosis around 3 (Snedecor and Cochran 1980:78-79). Table 4.6 indicates that after log-transformation the data became closer to normality as judged by measures of skewness and kurtosis.

Table 4.3 Distribution of anthropometric variables

Variable	Range	Mean	SD	Skewness	Kurtosis	n
(1) Male						
Weight (kg)	42-118	70.21	13.10	1.13	4.58	160
Height (cm)	148-192	165.35	6.99	0.42	4.24	160
BMI	17-39	25.62	4.08	1.01	3.99	160
Waist (cm)	62-131	87.99	12.10	0.91	3.85	155
Hips (cm)	83-124	99.01	7.18	0.80	3.96	155
WHR	0.73-1.15	0.89	0.07	0.52	3.41	155
Tri SF (mm)	3-42	14.97	9.27	0.73	2.54	151
Sub SF (mm)	3.5-46.0	15.24	9.09	1.34	4.26	151
SUM (mm)	8.5-88.0	30.21	15.44	1.06	4.46	151
STR	0.21-4.71	1.28	0.74	1.03	5.28	151
(2) Female						
Weight (kg)	41-104	63.29	12.53	0.86	3.22	208
Height (cm)	140-179	153.66	5.43	0.28	4.32	211
BMI	19-43	26.79	4.99	0.77	2.96	208
Waist (cm)	61-116	86.61	12.52	0.46	2.24	200
Hips (cm)	86-129	101.72	9.51	0.62	2.73	201
WHR	0.69-1.03	0.85	0.07	0.38	2.91	200
Tri SF (mm)	6-54	23.27	9.20	0.62	3.43	202
Sub SF (mm)	5.5-59.0	23.62	12.42	0.81	2.89	201
SUM (mm)	12.5-113	46.99	20.52	0.74	3.09	200
STR	0.41-1.94	1.02	0.32	0.36	2.71	200

Notes: SD standard deviation n valid number of cases in sample
 BMI body mass index WHR waist/hip ratio
 SF skinfold thickness Tri triceps
 Sub subscapular SUM sum of two skinfolds
 STR sub/tri skinfold ratio

Table 4.4 Correlation matrices for anthropometric variables

	Wt	Ht	BMI	Waist	Hips	WHR	Tri	Sub	STR
Ht	0.56	-							
BMI	0.87	0.09	-						
Waist	0.89	0.29	0.91	-					
Hips	0.89	0.43	0.82	0.87	-				
WHR	0.68	0.10	0.76	0.88	0.54	-			
Tri	0.35	0.10	0.38	0.37	0.37	0.27	-		
Sub	0.74	0.19	0.78	0.76	0.68	0.64	0.41	-	
STR	0.24	0.10	0.22	0.19	0.19	0.16	-0.57	0.38	-
SUM	0.65	0.17	0.69	0.67	0.62	0.55	0.84	0.84	-0.12
Ht	0.36	-							
BMI	0.90	-0.07	-						
Waist	0.85	0.01	0.90	-					
Hips	0.92	0.21	0.89	0.85	-				
WHR	0.46	-0.21	0.58	0.80	0.37	-			
Tri	0.74	0.07	0.76	0.72	0.75	0.42	-		
Sub	0.83	0.09	0.84	0.79	0.79	0.49	0.78	-	
STR	0.39	0.04	0.39	0.36	0.31	0.27	-0.01	0.58	-
SUM	0.84	0.09	0.85	0.81	0.82	0.49	0.93	0.96	0.35

Notes:

BMI body mass index

WHR waist/hip ratio

Ht Height (cm)

Wt Weight (kg)

Tri triceps (mm)

Sub subscapular (mm)

SUM sum of two skinfolds (mm)

STR sub/tri skinfold ratio

Numbers in table refer to Pearson correlation coefficients

Table 4.5 Distribution of metabolic and physiologic variables

Variable	Range	Mean	SD	Skewness	Kurtosis	n
(1) Male						
SBP (mmHg)	85-202	124.41	17.08	1.31	6.45	160
DBP (mmHg)	53-101	78.28	10.17	0.20	2.46	160
FG (mmol/L)	2.8-10	5.83	1.02	0.37	5.03	115
2hG (mmol/L)	2.40-13.3	6.01	1.84	1.05	5.01	107
F-ins (pmol/L)	19-280	53.12	39.75	3.65	19.31	59
TC (mmol/L)	2.67-8.08	4.85	1.08	0.48	3.15	112
HDL (mmol/L)	0.74-2.54	1.37	0.37	1.04	3.92	112
LDL (mmol/L)	1.27-6.00	3.05	0.97	0.70	3.47	112
TG (mmol/L)	0.40-3.12	0.96	0.44	1.87	8.24	112
(2) Female						
SBP (mmHg)	82-169	115.10	15.66	0.90	3.91	211
DBP (mmHg)	49-103	72.88	9.41	0.48	3.19	211
FG (mmol/L)	1.5-10.9	6.44	1.17	0.04	6.47	141
2hG (mmol/L)	3.6-17.5	7.59	2.46	1.26	5.49	135
F-ins (pmol/L)	25-415	63.13	49.49	5.65	40.08	64
TC (mmol/L)	3.2-8.28	5.12	0.99	0.67	3.38	141
HDL (mmol/L)	0.77-3.22	1.51	0.42	0.90	4.39	141
LDL (mmol/L)	1.14-5.85	3.09	0.88	0.64	3.69	141
TG (mmol/L)	0.40-3.52	1.15	0.61	1.62	5.90	141

Notes:

SD	standard deviation	SBP	systolic blood pressure
DBP	diastolic blood pressure	FG	fasting glucose
2hG	2-hr glucose	F-Ins	fasting insulin
TC	total cholesterol	HDL	high-density-lipoprotein
TG	triglycerides	LDL	low-density-lipoprotein
n	valid number of cases in sample		

Table 4.6 Distribution of log-transformed continuous variables

Variable	Range	Mean	SD	Skewness	Kurtosis
(1) Male					
■ Obesity/fat patterning					
Weight	3.74-4.77	4.24	0.18	0.53	3.60
Height	5.00-5.26	5.11	0.04	0.23	3.99
Body mass index	2.83-3.66	3.23	0.15	0.57	3.25
Waist circumference	4.13-4.88	4.47	0.13	0.53	3.14
Hips circumference	4.42-4.82	4.59	0.07	0.56	3.51
Waist-hip ratio	-0.32-0.14	-0.12	0.08	0.28	3.01
Triceps skinfold	1.10-3.74	2.50	0.66	-0.07	1.85
Subscapular skinfold	1.25-3.83	2.57	0.55	0.30	2.43
Sum of skinfolds	2.14-4.48	3.28	0.52	-0.13	2.34
Subscapular/triceps ratio	-1.58-1.55	0.07	0.64	-0.44	2.36
■ Blood pressure					
Systolic	4.44-5.31	4.82	0.13	0.66	4.58
Diastolic	3.97-4.62	4.35	0.13	-0.01	2.62
■ Glucose metabolism					
Fasting glucose	1.03-2.30	1.75	0.18	-0.58	4.90
2-hr glucose	0.88-2.59	1.75	0.30	0.02	3.21
Fasting insulin	2.94-5.64	3.82	0.49	1.30	5.25
■ Lipids					
Total cholesterol	0.98-2.09	1.56	0.22	-0.15	2.94
HDL-cholesterol	-0.30-0.93	0.28	0.25	0.39	2.84
LDL-cholesterol	0.24-1.06	1.06	0.32	-0.19	2.93
Triglycerides	-0.92-1.14	-0.13	0.40	0.44	3.17
(2) Female					
■ Obesity/fat patterning					
Weight	3.71-4.64	4.13	0.19	0.46	2.59
Height	4.94-5.19	5.03	0.04	0.12	3.88

Body mass index	2.94-3.76	3.27	0.18	0.43	2.42
Waist circumference	4.11-4.75	4.45	0.14	0.23	2.13
Hips circumference	4.45-4.86	4.62	0.09	0.46	2.48
Waist-hip ratio	-0.38-0.03	-0.17	0.08	0.17	2.83
Triceps skinfold	1.79-3.99	3.06	0.43	-0.57	3.32
Subscapular skinfold	1.71-4.08	3.02	0.54	-0.07	2.18
Sum of skinfolds	2.53-4.73	3.75	0.45	-0.27	2.73
Subscapular-triceps ratio	-0.90-0.66	-0.04	0.34	-0.38	2.62
■ Blood pressure					
Systolic	4.41-5.13	4.74	0.13	0.50	3.29
Diastolic	3.89-4.64	4.28	0.13	0.10	3.09
■ Glucose metabolism					
Fasting glucose	0.41-2.39	1.84	0.21	-2.47	18.5
2-hr glucose	1.28-2.86	1.98	0.31	0.15	3.24
Fasting insulin	3.22-6.03	4.02	0.45	1.32	7.77
■ Lipids					
Total cholesterol	1.16-2.11	1.62	0.19	0.16	2.77
HDL-cholesterol	-0.26-1.17	0.37	0.27	0.03	2.97
LDL-cholesterol	0.13-1.77	1.09	0.29	-0.36	3.61
Triglycerides	-0.92-1.26	0.03	0.47	0.41	2.72

Sociodemographic Data

The frequency distribution of different categories of the **community, sex, age group** and **ethnic group** variables is shown in Table 4.7. Only those who identified themselves as full- or mixed-heritage Inuit were analyzed. The 47 non-Inuit (11% of the sample) were excluded as the focus of the study was on Arctic adaptation among the indigenous population. The Euro-Canadian migrants to the Arctic were usually short-term residents, predominantly young male, fully employed, with the highest level of income and education. For the purpose of comparison,

an external non-indigenous population, that of the Province of Manitoba, was chosen instead (Section 5.3).

Table 4.7 Frequency distribution of selected sociodemographic variables, all ethnic groups combined

Variable	Category	Number	%
Community	Rankin Inlet	97	22.4
	Arviat	84	19.4
	Chesterfield Inlet	22	5.1
	Repulse Bay	30	6.9
	Coral Harbour	42	9.7
	Whale Cove	15	3.5
	Baker Lake	95	21.9
	Sanikiluaq	49	11.3
Sex	Male	199	45.9
	Female	235	54.2
Age Group	18-34	236	54.4
	35-54	134	30.9
	55+	64	14.8
Ethnic Group	Inuit	276	63.6
	Mixed	111	25.6
	Other	47	10.8

For the variables marital status, education, employment status, income, and proficiency in Inuktitut, some categories of response were collapsed because of small cell size.

For marital status, *single*, *separated*, *divorced* and *widowed* were combined into a *not married* category. In the analysis it would be understood that *not married* among the young generally meant *single*. "Married" also includes cohabitation or common-law marriage. For education, individual years of completed formal schooling were combined into two categories: 0-6 years as *primary education only*, and 7 or more years as *some secondary education*. The large number of categories in response to the question "Are you employed during the past 12 months?" were combined into three categories: *not employed*, *seasonal or part-time* and *full-time*. In the analysis, consideration was given to the fact that the *not employed* included housewives among women and the retired among the elderly. In certain analyses, seasonal/part-time and full-time employment were further combined into a single *employed* category.

Table 4.8 **Frequency distribution of selected sociodemographic variables among Inuit**

Variable	Category	Number	%
Marital status	not married	168	43.4
	married	219	56.6
Education	primary only	215	55.6
	some secondary	172	44.4
Employment	not employed	223	57.6
	seasonal/part-time	61	15.8
	full-time	103	26.6
Personal income	< \$10,000	163	42.1
	\$10,000+	85	22.0
	missing	139	35.9
Inuktitut proficiency	not fluent	109	28.2
	fluent	278	71.8

The income variable was dichotomized into $< \$10,000$ and $\$10,000+$, in accordance with the "poverty" line. However, a large number of people did not respond, and a category of *missing values* was retained. It was evident that this category was demographically similar to the lower income group, and behaved similarly in terms of the distribution of some of the anthropometric and metabolic variables. For proficiency in spoken Inuktitut, which served as one measure of adherence to a traditional lifestyle, or degree of acculturation, the great majority of Inuit spoke it very well, necessitating the combining of *fairly well*, *very little* and *none at all* into one category of *not fluent*. Table 4.8 summarized the frequency distribution of these variables after recoding and deletion of the 47 non-Inuit.

Smoking

The survey identified *never smokers*, *past smokers* and *current smokers* of cigarettes. Among current smokers, *regular smokers* were defined as those who smoked at least one cigarette daily and *occasional smokers* those who smoked less often than daily. The number of cigarettes smoked per day by regular smokers was also recorded (Table 4.9). Only a handful of Inuit smoked cigars or pipes, and 30 individuals (8%) chewed tobacco. Among the latter, 18 were also current cigarette smokers. In the analysis, only cigarette smoking was considered. In some analyses, never smokers and past smokers were combined into a single category of *non-current smokers*.

Table 4.9 Frequency distribution of cigarette smoking status

Smoking Category	Number	Percent
Current cigarette smokers	256	71.7
Regular smokers	224	62.7
1-5 cigarettes/day	46	12.9
6-10 cigarettes/day	56	15.7
11-15 cigarettes/day	63	17.6
16-20 cigarettes/day	12	3.4
21-50 cigarettes/day	47	13.2
Occasional smokers	32	9.0
Past cigarette smokers	65	18.2
Regular smokers	53	14.8
Occasional smokers	12	3.4
Never cigarette smokers	36	10.1
Total	357	100
(No response)	30	

Physical Activity

Two questions dealt with the frequency and intensity of physical activity:

- How would you rate the level of physical exertion in your daily activities, would you say: (1) hard (2) medium (3) low?
- How often do you engage in sporting activities? By sports, I mean vigorous activities such as [list]. How many times per week do you do these kinds of activities: (1) daily (2) 4-6 times per week (3) 1-3 times per week (4) less than once per week.

The first question required a self-rating of overall daily activity level, including both at work and so-called "leisure-time" activity. The second covered the following strenuous sports: hockey, jogging, square dancing, fast walking, bicycling, swimming, curling, skating, softball, soccer, paddling, basketball, volleyball, aerobics, skiing, weight lifting, Inuit games such as high kick

Where these two variables were cross-tabulated, it was evident that the two did not coincide, as many individuals who rated their daily activities as involving "hard" physical exertion did not engage in active sports. For example, of 56 people in the "hard" category, 34 (61%) said they "never" took part in any active sports. A new composite variable was created by combining responses to these two questions, as indicated in Table 4.10.

Three categories were created in the new variable on **total physical activity**:

- (1) *Very active*: those who answered "hard" to the question on degree of physical exertion in their daily activities OR "daily" or "4-6 times/week" to the question on sports [cells bounded by thick borders in Table 4.10];
- (2) *Moderately active*: an intermediate category to cover individuals who did not fall into categories (1) or (3);
- (3) *Inactive*: those who answered "low" to the question on daily physical exertion AND "never" to the question on sports [cells bounded by dotted lines in Table 4.10].

The frequency distribution was as follows: (1) very active: 102/331 (30.8%); (2) moderately active: 185/331 (55.9%); and (3) inactive: 44/331 (13.3%). In situations which required a dichotomous variable, (2) and (3) were combined into a *non-active* category. It should be recognized that this variable provided only a qualitative or ordinal level measure, based on

self-reports. The degree of detail in the questionnaire was not sufficient for conversion to quantitative measures of activity expressed in units of energy expenditure. It should also be emphasized that physical *activity* should not be equated with physical *fitness*, which is a physiologically distinct entity.

Table 4.10 Cross-tabulation of two variables measuring physical activity

Self-rated daily activity	Frequency of vigorous sports						Total
	daily	4-6/wk	1-3/wk	<1/wk	never	no response	
hard	2	2	10	7	34	1	56
medium	15	19	39	37	76	6	192
low	4	4	14	7	38	2	69
no response	4	0	4	2	4	56	70
total	25	25	67	53	152	65	387

Time Spent on the Land

Two identical questions covered the autumn/winter and spring/summer seasons:

- In the past year, how often did you go on the land? Specifically, in [season] would you say: (1) more than once a week (2) once a week (3) 2-3 times a month (4) once a month (5) less than once a month (6) never.

Certain individuals spent a considerable amount of time "on the land" in both seasons.

To create an overall, year-round measure of time spent on the land, the two variables were

cross-tabulated (Table 4.11). This could be used as a measure of adherence to a "traditional" Inuit lifestyle to supplement the question on the ability to speak the Inuktitut language.

Table 4.11 Cross-tabulation of time spent on the land during spring/summer and autumn/winter

Spring/ summer	Autumn/winter on the land							Total
	> 1/wk	1/wk	2-3/mo	1/mo	< 1/mo	never	no response	
> 1/wk	34	2	2	1	0	2	1	42
1/wk	25	8	6	2	0	1	0	42
2-3/mo	17	15	24	1	2	2	0	61
1/mo	11	4	8	1	1	0	0	25
< 1/mo	10	4	11	7	10	3	0	45
never	46	16	14	13	13	31	1	134
no response	0	0	1	0	0	1	36	38
Total	143	49	66	25	26	40	38	387

Three categories were recognized in the new composite variable **total time spent on the land year round**:

- (1) *Frequently*: those individuals who spent > 1/wk or 1/wk EITHER during spring/summer OR autumn/winter [cells bounded by thick borders in Table 4.11];
- (2) *Sometimes*: an intermediate category which included individuals belonging to neither (1) nor (3);

- (3) *Rarely*: those individuals who spent < 1/month or "never" during BOTH spring/summer AND autumn/winter [cells bounded by dotted lines in Table 4.11]. The frequency distribution was as follows: (1) Frequently: 207/351 (59.0%); (2) sometimes: 85/351 (24.2%); and (3) rarely: 59/351 (16.8%). In some analyses, (2) and (3) were further combined to produce a category of *infrequently*.

Past Medical History

Survey participants were asked regarding any past history of heart disease, diabetes and hypertension and the type of treatment received. No attempt was made to check the medical records to corroborate the respondents' recall (Table 4.12).

Table 4.12 Frequency distribution of past medical history

Condition	Sample size	no. with positive history	%	Type of treatment	
Heart disease	346	18	5.2		
Diabetes	346	8	2.3	insulin	0
				oral agents	1
				diet only	7
Hypertension	356	64	18.0	not treated	39
				treated	25
				drug	15
				non-drug*	10

Note: * included salt-free diet, stress reduction, smoking cessation and other non-pharmacologic treatment

Family History

Participants were asked if anyone in the family was known to have suffered from heart disease, stroke, diabetes or hypertension. No attempt was made to validate the accuracy of the diagnosis. Responses were categorized into no one, one parent, both parents, one sibling, more than one siblings, children, and grandparents. In some analyses, all first-degree relatives were combined into a *positive* category and all others were considered *negative*. A composite variable of family history involving any one of the four conditions was also created. In the sample, 21% of participants reported at least one first-degree relative with one or more of the four conditions, and 79% with a negative history.

Table 4.13 Family history of selected chronic diseases

Family history	Heart disease	%	Stroke	%	Diabetes	%	Hypertension	%
negative	246	70.7	271	77.9	292	83.9	267	76.7
parent(s)	30	8.6	20	5.8	4	1.2	11	3.2
sibling(s)	16	4.6	9	2.6	4	1.2	6	1.7
child(ren)	7	2.0	2	0.6	1	0.3	1	0.3
other	7	2.0	6	1.7	1	0.3	4	1.2
don't know	42	12.1	40	11.5	46	13.2	59	17.0
Total	348	100	348	100	348	100	348	100

CHAPTER FIVE COMPARISON POPULATIONS

To serve as comparisons with the Inuit data from the Keewatin Health Assessment Study (KHAS), three populations were chosen: Siberian Eskimos and Chukchi, Subarctic Cree and Ojibwa Indians, and the general population (predominantly Caucasian) of the province of Manitoba. Their choice was primarily based on availability of recent survey data which were very comparable with those from KHAS, and the fact that the author was personally involved in the planning and/or execution of all these studies. This chapter provides brief summaries of the background information about the study populations and settings, and the design and methods used.

5.1 Siberian Eskimos and Chukchi

As part of a research collaboration agreement between the University of Manitoba and the Siberian Branch of the [formerly Soviet] Academy of Medical Sciences (SB-AMS) in Novosibirsk, a health survey was conducted by the Academy's Institute of Internal Medicine among the indigenous Chukchi and Eskimo peoples in the Chukotka region, concurrently with the KHAS in the Northwest Territories. The Chukotka Chronic Diseases Study (CCDS) shared a core group of survey variables with the KHAS and the field methods used in the two studies were comparable.

Since 1979 the Institute of Internal Medicine, under the direction of Academician Yuri P. Nikitin, has been engaged in a series of epidemiological studies of the health status of the Native and newcomer populations of Chukotka. A previous study of the prevalence of ischemic

heart disease and cardiovascular risk factors included only men aged 30-59 (Nikitin 1988, Astakhova et al 1991, Stepanova and Shubnikov 1991). The more recent CCDS included adults of both sexes aged 25-64.

Study Population and Setting

The Chukotka Autonomous Region (*okrug*), located at the northeasternmost extreme of the Asian continent, is a sparsely populated region with physical features typical of the Arctic tundra and tundra-taiga transitional zones. The population is approximately 160,000, 10% of whom are aboriginal, including about 12,200 Chukchi, 1400 Eskimos, 1100 Evens and smaller numbers of Yukagir, Koryak, Chuvan and others. The rest of the population is predominantly Russians and other nationalities from various parts of the former Soviet Union (referred to as "newcomers"). Considerable genetic admixture has occurred, both between Natives and newcomers and between different Native groups, especially between Chukchi and Eskimos.

The Chukchi and Eskimos are two of some 26 officially designated "northern minorities" which inhabit the vast expanse of the Far North and Far East. [These are also called "numerically small peoples", to be distinguished from other larger non-Slavic minorities such as the Komi, Yakuts and Buryats]. The Soviet censuses of 1926, 1959 and 1989 enumerated respectively 1,293, 1,118 and 1,719 Eskimos, and 12,332, 11,727 and 15,184 Chukchi in the USSR. The total population of the 26 northern minorities in 1989 was only 183,700, less than 0.1% of the total population of the former USSR (Vakhtin 1992).

A limited amount of data on the human biology of these northern Natives collected by Soviet scientists are available in English translation, for example on anthropometry and craniometry (Alexeev, in Laughlin and Harper 1979), population genetics (Rychkov and

Sheremetyeva, in Harrison 1977, and in Milan 1980), and demography (Krupnik 1985). The genetic affinity of Eskimos, Athapaskan Indians and Chukchi was discussed in Section 3.2, based on studies on blood group, serum protein, enzyme and immunoglobulin polymorphisms (Szathmary 1981, Schanfield et al 1990 and Nazarova 1989), mitochondrial DNA (Shields et al 1992) and dermatoglyphics (Crawford and Duggirala 1992). Siberian Eskimos speak a variety of Yupik, a language that is also spoken in central Alaska and St. Lawrence Island.

Few English-language publications on the social, cultural and political history of the northern Natives, including translations of Soviet sources, are available. [See, for example Dunn and Dunn (1963), Hughes (1965), Armstrong (1966) and Levin and Potapov (1956, trans 1964). For a fresh, post-Soviet perspective which differs from the officially sanctioned ethnographic and historical literature, see Vakhtin (1992)].

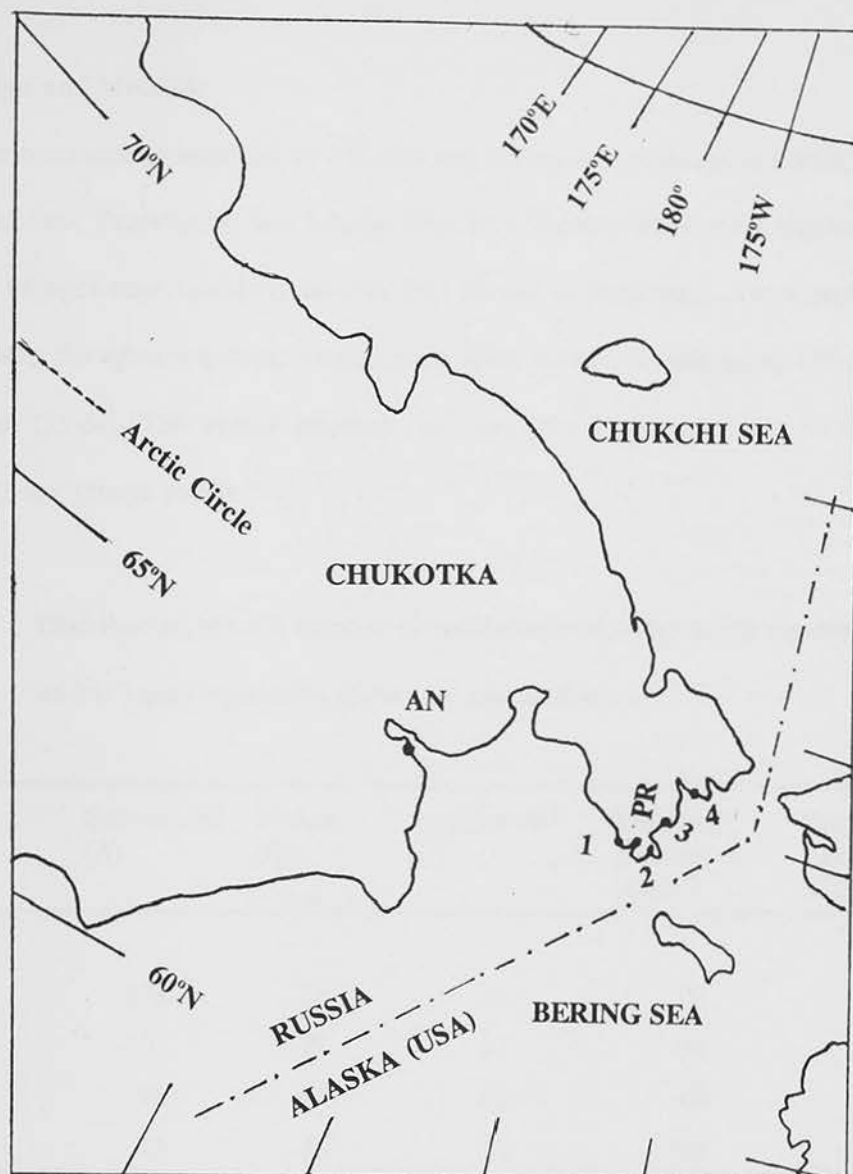
The nomadic hunting and fishing life of the northern Natives became irrevocably altered with the arrival of the first fur traders and cossacks across the Urals in the mid-16th century. New economies based on reindeer herding and fur trapping were developed. The extension of Soviet state power into the remote northern regions has often been portrayed officially as "progress towards socialism" but the Native populations suffered severely from the loss of their language and cultural identity and the effects of environmental despoliation under enforced industrialization, urbanization, collectivization and Russification. The tragic history of the relocation under coercion of the Eskimos of Naukan, first in 1958 and again in 1976, to larger centres dominated by Russians and Chukchi, was documented by the French anthropologist Chichlo (1981).

Illustrative of the prevailing Soviet view of the culture of indigenous peoples is the following quotation from the book *Narody Sibiri* [The Peoples of Siberia] by Levin and Potapov:

... The Soviet system ensures the betterment of the material conditions of life for the Eskimos and the liquidation of their cultural backwardness (translated and cited in Hughes 1963)

Writing in the Smithsonian volume *Crossroads of Continents* (Fitzhugh and Crowell 1988), the Soviet ethnographer Lebedev summarized the post-1917 development of northern Natives as follows:

In 70 years of Soviet administration, [Siberian Native peoples] advanced from patriarchal social and economic organization to contemporary socialism... [During the] economic reorganization and development of central villages [and] ... the creation of the kolkhoz [collective farms] and sovkhoz [state farms], the various ethnic groups began working together in brigades, and a process of unification commenced. Installation of the Soviet political system also brought these groups together to participate in important regional social and economic decisions. Growth of larger settlements, modern social structures, and the development of education allowed previously separated groups to interrelate freely... (Lebedev 1988)



AN	Anadyr	PR	Provideniya
1	Sireniki	2	Novoye Chaplino
3	Yanrakynnot	4	Lorino

Fig.5.1 Map of Chukotka showing communities participating in the Chukotka Chronic Diseases Study

Study Design and Methods

Four communities inhabited by Chukchi and Eskimos participated in the CCDS: Lorino, Novoye Chaplino, Yanrakynnot and Sireniki (Fig.5.1). Overall, 55% of the eligible population in the four villages were randomly selected and invited to participate. The sampling fraction differed among the age-sex groups, ranging from 25% in the youngest group (25-34) to 100% in the oldest (55-64). The overall response rate was 65.5%, ranging from 53% to 79% in different age-sex groups (Table 5.1).

Table 5.1 **Distribution of total number of individuals (A) eligible (B) randomly selected and (C) participated in survey by age and sex**

Age-sex group	Enumerated (A)	Invited (B)	Participated (C)	Sampling Fraction % (B)/(A)	Response Rate % (C)/(B)
(1) Male					
25-34	238	68	54	29	79
35-44	101	79	42	78	53
45-54	84	84	46	100	55
55-64	43	43	28	100	65
Total	466	274	170	59	62
(2) Female					
25-34	252	59	46	23	78
35-44	123	86	52	70	60
45-54	103	71	52	69	73
55-64	63	63	42	100	67
Total	541	279	192	52	69

The survey was conducted with the full cooperation of the local health services. Subjects selected for examination were notified through the local authorities. A specially trained team of physicians and other health workers from the Institute conducted the interviews and examinations in the local health facilities. The field work was completed between April and May of 1991.

The different components of the CCDS are summarized in Table 5.2. Those variables which were identical to both the CCDS and KHAS were indicated by italics. To ensure maximal comparability, the Canadian and Russian study teams visited each other during both the planning and execution stages of the research. At the conclusion of the field work and data entry, the datafiles from the two studies were exchanged to enable independent analyses.

The technical procedures were conducted in accordance with internationally standardized protocol of the multicentre study called *Monitoring of Trends and Determinants in Cardiovascular Diseases* (WHO-MONICA 1989). The Institute of Internal Medicine has been one of the World Health Organization collaborating centres in both MONICA and CINDI (Countrywide Integrated Non-Communicable Diseases Intervention) programs and has accumulated extensive experience during previous population surveys in Novosibirsk and in Chukotka. All quality control procedures stipulated by MONICA were adhered to.

All subjects underwent venipuncture after overnight (at least 12-16 hours) fasting. Sera were frozen and shipped to Novosibirsk for analysis. All laboratory procedures for lipids were standardized to the Lipid Reference Centre in Moscow and the MONICA Quality Control Centre in Prague. A two-hour glucose tolerance test was performed on capillary samples after a standard 75-gm glucose load, using an Ames Glucometer.

Table 5.2 Components of the Chukotka Chronic Diseases Study

Personal Interview

- Demographic Data: *Age, Sex, Ethnic Group, Education*
- Family History: *Cardiovascular Diseases, Diabetes*
- *Tobacco Use, Alcohol Consumption, Flush Syndrome*
- Rose Questionnaire for Ischemic Heart Disease
- 24-Hour Dietary Recall
- Screening Questionnaires for Diabetes, Anemia, and Stroke

Physical Examination

- Anthropometry: *Height, Weight, Hip and Waist Circumference, Subscapular and Triceps Skinfolds*
- *Blood Pressure*
- *12-Lead Resting Electrocardiography*
- Clinical and Neurological Examination
- Ultrasonography of Heart and Abdominal Organs
- Doppler Examination of Carotid Arteries

Laboratory Analyses

- Lipids and Lipoproteins: *Total Cholesterol, HDL-, LDL-Cholesterol, Triglycerides, Apolipoproteins A, B, Lp1*
 - *Glucose and Insulin: Fasting and 2-hour*
 - Hepatic Enzymes: ALAT, ASAT, GGT
 - Haematologic Indices: *Haemoglobin, Red Blood Cells, Haematocrit, Leucocytes, Serum Iron and Ferritin*
-

Note: Variables identical to those used in the Keewatin Health Assessment Study indicated in *italics*

A standard 12-lead ECG was recorded and coded independently by two coders using the Minnesota Code. These coders were trained and periodically re-certified by the MONICA Quality Control Centre for ECG Coding in Budapest.

Dietary intake was obtained using the 24-hour recall method, similar to the one used in a previous study described in detail elsewhere (Nikitin et al 1991). The Rose Questionnaire for the detection of symptoms of ischemic heart disease was used (Rose et al 1982). Other screening questionnaires for diabetes, stroke, alcohol intake and anemia were specially designed for this study.

5.2 Subarctic Cree and Ojibwa Indians

The Northern Indians Chronic Disease Study (NICDS) was designed to determine the prevalence of existing chronic diseases and risk factors in a geographically defined northern Indian population undergoing rapid social change. The study was conducted in 4 Cree and Ojibwa communities in northwestern Ontario and 2 in northeastern Manitoba (Fig.5.2). The total population of the 6 study communities in the mid-1980s was estimated to be about 5,000, 40% of which fell within the 20-64 age-group.

Study Population and Setting

The region lies within the subarctic boreal forest zone of central Canada, a vast expanse of forest, lakes and swamps, criss-crossed by numerous rivers and streams. The Indians in the central subarctic belong to two genetically and culturally related groups - the Cree and Ojibwa, members of the Algonkian language family. A considerable body of data on genetic markers is available [reviewed in Szathmary and Auger (1983)].

Prior to European contact in the early 17th century, with the arrival of traders from the Hudson's Bay Company, the aboriginal culture was centred around the hunting of large game such as moose and caribou. From the late 17th century onwards, the history and economy of the Indians were dominated by the fur trade. From coastal forts along Hudson Bay, inroads into the interior were made and numerous trading posts were established. The Canadian government made its presence in the region towards the end of the 19th century, when several treaties were signed with the Cree and Ojibwa nations. In return for the surrender of their lands, Indian Reserves were established and certain rights and benefits guaranteed by the government. Large-scale social changes occurred after World War II, when permanent communities were established, with schools, churches, nursing stations and other government services. With the decline of the fur trade and land-based subsistence activities, the people became increasingly dependent on social assistance and government-sponsored economic development projects. Vastly improved air transportation and telecommunications reduced the isolation and exposed the people to the lifestyle of the larger, Euro-Canadian society. The general pattern of health and disease and the health care delivery system in this population has been studied extensively in the past, summarized in the book *Health Care and Cultural Change: The Indian Experience in the Central Subarctic* (Young 1988).

From the perspective of a population-based study, the advantages of the region include its well defined geographical boundaries, isolation and remote location, relatively homogeneous cultural and linguistic background, and the existence of a single source of community-based primary care with well maintained and comprehensive medical records.

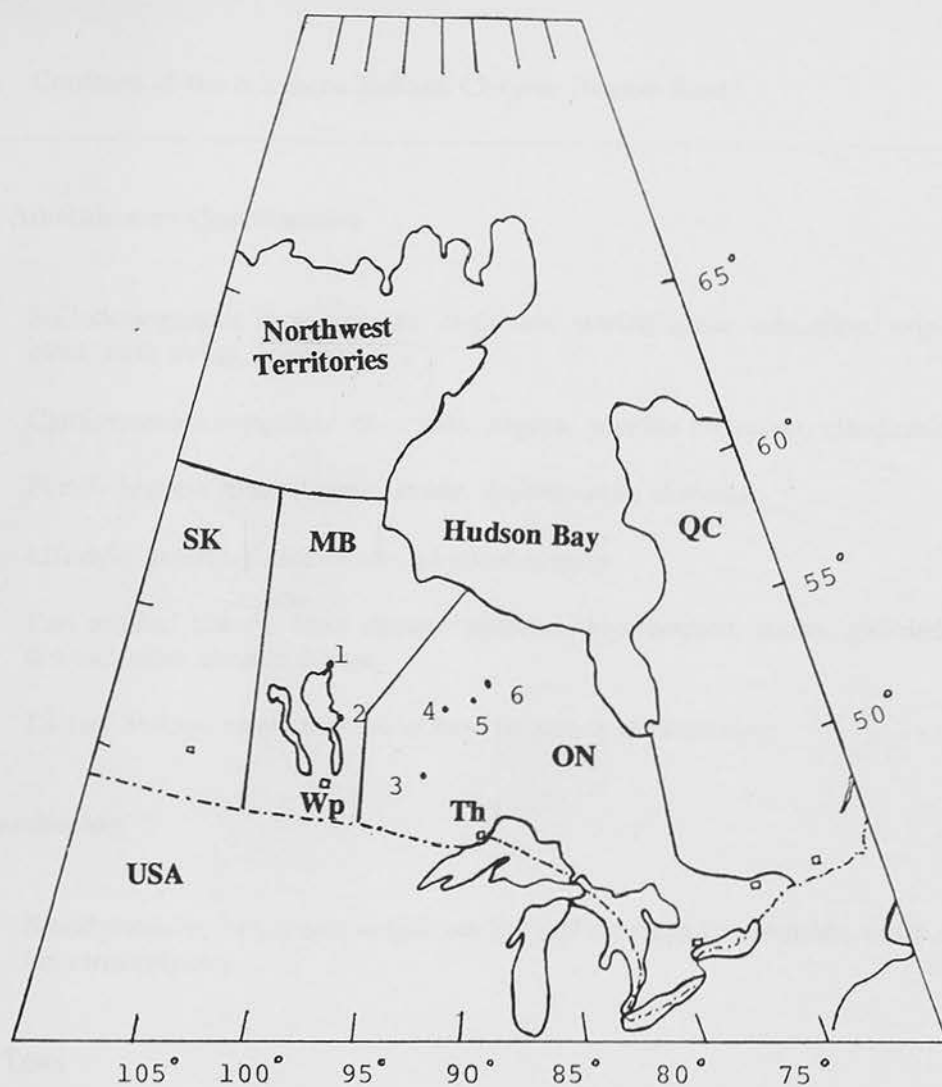


Fig.5.2 Map of northwestern Ontario and northeastern Manitoba showing communities participating in the Northern Indians Chronic Disease Study

MB Manitoba
 ON Ontario
 SK Saskatchewan
 QC Québec
 Wp Winnipeg
 Th Thunder Bay

Study Communities:

1 Norway House
 2 Berens River
 3 Lac Seul
 4 Weagamow Lake
 5 Kingfisher Lake
 6 Kasabonika Lake

Table 5.3 Contents of the Northern Indians Chronic Disease Study

Interviewer-Administered Questionnaire

- Sociodemographic data: *age, sex, residence, marital status, education, employment, bush living, housing*
- Cardiovascular symptoms: chest pain, angina, possible infarction, claudication
- Family history: *heart disease, stroke, hypertension, diabetes*
- Lifestyle: *smoking, alcohol use, physical activity*
- Past medical history: *heart disease, diabetes, hypertension, stroke, gallbladder disease, other chronic diseases*
- Dietary history: semi-quantitative food frequency questionnaire

Physical Examination

- *Blood pressure, height and weight, triceps and subscapular skinfolds, waist and hip circumference*

Laboratory Tests

- *Total cholesterol, HDL, LDL, triglycerides, fasting glucose*
 - *Electrocardiography*
-

Note: Variables identical to those used in the Keewatin Health Assessment Study indicated in *italics*

Study Design and Methods

In the four Ontario communities, all eligible adults were invited to participate, with response rates which ranged from 60% to 73%. In the two Manitoba communities, which were larger in size, a 15% random sample of eligible adults were selected, with response rate about 50%. The final sample consisted of 704 adults. Table 5.3 lists the types of data collected, with those variables which were comparable to those in the KHAS printed in italics.

Details on the methodology of this study and results relating to obesity, diabetes, gallbladder disease and hypertension have been published (Young and Sevenhuysen 1989, Young and Roche 1990, Young, Sevenhuysen, Ling and Moffatt 1990, Young 1991).

5.3 General Population of Manitoba

The Manitoba Heart Health Survey (MHHS), a survey of cardiovascular risk factors and knowledge and practices related to health promotion, was conducted in a representative sample of adult Manitobans aged 18-74 during 1989-90. It was one in a series of surveys conducted in all provinces of Canada (MacLean et al 1992).

Study Population and Setting

The province of Manitoba is located in the geographical centre of Canada, with a population of 1,091,000 inhabitants according to the 1991 Census, 60% (652,000) of whom reside in the capital city of Winnipeg. Ecologically the province can be divided into three zones - the boreal forest to the north, which blends into the tundra near the border with the Northwest Territories, the prairie grasslands to the south, and a transitional zone in between. Agriculture

has been the mainstay of the economy, the Prairie provinces being the breadbasket for much of the world, while mining and forestry are important industries in the north.

The population of the province, while predominantly Caucasian, is ethnically mixed. The original inhabitants - Algonkian, Siouan, and Athapaskan speaking Indians, and later the Metis, progeny of mixed Indian and white (mostly French-Canadian) marriages - were soon outnumbered by settlers of European origins. Several waves of European immigration beginning since the late 19th century brought in English-Canadians from Ontario, Scots (the "Selkirk settlers" who were enticed by Lord Selkirk), Ukrainians, Germans (particularly those belonging to the Mennonite faith), Poles and Jews, among others. The province also has one of the largest French-Canadian population outside of Québec. Since the 1960s, the sources of immigration have shifted from Europe to East, South and Southeast Asia.

Study Design and Methods

The sampling frame for the MHHS was the registry of beneficiaries of the Manitoba Health Services Commission, the provincial agency responsible for administering the universal health insurance plan, as of June 1, 1989. As there is no premium for enrolling in the provincial health insurance plan, coverage is virtually complete. The total eligible adult population aged 18-74 identified was 754,664 persons, each residing in one of 269 non-overlapping administrative units (municipalities, towns, villages, etc), uniquely identified by a "municipality code" by the Health Commission.

The target population was subdivided into three geographic strata on the basis of community type:

- (1) The City of Winnipeg and its adjacent rural municipalities: adult population 464,195 (62% of total province);
- (2) Six regional centers, each with its respective surrounding rural municipality, and larger towns with adult population >2,000: total population 113,998 (15%), and
- (3) Small rural communities, including Indian Reserves, local government districts, all other rural municipalities not included under (1) and (2) above, small towns and villages: population 176,471 (23%).

Equal size samples were drawn from each of these strata. Each geographical stratum was further subdivided into 6 age-sex groups of equal sizes: 3 age groups (18-34, 35-64, and 65-74) in both sexes. Forty-six communities (Fig.5.3) were selected from among the list of 269 municipality codes using a random number generator: 10 in Stratum (1), 16 in Stratum (2) and 20 in Stratum (3). The Health Commission was then requested to generate from its population registry the names and addresses of the required number of individuals from within the selected communities with the appropriate age and sex.

A total of 4,080 names and addresses were initially generated. The large number of non-located subjects, primarily the result of outdated or incomplete addresses in certain municipalities, and the low response of certain age-sex groups (particularly young males) necessitated the drawing of additional names. This second sample consisted of 510 individuals. All such individuals were contacted and those who agreed to participate were included with the first sample. Within any stratum, individuals from the replacement sample were considered to have the same probability of selection as individuals in the first sample.

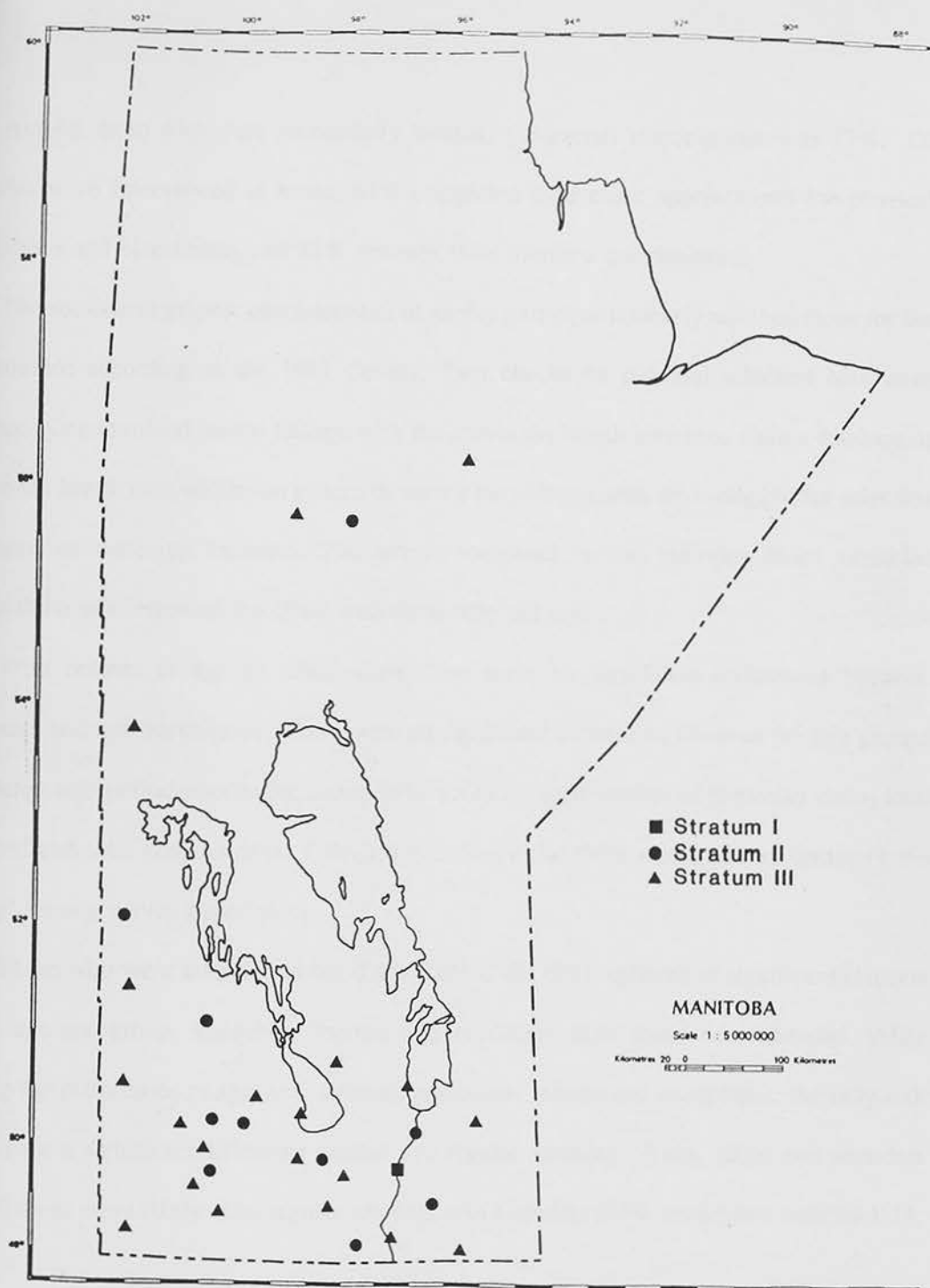


Fig.5.3 Map of Manitoba showing communities included in the Manitoba Heart Health Survey

Among those who were successfully located, the overall response rate was 77%. Of those who were interviewed at home, 84% completed their clinic appointments for physical examinations and blood tests, and 88% returned their nutrition questionnaire.

The sociodemographic characteristics of survey participants closely matched those for the total province according to the 1991 Census. Two checks on potential selection bias were conducted. One involved record linkage with the provincial health insurance claims database to compare the health care utilization pattern of survey participants with those eligible for selection but refused or were not locatable. The second compared various interview-based variables between those who attended the clinic with those who did not.

With respect to age-sex distribution there were no significant differences between participants and non-participants. There were no significant differences between the two groups over a three year period prior to the survey in terms of the total number of physician visits, total fees billed and total hospital days. Thus it was unlikely that there was a bias in favour of the "healthy" among survey participants.

Those who were interviewed but did not attend the clinic differed in significant respects such as age-sex group, residence, income and education from those who attended. After adjusting for differences in age, sex, stratum, education, income and occupation, the only risk factor where a significant difference existed was regular smoking. Thus, clinic non-attenders were 1.5 times more likely to be regular smokers than attenders (95% confidence interval 1.14, 2.03)

Since the sample design dictated the selection of 3 age groups within each of the 3 strata of equal size, contrary to the actual population distribution in the province, individuals in

specific age-sex-strata were assigned different weights, based on their probability of inclusion in the sample. This would allow the projection of sample-based prevalence estimates to the total province as a whole, with appropriate adjustment for the sample's actual demographic composition. The sampling weight is the inverse of the probability of selection, which is represented by the proportion of actual number of respondents in a substratum among the total age-sex-specific population in that substratum.

The Manitoba Heart Health Survey comprised three separate elements: a risk factor questionnaire, a nutrition questionnaire, and clinical and laboratory studies.

The Risk Factor Questionnaire was administered in the respondents' homes by trained nurse interviewers. This interview elicited information pertaining to cardiovascular disease awareness, knowledge and behaviours significant to the prevention of cardiovascular disease, and demographic data. During the home visit, the nurse also completed two blood pressure measurements, explained the Nutrition Questionnaire and arranged a clinic appointment for the respondent.

The Nutrition Questionnaire was designed to assess food frequencies. The nurse interviewer explained the questionnaire and demonstrated usual portion sizes using plastic food samples. The questionnaire was then left with the respondent to complete and return to the study at the time of their clinic appointment.

The clinic portion of the survey included anthropometric and further blood pressure measurements. Fasting blood specimens were drawn for the determination of total plasma cholesterol, high- and low-density- lipoproteins, triglyceride, glycosylated haemoglobin and fasting plasma glucose levels. The specimens for lipid analyses were analyzed at the Lipid

Research Laboratory in St. Michael's Hospital, Toronto, the only national reference lipid laboratory and the same one used by the Keewatin Study. The glucose analyses were performed at the Cadham Provincial Laboratory in Winnipeg.

PART THREE RESULTS AND DISCUSSION

CHAPTER SIX OBESITY AND REGIONAL FAT PATTERNING

6.1 Prevalence and Distribution

In Chapter Two the availability of several types of measures of obesity and fat patterning based on anthropometric surveys of large populations is discussed. Many of these measures are correlated with one another, as evident from the correlation matrix in Chapter Four (Table 4.4). While some consensus on the categorization of BMI and WHR exists (eg. Bray 1989), based on the relative risks of adverse health effects established in long-term prospective studies, no such standards exist for the other indices. Table 6.1 shows the variation in the mean of different anthropometric measures among six age-sex groups.

Table 6.1 Means (and standard errors) of anthropometric measures by age and sex among Keewatin Inuit

Anthropo- metric measure	Male			Female		
	< 35	35-49	≥ 50	< 35	35-49	≥ 50
Height (cm)	166.2 (0.73)	166.2 (1.18)	162.6 (1.10)	154.5 (0.50)	154.8 (0.72)	150.4 (0.72)
Weight (kg)	68.4 (1.16)	76.4 (3.14)	70.9 (2.35)	60.1 (1.10)	68.2 (1.77)	64.6 (1.84)
BMI	24.7 (0.37)	27.6 (0.90)	26.7 (0.71)	25.1 (0.41)	28.5 (0.72)	28.6 (0.73)
Waist (cm)	83.8 (1.06)	94.3 (2.69)	94.7 (1.87)	81.0 (0.98)	90.6 (1.72)	94.0 (1.81)
Hips (cm)	97.4 (0.67)	102.0 (1.52)	101.1 (1.27)	98.6 (0.82)	105.5 (1.30)	104.6 (1.50)
WHR	0.86 (0.006)	0.92 (0.018)	0.93 (0.009)	0.82 (0.005)	0.86 (0.009)	0.90 (0.009)
Triceps	15.2 (1.02)	16.3 (1.97)	13.5 (1.29)	21.1 (0.73)	25.7 (1.43)	25.4 (1.51)
Subscapular	13.9 (0.88)	20.2 (2.07)	15.5 (1.56)	21.0 (1.06)	27.1 (1.76)	25.4 (2.11)
SUM (mm)	29.1 (1.56)	36.4 (3.47)	28.9 (2.56)	42.1 (1.68)	52.8 (3.05)	50.8 (3.45)
STR	1.22 (0.08)	1.51 (0.15)	1.30 (0.12)	1.00 (0.03)	1.06 (0.04)	0.99 (0.05)

It is evident from Table 6.1 that height among Inuit adults remains relatively stable with increasing age. For weight and the various weight-, girth- and skinfold-based indicators of obesity and central fat patterning, there is an increase between the young adult group (under 35) and the middle age group (35-49). Beyond age 50, there is a decrease or levelling off. This phenomenon is not unique to the Inuit, and can be observed in various national surveys, such as the Canada Health Survey (Canada 1981) and the U.S. National Health and Nutrition Examination Survey (Frisancho 1990).

The data on mean BMI in Table 6.1 can be compared to historical data series from various Arctic regions provided in Chapter Three. Pre-1960 BMI values of men (Table 3.3, p.47, derived from Newman 1960) ranged from 23 to 28. These data refer to an imprecisely defined "adult" age group. Of interest are the 1939 data from Chesterfield Inlet ($n=30$), which is one of the communities in the Keewatin Health Assessment Study. The BMI reported was 25, comparable to the current Keewatin Inuit means of 23.7 to 24.8. Table 3.4 (p.48) summarizes more recent data. For men, BMI range from 23 to 28, and for women, from 23 to 30. It would seem that the current Keewatin BMIs are not substantially different from those reported in the older literature. Data from one community, Igloolik, spanning the period 1970-1990 (Fig.3.7, p.57, derived from Rode and Shephard 1992) indicate that the mean BMI has increased only slightly in most age-sex groups, except among women over the age of 40. Thus on the basis of BMI alone, one would be hard pressed to conclude that Canadian Inuit have become more "obese" in recent years. As pointed out by Garn et al (1986), the BMI does not completely reflect obesity since it also correlates with relative sitting height and lean body mass. The fact that mean BMI has not changed substantially among the Inuit does not preclude increasing

obesity masked by a change in body proportions (ie. less short-legged) or the replacement of lean tissues with adipose tissue.

Skinfold thicknesses are considered more "direct" measure of obesity than weight-based indices [see discussion in Chapter Two, p.10]. Here, Shephard's data from Igloolik, based on the sum of three skinfolds - triceps, subscapular and suprailiac - show a marked increase between 1970 and 1990 in all age-sex groups (Fig.3.7, p.57). Similarly the current Keewatin data for triceps skinfolds (14-16 mm in men and 21-26 mm in women) are substantially higher than the 6-7 mm (men) and 11-17 mm (women) reported by Schaefer (1977), based on surveys in various communities of the NWT during 1964-1970 (Table 3.4, p.48). A similar increase is observed for subscapular skinfolds, which is lower than 9 mm in men and range from 10 to 17 mm in women in Schaefer's series, compared to 14-16 mm (men) and 21-27 mm (women) in the Keewatin today. Thus there is some evidence that obesity, as measured by skinfolds, has increased among Canadian Inuit.

The Keewatin skinfold data can be compared with the U.S. White population, nationally representative surveys on whom have been conducted and reported. Data from the National Health and Nutrition Examination Surveys of 1971-74 and 1976-80 (NHANES I and II) were pooled and published in tables of anthropometric standards by Frisancho (1990). The Inuit mean triceps and subscapular skinfolds generally fall between the 50th and 75th percentile.

The Keewatin data can be compared internationally with other populations. Data on mean BMI are available from a variety of populations from the INTERSALT Study (Intersalt Cooperative Research Group 1988), the WHO-MONICA Study (World Health Organization 1989) and the *World Data Book of Obesity* (Baba and Zimmet 1990). The Keewatin Inuit BMIs

are comparable to ranges reported from many developed countries, but are generally higher than those from many developing countries in Asia and Africa and unacculturated Amazonian Indians. The Inuit BMIs are far below those of such highly obese populations as the Nauruans, among whom mean values ranging from 30 to 38 are reported in various adult age-sex groups (Dowse et al 1992).

More detailed and meaningful comparisons can be made with the three other populations which had undergone surveys similar to the Keewatin Study and using comparable methods. Background information on the Native population (Chukchi and Eskimos) in Chukotka, Russia, the general, all-race (but predominantly Caucasian) population of the Province of Manitoba, and the Cree-Ojibwa Indians of northern Ontario and Manitoba has been provided in Chapter Five.

Figs.6.1-6.4 compare the frequency distributions in BMI, WHR, waist and hip girths, and subscapular and triceps skinfolds between the four populations. For BMI, all four populations tend to skew to the right. Among men, the Indian curve is shifted to the right of the other three. Among women, the distributions are not sufficiently distinct. The distributions of WHR show close match between the Keewatin and Chukotka samples, distinct from the right-shifted Indian curve. Among men, the all-race Manitoba population occupies an intermediate position between the Eskimo/Chukchi and the Cree-Ojibwa, whereas among women, the Manitoba distribution is shifted to the left of the three aboriginal populations.

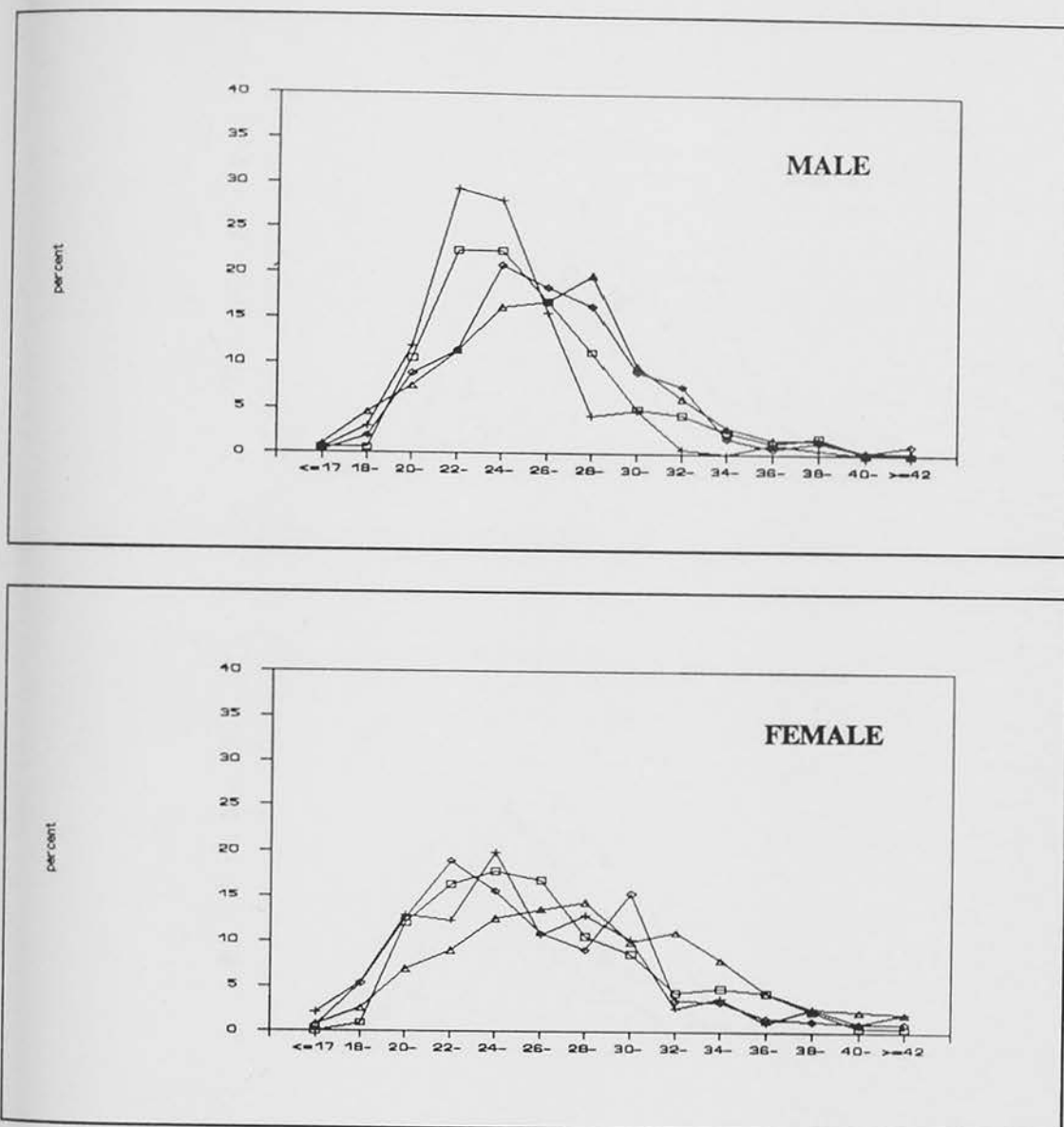
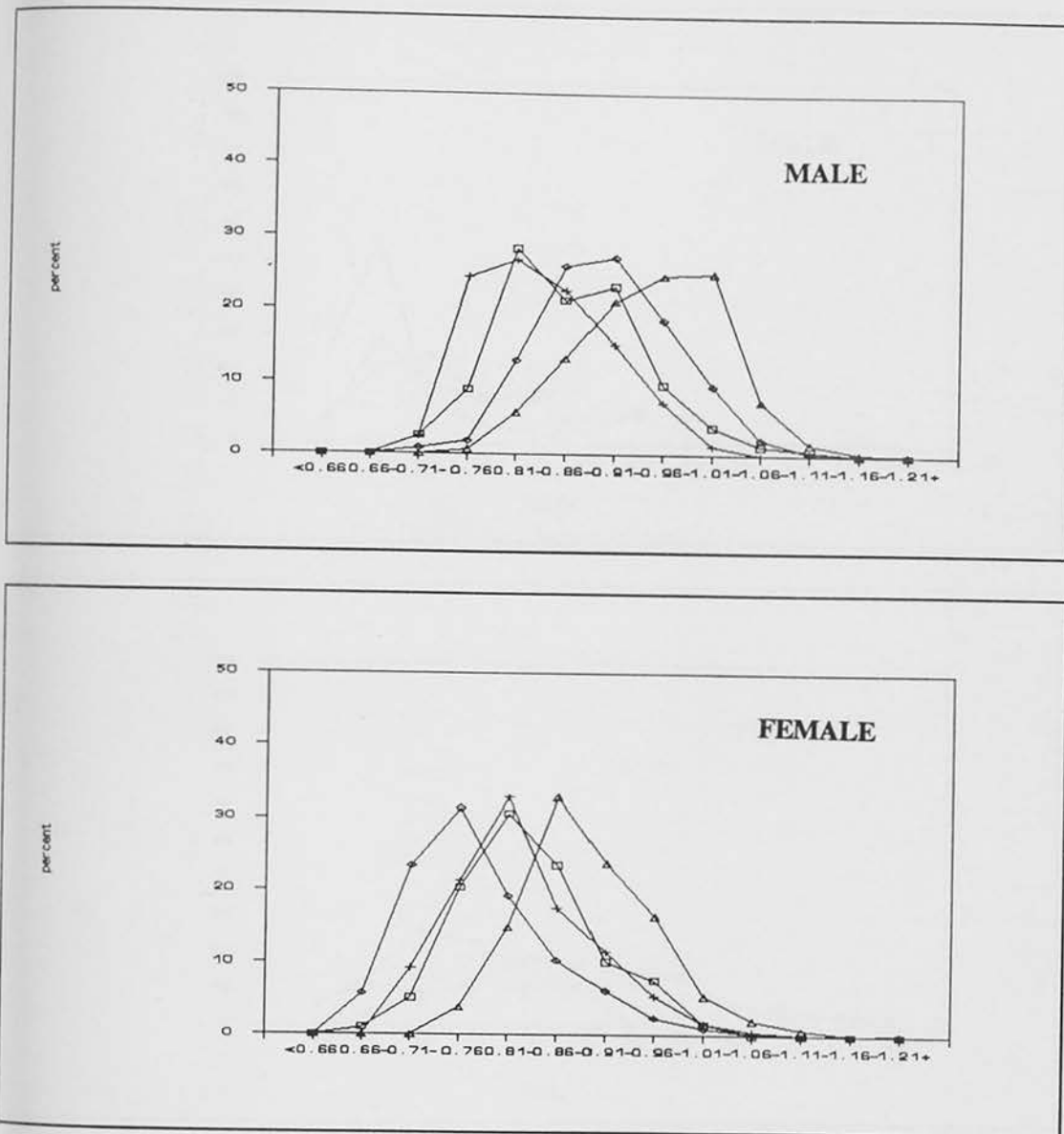


Fig.6.1 Frequency distribution of body mass index (BMI) by sex

□ Keewatin + Chukotka ◇ Manitoba △ Indians



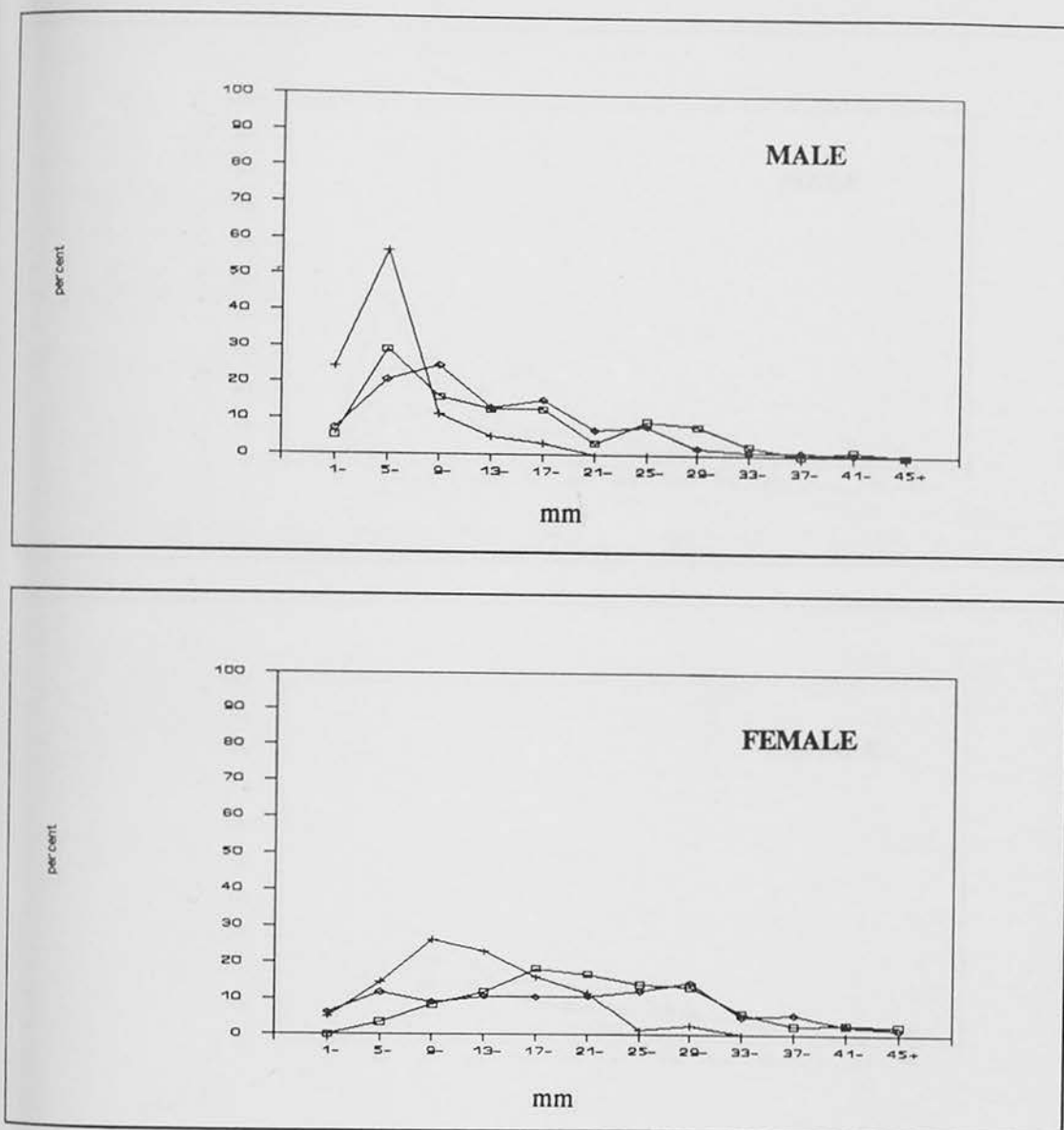


Fig.6.3 Frequency distribution of triceps skinfolds by sex

□ Keewatin + Chukotka ◇ Manitoba △ Indians

Note: [Skinfold data not available for Province of Manitoba]

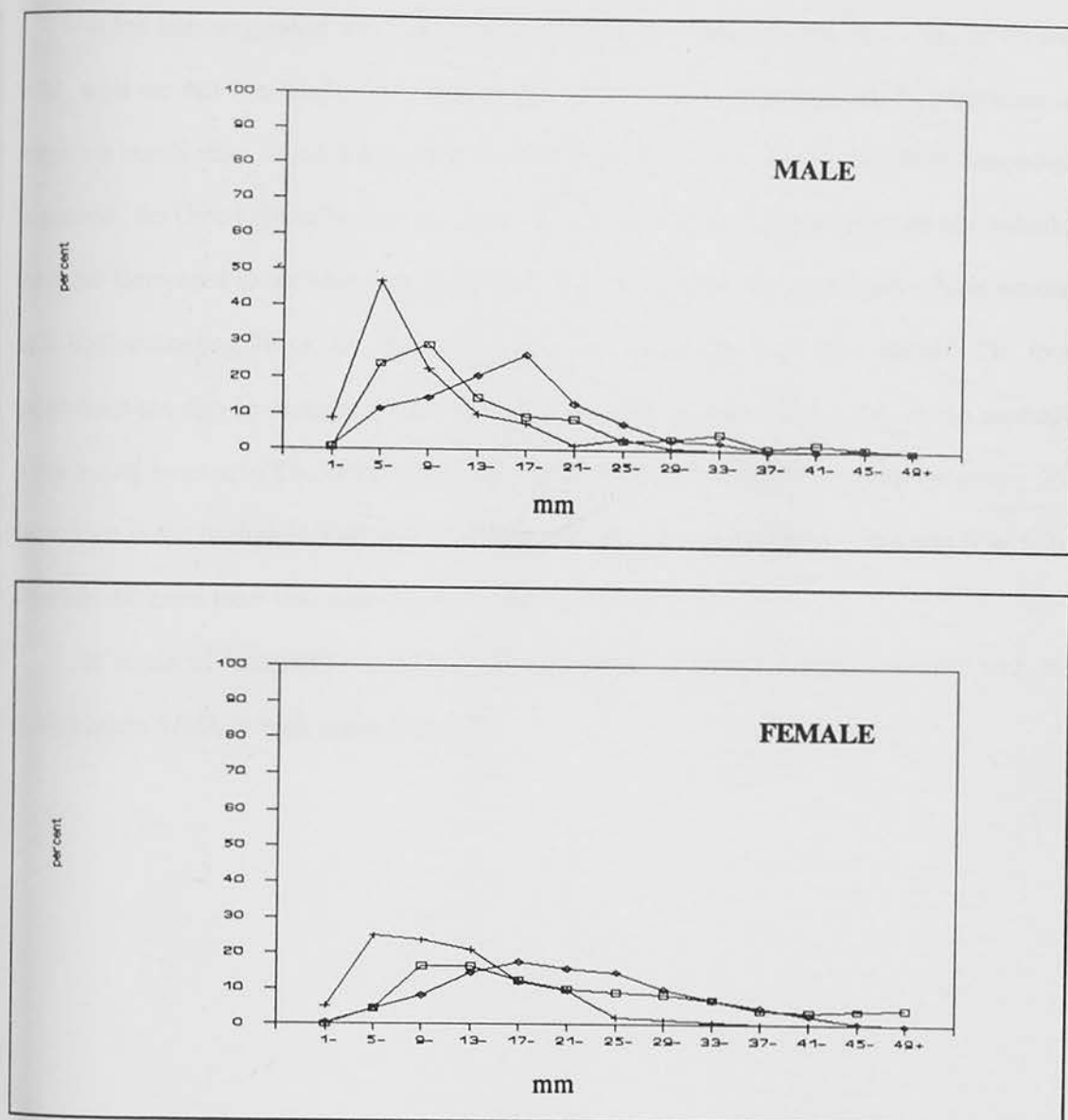


Fig.6.4 Frequency distribution of subscapular skinfold by sex

□ Keewatin + Chukotka ◇ Manitoba △ Indians

Note: [Skinfold data not available for Province of Manitoba]

It has been suggested (eg. Bray 1989) that BMI can be categorized into <26 , 26-30 and >30 , with the last two labelled as "overweight" and "obese", corresponding to gradations in long-term health risks. Fig.6.5 compares the four populations in terms of such BMI categories. In general, the Cree-Ojibwa Indians are the most "obese" and the Chukchi/Eskimo of Chukotka the least. Compared to the Manitoba population, a greater proportion of Keewatin Inuit women have BMI exceeding 26 or 30, whereas among men the difference is less marked. The four populations can also be compared in terms of age-sex-specific means (Fig.6.6-6.7). An anomaly is that young women in Chukotka have a high mean BMI and prevalence of BMI exceeding 26. Of interest is the decline in BMI and skinfolds with age among women in Chukotka (Fig.6.3), a pattern different from that observed in the other populations.

In terms of "centrality" measures, the Indians are clearly the most "central", with the highest mean WHR in both sexes (Fig.6.6).

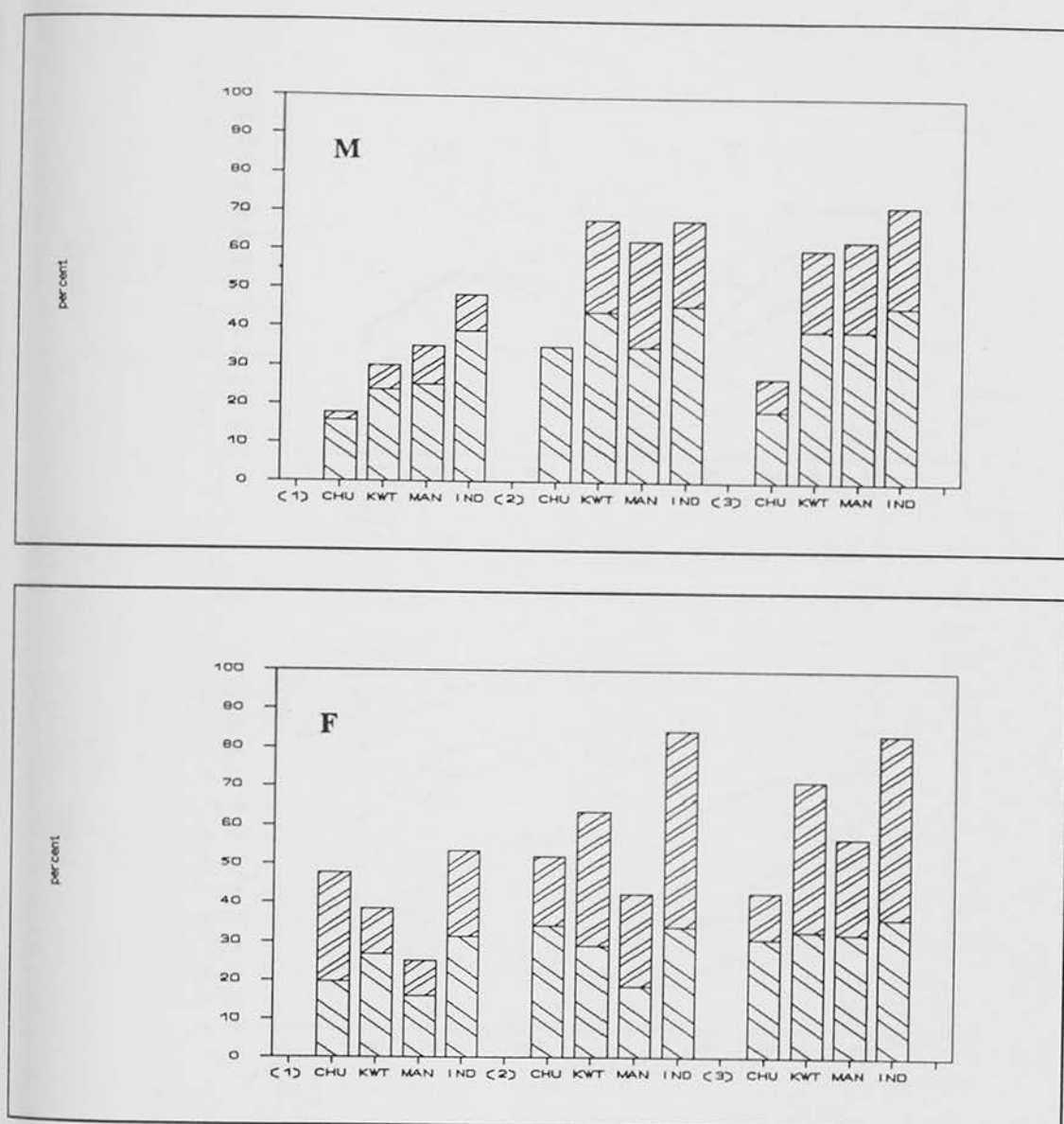


Fig.6.5 Prevalence of obesity (BMI 26-30 and BMI > 30) by age and sex

Notes: (1) age < 35 (2) 35-49 (3) ≥ 50

CHU Chukotka Natives
MAN Province of Manitoba

KWT Keewatin Inuit
IND Cree-Ojibwa Indians

BMI 26-30 

BMI > 30 

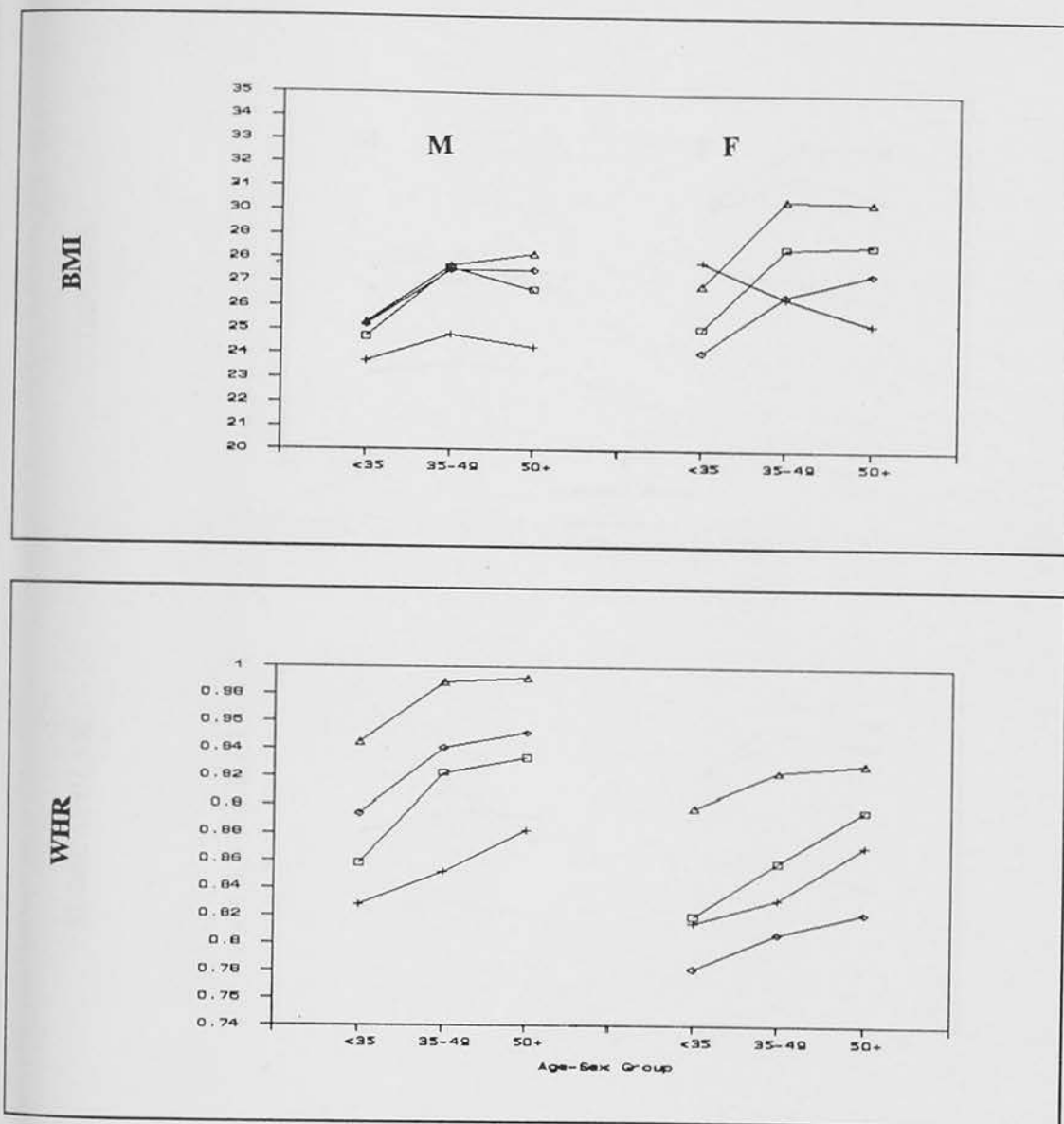


Fig.6.6 Mean body mass index (BMI) and waist-hip ratio (WHR) by age and sex

□ Keewatin + Chukotka ◇ Manitoba Δ Indians

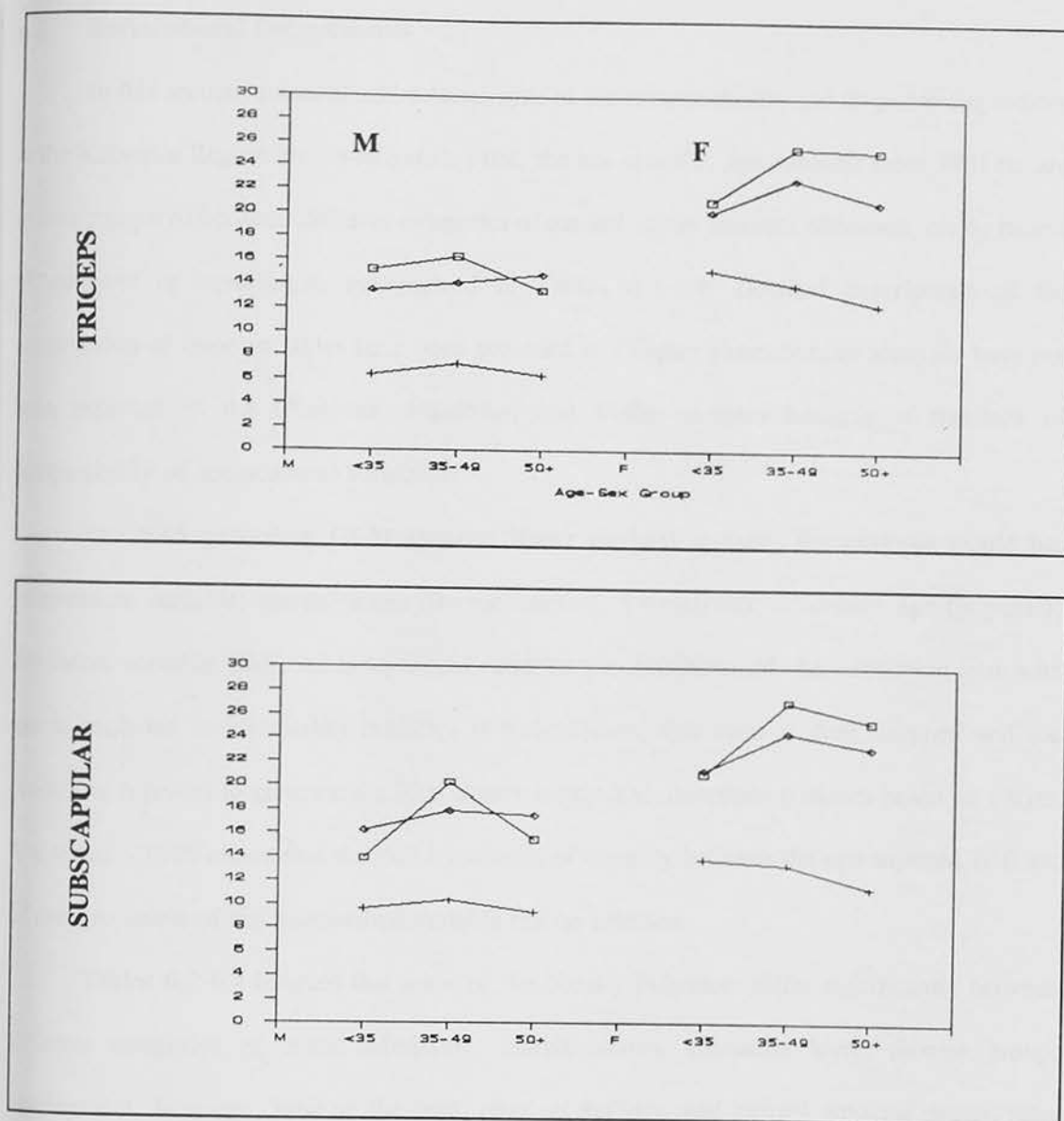


Fig.6.7 Mean subscapular and triceps skinfolds by age and sex

□ Keewatin + Chukotka ◇ Manitoba Δ Indians

6.2 Sociocultural Determinants

In this section, sociocultural determinants of the various obesity and fat patterning indices in the Keewatin Region are investigated. First, the sex-specific, age-adjusted mean BMI etc are in turn compared between different categories of marital status, income, education, etc by means of *analyses of covariance*, summarized in Tables 6.2-6.4. Detailed descriptions of the construction of these variables have been provided in Chapter Four. Similar analyses have not been repeated in the Chukotka, Manitoba, and Indian samples because of the lack of comparability of sociocultural variables.

The SAS procedure **GLM** (general linear models) is used. An example would be: *independent variable*: marital status (0=not married, 1=married); *covariate*: age (in years); *dependent variable*: BMI. All independent variables are dichotomized. An interaction term with age is included in all models initially. If insignificant, this term is then dropped and the procedure repeated to generate the least-square means and associated p values based on t-tests. A p value <0.05 means that the null hypothesis of equality between the age-adjusted BMI etc of the two levels of the independent variable can be rejected.

Tables 6.2-6.4 indicate that some of the obesity indicators differ significantly between different categories of ethnic admixture, marital status, education level, income group, employment, language, time on the land, physical activity, and current smoking status, after adjustment for age. The sexes, however, do differ: for example, female non-smokers are more obese (regardless of the indicator used) than smokers, while no significant difference can be detected among males; better educated males tend to be more obese than the less educated, while the reverse is true in females, although this trend is not statistically significant in all cases.

Table 6.2 Comparison of sex-specific and age-adjusted mean body mass index by selected sociocultural variables: Keewatin Inuit

Variable	Category	Body mass index			
		Male		Female	
		mean	p	mean	p
Marital status	not married	25.1	0.091	26.0	0.082
	married	26.2		27.3	
Race	mixed	25.8	0.747	26.7	0.900
	Inuit only	25.6		26.8	
Education	primary only	24.9	0.081	27.3	0.287
	some secondary	26.3		25.9	
Income	<\$10,000	25.3	0.137	26.7	0.543
	≥\$10,000	26.4		27.3	
Employment	not employed	25.0	0.051	26.7	0.632
	full/part-time	26.3		27.0	
Inuit language	not fluent	25.4	0.639	25.4	0.014
	fluent	25.7		27.3	
Time on land	infrequent	24.6	0.068	26.7	0.682
	frequent	25.9		27.0	
Current smoking	non-smoker	26.0	0.416	29.4	0.000
	smoker	25.4		25.9	
Physical activity	inactive	24.2	0.291	27.8	0.123
	active	25.6		26.5	
Family history	negative	25.7	0.470	27.0	0.479
	positive	25.1		26.4	

Table 6.3 Comparison of sex-specific and age-adjusted mean skinfolds by selected sociocultural variables: Keewatin Inuit

Variable	Category	Subscapular				Triceps			
		Male		Female		Male		Female	
		mean	p	mean	p	mean	p	mean	p
Marital status	not married	14.2	0.157	21.9	0.142	14.5	0.557	22.0	0.134
	married	16.3		24.6		15.4		24.0	
Race	mixed	16.9	0.256	23.0	0.579	18.5	0.017	23.0	0.792
	Inuit only	14.8		24.0		14.0		23.4	
Education	primary only	13.2	0.042	24.8	0.144	14.9	0.946	24.0	0.218
	some secondary	17.0		21.8		15.0		22.2	
Income	<\$10,000/yr	14.0	0.011	23.5	0.797	14.1	0.096	23.3	0.815
	≥\$10,000/yr	18.1		24.1		16.9		22.9	
Employment	not employed	13.7	0.046	23.1	0.441	13.8	0.130	23.5	0.605
	full/part-time	16.7		24.5		16.1		22.8	
Inuit language	not fluent	14.6	0.596	21.0	0.080	11.2	0.001	21.3	0.075
	fluent	15.5		24.5		16.6		24.0	
Time on land	infrequent	13.6	0.103	24.0	0.947	17.4	0.025	23.2	0.729
	frequent	16.2		23.8		13.7		23.6	
Current smoking	non-smoker	16.4	0.501	28.7	0.001	16.8	0.268	28.0	0.000
	smoker	15.2		21.9		14.7		21.5	
Physical activity	inactive	13.8	0.615	27.2	0.045	11.4	0.212	25.2	0.171
	active	15.6		22.6		15.7		22.8	
Family history	negative	16.0	0.277	24.4	0.340	15.7	0.420	23.9	0.199
	positive	13.5		22.4		13.9		21.8	

Table 6.4 Comparison of sex-specific and age-adjusted mean circumferences by selected sociocultural variables: Keewatin Inuit

Variable	Category	Waist				Hips			
		Male		Female		Male		Female	
		mean	p	mean	p	mean	p	mean	p
Marital status	not married	87.1	0.314	84.5	0.043	98.7	0.552	100.0	0.038
	married	88.9		87.9		99.4		102.8	
Race	mixed	89.0	0.579	85.8	0.502	99.5	0.636	102.1	0.724
	Inuit only	87.7		87.0		98.9		101.6	
Education	primary only	86.5	0.210	88.2	0.028	98.3	0.347	102.7	0.094
	some secondary	89.4		84.1		99.7		100.2	
Income	<\$10,000/yr	87.3	0.236	86.8	0.631	98.8	0.577	101.6	0.667
	≥\$10,000/yr	89.7		85.7		99.5		102.4	
Employment	not employed	86.8	0.173	86.9	0.658	98.3	0.236	101.7	0.966
	full/part-time	89.3		86.1		99.7		101.8	
Inuit language	not fluent	87.1	0.534	84.3	0.099	99.1	0.921	99.9	0.098
	fluent	88.4		87.4		99.0		102.4	
Time on land	infrequent	85.6	0.156	86.9	0.872	97.9	0.313	102.0	0.958
	frequent	88.4		86.6		99.1		101.9	
Current smoking	non-smoker	90.3	0.121	91.8	0.000	100.4	0.126	107.0	0.000
	smoker	86.9		84.8		98.3		99.9	
Physical activity	inactive	80.3	0.061	88.3	0.344	94.4	0.051	104.3	0.070
	active	87.9		86.2		99.2		101.1	
Family history	negative	88.2	0.364	86.8	0.888	99.4	0.190	102.1	0.670
	positive	85.8		86.5		97.3		101.4	

The next step involves a series of stepwise multiple regression analyses, with the independent variable list including all the sociocultural variables listed in Tables 6.2-6.4 as well as age as a continuous variable. Each of the obesity indices serves in turn as the dependent variable. Since the distribution of these variables has been shown earlier (Table 4.3 in Chapter Four, p.119) to deviate from "normality", their logarithm-transformed values are used in the regressions (Table 6.5). To facilitate comparison on the impact of the predictors, the standardized regression coefficients are also included.

Table 6.5 shows that age, education and smoking status are consistently identified as predictors of most measures of obesity among women. Among men, apart from age, various predictors such as education, non-Inuit admixture, income, language and time spent on the land are associated with some measures of obesity. It should be noted that education has opposite effects in the two sexes. Among men, those with some secondary education are more obese than those with primary education only, whereas among women, the opposite is observed. Current smokers have lower measures of obesity than non-smokers, but among women only. For measures of "acculturation" the data are somewhat conflicting: men who are fluent in the Inuit language (hence "less acculturated") and those who spend less time on the land (hence "more acculturated") are more likely to have higher triceps skinfolds. These variables are not important in predicting subscapular skinfolds. It is possible that fluency in the Inuit language is highly sought after as interpreter and would thus be a marker of acculturation. Those with mixed Inuit/non-Inuit heritage, to the extent known to and reported by the respondents, have higher triceps skinfold than unadmixed Inuit. Note that "ethnocultural" variables such as admixture, language and time on the land are associated only with triceps skinfold among men.

Table 6.5 Significant independent predictors of obesity/central fat patterning indices established by stepwise multiple regression analyses: Keewatin Inuit

Dependent variable (log transformed)	Independent predictors [standardized regression coefficients]	Model R ²
(1) Male		
Body mass index	Age [0.398], Education [0.255]	0.108
Waist	Age [0.408]	0.166
Hips	Age [0.304]	0.092
Waist/hip ratio	Age [0.417]	0.174
Triceps skinfold	Race [-0.196], Language [0.228], Time on land [-0.206]	0.131
Subscapular skinfold	Income [0.212]	0.045
Sum of skinfolds	Income [0.214]	0.046
Subscapular/triceps ratio	Age [0.310], Language [-0.312], Time on land [0.239], Education [0.199]	0.189
(2) Female		
Body mass index	Age [0.320], Smoking [-0.239]	0.192
Waist	Age [0.355], Education [-0.191], Smoking [-0.233]	0.322
Hips	Age [0.215], Education [-0.160], Smoking [-0.298]	0.224
Waist/hip ratio	Age [0.424], Education [-0.161]	0.272
Triceps skinfold	Education [-0.178], Smoking [-0.288]	0.122
Subscapular skinfold	Age [-0.186], Smoking [-0.177]	0.080
Sum of skinfolds	Smoking [-0.260], Education [-0.194]	0.113
Subscapular/triceps ratio	nil significant	

Note: The independent variables in all models included:

age (in years); race (0=mixed, 1=Inuit only); marital status (0=not married, 1=married); education (0=primary only, 1=some secondary); income (0=<\$10,000, 1=>\$10,000 per year); employment (0=not employed, 1=employed full/part-time); language (0=not fluent, 1=fluent in Inuktitut); time on land (0=infrequent, 1=frequent); current smoking (0=non-smoker, 1=smoker); physical activity (0=inactive, 1=active); family history (0=negative, 1=positive).

6.3 Metabolic Consequences

One approach to understanding if the "obesity" observed among the Inuit has the same biological significance as that observed in other populations is to determine if it is associated with metabolic disturbances as reflected in changes in the levels of blood pressure, glucose, insulin and lipids.

The analytic approach here differs from that used in Section 6.2. Obesity is now the independent rather than dependent variable. The dependent variables are mean systolic and diastolic blood pressure (**SBP, DBP**), fasting and 2-hour post-load glucose (**FG, 2hG**), fasting insulin (**FIns**), total cholesterol (**TC**), HDL- and LDL-cholesterol (**HDL, LDL**), and triglycerides (**TG**).

The independent variables are initially analysed as categorical variables in analyses of covariance. For BMI and WHR, cut-off points associated with increased disease risks established in various epidemiological studies can be used. For BMI, three categories (<26, 26-30 and >30) with increasing risk are used; for WHR, "at risk" in men refers to those with values exceeding 0.9 and for women, 0.8 (Bray 1989). Table 6.6A compares the age-adjusted means of various metabolic variables for each sex separately, between categories of BMI. Table 6.6B compares high and low categories of WHR, adjusting for both age *and* BMI. This is to determine if "centrality", as indicated by WHR, has an effect on metabolic variables independent of "overall" obesity, as represented by BMI. STR are not analysed as risk categories as these are not as well established as categories of BMI and WHR. However, individual skinfolds and STR are used in various regression models where appropriate.

Table 6.6 Comparison of sex-specific and age-adjusted means of selected metabolic variables according to risk categories of body mass index (BMI) and waist-hip ratio (WHR): Keewatin Inuit

	Male				Female			
(A) Independent variable: BMI categories; covariate: Age								
	BMI <26	26-30	> 30	p	BMI <26	26-30	> 30	p
SBP	121.3	126.3	133.8	0.008	111.9	115.6	121.0	0.001
DBP	75.1	80.2	88.0	0.000	69.9	73.3	78.5	0.000
FG	5.82	5.85	5.86	0.983	6.25	6.74	6.48	0.120
2hG	6.09	5.86	5.56	0.590	7.41	7.55	7.74	0.836
FIns	44.8	61.7	69.0	0.242	64.1	55.1	71.8	0.617
TC	4.54	5.05	5.44	0.001	5.13	4.96	5.22	0.382
HDL	1.40	1.39	1.17	0.057	1.66	1.45	1.33	0.001
LDL	2.74	3.24	3.68	0.000	3.05	2.97	3.26	0.286
TG	0.87	0.93	1.30	0.004	0.93	1.17	1.38	0.002
(B) Independent variable: WHR categories; covariates: Age and BMI								
	Low WHR	High WHR	p	Low WHR	High WHR	p		
SBP	126.7	121.8	0.200	115.3	115.4	0.976		
DBP	78.1	78.8	0.753	75.1	72.4	0.093		
FG	5.69	6.01	0.263	6.25	6.55	0.286		
2hG	5.82	6.14	0.547	7.84	7.46	0.520		
FIns	68.6	40.6	0.059	73.3	62.1	0.550		
TC	4.63	5.09	0.069	4.95	5.10	0.457		
HDL	1.38	1.37	0.912	1.55	1.48	0.466		
LDL	2.84	3.27	0.067	2.96	3.09	0.490		
TG	0.92	0.99	0.558	0.97	1.16	0.133		

Notes: WHR risk categories (HIGH: > 0.9 in males and > 0.8 in females)

It can be seen that higher blood pressure levels and an unfavourable lipid profile (high TC, TG, LDL and/or low HDL) are present among those with BMI exceeding 30 than those in the intermediate (26-30) and lowest category (<26). For insulin, there is a trend to an increase, although the magnitude is not sufficiently large to be statistically significant. No significant difference in glucose levels between BMI categories can be detected. None of the metabolic indicators, however, differ between high and low categories of WHR, when both age and BMI have been adjusted for.

Note that the categories used are based on external standards. The categories thus defined indicate that in some other populations they are predictive of long-term health risks. A different analytical approach is to group the data according to quartiles (Table 6.7). "High" and "low" categories are then specific to the distribution in each sample, and vary from population to population. A similar pattern is observed, ie. increased BP and a more unfavourable lipid profile at higher quartile of BMI, waist, hips and subscapular skinfolds but little change in FG, 2hG and FIns. An increase in triceps skinfold is associated with an increase only in BP. As an alternative measure of "overall" obesity, the sum of skinfolds "behaves" in a fashion similar to that of BMI, with significant effects on blood pressure and lipids. As a centrality index, WHR independent of BMI has little effect on any of the metabolic indices. STR independent of the sum of skinfolds, on the other hand, has an effect on some lipid measures among women.

Note that there appears to be a reverse-L or U-shaped relationship between the obesity indices and the various metabolic indicators among males, whereas in females, the relationship is more linear. This is due to the fact that in the latter, the mean values of obesity indicators are generally higher, such that even the lower quartiles are at the "high end" of the curve in males.

Table 6.7 Comparison of sex-specific and age-adjusted means of selected metabolic variables according to quartiles of obesity indices: Keewatin Inuit

	Male					Female				
	Min-Q1	Q1-Q2	Q2-Q3	Q3-Max	p	Min-Q1	Q1-Q2	Q2-Q3	Q3-Max	p
(1) Body mass index										
SBP	122.3	119.3	122.6	132.8	0.001	110.4	113.5	115.6	121.0	0.002
DBP	76.1	74.8	76.9	84.8	0.000	69.1	70.6	73.3	78.5	0.000
FG	5.78	5.68	5.80	6.03	0.622	6.39	6.13	6.74	6.48	0.175
2hG	6.17	5.89	5.76	6.10	0.826	6.92	7.91	7.55	7.73	0.429
FIns	36.9	52.9	44.4	76.8	0.066	71.6	51.5	55.1	71.9	0.609
TC	4.26	4.56	4.96	5.25	0.002	5.00	5.26	4.96	5.22	0.369
HDL	1.41	1.35	1.48	1.24	0.027	1.75	1.58	1.45	1.33	0.001
LDL	2.47	2.80	3.09	3.49	0.001	2.85	3.24	2.97	3.26	0.123
TG	0.83	0.90	0.88	1.14	0.040	0.89	0.98	1.17	1.38	0.004
(2) Waist circumference										
SBP	122.7	121.3	120.4	134.4	0.001	109.7	114.9	116.1	121.6	0.001
DBP	74.9	74.9	78.1	86.2	0.000	69.0	70.4	74.4	78.8	0.000
FG	5.82	5.78	5.80	5.94	0.948	6.32	6.27	6.67	6.64	0.469
2hG	6.25	6.06	5.74	5.83	0.809	7.44	6.78	8.04	7.81	0.204
FIns	49.4	38.9	61.6	65.1	0.482	72.2	53.8	55.9	74.4	0.620
TC	4.48	4.59	4.77	5.45	0.001	4.87	5.21	5.05	5.11	0.489
HDL	1.48	1.44	1.34	1.24	0.086	1.71	1.56	1.52	1.24	0.000
LDL	2.63	2.75	3.04	3.66	0.000	2.78	3.20	3.03	3.20	0.171
TG	0.82	0.90	0.85	1.21	0.004	0.84	1.00	1.10	1.47	0.000
(3) Hips circumference										
SBP	122.2	118.3	129.7	129.7	0.004	110.1	113.6	117.5	121.0	0.001
DBP	76.4	72.7	79.7	85.3	0.000	69.1	70.2	75.0	78.4	0.000
FG	5.93	5.85	5.83	5.74	0.932	6.30	6.20	6.75	6.57	0.205
2hG	6.35	6.17	5.71	5.59	0.421	7.26	7.39	7.71	7.94	0.710

FIns	44.9	47.3	50.0	86.4	0.063	70.8	50.9	58.6	78.0	0.561
TC	4.64	4.80	4.63	5.26	0.055	5.00	5.34	4.88	5.25	0.155
HDL	1.40	1.41	1.45	1.23	0.069	1.67	1.62	1.42	1.31	0.001
LDL	2.84	3.00	2.73	3.54	0.004	2.90	3.29	2.92	3.31	0.075
TG	0.89	0.84	1.00	1.07	0.225	0.96	0.95	1.20	1.39	0.010

(4) Waist-hip ratio, adjusted for BMI

SBP	128.5	127.3	121.1	122.0	0.319	116.0	114.6	116.1	115.0	0.932
DBP	78.5	77.9	78.7	78.4	0.987	75.1	71.6	74.1	71.4	0.110
FG	5.85	6.23	6.08	5.75	0.812	6.23	6.36	6.54	6.71	0.607
2hG	5.72	5.86	5.62	6.15	0.345	7.91	7.09	8.03	7.13	0.259
FIns	84.1	66.1	48.6	31.3	0.066	70.4	54.0	62.9	67.2	0.897
TC	4.77	4.67	5.04	4.82	0.509	4.84	4.96	4.92	5.46	0.561
HDL	1.45	1.32	1.35	1.38	0.598	1.53	1.43	1.49	1.52	0.796
LDL	2.91	2.89	3.27	3.01	0.358	2.89	3.05	2.90	3.36	0.130
TG	0.91	1.01	0.92	0.95	0.838	0.94	1.05	1.16	1.27	0.259

(5) Triceps skinfolds

SBP	121.9	123.0	124.6	128.2	0.366	110.8	114.9	116.3	118.8	0.024
DBP	76.5	79.6	75.8	82.1	0.023	70.2	70.9	73.2	77.9	0.000
FG	5.68	5.50	5.92	6.10	0.186	6.26	6.42	6.72	6.34	0.362
2hG	5.70	6.54	6.00	5.89	0.480	7.36	7.38	7.72	7.93	0.757
FIns	57.8	45.0	60.7	70.9	0.728	70.1	57.9	52.2	72.7	0.662
TC	4.96	4.66	4.86	4.84	0.768	5.34	5.15	4.94	5.08	0.339
HDL	1.52	1.39	1.29	1.32	0.057	1.64	1.55	1.47	1.37	0.082
LDL	3.03	2.86	3.13	3.07	0.783	3.20	3.10	2.97	3.10	0.715
TG	0.91	0.90	0.97	0.99	0.857	1.11	1.11	1.10	1.31	0.441

(6) Subscapular skinfolds

SBP	121.1	121.8	125.6	129.4	0.111	108.3	115.9	116.7	120.0	0.000
DBP	75.1	77.3	78.2	83.4	0.002	69.1	70.5	75.2	77.4	0.000
FG	5.93	5.72	5.86	5.81	0.908	6.53	6.30	6.50	6.55	0.788
2hG	5.73	5.53	6.42	6.23	0.312	7.23	7.90	7.43	7.79	0.705
FIns	39.0	45.5	60.2	75.4	0.111	80.1	53.5	50.2	70.3	0.418
TC	4.54	4.74	4.97	5.14	0.098	5.00	5.06	5.33	5.04	0.431

HDL	1.47	1.48	1.31	1.25	0.029	1.71	1.57	1.46	1.32	0.001
LDL	2.68	2.86	3.23	3.39	0.013	2.91	3.04	3.25	3.09	0.424
TG	0.86	0.88	0.94	1.11	0.177	0.83	0.99	1.36	1.38	0.000

(7) Sum of skinfolds

SBP	122.3	120.6	126.5	127.9	0.179	108.0	116.1	116.8	120.2	0.000
DBP	76.4	76.0	78.4	82.4	0.023	69.0	70.6	74.4	78.1	0.000
FG	5.72	5.87	5.75	5.98	0.767	6.38	6.40	6.36	6.64	0.736
2hG	5.46	6.43	6.02	5.94	0.373	7.19	7.96	7.39	7.87	0.578
FIns	40.8	54.8	77.2	61.5	0.156	76.0	54.8	47.1	71.5	0.414
TC	4.81	4.73	4.76	5.03	0.604	5.06	5.21	5.22	4.96	0.597
HDL	1.55	1.37	1.39	1.24	0.014	1.71	1.54	1.44	1.37	0.006
LDL	2.86	2.94	3.02	3.27	0.367	2.97	3.15	3.19	3.00	0.617
TG	0.90	0.93	0.78	1.15	0.009	0.83	1.13	1.29	1.31	0.005

(8) Subscapular/triceps skinfold ratio, adjusted for sum of skinfolds

SBP	123.6	125.0	123.8	124.9	0.980	115.6	114.2	114.5	116.5	0.795
DBP	76.7	78.0	78.2	80.4	0.435	72.5	72.5	73.8	73.3	0.864
FG	5.91	6.22	5.67	5.51	0.104	6.60	6.53	6.30	6.35	0.721
2hG	5.73	5.80	6.39	6.06	0.624	7.84	7.53	7.58	7.50	0.949
FIns	28.9	58.5	47.0	65.1	0.439	46.9	57.6	73.4	67.3	0.715
TC	4.50	5.05	4.93	4.94	0.164	4.76	4.86	5.34	5.49	0.004
HDL	1.43	1.41	1.27	1.36	0.340	1.55	1.54	1.47	1.45	0.723
LDL	2.69	3.22	3.17	3.12	0.119	2.76	2.85	3.32	3.40	0.005
TG	0.84	0.92	1.07	0.99	0.317	0.98	1.03	1.20	1.41	0.022

Notes: Min - minimum; Q1 Q2 Q3 - first/second/third quartile; Max - maximum

All obesity and centrality measures are further analysed as continuous variables in stepwise multiple regression analyses to predict blood pressure, lipids and glucose, etc (Table 6.8). Five separate analyses are conducted:

- (1) $\log DV = \text{Age} + \log \text{BMI} + \log \text{WHR}$
- (2) $\log DV = \text{Age} + \log \text{Triceps} + \log \text{Subscap}$
- (3) $\log DV = \text{Age} + \log \text{Waist} + \log \text{Hips}$
- (4) $\log DV = \text{Age} + \log \text{BMI} + \log \text{Triceps} + \log \text{Subscap}$
- (5) $\log DV = \text{Age} + \log \text{BMI} + \log \text{Triceps} + \log \text{Subscap} + \log \text{Waist} + \log \text{Hips}$

[DV = dependent variable, ie. BP, glucose, insulin, and lipids]

Table 6.8. Significant independent predictors of selected metabolic variables established by stepwise multiple regression analyses: Keewatin Inuit

Dependent Variable	Independent predictor [standardized regression coefficient]			
	Male	Model R ²	Female	Model R ²
(1) Independent variable list: age, log BMI, log WHR				
log SBP	Age [0.200] log BMI [0.273]	0.137	Age [0.379] log BMI [0.303]	0.303
log DBP	Age [-0.196] log BMI [0.355]	0.137	log BMI [0.396]	0.157
log FG	[nil significant]	0.002	[nil significant]	0.026
log 2hG	Age [0.201]	0.040	Age [0.182]	0.033
log FIns	log BMI [0.617] log WHR [-0.369]	0.188	[nil significant]	0.050
log TC	Age [0.413] log BMI [0.265]	0.293	Age [0.442]	0.196
log HDL	Age [0.511] log BMI [-0.250]	0.262	Age [0.264] log BMI [-0.389]	0.147
log LDL	Age [0.226] log WHR [0.301]	0.062	Age [0.375]	0.141
log TG	log BMI [0.248]	0.062	log BMI [0.394]	0.156

(2) Independent variable list: age, log TRIceps, log SUBscapular

log SBP	Age [0.214] log SUB [0.226]	0.103	Age [0.429] log SUB [0.307]	0.305
log DBP	Age [-0.157] log SUB [0.318]	0.120	log SUB [0.400]	0.160
log FG	[nil significant]	0.055	[nil significant]	0.017
log 2hG	[nil significant]	0.049	[nil significant]	0.028
log FIns	log SUB [0.403]	0.162	[nil significant]	0.030
log TC	Age [0.496]	0.246	Age [0.420]	0.176
log HDL	Age [0.474] log SUB [-0.307]	0.290	log SUB [-0.254]	0.065
log LDL	Age [0.373] log SUB [0.183]	0.186	Age [0.375]	0.140
log TG	log SUB [0.225]	0.051	log SUB [0.616] log TRI [-0.278]	0.200

(3) Independent variable list: age, log WAIST, log HIPS

log SBP	log WAIST [0.323]	0.104	Age [0.400] log HIPS [0.266]	0.286
log DBP	Age [-0.210] log HIPS [0.408]	0.176	log HIPS [0.380]	0.144
log FG	[nil significant]	0.006	[nil significant]	0.021
log 2hG	Age [0.201]	0.040	Age [0.182]	0.033
log FIns	log HIPS [0.353]	0.125	[nil significant]	0.066
log TC	Age [0.372] log WAIST [0.250]	0.278	Age [0.442]	0.196
log HDL	Age [0.570] log WAIST [-0.285]	0.271	Age [0.245] log HIPS [-0.381]	0.145
log LDL	Age [0.238] log WAIST [0.304]	0.209	Age [0.375]	0.141
log TG	Age [-0.209] log WAIST [0.318]	0.090	log WAIST [0.427]	0.183

(4) Independent variable list: age, log BMI, log TRIceps, log SUBscapular

log SBP	Age [0.160] log BMI [0.287]	0.130	Age [0.427] log SUB [0.306]	0.304
log DBP	Age [-0.157] log SUB [0.318]	0.120	log SUB [0.401]	0.161
log FG	[nil significant]	0.056	[nil significant]	0.021
log 2hG	[nil significant]	0.133	[nil significant]	0.036
log FIns	log SUB [0.403]	0.162	[nil significant]	0.063
log TC	Age [0.422] log BMI [0.251]	0.304	Age [0.447]	0.200
log HDL	Age [0.474] log SUB [-0.307]	0.290	Age [0.257] log BMI [-0.366]	0.134
log LDL	Age [0.302] log BMI [0.300]	0.235	Age [0.384]	0.148
log TG	log BMI [0.229]	0.052	log SUB [0.412]	0.169

(5) Independent variable list: age, log BMI, log WAIST, log HIPS, log TRiceps, log SUBscapular

log SBP	Age [0.163] log BMI [0.286]	0.131	Age [0.428] log SUB [0.311]	0.309
log DBP	Age [-0.223] log HIPS [0.397]	0.167	log SUB [0.401]	0.161
log FG	[nil significant]	0.068	[nil significant]	0.085
log 2hG	[nil significant]	0.166	Age [0.178]	0.032
log FIns	log SUB [0.403]	0.162	[nil significant]	0.004
log TC	Age [0.422] log BMI [0.251]	0.304	Age [0.454]	0.206
log HDL	Age [0.474] log SUB [-0.307]	0.290	Age [0.245] log HIPS [-0.378]	0.146
log LDL	Age [0.260] log WAIST [0.287]	0.218	Age [0.386]	0.149
log TG	log BMI [0.229]	0.052	log SUB [0.433]	0.188

Several observations can be made from Table 6.8. Again, obesity is shown to have little effect on fasting or 2-hour glucose levels, although a significant effect on fasting insulin, blood pressure and lipid levels can be seen. In the presence of BMI, WHR rarely emerges as an independent predictor of metabolic effects. SUB is clearly the more important predictor than TRI when both are present in a regression model; when TRI does appear together with SUB, as in the regression equation for logTG, it is negative. When both waist and hips are present, waist is more important among males and hips among females. Regression equations (4) and (5), when BMI is present together with both skinfolds, and with skinfolds and circumferences, respectively, are intended to isolate the effect of "internal" or intraabdominal fat. While subscapular skinfold and waist circumference indicate "central" fat, they actually reflect only the "subcutaneous" component. Thus theoretically, the effect of BMI exclusive of all measures of subcutaneous fat should indicate the effect of internal fat. Regressions (4) and (5) are generally similar. BMI

independent of subcutaneous fat measures is a significant predictor of blood pressure and some lipids in men more so than in women.

6.4 Between-Population Comparisons

Among Keewatin Inuit, increased measures of obesity and/or central fat patterning have been found to be associated with blood pressure, plasma lipids and lipoproteins and fasting insulin, but not with plasma glucose levels. Whether this phenomenon is unique to the Inuit can be investigated by repeating the analyses in the other three comparison populations. Table 6.9 summarizes data from these analyses and shows the significant differences in age-adjusted means between categories of BMI or WHR, defined according to disease risk, while Table 6.10 compares quartiles of various obesity indicators.

Table 6.11 summarizes the stepwise multiple regression analyses where the logarithm-transformed values of both dependent and independent continuous variables are used. Four regression models are fitted:

$$(1) \log DV = \text{Age} + \log \text{BMI} + \log \text{WHR}$$

$$(2) \log DV = \text{Age} + \log \text{Triceps} + \log \text{Subscap}$$

$$(3) \log DV = \text{Age} + \log \text{Waist} + \log \text{Hips}$$

$$(4) \log DV = \text{Age} + \log \text{BMI} + \log \text{Triceps} + \log \text{Subscap} + \log \text{Waist} + \log \text{Hips}$$

[DV = dependent variable, ie. BP, glucose, insulin, and lipids]

Table 6.9 Summary of comparison of sex-specific, age-adjusted means of selected metabolic variables according to risk categories of BMI and WHR:
Comparison populations

Metabolic variables	Keewatin		Chukotka		Manitoba		Indians	
	M	F	M	F	M	F	M	F
(1) Age-adjusted means between BMI categories (<26, 26-30, >30):								
Systolic BP	**	**	*	**	**	**	NS	**
Diastolic BP	**	**	*	**	**	**	**	**
Fasting glucose	NS	NS	NS	NS	**	**	NS	*
2-hr glucose	NS	NS	NS	NS	-	-	-	-
Fasting insulin	NS	NS	NS	**	-	-	-	-
Total cholesterol	**	NS	NS	NS	**	**	**	*
HDL-cholesterol	NS	**	NS	*	**	**	**	**
LDL-cholesterol	**	NS	NS	NS	**	**	**	*
Triglycerides	**	**	NS	NS	**	**	**	**
(2) Age- and BMI-adjusted means between HIGH/LOW categories of WHR								
Systolic BP	NS	NS	NS	*	**	**	NS	NS
Diastolic BP	NS	NS	NS	NS	**	NS	NS	NS
Fasting glucose	NS	NS	NS	NS	NS	*	NS	NS
2-hr glucose	NS	NS	NS	NS	-	-	-	-
Fasting insulin	NS	NS	NS	NS	-	-	-	-
Total cholesterol	NS	NS	NS	NS	**	**	NS	NS
HDL-cholesterol	NS	NS	NS	NS	*	*	NS	NS
LDL-cholesterol	NS	NS	NS	NS	*	*	NS	NS
Triglycerides	NS	NS	NS	NS	**	**	NS	NS

Notes: * $p < 0.05$; ** $p < 0.01$; NS not significant $p > 0.05$;
- not available

Table 6.10 Comparison of sex-specific and age-adjusted means of selected metabolic variables according to quartiles of obesity indices: Comparison populations

Metabolic variables	Keewatin		Chukotka		Manitoba		Indians	
	M	F	M	F	M	F	M	F
(1) Age-adjusted means between quartiles of BMI:								
Systolic BP	**	**	NS	*	**	**	NS	**
Diastolic BP	**	**	NS	**	**	**	**	**
Fasting glucose	NS	NS	NS	NS	**	**	*	*
2-hour glucose	NS	NS	NS	NS	-	-	-	-
Fasting insulin	NS	NS	NS	**	-	-	-	-
Total cholesterol	**	NS	NS	NS	**	**	**	*
HDL-cholesterol	*	**	NS	*	**	**	**	**
LDL-cholesterol	**	NS	NS	NS	**	**	*	*
Triglycerides	*	**	NS	NS	**	**	**	**
(2) Age and BMI-adjusted means between quartiles of WHR:								
Systolic BP	NS	NS	NS	NS	NS	**	NS	NS
Diastolic BP	NS	NS	NS	NS	**	NS	*	NS
Fasting glucose	NS	NS	NS	NS	NS	*	NS	NS
2-hour glucose	NS	NS	NS	NS	-	-	-	-
Fasting insulin	NS	NS	NS	NS	-	-	-	-
Total cholesterol	NS	NS	NS	NS	*	*	NS	NS
HDL-cholesterol	NS	NS	*	NS	**	**	*	**
LDL-cholesterol	NS	NS	NS	NS	NS	*	NS	NS
Triglycerides	NS	NS	NS	NS	**	**	**	**
(3) Age-adjusted means between quartiles of SUM:								
Systolic BP	NS	**	*	NS	-	-	*	**
Diastolic BP	*	**	*	*	-	-	**	**

Fasting glucose	NS	NS	NS	NS	-	-	NS	NS
2-hour glucose	NS	NS	NS	NS	-	-	-	-
Fasting insulin	NS	NS	NS	NS	-	-	-	-
Total cholesterol	NS	NS	NS	NS	-	-	**	NS
HDL-cholesterol	*	**	NS	*	-	-	**	*
LDL-cholesterol	NS	NS	NS	*	-	-	**	NS
Triglycerides	**	**	NS	NS	-	-	**	NS

(4) Age and SUM-adjusted means between quartiles of STR:

Systolic BP	NS	NS	NS	NS	-	-	NS	NS
Diastolic BP	NS	NS	NS	NS	-	-	NS	NS
Fasting glucose	NS	NS	NS	NS	-	-	NS	NS
2-hour glucose	NS	NS	NS	NS	-	-	-	-
Fasting insulin	NS	NS	NS	NS	-	-	-	-
Total cholesterol	NS	**	NS	NS	-	-	NS	NS
HDL-cholesterol	NS	NS	NS	NS	-	-	**	**
LDL-cholesterol	NS	**	NS	NS	-	-	NS	NS
Triglycerides	NS	*	NS	NS	-	-	NS	**

Notes: * p < 0.05; ** p < 0.01; NS not significant p > 0.05;

- not available: 2-hour glucose and fasting insulin not available from MANITOBA and INDIANS samples; skinfolds not available from MANITOBA sample

Regardless of the analytical approach - whether analysis of covariance or multiple linear regression - the Inuit in Keewatin and the Eskimo/Chukchi in Chukotka differ from the Indians and all-race population of Manitoba in the absence of an association between obesity and plasma glucose.

Table 6.11 Summary of significant independent predictors of selected metabolic variables established by stepwise multiple regression analyses: Comparison populations

DV	Keewatin		Chukotka		Manitoba		Indians	
	M	F	M	F	M	F	M	F
(1) log DV = age + log BMI + log WHR								
SBP	Age BMI	Age BMI	WHR	Age BMI	Age BMI	WHR	Age WHR	Age BMI
DBP	Age BMI	BMI	WHR	BMI	Age BMI WHR	Age BMI	Age WHR	Age BMI WHR
FG	nil	nil	Age	Age	Age BMI WHR	Age BMI WHR	Age WHR	Age BMI
2hG	Age	Age	nil	nil	-	-	-	-
FIns	BMI WHR	nil	WHR	BMI	-	-	-	-
TC	Age BMI	Age	Age	Age	Age BMI WHR	Age BMI WHR	Age BMI	Age
HDL	Age BMI	Age BMI	nil	BMI	BMI WHR	BMI WHR	Age BMI	Age BMI WHR
LDL	Age WHR	Age	nil	Age BMI	Age BMI WHR	Age BMI	Age BMI	Age
TG	BMI	BMI	Age	nil	Age BMI WHR	Age BMI WHR	Age BMI WHR	Age BMI WHR
(2) log DV = age + log TRI + log SUB								
SBP	Age SUB	Age SUB	SUB	Age	-	-	Age TRI	Age SUB
DBP	Age SUB	SUB	SUB	SUB	-	-	Age TRI	Age SUB
FG	nil	nil	Age	Age	-	-	Age TRI	Age SUB
2hG	nil	nil	nil	nil	-	-	-	-
FIns	SUB	nil	nil	nil	-	-	-	-

TC	Age	Age	Age	Age TRI	-	-	Age TRI	Age
HDL	Age SUB	SUB	nil	nil	-	-	nil	TRI SUB
LDL	Age SUB	Age	TRI	Age TRI	-	-	Age SUB	Age
TG	SUB	SUB TRI	Age	nil	-	-	SUB	Age TRI SUB

(3) log DV = age + log WAI + log HIP

SBP	WAI	Age HIP	HIP	Age HIP	Age WAI	Age WAI	Age	Age HIP
DBP	Age HIP	HIP	WAI	HIP	Age WAI	Age WAI HIP	WAI	Age HIP
FG	nil	nil	Age	Age	Age WAI	Age WAI	Age WAI	Age WAI
2hG	Age	Age	nil	nil	-	-	-	-
FIns	HIP	nil	WAI	HIP	-	-	-	-
TC	Age WAI	Age	Age	Age HIP	Age WAI HIP	Age WAI	Age WAI	Age
HDL	Age WAI	Age HIP	nil	HIP	WAI	Age WAI	Age WAI	Age WAI HIP
LDL	Age WAI	Age	HIP	Age HIP	Age WAI HIP	Age WAI	Age WAI	Age
TG	Age WAI	WAI	Age	nil	Age WAI	Age WAI	Age WAI HIP	WAI HIP

(4) log DV = age + BMI + log WAI + log HIP + log TRI + log SUB

SBP	Age BMI	Age SUB	HIP	Age	-	-	Age	Age SUB
DBP	Age HIP	SUB	WAI	HIP	-	-	WAI	Age HIP TRI
FG	nil	nil	Age	Age	-	-	Age WAI	Age WAI
2hG	nil	Age	nil	Age	-	-	-	-
FIns	SUB	nil	WAI	HIP	-	-	-	-
TC	Age BMI	Age	Age	Age TRI	-	-	Age WAI TRI	Age WAI
HDL	Age SUB	Age HIP	nil	HIP	-	-	Age WAI TRI	HIP TRI

LDL	Age WAI	Age	HIP	Age TRI	-	-	Age WAI	Age
TG	BMI	SUB	Age	nil	-	-	Age WAI HIP	Age HIP

Notes:

SBP	systolic blood pressure	BMI	body mass index
DBP	diastolic blood pressure	WHR	waist-hip ratio
FG	fasting glucose	TRI	triceps skinfold
2hG	2-hour glucose	SUB	subscapular skinfold
FIns	fasting insulin	WAI	waist circumference
TC	total cholesterol	HIP	hip circumference
HDL	HDL-cholesterol		
LDL	LDL-cholesterol		
TG	triglycerides		

- not available: 2hG and FIns not available from MANITOBA and INDIANS samples; TRI and SUB not available from MANITOBA sample

nil no independent variable significant on stepwise multiple regression

Even though BP, TC, LDL and TG tend to increase and HDL decrease with increasing levels of an obesity indicator in all populations, the pattern of response is not identical. Fig.6.8 shows the relationship between blood pressure and the three levels of BMI in the four populations. For diastolic pressure, the rise-with-BMI curves are quite close together. For systolic BP, female Keewatin Inuit have the lowest BP at each category of BMI. The divergence, particularly between Keewatin and Chukotka Natives on the one hand, and the Manitoba and Indian samples on the other, is more clearly evident in the lipid/BMI curves, as shown in Fig.6.9. At each category of BMI, the Keewatin/Chukotka samples have lower triglycerides and higher HDL than the other two samples. The Chukotka Natives also differ from the Keewatin Inuit in that there is no rise in triglycerides with BMI at all, and among men, the relation between HDL and BMI is in fact U-shaped.

Table 6.12 compares the standardized coefficients of the regression of BMI on the various metabolic variables in the four populations. In general, the Chukotka and Keewatin coefficients are lower than the Indian and Manitoba ones, particularly with regard to glucose, HDL and triglycerides. The relationship is less clearcut for blood pressure.

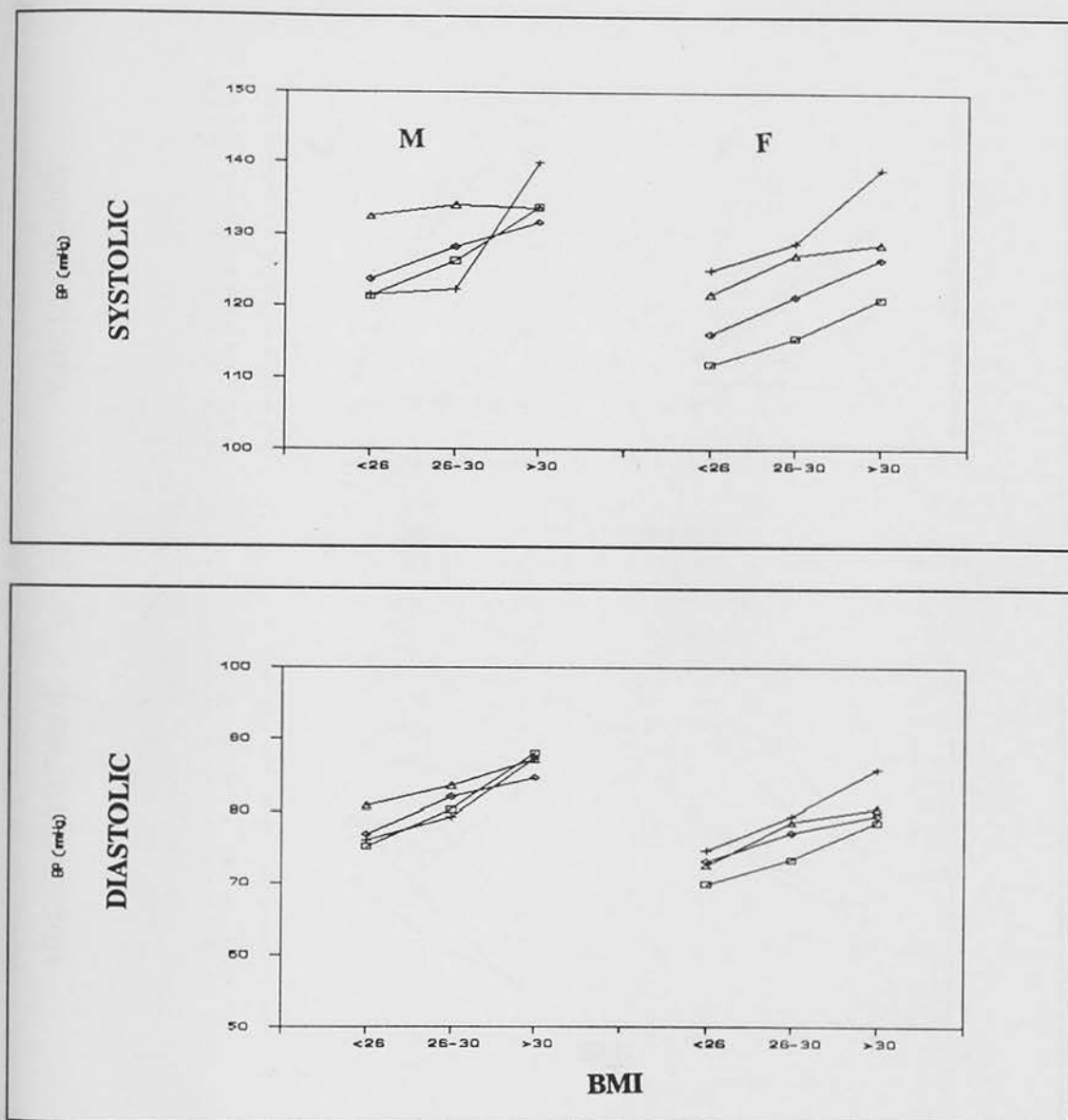


Fig.6.8 Mean systolic and diastolic blood pressure levels by categories of BMI

□ Keewatin + Chukotka ◇ Manitoba △ Indians

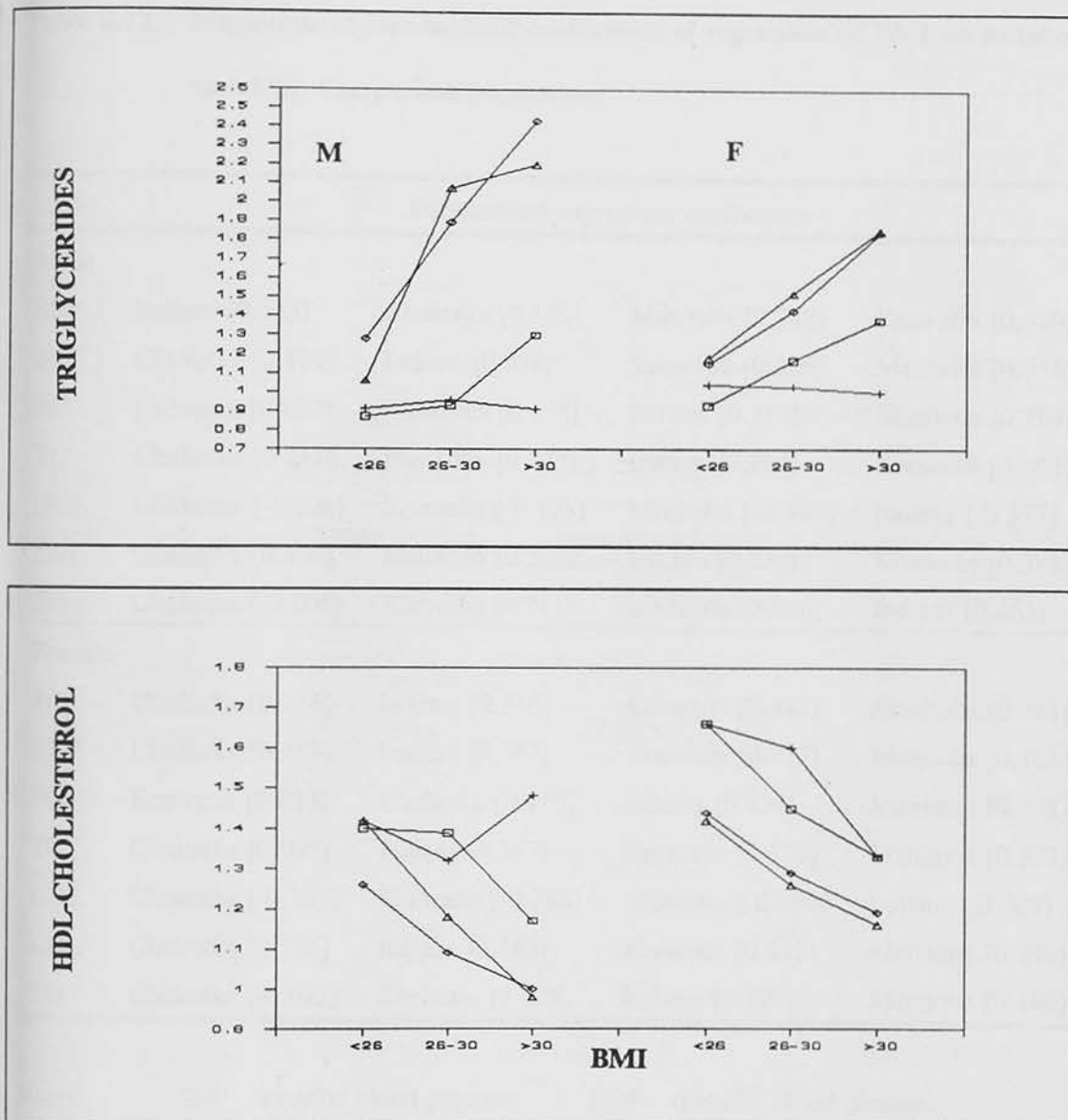


Fig.6.9 Mean triglycerides and HDL-cholesterol levels by categories of BMI

□ Keewatin + Chukotka ◇ Manitoba Δ Indians

Table 6.12 Magnitude of standardized coefficients of regression of BMI on metabolic variables: Comparison populations

DV		Standardized regression coefficients		
Male				
SBP	Indians [0.133]	Chukotka [0.153]	Manitoba [0.298]	Keewatin [0.310]
DBP	Chukotka [0.187]	Indians [0.256]	Keewatin [0.326]	Manitoba [0.356]
FG	Keewatin [0.027]	Chukotka [0.138]	Indians [0.212]	Manitoba [0.264]
TC	Chukotka [0.041]	Manitoba [0.223]	Indians [0.289]	Keewatin [0.362]
HDL	Chukotka [-0.106]	Keewatin [-0.125]	Manitoba [-0.306]	Indians [-0.397]
LDL	Chukotka [0.104]	Manitoba [0.208]	Indians [0.258]	Keewatin [0.388]
TG	Chukotka [-0.005]	Keewatin [0.241]	Manitoba [0.386]	Indians [0.463]
Female				
SBP	Chukotka [0.216]	Indians [0.316]	Keewatin [0.382]	Manitoba [0.391]
DBP	Chukotka [0.359]	Indians [0.367]	Keewatin [0.377]	Manitoba [0.401]
FG	Keewatin [0.031]	Chukotka [0.110]	Indians [0.224]	Manitoba [0.301]
TC	Chukotka [0.095]	Indians [0.167]	Keewatin [0.179]	Manitoba [0.237]
HDL	Chukotka [-0.183]	Keewatin [-0.284]	Manitoba [-0.296]	Indians [-0.304]
LDL	Chukotka [0.136]	Indians [0.162]	Keewatin [0.213]	Manitoba [0.248]
TG	Chukotka [-0.033]	Keewatin [0.375]	Indians [0.399]	Manitoba [0.409]

Notes: SBP systolic blood pressure DBP diastolic blood pressure
 FG fasting glucose TC total cholesterol
 HDL HDL-cholesterol LDL LDL-cholesterol
 TG triglycerides

The behaviour of the Manitoba sample, derived from a "typically" North American, predominantly Caucasian population, is similar to that reported from other studies in the literature [see the reviews by Björntorp (1988) and Kissebah and Peiris (1989)]. Overall, with increasing level of obesity, there is a corresponding change in various metabolic markers - increase in BP, FG, TC, LDL and TG and decrease in HDL. In general the Cree and Ojibwa Indians follow this pattern. The Inuit in the Keewatin, and their fellow Arctic dwellers the Eskimo/Chukchi in Chukotka, differ significantly in not showing any effect of increasing obesity on glucose (both fasting and 2-hour) levels. Even when an obesity-induced change is observed, particularly with respect to lipids, the size of the impact also appears to be less than that observed in the Manitoba and Indian samples.

Unfortunately fasting insulin levels were not collected in the Manitoba and Indian samples. However, it is well documented in the clinical literature that plasma insulin correlates with various measures of obesity in studies involving Europeans, White Americans, and Pima Indians [see review in Felber et al 1993:163ff]. Fasting insulin was measured only in a subsample of the Keewatin sample ($n=118$). On analysis of covariance, the age-adjusted mean FIns increases at higher quartiles of various obesity measures, though not with statistical significance, attributable to the smaller sample size (Table 6.10). On stepwise multiple regression, however, some measures of obesity do predict FIns (Table 6.11). A similar situation applies in the Chukotka sample. Thus one can conclude that among the Inuit, obesity is associated with high fasting insulin levels, as have been observed in other populations.

CHAPTER SEVEN CONCLUSIONS AND SYNTHESIS

In Chapter One, three questions are posed:

- (1) Is obesity prevalent among Inuit today and has it significantly increased?
- (2) Is such obesity as occurs metabolically "harmful"?
- (3) What are the evolutionary implications of the emergence of obesity and associated diseases in this Arctic population?

In this final chapter, an attempt will be made to provide answers to these questions, based on the data presented in the previous chapter, interpreted in the light of existing knowledge available from the published literature.

First of all, the main findings from the Keewatin Study and studies from the three comparison populations are recapitulated.

7.1 Epidemiological Patterns

The results presented in Chapter Six relating to the prevalence and determinants of obesity can be summarized as follows:

- The mean BMI among adult Inuit falls within the ranges reported in the published literature for most developed countries, is higher than those of transitional societies in Asia and Africa and unacculturated Amazonian Indians, but below those of some populations characterized by extreme prevalence of diabetes such as the Pima Indians and the Nauruans in the Pacific. The international comparison of Inuit (p.154-155) is illustrated in Fig.7.1-7.2, based on data from the MONICA and INTERSALT studies.

[Note that the former is composed primarily of Europeans and Asians, while the latter includes several "traditional" societies in various stages of sociocultural transition, eg. Amazonian Indians in Brazil and the Masai of Kenya].

- The proportion of Inuit women with BMI exceeding 26 or 30 is higher than that for a representative sample of all women in Manitoba in all age groups; among men, the proportion is similar in the two populations (p.160).
- The Inuit mean triceps and subscapular skinfold thicknesses generally fall between the 50th and 75th percentile of U.S. national standards (p.154).
- The distribution of the waist-hip ratio, used as a measure of central fat patterning, is lower in Inuit men, and higher in Inuit women, than the general population of Manitoba (p.155).
- In general, the Keewatin Inuit resembles the Chukotka Native (Chukchi and Eskimo) but differs from the Cree-Ojibwa Indians, who tend to be the most obese (both overall and central) of the comparison populations (p.155).
- The current Inuit BMI values do not differ substantially from those reported in the historical literature (p.47-50), whereas skinfold values have increased substantially from the 1960s and early 1970s (p.153).

At this point, Question (1) "*Is obesity prevalent among Inuit today?*" can be answered.

Judged by both BMI and skinfolds, obesity among the Inuit in the Keewatin region is as prevalent as it is in the general North American population. At least for skinfolds, there has been an increase over the past two or three decades. Obesity is more prevalent among women, among whom there is also a higher degree of central fat patterning.

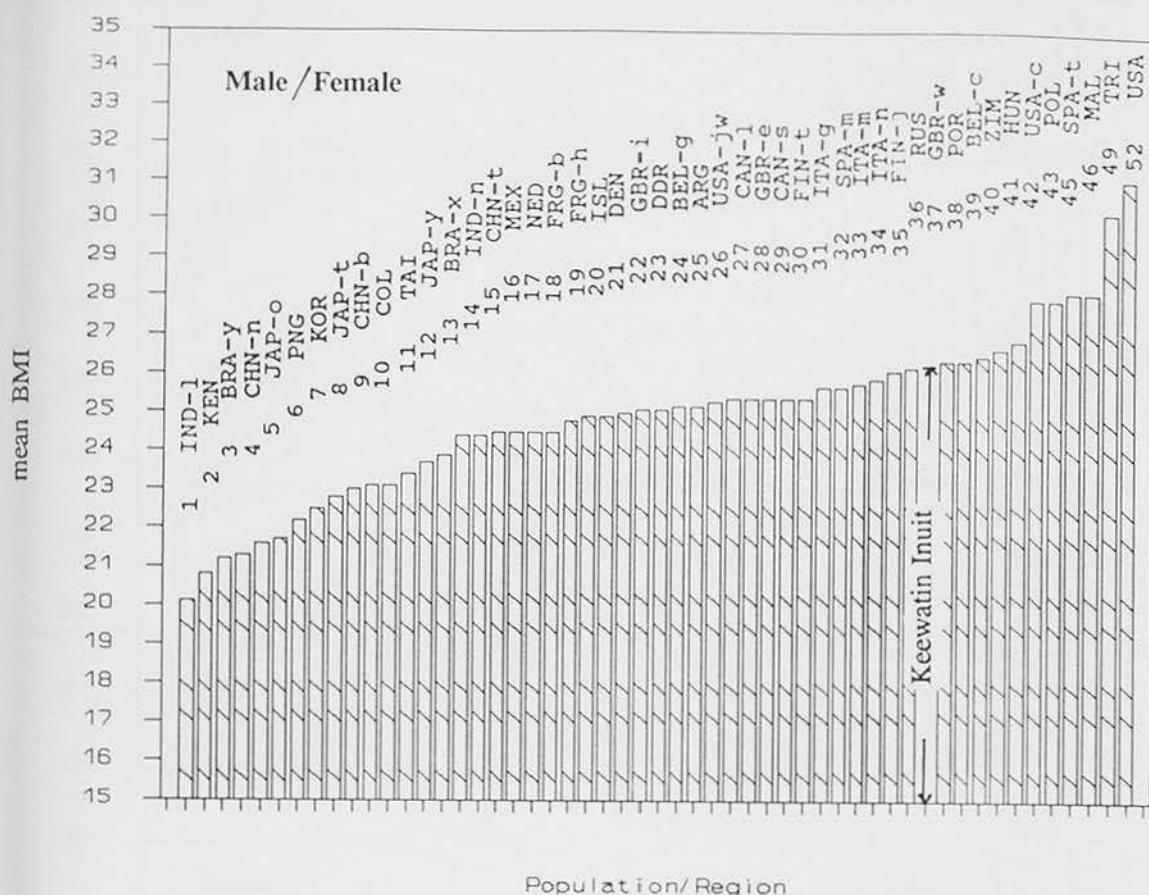


Fig.7.1 Relative ranking of body mass index of Keewatin Inuit among populations in INTERSALT STUDY

Key: Code: Population (mean BMI)

1 IND-l: Ladakh, India (20.1); 2 KEN: Masai, Kenya (20.8); 3 BRA-y: Yanomano, Brazil (21.2); 4 CHN-n: Nanning, China (21.3); 5 JAP-o: Osaka, Japan (21.6); 6 PNG: Papua-New Guinea (21.7); 7 KOR: South Korea (22.2); 8 JAP-t: Tochigi, Japan (22.5); 9 CHN-b: Beijing, China (22.8); 10 COL: Colombia (23.0); 11 TAI: Taiwan (23.1); 12 JAP-y: Toyoma, Japan (23.1); 13 BRA-x: Xingu, Brazil (23.4); 14 IND-n: New Delhi, India (23.7); 15 CHN-t: Tianjin, China (23.9); 16 MEX: Mexico (24.4); 17 NED: Netherlands (24.4); 18 FRG-b: Bernried, W. Germany (24.5); 19 FRG-h: Heidelberg, W. Germany (24.5); 20 ISL: Iceland (24.5); 21 DEN: Denmark (24.5); 22 GBR-i: Belfast, N. Ireland (24.8); 23 DDR: East Germany (24.9); 24 BEL-g: Ghent, Belgium (24.9); 25 ARG: Argentina (25.0); 26 USA-jw: Jackson (Whites), USA (25.1); 27 CAN-l: Labrador, Canada (25.1); 28 GBR-e: Birmingham, England (25.2); 29 CAN-s: St. John's, Newfoundland, Canada (25.2); 30 FIN-t: Turku, Finland (25.3); 31 ITA-g: Gubbio, Italy (25.4); 32 SPA-m: Manreva, Spain (25.4); 33 ITA-m: Mirano, Italy (25.4); 34 ITA-n: Naples, Italy (25.4); 35 FIN-j: Joensuu, Finland (25.4); 36 RUS: Russia (25.7); 37 GBR-w: South Wales (25.7); 38 POR: Portugal (25.8); 39 BEL-c: Charlevoix, Belgium (25.9); 40 ZIM: Zimbabwe (26.1); 41 HUN: Hungary (26.2); 42 USA-c: Chicago, USA (26.4); 43 POL-k: Krakow, Poland (26.4); 44 POL-w: Warsaw, Poland (26.5); 45 SPA-t: Torrejon, Spain (26.7); 46 MAL: Malta (26.9); 47 ITA-b: Bassiano, Italy (28.0); 48 USA-jb: Jackson (Blacks), USA (28.0); 49 TRI: Trinidad and Tobago (28.2); 50 USA-gw: Goodman (Whites), USA (28.2); 51 USA-gb: Goodman (Blacks), USA (30.3); 52 USA-h: Hawaii, USA (31.2)

median BMI

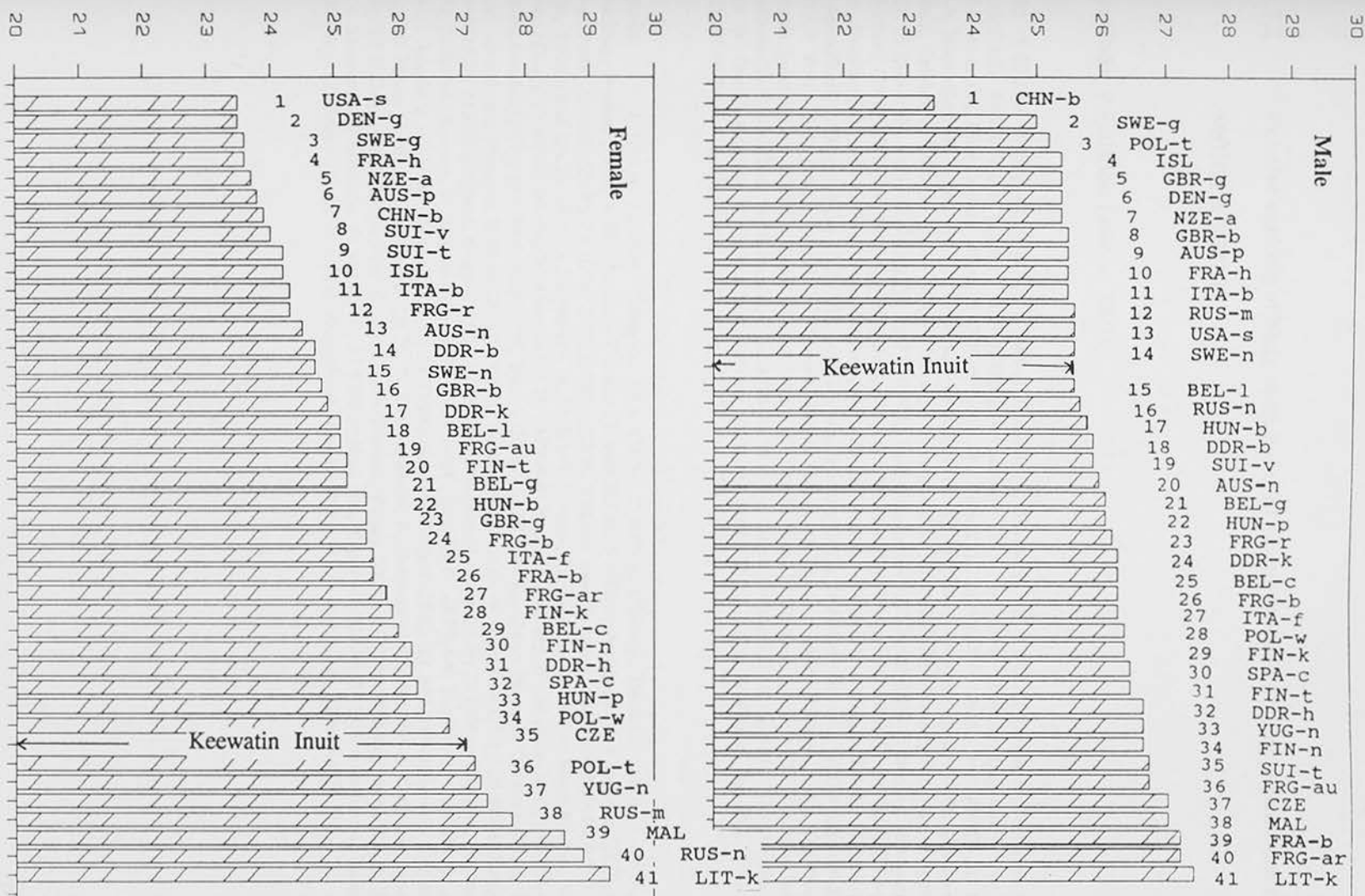


Fig.7.2 **Relative ranking of body mass index of Keewatin Inuit among populations in**
MONICA STUDY

Key: Code: Population (median BMI)

Male 1 CHN-b: Beijing, China (23.4); 2 SWE-g: Gothenburg, Sweden (25.0); 3 POL-t: Tarnobrzeg Voivodship, Poland (25.2); 4 ISL: Iceland (25.4); 5 GBR-g: Glasgow, Scotland (25.4); 6 DEN-g: Glostrup, Denmark (25.4); 7 NZE-a: Auckland, New Zealand (25.4); 8 GBR-b: Belfast, N. Ireland (25.5); 9 AUS-p: Perth, Australia (25.5); 10 FRA-h: Haute-Garonne, France (25.5); 11 ITA-b: Brianza, Italy (25.5); 12 RUS-m: Moscow, Russia (25.6); 13 USA-s: Stanford, California, USA (25.6); 14 SWE-n: Northern Sweden (25.6); 15 BEL-l: Luxembourg Province, Belgium (25.6); 16 RUS-n: Novosibirsk, Russia (25.7); 17 HUN-b: Budapest, Hungary (25.8); 18 DDR-b: Berlin-Lichtenberg, E. Germany (25.9); 19 SUI-v: Vaud-Fribourg, Switzerland (25.9); 20 AUS-n: Newcastle, Australia (26.0); 21 BEL-g: Ghent, Belgium (26.1); 22 HUN-p: Pecs, Hungary (26.1); 23 FRG-r: Rhein-Neckar, W. Germany (26.2); 24 DDR-k: Karl-Marx-Stadt, E. Germany (26.3); 25 BEL-c: Charlevoix, Belgium (26.3); 26 FRG-b: Bremen, W. Germany (26.3); 27 ITA-f: Friuli, Italy (26.3); 28 POL-w: Warsaw, Poland (26.4); 29 FIN-k: Kuopio, Finland (26.4); 30 SPA-c: Catalonia, Spain (26.5); 31 FIN-t: Turku, Finland (26.5); 32 DDR-h: Halle, E. Germany (26.7); 33 YUG-n: Novi Sad, Yugoslavia (26.7); 34 FIN-n: North Karelia, Finland (26.7); 35 SUI-t: Ticino, Switzerland (26.8); 36 FRG-au: Augsburg urban, W. Germany (26.8); 37 CZE: Czechoslovakia (27.1); 38 MAL: Malta (27.1); 39 FRA-b: Bas-Rhin, France (27.3); 40 FRG-ar: Augsburg rural, W. Germany (27.3); 41 LIT-k: Kaunas, Lithuania (27.5)

Female 1 USA-s: Stanford, California, USA (23.5); 2 DEN-g: Glostrup, Denmark (23.5); 3 SWE-g: Gothenburg, Sweden (23.6); 4 FRA-h: Haute-Garonne, France (23.6); 5 NZE-a: Auckland, New Zealand (23.7); 6 AUS-p: Perth, Australia (23.8); 7 CHN-b: Beijing, China (23.9); 8 SUI-v: Vaud-Fribourg, Switzerland (24.0); 9 SUI-t: Ticino, Switzerland (24.2); 10 ISL: Iceland (24.2); 11 ITA-b: Brianza, Italy (24.3); 12 FRG-r: Rhein-Neckar, W. Germany (24.3); 13 AUS-n: Newcastle, Australia (24.5); 14 DDR-b: Berlin-Lichtenberg, E. Germany (24.7); 15 SWE-n: Northern Sweden (24.7); 16 GBR-b: Belfast, N. Ireland (24.8); 17 DDR-k: Karl-Marx-Stadt, E. Germany (24.9); 18 BEL-l: Luxembourg Province, Belgium (25.1); 19 FRG-au: Augsburg urban, W. Germany (25.1); 20 FIN-t: Turku, Finland (25.2); 21 BEL-g: Ghent, Belgium (25.2); 22 HUN-b: Budapest, Hungary (25.5); 23 GBR-g: Glasgow, Scotland (25.5); 24 FRG-b: Bremen, W. Germany (25.5); 25 ITA-f: Friuli, Italy (25.6); 26 FRA-b: Bas-Rhin, France (25.6); 27 FRG-ar: Augsburg rural, W. Germany (25.8); 28 FIN-k: Kuopio, Finland (25.9); 29 BEL-c: Charlevoix, Belgium (26.0); 30 FIN-n: North Karelia, Finland (26.2); 31 DDR-h: Halle, E. Germany (26.2); 32 SPA-c: Catalonia, Spain (26.3); 33 HUN-p: Pecs, Hungary (26.4); 34 POL-w: Warsaw, Poland (26.8); 35 CZE: Czechoslovakia (27.2); 36 POL-t: Tarnobrzeg Voivodship, Poland (27.3); 37 YUG-n: Novi Sad, Yugoslavia (27.4); 38 RUS-m: Moscow, Russia (27.8); 39 MAL: Malta (28.6); 40 RUS-n: Novosibirsk, Russia (28.9); 41 LIT-k: Kaunas, Lithuania (29.3)

How has this state of affairs arisen? Chapter Three provides a brief history of the Inuit since the time of contact with Europeans. Despite the introduction of various items of European material culture and a fur-based economy, the lifestyle of the Inuit remained relatively unchanged until the middle of the twentieth century. Since then, there has been a substantial shift from a physically highly demanding hunting, trapping and fishing life to a sedentary life in permanent settlements, engagement in the wage economy and dependence on government subsidies. While longitudinal data on changes in physical fitness are available from one community only, from the work in Igloodik by Shephard and colleagues (p.60-61), it would be reasonable to assume that similar changes also occurred elsewhere in the Arctic. While at the organism level obesity is considerably more complex than simply the excess of energy intake over expenditure, at the population level, the general prevalence of obesity can be expected to increase in tandem with a decline in the overall physical activity level.

Another major change that the Inuit have experienced is in the diet and nutritional status of the people. Again, viewed from a population perspective, "overnutrition" is a plausible cause of increasing obesity. There is ethnohistorical evidence of periodic starvation associated with hunting failures (p.35), and as recently as the 1940s, malnutrition was widespread and an important factor in the prevalence and severity of many infectious diseases. With an assured supply of food, and a shift from land and marine-based food sources to store-bought, imported foods, the dietary pattern of the Inuit underwent substantial changes. Schaefer has documented such changes in various communities during the 1950s to 1970s (p.62-63), particularly in the proportion of energy derived from carbohydrates in general and refined sugar in particular. The rampant dental caries among the young attest to the intensity of such dietary change. While it

would be difficult to pinpoint a specific nutrient as responsible for the development of obesity, and sugar is by no means even the chief culprit, its use and distribution can be regarded as a marker of the overall process of dietary change. While the biochemical relationship between marine mammal and fish-based fatty acids, serum lipids, and obesity is complex, a decline in the diet of "country foods" may also operate in reducing the relative protection from cardiovascular diseases and diabetes that the Inuit have enjoyed until relatively recently.

Although not the focus of this dissertation, we did collect food frequency data in the Keewatin Study. At the time of the survey, more than half of the adults ate meat from the land (eg. caribou) on a daily basis and about 50% ate locally caught fish (eg. Arctic char) at least weekly. While sea mammals (eg. seals and walrus) were not available in all communities, 20% were able to obtain such meat more than once a week. On the other hand, "junk" snacks such as chocolate bars, cookies and potato crisps were eaten by more than half of the adults at least once a week. Carbonated drinks were consumed twice or more per day by 28% of the adults. Thus the picture at the beginning of the 1990s in the Keewatin Region is one of dietary transition, although it is not simply a matter of substitution of "modern" for "traditional" foods, but more like one of addition of new food items.

The availability of the Keewatin Survey data can further our understanding of the development of obesity among modern Inuit. By asking "within this population, who are more likely to be obese?" one can identify various sociocultural determinants of obesity. In Chapter Six, it was reported that:

- Mean BMI, waist and hip girths, and subscapular and triceps skinfolds differ significantly between different categories of such sociocultural variables as ethnic admixture, marital

status, education level, income group, employment, fluency in the Inuit language, time spent "on the land", physical activity and smoking status, after adjustment for age only.

- On multivariate analysis, age, education and smoking status are consistently identified as independent predictors of various obesity indices. Non-smoking women are more obese than current smokers, regardless of the obesity measure used. Women with only primary education have higher waist and hip circumference, waist-hip ratio, and triceps skinfolds.
- Among men, age and education are independently predictive of BMI. However, it is better educated men (those with some secondary education) are more likely to be obese, a situation which is the reverse of that seen among women. Income is positively correlated with subscapular skinfold. Smoking is not associated with any obesity index in men.
- Inuit men with some non-Inuit admixture have higher triceps skinfold than the unadmixed. Two measures of "acculturation" are also independent predictors of triceps skinfold but in opposite direction: those who infrequently spend time "on the land" pursuing traditional activities (hence more "acculturated") and those who are fluent in the Inuit language (hence less "acculturated") are more likely to have higher triceps skinfold.

Some of these findings, such as the positive association of obesity with increasing age and non-smoking status, are well-known and frequently found in many population surveys. The sexual difference with regards to the effect of education as well as the conflicting observations relating to measures of acculturation cannot be readily explained on the basis of the data. Note that in the multiple regression models, income, employment status and physical activity have

already been controlled for. One may perhaps seek an explanation in the different sex roles and their associated stress levels in a rapidly acculturating and modernizing society. Such data are more fruitfully to be obtained from qualitative, ethnographic research than quantitative data derived from relatively "blunt" survey instruments. Nevertheless, these results do indicate that obesity is not evenly distributed among the population, but depends on the presence or absence of various behavioural and lifestyle attributes.

Are obesity criteria based on studies in other populations applicable to the Inuit? Such a question cannot be readily answered without a long term prospective cohort study. However, even using our cross-sectional data, one can compare "obese" and "non-obese" Inuit in terms of the associated metabolic effects. Thus, if Inuit who would otherwise be considered "obese" using current criteria in fact do not have the metabolic effects associated with obesity observed in most other populations, perhaps they should not be considered "obese". If obesity is to be considered a biological concept, rather than simply a statistical one dependent on the method and criteria of measurement, then the second of the three questions posed at the beginning of Chapter One is the more meaningful one.

7.2 Metabolic Pathways

By looking at the effect of obesity on such physiological and biochemical markers as blood pressure, glucose, insulin and lipids, it is possible to address Question (2): *Is such obesity as occurs among the Inuit metabolically "harmful"?* In Chapter Six, the following results were presented:

- Comparing the three risk categories of BMI (<25 , 26-30 and >30), and adjusting for age, significant differences are found in systolic and diastolic blood pressure in both sexes, total cholesterol, LDL and triglycerides in men, and HDL and triglycerides in women. No differences are found in mean fasting and hour-2 post-load glucose and fasting insulin.
- Comparing "high" and "low" waist-hip ratios (cut-off points at 0.9 for men and 0.8 for women), and adjusting for both age and BMI, reveals no significant differences in any of the metabolic markers in either sex.
- Comparing quartiles of BMI, the two circumferences and the two skinfolds, and adjusting for age, shows a consistent pattern of an increasing trend in blood pressure and one or more of the lipids but no significant change in glucose or insulin.
- Using multiple regression analyses, various combinations of obesity indices are found to correlate with blood pressure and one or more of the lipids but not with glucose. Some obesity indices, however, do predict fasting insulin.
- In the presence of BMI (an "overall" measure), WHR (a "centrality" measure) rarely emerges as an independent predictor. When only triceps and subscapular skinfolds, or waist and hip circumference are present in a model, the more "central" indicator (ie. subscapular skinfold and waist circumference) tends to be the stronger determinant of metabolic effects.
- The relationship between obesity and metabolic effects in the four populations differ in significant respects. The pattern among Chukotka Natives is very similar to that seen in the Keewatin Inuit. The two Arctic groups, however, differ from the general population

of Manitoba and subarctic Cree-Ojibwa Indians. Among the latter two groups, increasing obesity tends to be associated with higher levels of fasting glucose [2hG and FIn were not measured].

- Even though there is generally an increasing trend in blood pressure and lipids with obesity in all four populations, the magnitude of response also differs, particularly with respect to triglycerides and HDL-cholesterol. At each level of obesity, Manitobans and Indians have higher mean triglycerides and lower mean HDL levels than the Inuit and Chukchi. This pattern is seen regardless of whether categories of BMI, skinfolds or circumferences are being compared.

These results indicate that there are significant and interesting differences in the metabolic effects of obesity between the Inuit and non-Inuit populations. The answer to Question (2) is then: obesity appears to be less "harmful", if "harm" is defined as an unfavourable cardiovascular risk factor profile. How such observations fit (or not fit) with existing notions of the metabolic basis of obesity and associated diseases is explored in the following section. Alternatively the measures of obesity are unsatisfactory when applied to the Inuit.

In Chapter Two the current understanding of the etiology and pathogenesis of obesity and its associated diseases was reviewed. It was pointed out that **insulin resistance** is increasingly seen as the key metabolic defect, if not causally related to, at least closely associated with, obesity. Insulin resistance is a constellation of metabolic processes manifested by various plasma markers, among which hyperinsulinaemia is the most characteristic. The differential effect of obesity on glucose, blood pressure and lipids in Inuit vis à vis non-Inuit suggests that perhaps the Inuit, for both genetic and non-genetic reasons, do not possess the entire "package". A

population survey such as the Keewatin Study obviously is not designed to examine underlying mechanisms at the cellular and molecular levels. However, a review of the key metabolic pathways involved in obesity and insulin resistance may indicate possible site(s) where population differences can occur and account for the observed pattern.

The metabolic patterns associated with obesity observed in this study bear some similarity to older published reports in the western NWT and Alaska, although direct comparability is difficult due to the different statistical approaches and the method of data presentation. In their study of hospitalized Inuit, Schaefer et al (1972) compared the oral glucose tolerance test response across three categories of sum of three skinfolds and found no difference in either the total glucose area or total insulin area. In Mouratoff and Scott's study (1973) they used as an index of obesity "differential weight", defined as the excess weight over a white American standard of the same age, sex and height. Fasting blood glucose was found to correlate with differential weight in men but not women while no correlation was found with the 2-hour values. Of interest is Szathmary's study on the Dogrib Indians, who belong to the Athapaskan language family believed to be genetically closer to the Inuit than to other North American Indians. While the study dealt specifically with the effect of carbohydrate intake on insulin response, it was reported that BMI was an independent predictor of fasting insulin but not 2-hour glucose, with carbohydrate intake, age and sex adjusted for (Szathmary 1989).

Any explanation for a distinctively Inuit metabolism must take into account the unique Inuit diet which has been in place for thousands of years until relatively recently. The traditional Inuit diet, in terms of sources of energy, is composed predominantly of proteins and fats, with carbohydrates playing an insignificant role. [While tropical hunters also consume a high level

of proteins, they engaged much more in foraging for plant foods]. This key feature means that the metabolic mechanisms in place to handle the post-prandial upsurge in glucose - insulin binding, glucose uptake, glucose transport - would be much less active. The main source of glucose, which the brain and erythrocytes cannot do without, must be derived endogenously through gluconeogenesis in the liver. The substrates are amino acids from proteolysis and glycerol from lipolysis, the availability of which is largely dependent on dietary intake of proteins and fats.

This study shows that obesity is associated with some degree of hyperinsulinaemia (which can be interpreted as a manifestation of insulin resistance) but not at all with fasting or post-load hyperglycaemia, a situation at variance with that seen in whites and Indians. One could speculate that for Inuit, the pathologic progression from insulin resistance to impaired glucose tolerance is not as well established as in other populations .

Insulin resistance has two "divisions": one involving glucose disposal and the other lipid oxidation. Studies in Pima Indians have shown that glucose disposal is impaired in obese subjects whereas insulin's inhibitory action on lipolysis is little affected (Howard et al 1991). It is possible that among the Inuit the situation is reversed, ie. glucose disposal is not impaired (thus accounting for the absence of hyperglycaemia accompanying obesity), whereas increased lipid oxidation (due to decreased inhibition of lipolysis) constitutes the mainstay of insulin resistance. There is an overall reduction in the impact of IR. This would account for the rise in plasma lipids with increasing obesity which is observed in the Inuit, although to a much reduced extent. [Note that the measurement of FFA in the Keewatin Study would have been extremely

informative. The only study which measured FFA in Arctic Eskimos is from Point Hope, Alaska (Ho et al 1972), which showed an average level higher than in the American population].

A study of non-diabetic non-obese first-degree relatives of NIDDM patients in Finland found that glucose storage (ie. glycogen synthesis) is impaired but not oxidation of glucose or lipids (Eriksson et al 1991), thus suggesting that some aspects of IR manifest early and may reflect a genetic defect. In the Pima, insulin action has been found to be familial, independent of age, sex and obesity (Knowler et al 1990). There are several potential candidates for the genetic defect(s) in insulin resistance, including the insulin receptor, various phosphatases relating to insulin action, glycogen synthase and the glucose transporters (Häring and Mehnert 1993). If glucose disposal is not impaired in Inuit with insulin resistance, one can speculate that the frequency of such genetic defects may have been very low in the Inuit.

Another observation from the Keewatin Study is the lower prevalence of central fat patterning in men and higher prevalence in women, compared to whites and Indians. The central fat that is present among the Inuit does not appear to have an independent effect on any of the metabolic markers over and above that attributable to overall obesity. The reduced effect of obesity on plasma lipids between Inuit and non-Inuit is seen when BMI, skinfolds, or either of waist or hips is compared. Since central fat is supposed to be more active metabolically (Björntorp 1988), particularly in the mobilization of fat stores, its relative "inertness" in the Inuit may explain the generally lower level of triglycerides, total and LDL-cholesterol (and the opposite in HDL) at each category of obesity. The high content of eicosapentaenoic acid (EPA) in the Inuit diet also exerts a lipid-lowering effect measurable in the plasma.

The high protein, low-carbohydrate diet of the Inuit can also be expected to have an effect on metabolism. Schaefer et al (1968, 1972) found that over 50% of hospitalized Inuit patients and about a quarter of a community sample had abnormally sustained post-prandial hyperglycaemia. However, these reverted to normal if the oral glucose tolerance test was preceded by a meat meal or if the glucose was administered intravenously. Schaefer hypothesized that Inuit could have a lower sensitivity of insulinogenic gut hormones to ingested glucose. Such gut hormones are now called incretins generically, one of which being the gastric inhibitory polypeptide (GIP). A variety of protein foods have also been found to have comparable potency to glucose in stimulating insulin secretion in diabetics but less so than glucose among normal subjects. The presence of protein products in the gut and not plasma amino acid level appears to stimulate GIP secretion (Gannon et al 1988).

It should be noted that Schaefer's observations have not been replicated or substantiated elsewhere in the Arctic (Bell et al 1973). The observation that healthy Inuit are unable to "handle" orally ingested glucose but can respond normally to intravenously delivered glucose is compatible with the finding in the Keewatin Study that, regardless of the degree of obesity, the disposal of glucose in the plasma is not impaired.

The glucose "malabsorption" problem resembles two other carbohydrate malabsorption syndromes which are well recognized to be more prevalent among Inuit than in other populations - lactose and sucrose intolerance (Bell et al 1973, Ellestad-Sayed et al 1978). These operate from different mechanisms, that of specific deficiency of the intracellular enzymes lactase and sucrase, rather than impaired hormonal action at the gut mucosa.

The similarity in blood pressure increase with obesity between Inuit and non-Inuit suggests that the mechanism may be quite separate from glucose and fat metabolism. The epidemiologic evidence linking hyperinsulinaemia and hypertension is quite consistent and it is believed that it is mediated through an increase in circulating epinephrine and norepinephrine and increase sodium reabsorption by distal renal tubules (Donahue et al 1990). In this respect, there may be little difference between Inuit and other populations.

7.3 Evolutionary Perspectives

It has been pointed out that throughout much of human history, obesity had never occurred as a common health problem. Rather, scarcity of food had acted as an agent of natural selection and was ubiquitous among preindustrial societies (Brown and Konner 1987). The widespread development of obesity is a relatively recent phenomenon, as part and parcel of the health consequences of "Westernization" (eg. Trowell and Burkitt 1981) and the last stage of the "epidemiologic transition" (Omran 1971). While a variety of studies on the development of obesity in "modernizing" populations have been done, the relationship is by no means consistent, which may reflect differences in the duration and pace of exposure and the specific types of environmental changes that have occurred (McGarvey et al 1989).

Modern man has been referred to as "stone agers in the fast lane". While still possessing the genetic make-up of the late Pleistocene hunter-gatherers, which has hardly changed in 10,000 years, modern man lives in a nutritional environment very unlike that to which early man has long adapted. The new dietary regime promotes chronic, degenerative diseases, which because of their late onset in life, have little effect on reproductive success (Eaton and Konner 1985,

Eaton et al 1988). Arguments such as these relied on both archaeological data and contemporary hunter-gatherers, with whom the Eskimo is usually included. [It has been pointed out that early hominids by no means lived in a nutritional nirvana - there were frequent deficiencies and food preparation methods may well have made eating a hazardous activity (Garn and Leonard 1989)].

Much of what we know about the human biology of the Inuit suggests that, for the Inuit to claim the distinction of having successfully adapted to one of the coldest and most inhospitable environments on earth, significant metabolic differences from the rest of humanity who remained in the temperate and warm climates could have evolved. Could obesity have played a part in this adaptation?

It is now generally agreed that hominid evolution originated in the tropical savannas of East Africa. The types of habitats proliferated subsequent to the outmigration from Africa, followed by biological diversification and cultural innovation. *Homo sapiens* by and large is still genetically adapted to a hot dry climate and still have only a rather narrow physiological response to climatic extremes, especially cold.

Of the several possible cold adaptation strategies available to the Inuit, such regulatory changes as increasing metabolic rate through exercise, shivering and restricting blood flow to the extremities are costly and impractical in the long run. Morphological changes through the deposition of fat are the only viable long-term adaptation for survival in the Arctic (Shephard 1991:246-7). Shephard suggested that adaptation favours the deposition of intraabdominal fat which stores quickly available fuel for heat production in response to cold-induced catecholamine stimulation, whereas subcutaneous fat's primary purpose is insulation (Shephard 1991:198). While subcutaneous fat offers effective insulation it has a low metabolism and limited blood

supply and is thus relatively inflexible when responding to increases in heat flux generated by physical activity. Shephard maintained that it is intraabdominal rather than subcutaneous fat that has traditionally been associated with the Inuit. However, this cannot be established since the only measurements available were skinfold thicknesses. From this study as well as Shephard's own data from Igloolik, we do know that subcutaneous skinfolds had increased over the last three decades. Furthermore currently the distribution of waist-to-hip ratio in Inuit men is lower than in other populations, and that the amount of central fat that is present seems to be less rather than more active metabolically. It is possible that the excessive central fat that is identified by a high WHR among the Inuit is predominantly subcutaneous rather than intraabdominal. In Bouchard's scheme (p.12), Inuit obesity may be predominantly type II rather than type III.

As pointed out earlier, obesity, or at least insulin resistance, has a large genetic component. The gene(s) for obesity conceivably could be very old and had been selected for among early hominids as they penetrated the temperate and subarctic biomes - where it might have been selected for because of its value as insulation and fuel storage. However, as human beings advanced into the Arctic, no amount of obesity could have been up to the task and technological and cultural innovations in clothing and shelter "saved the day". Even though there was no "need" for obesity it could have perpetuated in the population, particularly if the type of obesity was metabolically "inert" and its continued presence would not have been detrimental. An analogy can be drawn with the high prevalence of sucrose intolerance (sucrase deficiency) among the Inuit. In the absence of sugar in the traditional diet, the persistence of sucrase activity into adulthood would not have been selected for (Stinson 1992). The effect of sucrase deficiency

is essentially neutral and its original appearance and continuation in the population can be accounted for by genetic drift.

In most other populations obesity, especially central fat, can cause disease. That such obesity-related diseases have not been prominent among the Inuit (Chapter Three, esp. pp.89-93) lends support to the assertion earlier in this chapter (p.199) that the Inuit may be statistically but not biologically "obese". It is only recently that factors related to acculturation, particularly diet and physical activity, have emerged. Such factors could be considered as promoting factors which can "overcome" the metabolic protection that has existed for several thousand years.

Much of the discussion until now has focused on obesity and its metabolic effects as expressed in hypertension, diabetes and dyslipidaemia. It is also known that obesity has an impact on fertility. Studies by Frisch (1988) have shown that fat has a regulatory effect on female fecundity - excessive leanness (BMI <20) or obesity (>33) is associated with lower fecundity. A recent study on Dutch women undergoing artificial insemination in an infertility clinic showed that WHR is negatively associated with the probability of conception, and is a more important factor than age or BMI (Zaadstra et al 1993). Thus it would seem that excessive obesity, especially of the central type, could reduce reproductive fitness and be selected against. On the other hand, moderate obesity in women of childbearing age could protect the newborn and unborn against food shortages during pregnancy and lactation.

The study of obesity among the Inuit, and its metabolic differences with other populations, can shed some light into the still unsettled and controversial issues surrounding the peopling of the Americas. A low prevalence of diabetes has been demonstrated among Inuit on both sides of Bering Straits and the northern Athapaskans, relative to North American whites

and most of the so-called "Paleo-Indians" (Young, Szathmary et al 1990; Young, Schraer et al 1992). Can metabolic differentiation be used to determine the timing of the genetic differentiation of the major population groups and their separate migrations into the New World?

That diabetes has within the past four or five decades become so prevalent in many North American Indian populations has spawned various theories by geneticists and anthropologists. Neel first proposed the "thrifty genotype" hypothesis in 1962 and subsequently revised it in 1982. Neel noted that, for it to persist over time, the diabetic genotype must have conferred survival advantage under previously prevailing "feast or famine" environmental conditions. The thrifty gene enabled an individual to produce insulin rapidly in response to rising blood glucose levels (the "quick insulin trigger"), which facilitates the storage of glucose in the form of triglycerides in fat cells. In times of food scarcity, such individuals would have fat stores from which to derive energy. However, with sedentarization, the development of agricultural societies, and the assurance of a continuous and ample food supply, the quick insulin trigger would result in too much insulin, hyperglycaemia, obesity and diabetes.

Szathmary (1990) noted that the thrifty gene model assumed a nutritional environment in which carbohydrate intake exceeded daily energy requirements. This was not the situation when the ancestors of modern Native Americans first occupied the continent, where the diet was one based predominantly on meat and fat. Survival advantage in a low carbohydrate, arctic/subarctic environment was provided by having an efficient formation of endogenous glucose through gluconeogenesis, efficient use of free fatty acids to provide for the energy needs of non-glucose dependent tissues, and the efficient use of ketone bodies as fuel. Under such conditions, selection should have favoured individuals in whom gluconeogenesis and free fatty

acid release and use were enhanced. Phrased in another way and invoking the concept of insulin resistance, Szathmary's hypothesis is congruent with the conclusions arrived at in Section 7.2. One could say that insulin resistance - the component involving increased lipid oxidation but not the component involving reduced glucose disposal - would have been advantageous. While Neel did not detail the metabolic pathways involved in the "thrifty genotype", it is implied that during the "feast" phase, excess calories are stored as fat. Generally speaking, little glucose is turned into fat (and there is not much glucose of dietary origin anyway) and excess dietary fat is the main cause of increased fat stores. Furthermore, insulin secretion is not affected by plasma fatty acid levels (Felber et al 1993:33). While proteins in combination with glucose are potent stimulant of insulin secretion, this occurs in diabetics but not in normoglycaemic persons. Thus, with mostly proteins and fat in the diet during an Inuit "feast" there would not have been much of a "quick insulin trigger" to speak of.

Ritenbaugh and Gooby (1989) also emphasized the physiologic consequences of the northern hunting lifestyle: low carbohydrate availability, intermittent lipid storage, protein sufficiency, and high energy demands for activity and body warmth. With the transition to agriculturally-based subsistence systems and modern industrial societies, genetically controlled modifications could have occurred in several enzymatically mediated pathways in lipid and glucose metabolism.

Wendorf and Goldfine (1991) attempted to "validate" the thrifty genotype hypothesis with archaeological evidence for big-game hunting (primarily bison) among ancestral Indian groups who had migrated into temperate North America between 12,000 and 11,000 years ago. Despite the changes in climate, flora and fauna, these groups continued to practise the food gathering

strategy more suited to the arctic environment. They were thus subject to an unpredictable food supply, for which a thrifty genotype would offer some selective advantage. Subscribing to the theory that Asiatic peoples migrated to North America in three separate waves, these authors believed that the Na-Dene and Eskimos, who migrated around 10,000 and 5,000 years ago respectively, much later than the Paleo-Indians (20-25,000 years ago), did not develop the thrifty genotype. This accounted for their still much lower prevalence of diabetes.

Weiss and others (1984) noted the high prevalence and co-occurrence of gallbladder disease, obesity and diabetes in diverse Native American populations, and referred to them collectively as the "New World Syndrome". Their occurrence appears to be over and above what could be expected solely from "westernization". They postulated the existence of susceptibility genes involved in some fundamental aspect of lipid metabolism, which are expressed phenotypically in the constellation of diseases. Such genes arose because of selective advantage during or before the initial peopling of America. Three sets of conditions promote the spread of these genes throughout the small population at the Beringia corridor:

- (1) small, scattered, hunting-gathering demes;
- (2) population bottleneck, favourable to genetic drift and founder effect;
- (3) arduous environmental conditions.

Due to insufficient data, Weiss and colleagues did not include the Inuit among those susceptible to the New World Syndrome. However, if the Inuit were exempt, it would imply that these susceptibility genes must have arisen in North America before the arrival of Inuit forebears among the early Asiatic migrants.

Hales and Barker (1992), proposed a variation on the thrifty genotype theme. Called "thrifty phenotype", this hypothesis states that poor maternal-fetal and early infancy nutrition imposes nutritional "thrift" on the rapidly developing pancreas, resulting in reduced beta cell mass and islet function. As long as the individual persists in an undernourished state, there is not much need for insulin. However, a sudden shift to overnutrition exposes the pre-existing state of insulin deficiency, leading to diabetes. This is unlikely to have been the case among the Inuit, who were exposed to widespread under-nutrition in the pre-World War II years. There has been no diabetes epidemic among the cohort that was born during that period.

North American Indians are not the only population which has undergone an explosive increase in the prevalence of obesity and diabetes. The Nauruans in the Pacific (Zimmet et al 1990) and Australian Aborigines (O'Dea 1992) are other well-studied cases, often also used to furnish support to the thrifty genotype hypothesis. "Feast or famine" conditions are believed to have prevailed during the early peopling of the Pacific, which necessitated long sea voyages. Interestingly among the Nauruans, it has also been observed that, after an early explosive epidemic during the 1970s, the incidence of new cases of impaired glucose tolerance and diabetes among normal individuals has declined in the 1980s, even though there has been no change in the prevalence of behavioural risk factors (Dowse et al 1991). This phenomenon was attributed to the decreased fertility and increased mortality of diabetics. Also the earlier epidemic depleted the pool of susceptible individuals from the normal glucose-tolerant population, resulting in a fall in the frequency of the thrifty genotype.

The Inuit differ fundamentally from many other indigenous populations which have experienced widespread obesity and diabetes in recent years in their adaptation to cold and the

near-absence of carbohydrates in the diet. Of these, nutritional adaptation probably has the stronger impact on the pattern of health and disease. The concept of "selective insulin resistance" has been proposed for such groups as the Pima (Knowler et al 1983) and Australian Aborigines (O'Dea 1992). Basically, it is hypothesized that the glucoregulatory action of insulin is impaired while insulin's inhibitory action on lipolysis and stimulatory action on lipogenesis remain unaffected. [Refer to Fig.2.4, pp.18-19 for a schematic view of insulin action]. Hepatic gluconeogenesis is no longer suppressed by insulin whereas lipogenesis continues to be stimulated by insulin. The end result is excessive peripheral glucose and increased fat stores. Among the Inuit, it would appear that insulin resistance, if it occurs, operates in the opposite manner. That increasing body mass has no effect on fasting glucose or 2-hour glucose level indicates that the ability to dispose of glucose seems to be unimpaired. The reduced production of triglycerides in the liver and release into the plasma, manifested by a reduced response in plasma triglycerides to increasing obesity, indicates that lipogenesis promoted by insulin (and lipolysis inhibited by insulin) is affected.

The state of current research, which must range from broad evolutionary strokes to intracellular enzymatic processes, has not yet reached a stage where an all-encompassing theory can account for the observed metabolic differences between populations and throw light on the peopling of the New World. It is clear that the Inuit, as the most recent arrivals, must still hold the key to an improved understanding of microevolution, adaptation, and the distribution of diseases.

What can one expect of the future in terms of the people's health? Should the Inuit people be alarmed by the recent increase in obesity over a relatively short period? Is the current low

prevalence of diabetes and cardiovascular diseases likely to change? While the data from the Keewatin Study suggest that the obesity detected is metabolically less "harmful" than that seen in other populations, only a long-term prospective study can determine the true risk of future disease. On the other hand, the data also suggest that lifestyle and behavioural factors do affect the distribution of obesity within the population. Compared to many North American Indian groups, the Inuit are metabolically "different". If this is predominantly an effect of acculturation, it would suggest that Inuit are either not subjected to acculturation long enough, or the acculturation that they have been subjected to has not been severe enough. Alternatively the Inuit may be more "resistant" to the influence of acculturation. While genetic susceptibility may be invoked, the continuing (albeit much reduced) importance of the traditional diet and physical activity pattern may also be responsible.

While more research, as is customarily recommended at the end of a study, would help elucidate the problem further and add to the pool of scientific knowledge, the applied health scientist, with whom human biologists may claim solidarity, must also recommend courses of action based on incomplete data. It would only be prudent that one should not count on the genetic "protection" from the health consequences of obesity to be maintained long into the future. Enough is known about the important role of gene-environment interactions in myriad diseases that one would want to monitor and ameliorate the detrimental effects of rapid sociocultural change. While individual health promotion activities (eg. diet, physical activity) and broad socioeconomic strategies (eg. employment, income) may do some good in safeguarding the people's health, they certainly could do little harm.

BIBLIOGRAPHY

- Adamson JD, Moody JP, Peart AF et al. Poliomyelitis in the Arctic. *Can Med Assoc J* 1949;61:339-48.
- Alberti KG, Defronzo RA, Keen H, Zimmet P, eds. *International Textbook of Diabetes Mellitus*. New York: John Wiley; 1992.
- Alexseev VP. Anthropometry of Siberian peoples. In: Laughlin WS, Harper AB, eds. *The First Americans: Origins, Affinities, and Adaptations*. Stuttgart: Gustav Fischer; 1979:57-90.
- Alland A. *Adaptation in Cultural Evolution: An Approach to Medical Anthropology*. New York: Columbia University Press; 1970.
- Arima EY. Caribou Eskimos. In: Damas D, ed. *Arctic*. Washington, DC: Smithsonian Institution; 1984:447-62.
- Armstrong AR, Peart HE. A comparison between the behaviour of Eskimos and non-Eskimos to the administration of isoniazid. *Am Rev Resp Dis* 1960;81:588-94.
- Armstrong T. The administration of northern peoples: the USSR. In: Macdonald RS, ed. *The Arctic Frontier*. Toronto: University of Toronto Press; 1966:57-88.
- Arthaud JB. Causes of death in 339 Alaskan Eskimos as determined by autopsy. *Arch Pathol* 1970;90:433-8.
- Ashwell M, Cole TJ, Dixon AK. New insight into the anthropometric classification of fat distribution shown by computed tomography. *Br Med J* 1985;290:1692-4.
- Astakhova T, Rjabikov A, Astakhov V et al. Risk factors and chronic non-communicable diseases in Native residents and newcoming population of Chukotka. In: Postl BD, Gilbert P, Goodwill J et al., eds. *Circumpolar Health 90*. Winnipeg: University of Manitoba Press; 1991:408-9.
- Auger F, Jamison PL, Balslev-Jorgensen J et al. Anthropometry of circumpolar populations. In: Milan FA, ed. *The Human Biology of Circumpolar Populations*. Cambridge: Cambridge University Press; 1980:213-55.
- Baba S, Zimmet P, eds. *World Data Book of Obesity*. Amsterdam: Excerpta Medica, 1990.
- Bachorik PS, Walker RE, Virgil DG. High-density-lipoprotein cholesterol in heparin $MnCl_2$ supernates determined with the Dow enzymic method after precipitation of Mn^{+2} with HCO_3^- . *Clin Chem* 1984;30:839-42.

- Baikie M, Ratnam S, Bryant DG, Jong M, Bokhout M. Epidemiologic features of hepatitis B virus infection in northern Labrador. *Can Med Assoc J* 1989;141:791-5.
- Baker PT. The adaptive limits of human populations. *Man* 1984;19:1-14.
- Baker PT, Weiner JS, eds. *Biology of Human Adaptability*. Oxford: Clarendon Press; 1967.
- Balikei A. Female infanticide on the Arctic coast. *Man* 1967;2:615-25.
- Bang HO, Dyerberg J. Plasma lipids and lipoproteins in Greenlandic west coast Eskimos. *Acta Med Scand* 1972;192:85-94.
- Bang HO, Dyerberg J, Hjørne N. The composition of food consumed by Greenland Eskimos. *Acta Med Scand* 1976;200:69-73.
- Bang HO, Dyerberg J, Sinclair HM. The composition of the Eskimo food in northwestern Greenland. *Am J Clin Nutr* 1980;33:2657-61.
- Barrett-Connor E. Epidemiology, obesity, and non-insulin-dependent diabetes mellitus. *Epidemiol Rev* 1989;11:172-81.
- Bell R, Draper H, Bergan JG. Sucrose, lactose and glucose tolerance in northern Alaskan Eskimos. *Am J Clin Nutr* 1973;26:1185-90.
- Bell RR, Heller CA. Nutrition studies: an appraisal of the modern Alaskan Eskimo diet. In: Jamison PL, Zegura SL, Milan FA, eds. *Eskimos of Northwestern Alaska: A Biological Perspective*. Stroudsburg, PA: Dowden, Hutchinson and Ross; 1978:145-56.
- Bender TR, Jones TS, DeWitt WE et al. Salmonellosis associated with whale meat in an Eskimo community: serologic and bacteriologic methods as adjuncts to an epidemiologic investigation. *Am J Epidemiol* 1972;96:153-60.
- Birket-Smith K. *The Caribou Eskimo: Material and Social Life and Their Cultural Position*. Report of the 5th Thule Expedition, 1921-24, vol 5, parts 1 and 2. Copenhagen: 1929.
- Birket-Smith K. *The Eskimos*. London: Methuen; 1936. Trans from Danish by Calvert WE.
- Bjerager P, Kromann N, Thygesen K, Harvald B. Blood pressure in Greenland Eskimos. In: Harvald B, Hansen JP, eds. *Circumpolar Health 81*. Oulu, Finland: Nordic Council for Arctic Medical Research; 1982:317-20.
- Bjerregaard P. Housing standards, social group, and respiratory infections in children of Upernavik, Greenland. *Scand J Soc Med* 1983;11:107-11.

- Bjerregaard P. Fatal accidents in Greenland. *Arct Med Res* 1990;49:132-41.
- Bjerregaard P. Disease pattern in Greenland: studies on morbidity in Upernavik 1979-1980 and mortality in Greenland 1968-1985. *Arct Med Res* 1991;50(Suppl 4):1-62.
- Bjerregaard P, Dyerberg J. Mortality from ischemic heart disease and cerebrovascular disease in Greenland. *Int J Epidemiol* 1988;17:514-9.
- Björntorp P. The associations between obesity, adipose tissue distribution and disease. *Acta Med Scand* 1988;(Suppl 723):121-34.
- Björntorp P, Brodoff BN, eds. *Obesity*. Philadelphia: J.B. Lippincott; 1992.
- Björntorp P, Smith U, Lonnroth P, eds. *Health Implications of Regional Obesities*. *Acta Med Scand* 1988;(Suppl 723).
- Blackwood L. Alaska Native fertility trends, 1950-1978. *Demography* 1981;18:173-9.
- Boas F. *The Central Eskimo*. 6th Annu Rep Am Bur Ethnol 1884-1885. Washington, DC: Government Printing Office; 1888:399-669. [Reprinted by the University of Nebraska Press, Lincoln, 1964].
- Boas F. The Eskimos of Baffin Land and Hudson Bay. *Bull Am Mus Nat Hist* 1901;15:4-370.
- Bone RM. *The Geography of the Canadian North: Issues and Challenges*. Toronto: Oxford University Press; 1992.
- Booyens J, Louwrens CC, Katzeff IE. The Eskimo diet: prophylactic effects ascribed to the balanced presence of natural cis unsaturated fatty acids and to the absence of unnatural trans and cis isomers of unsaturated fatty acids. *Med Hypotheses* 1986;21:387-408.
- Borre K. Seal blood, Inuit blood, and diet: a biocultural model of physiology and cultural identity. *Med Anthropol Q* 1991;5:48-62.
- Bouchard C. Current understanding of the etiology of obesity: genetic and nongenetic factors. *Am J Clin Nutr* 1991;53:1561S-5S.
- Bouchard C, Pérusse L. Heredity and body fat. *Annu Rev Nutr* 1988;8:259-77.
- Bouchard C, Pérusse L. Genetics of obesity. *Annu Rev Nutr* 1993;13:337-54.
- Bradley PJ. Is obesity an advantageous adaptation? *Int J Obesity* 1982;6:43-52.
- Bray GA. Classification and evaluation of the obesities. *Med Clin North Am* 1989;73:161-84.

- Brody H. *The People's Land: Eskimos and Whites in the Eastern Arctic*. Harmondsworth, UK: Penguin; 1975.
- Broudy DW, May PA. Demographic and epidemiologic transition among the Navajo Indians. *Soc Biol* 1983;30:1-16.
- Broussard BA, Johnson A, Himes JH et al. Prevalence of obesity in American Indians and Alaska Natives. *Am J Clin Nutr* 1991;53:153S-42S.
- Brown PJ, Konner M. An anthropological perspective on obesity. *Ann NY Acad Sci* 1987;499:29-46.
- Burch ES. The Caribou Inuit. In: Morrison RB, Wilson CR, eds. *Native Peoples: The Canadian Experience*. Toronto: McClelland and Stewart; 1986:106-33.
- Canada, Department of National Health and Welfare. *Nutrition Canada: Food Consumption Patterns Report*. Ottawa: DNHW; 1975.
- Canada, Department of National Health and Welfare. *Nutrition Canada: Eskimo Survey Report*. Ottawa: DNHW; 1975.
- Canada, Department of National Health and Welfare. *Nutrition Canada: Anthropometry Report*. Ottawa: DNHW; 1980.
- Canada, Department of National Health and Welfare. *The Health of Canadians: Report of the Canada Health Survey*. Ottawa: DNHW; 1981.
- Canada, Department of National Health and Welfare. *Guide for Anthropometric Classification of Canadian Adults for Use in Nutritional Assessment*. Ottawa: DNHW; 1983.
- Carvalho JM, Baruzzi RG, Howard PF et al. Blood pressure in four remote populations in the INTERSALT Study. *Hypertension* 1989;14:238-46.
- Cavalli-Sforza LL, Piazza A, Menozzi P, Mountain JL. Reconstruction of human evolution: bringing together genetic, archaeological and linguistic data. *Proc Nat Acad Sci* 1988;85:6002-6.
- Chakraborty R, Szathmary EJ, eds. *Diseases of Complex Etiology in Small Populations: Ethnic Differences and Research Approaches*. New York: Alan R. Liss; 1985.
- Chapman M. Infanticide and fertility among Eskimos: a computer simulation. *Am J Phys Anthropol* 1980;53:317-27.
- Chichlo B. Les Nevuqaghmiit ou la fin d'une ethnie. *Études Inuit Studies* 1981;5(2):29-47.

- Chown B, Lewis M. The blood group genes of the Copper Eskimo. *Am J Phys Anthropol* 1959;17:13-8.
- Chown B, Lewis M. The blood group and secretor genes of the Eskimo on Southampton Island. *Bull Nat Mus Can* 1960;180(pt i):181-90.
- Ciba Foundation Symposium. *Health and Disease in Tribal Societies*. Amsterdam: Excerpta Medica; 1977.
- Cohen JB, Baskett TF. Sterilization patterns in a northern Canadian population. *Can J Public Health* 1978;69:222-4.
- Colbert MJ, Mann GV, Hursh LM. Nutrition studies: clinical observations on nutritional health. In: Jamison PL, Zegura SL, Milan FA, eds. *Eskimos of Northwestern Alaska*. Stroudsburg, PA: Dowden; 1978:162-73.
- Condon RG. Inuit natality rhythms in the central Canadian Arctic. *J Biosoc Sci* 1982;14:167-77.
- Connelly PW, MacLean DR, Horlick L et al. Plasma lipids and lipoproteins and the prevalence of risk for coronary heart disease in Canadian adults. *Can Med Assoc J* 1992;146:1977-88.
- Corcoran AC, Rabinowitch IM. A study of the blood lipids and blood proteins in Canadian Eastern Arctic Eskimos. *Biochem J* 1937;31:343-8.
- Crawford MH, Duggirala R. Digital dermatoglyphic patterns of Eskimo and Amerindian populations: relationship between geographic, dermatoglyphic, genetic, and linguistic distances. *Hum Biol* 1992;64:683-704.
- Crawford MH, Enciso VB. Population structure of circumpolar groups of Siberia, Alaska, Canada and Greenland. In: Crawford MH, Mielke JH, eds. *Current Developments in Anthropological Genetics*. New York: Plenum Press; 1982;vol 2:51-91.
- Crile GW, Quiring DP. Indian and Eskimo metabolism. *J Nutr* 1939;18:361-8.
- Curtis MA, Bylund G. Diphyllbothriasis: fish tapeworm disease in the circumpolar North. *Arct Med Res* 1991;50:18-25.
- Dahlberg AA. Craniofacial studies. In: Milan FA, ed. *The Human Biology of Circumpolar Populations*. Cambridge: Cambridge University Press; 1980:169-192.
- Damas D, ed. *Arctic. Handbook of North American Indians*, Vol 5. Washington, DC: Smithsonian Institution; 1984.

- Damon A. *Physiological Anthropology*. New York: Oxford University Press; 1975.
- Davidson M, Schraer CD, Parkinson AJ et al. Invasive pneumococcal disease in an Alaska Native population, 1980 through 1986. *JAMA* 1989;261:715-8.
- Davies LE, Hanson S. The Eskimos of the Northwest Passage: a survey of dietary composition and various blood and metabolic measurements. *Can Med Assoc J* 1965;92:205-16.
- Dean HJ, Carson J. Insulin-dependent diabetes mellitus in an Inuit child. *Can Med Assoc J* 1989;140:527-8.
- DeFronzo RA, Bonadonna RC, Ferrannini E. Pathogenesis of NIDDM: a balanced overview. *Diabetes Care* 1992;15:318-68.
- DeFronzo RA, Ferrannini E. Insulin resistance: a multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care* 1991;14:173-94.
- Desrochers F, Curtis MA. The occurrence of gastrointestinal helminths in dogs from Kuujuaq (Fort Chimo), Quebec, Canada. *Can J Public Health* 1987;78:403-6.
- Dinneen S, Gerich J, Rizza R. Carbohydrate metabolism in non-insulin-dependent diabetes mellitus. *N Engl J Med* 1992;327:707-13.
- Dippe SE, Bennett PH, Dippe DW et al. Glucose tolerance among Aleuts on the Pribilof Islands. In: Shephard RJ, Itoh S, eds. *Circumpolar Health: Proceedings of the 3rd International Symposium*. Toronto: University of Toronto Press; 1976:156.
- Donahue RP, Skyler JS, Schneiderman N, Prineas RJ. Hyperinsulinemia and elevated blood pressure: cause, confounder or coincidence. *Am J Epidemiol* 1990;132:827-36.
- Dossetor JB, McConnachie PR, Stiller CR et al. The major histocompatibility complex in Eskimos. *Transplant Proc* 1973;5:209-13.
- Dowse G, Zimmet P, Collins V, Finch C. Obesity in Pacific populations. In: Björntorp P, Brodoff BN, eds. *Obesity*. Philadelphia: J.B.Lippincott; 1992:619-39.
- Dowse GK, Zimmet PZ, Finch CF, Collins VR. Decline in incidence of epidemic glucose intolerance in Nauruans: implication for the "thrifty genotype". *Am J Epidemiol* 1991;133:1093-1104.
- Draper HH. The aboriginal Eskimo diet in modern perspective. *Am Anthropologist* 1977;79:309-16.

- Draper HH. Nutrition. In: Milan FA, ed. *The Human Biology of Circumpolar Populations*. Cambridge: Cambridge University Press; 1980:257-284.
- Driver HE. *Indians of North America*. 2nd ed. Chicago: University of Chicago Press; 1969.
- Dunn SP, Dunn E. The transformation of economy and culture in the Soviet North. *Arctic Anthropol* 1963;1(2):1-28.
- Dyerberg J. Coronary heart disease in Greenland Inuit: a paradox. *Arct Med Res* 1989;48:47-54.
- Dyerberg J, Bang HO, Hjørne N. Fatty acid composition of the plasma lipids in Greenland Eskimos. *Am J Clin Nutr* 1975;28:958-66.
- Dyerberg J, Bang HO. Hemostatic function and platelet polyunsaturated fatty acids in Eskimos. *Lancet* 1979;ii:433-5.
- Eaton SB, Konner M. Paleolithic nutrition: a consideration of its nature and current implications. *N Engl J Med* 1985;312:283-9.
- Eaton SB, Konner M, Shostak M. Stone agers in the fast lane: chronic degenerative diseases in evolutionary perspective. *Am J Med* 1988;84:739-49.
- Editorial. Eskimo diets and diseases. *Lancet* 1983;i:1139-41.
- Ehrstrom MC. Medical studies in north Greenland 1948-1949. VI: Blood pressure, hypertension and atherosclerosis in relation to food and mode of living. *Acta Med Scand* 1951;140:416-22.
- Ekoé JM, Thouez JP, Petitclerc C, Foggin PM, Ghadirian P. Epidemiology of obesity in relationship to some chronic medical conditions among Inuit and Cree Indian populations in New Quebec, Canada. *Diab Res Clin Pract* 1990;10:S17-S27.
- Ellestad-Sayed JJ, Haworth JC, Hildes JA. Dissacharide malabsorption and dietary patterns in two Canadian Eskimo communities. *Am J Clin Nutr* 1978;31:1473-8.
- Eriksson AW, Lehmann W, Simpson NE. Genetic studies on circumpolar populations. In: Milan FA, ed. *The Human Biology of Circumpolar Populations*. Cambridge: Cambridge University Press; 1980:81-168.
- Eriksson J, Saloranta C, Widén E et al. Non-esterified fatty acids do not contribute to insulin resistance in persons at increased risk of developing type 2 (non-insulin-dependent) diabetes mellitus. *Diabetologia* 1991;34:192-7.

- Fagan BM. *The Great Journey: The Peopling of Ancient America*. New York: Thames and Hudson; 1987.
- Felber J-P, Acheson KJ, Tappy L. *From Obesity to Diabetes*. Chichester: John Wiley and Sons, 1993.
- Feldman SA, Rubenstein AH, Ho KJ et al. Carbohydrate and lipid metabolism in the Alaskan Arctic Eskimo. *Am J Clin Nutr* 1975;28:588-94.
- Fitzhugh WW, Crowell A, eds. *Crossroads of Continents: Cultures of Siberia and Alaska*. Washington, DC: Smithsonian Institution; 1988.
- Folk GE. *Introduction to Environmental Physiology*. Philadelphia: Lea and Febiger; 1966.
- Fortune R. *Chills and Fever: Health and Disease in the Early History of Alaska*. Fairbanks, AK: University of Alaska Press; 1989.
- Fortune R. The health of the Eskimos, as portrayed in the earliest written accounts. *Bull Hist Med* 1971;45:97-114.
- Fournelle HJ, Rader V, Allen C. A survey of enteric infections among Alaskan Indians. *Public Health Rep* 1966;81:797-803.
- Freeman MM. The significance of demographic changes occurring in the Canadian East Arctic. *Anthropologica* 1971;13:215-36.
- Freeman MM. A social and ecological analysis of systematic female infanticide among the Netsilik Eskimos. *Am Anthropologist* 1971;73:1011-8.
- Freeman MM. Arctic ecosystems. In: Damas D, ed. *Arctic*. Washington, DC: Smithsonian Institution; 1984:36-48.
- Friedewald T, Levy RI, Fredrikson DS. Estimation of the concentration of low-density-lipoprotein cholesterol in plasma without use of the preparative ultra-centrifuge. *Clin Chem* 1972;18:499-502.
- Frisancho AR. *Anthropometric Standards for the Assessment of Growth and Nutritional Status*. Ann Arbor, MI: University of Michigan Press, 1990.
- Frisch RE. Fatness and fertility. *Sci Am* 1988;258(3):70-7.
- Fujioka S, Matsuzawa Y, Tokunaga K, Tarui S. Contribution of intra-abdominal fat accumulation to the impairment of glucose and lipid metabolism in human obesity. *Metabolism* 1987;36:54-9.

- Gannon MC, Nuttall FQ, Neil BJ, Westphal SA. The insulin and glucose responses to meals of glucose plus various proteins in type II diabetic subjects. *Metabolism* 1988;37:1081-8.
- Garn SM, Leonard WR. What did our ancestors eat? *Nutr Rev* 1989;47:337-45.
- Garn SM, Leonard WR, Hawthorne VM. Three limitations of the body mass index. *Am J Clin Nutr* 1986;44:996-7.
- Garrow JS. *Obesity and Related Diseases*. Edinburgh: Churchill Livingstone, 1988.
- Gaudette LA, Dufour R, Freitag S, Miller AB. Cancer patterns in the Inuit population of Canada, 1970-1984. In: Postl BD, Gilbert P et al., eds. *Circumpolar Health 90: Proceedings of the 8th International Congress on Circumpolar Health*. Winnipeg: University of Manitoba Press; 1991:443-6.
- Gerasimova E, Perova N, Ozerova I, Polesky V et al. The effect of dietary n-3 polyunsaturated fatty acids on HDL cholesterol in Chukot residents vs Muscovites. *Lipids* 1991;26:261-5.
- Gerdes LU, Schmidt EB, Klausen IC et al. Plasma concentration of levels of apolipoprotein A-I, apolipoprotein B and lipoprotein (a) in Greenland Inuit (Eskimos). *J Intern Med* 1992; 231:623-5.
- Goldberg DP. *The Detection of Psychiatric Illness by Questionnaire*. London: Oxford University Press; 1972.
- Goto Y, Tamachi H, Moriguchi EH. Eicosapentaenoic acid and atherosclerosis. *Prostaglandin Leuk Essent Fatty Acid* 1993;48:337-42.
- Gottman AW. A report of 103 autopsies on Alaskan Eskimos. *Arch Path* 1960;70:117-24.
- Greenberg JH, Ruhlen M. Linguistic origins of Native Americans. *Sci Am* 1992;267(5):60-5.
- Greenberg JH, Turner CG, Zegura SL. The settlement of the Americas: a comparison of the linguistic, dental and genetic evidence. *Curr Anthropol* 1986;27:447-97.
- Grove O, Lynge I. Suicide and attempted suicide in Greenland. *Acta Psychiatr Scand* 1979;60:375-91.
- Grzybowski S, Styblo K, Dorken E. Tuberculosis in Eskimos. *Tubercle* 1976;57(Supp 4):S1-S58.
- Gurney M, Gorstein J. The global prevalence of obesity - an initial overview of available data. *World Health Stat Q* 1988;41:251-4.

- Haffner SM, Stern MP, Hazuda HP, Pugh J, Patterson JK. Do upper-body and centralized adiposity measure different aspects of regional body-fat distribution? Relationship to non-insulin-dependent diabetes mellitus, lipids, and lipoproteins. *Diabetes* 1987;36:43-51.
- Hales CN, Barker DJP. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia* 1992;35:595-601.
- Halli SS, Trovato F, Driedger L. *Ethnic Demography: Canadian Immigrant, Racial and Cultural Variations*. Ottawa: Carleton University Press; 1990.
- Hammond GW, Rutherford BE, Malazdrewicz R, MacFarlane N et al. Haemophilus influenzae meningitis in Manitoba and the Keewatin District, NWT: potential for mass vaccination. *Can Med Assoc J* 1988;139:743-7.
- Hansen JP, Hancke S, Møller-Petersen J. Atherosclerosis in Native Greenlanders: an ultrasonographic investigation. *Arct Med Res* 1990;49:151-6.
- Hansen JP, Meldgaard J, Nordqvist J. The mummies of Qilakitsoq. *Nat Geographic* 1985; 167:191-207.
- Harper AB. Origins and divergence of Aleuts, Eskimos and American Indians. *Ann Hum Biol* 1980;7:547-54.
- Häring HV, Mehnert H. Pathogenesis of type 2 (non-insulin-dependent) diabetes mellitus: candidates for a signal transmitter defect causing insulin resistance of the skeletal muscles. *Diabetologia* 1993;36:176-82.
- Harrison GA, ed. *Population Structure and Human Variation*. Cambridge: Cambridge University Press; 1977.
- Harrison GA, Boyce AJ, eds. *The Structure of Human Populations*. Oxford: Clarendon Press; 1972.
- Harrison GA, Tanner JM, Pilbeam DR, Baker PT. *Human Biology: An Introduction to Human Evolution, Variation, Growth, and Adaptability*. Oxford: Oxford University Press; 1988.
- Harrison GG, Ritenbaugh CK. Obesity among North American Indians. In: Bjorntorp P, Brodoff BN, eds. *Obesity*. Philadelphia: JB Lippincott; 1992:610-8.
- Hauschild AH, Gauvreau L. Food-born botulism in Canada, 1971-84. *Can Med Assoc J* 1985;133:1141-6.
- Heller CA, Scott EM. *The Alaska Dietary Survey 1956-1961*. Anchorage, AK: US Public Health Service; 1967. DHEW/PHS/Publ No 999-AH-2.

- Hildes JA. The circumpolar people - health and physiological adaptations. In: Baker PT, Weiner JS, eds. *The Biology of Human Adaptability*. Oxford: Clarendon Press; 1966:497-508.
- Hildes JA, Schaefer O. Health of Igloolik Eskimos and change with urbanization. *J Hum Evol* 1973;2:241-6.
- Hildes JA, Schaefer O. The changing picture of neoplastic disease in the western and central Canadian Arctic (1950-1980). *Can Med Assoc J* 1984;130:25-33.
- Hlady WG, Middaugh JP. Suicides in Alaska: firearms and alcohol. *Am J Public Health* 1988; 78:179-80.
- Ho KJ, Mikkelsen B, Lewis LA, Feldman SA, Taylor CB. Alaskan Arctic Eskimo: responses to a customary high fat diet. *Am J Clin Nutr* 1972;25:737-45.
- Hoffecker JF, Powers WR, Goebel T. The colonization of Beringia and the peopling of the New World. *Science* 1993;259:46-53.
- Hoppner K, McLaughlin JM, Shah BG et al. Nutrient levels of some foods of Eskimos from Arctic Bay, NWT, Canada. *J Am Diet Assoc* 1978;73:257-61.
- Horrobin DF. Low prevalence of coronary heart disease (CHD), psoriasis, asthma and rheumatoid arthritis in Eskimos: are they caused by high dietary intake of eicosapentaenoic acid (EPA), a genetic variation of essential fatty acid (EFA) metabolism or a combination of both? *Med Hypotheses* 1987;22:421-8.
- Howard BV, Bogardus C, Ravussin E, Foley JE et al. Studies of the etiology of obesity in Pima Indians. *Am J Clin Nutr* 1991;53:1577S-85S.
- Hrdlička A. *Anthropological Survey in Alaska*. 46th Annu Rep Bur Am Ethnol 1928-1929. Washington, DC: Government Printing Office; 1930:19-374.
- Hubert HB. The importance of obesity in the development of coronary risk factors and disease: the epidemiologic evidence. *Annu Rev Public Health* 1986;7:493-502.
- Hughes CC. Translation of chapter on Eskimos from *The Peoples of Siberia* edited by M.G. Levin and L.P. Potapov. *Anthropol Papers Univ Alaska* 1963;12(1):1-13.
- Hughes CC. Under four flags: recent culture change among the Eskimos. *Curr Anthropol* 1965; 6:3-69.
- Imrie R, Warren R. Health promotion survey in the Northwest Territories. *Can J Public Health* 1988;79:16-24.

- Innis SM, Kuhnlein HV, Kinloch D. The composition of red cell membrane phospholipids in Canadian Inuit consuming a diet high in marine mammals. *Lipids* 1988;23:1064-8.
- Innis SM, Kuhnlein HV. The fatty acid composition of northern Canadian marine and terrestrial mammals. *Acta Med Scand* 1987;222:105-9.
- Innis SM, Kuhnlein HV. Long-chain n-3 fatty acids in breast milk of Inuit women consuming traditional foods. *Early Hum Dev* 1988;18:185-9.
- Intersalt Cooperative Research Group. INTERSALT: an international study of electrolyte excretion and blood pressure. *Br Med J* 1988;297:319-28.
- Itoh S. Physiology of circumpolar peoples. In: Milan FA, ed. *The Human Biology of Circumpolar Populations*. Cambridge: Cambridge University Press; 1980:285-304.
- Jamison PL, Zegura SL, Milan FA, eds. *The Eskimo of Northwest Alaska: A Biological Perspective*. Stroudsburg, PA: Dowden, Hutchinson and Ross; 1978.
- Jenness D. *Physical Characteristics of the Copper Eskimos*. Report of the Canadian Arctic Expedition, 1913-18, Vol. 12, Part B. Ottawa: King's Printer; 1923.
- Johansson SR. The demographic history of the Native Peoples of North America: a selective bibliography. *Yearbk Phys Anthropol* 1982;25:133-52.
- Johnston FE. Health implications of childhood obesity. *Ann Intern Med* 1985;103:1068-72.
- Johnston FE, Laughlin WS, Harper AB, Ensworth AE. Physical growth of St. Lawrence Island Eskimos: body size, proportion, and composition. *Am J Phys Anthropol* 1982;58:397-401.
- Jones RH. Insulin action and metabolism. In: Besser GM, Bodansky HJ, Cudworth AG, eds. *Clinical Diabetes: An Illustrated Text*. Philadelphia: Lippincott, 1988; 5.1-5.16.
- Jørgensen KA, Nielsen AH, Dyerberg J. Hemostatic factors and renin in Greenland Eskimos on a high eicosapentaenoic acid intake: results of the 5th Umanak Expedition. *Acta Med Scand* 1986;219:473-9.
- Keenleyside A. Euro-American whaling in the Canadian Arctic: its effects on Eskimo health. *Arctic Anthropol* 1990;27(1):1-19.
- Ketl PA, Bixler EO. Suicide in Alaska Natives, 1979-1984. *Psychiatry* 1991;54:55-63.
- Kirk R, Szathmary EJ, eds. *Out of Asia: Peopling the Americas and the Pacific*. Canberra: Journal of Pacific History Special Publication; 1985.

- Kissebah AH, Peiris AN. Biology of regional body fat distribution: relationship to non-insulin-dependent diabetes mellitus. *Diab Metab Rev* 1989;5:83-109.
- Kjaer SK, Teisen C, Haugaard BJ et al. Risk factors for cervical cancer in Greenland and Denmark: population-based cross-sectional study. *Int J Cancer* 1989;44:40-7.
- Knowler WC, Pettitt DJ, Bennett PH, Williams RC. Diabetes mellitus in the Pima Indians: genetic and evolutionary considerations. *Am J Phys Anthropol* 1983;62:107-14.
- Knowler WC, Pettitt DJ, Saad MF, Bennett PH. Diabetes mellitus in the Pima Indians: incidence, risk factors and pathogenesis. *Diabetes Metab Rev* 1990;6:1-27.
- Knowler WC, Pettitt DJ, Saad MF, Charles MA et al. Obesity in the Pima Indians: its magnitude and relationship with diabetes. *Am J Clin Nutr* 1991;53:1543S-51S.
- Kraus RF, Buffler PA. Sociocultural stress and the American Native in Alaska: an analysis of changing patterns of psychiatric illness and alcohol abuse among Alaska Natives. *Cult Med Psychiat* 1979;3:111-51.
- Kroman N, Green A. Epidemiological studies in the Upernavik District: incidence of some chronic diseases, 1950-1974. *Acta Med Scand* 1980;208:401-6.
- Krupnik II. The male-female ratio in certain traditional populations of the Siberian Arctic. *Études Inuit Studies* 1985;9:115-40.
- Kunitz SJ. *Disease Change and the Role of Medicine: The Navajo Experience*. Berkeley, CA: University of California Press; 1983.
- Lanier AP, Bulkow LR, Ireland B. Cancer in Alaskan Indians, Eskimos, and Aleuts, 1969-83: implications for etiology and control. *Public Health Rep* 1989;104:658-64.
- Larke RB, Froese GJ, Devine RD, Petruk MW. Extension of the epidemiology of hepatitis B in circumpolar regions through a comprehensive serologic study in the Northwest Territories of Canada. *J Med Virol* 1987;22:269-76.
- Laughlin WS. Eskimos and Aleuts: their origins and evolution; physiological and cultural adaptation to facilitate the evolutionary success of Eskimo-Aleut stock. *Science* 1963;142:633-45.
- Laughlin WS. Genetical and anthropological characteristics of Arctic populations. In: Baker PT, Weiner JS, eds. *The Biology of Human Adaptability*. Oxford: Clarendon Press; 1966: 469-95.

- Laughlin WS, Harper AB, eds. *The First Americans: Origins, Affinities, and Adaptations*. New York: Gustav Fischer; 1979.
- Lebedev VV. Siberian peoples: a Soviet view. In: Fitzhugh WW, Crowell A, eds. *Crossroads of Continents: Cultures of Siberian and Alaska*. Washington, DC: Smithsonian Institution; 1988:314-8.
- Lederman JM, Wallace AC, Hildes JA. Arteriosclerosis and neoplasms in Canadian Eskimos. In: *Biological Aspect of Aging: Proceedings of the Fifth International Congress on Gerontology*. New York: Columbia University Press; 1962:201-7.
- Leighton AH, Hughes CC. Notes on Eskimo patterns of suicide. *Southwest J Anthropol* 1955; 11:327-8.
- Levin MG, Potapov LP, eds. *The Peoples of Siberia*. Chicago: University of Chicago Press; 1964:799-835. [Originally published by USSR Academy of Sciences, Moscow, 1956].
- Little MA, Haas JD, eds. *Human Population Biology: A Transdisciplinary Science*. New York: Oxford University Press; 1989.
- Lukaski HC. Methods for the assessment of human body composition: traditional and new. *Am J Clin Nutr* 1987;46:537-56.
- Lynge I. Suicide in Greenland. *Arct Med Res* 1985;40:53-60.
- MacLean DR, Petrasovits A, Nargundkar M et al. Canada heart health surveys: a profile of cardiovascular risk. Survey methods and data analysis. *Can Med Assoc J* 1992;146: 1969-76.
- Mann GV, Scott EM, Hursh LM et al. The health and nutritional status of Alaskan Eskimos: a survey of the Interdepartmental Committee on Nutrition for National Defence - 1958. *Am J Clin Nutr* 1962;11:31-75.
- Marchand JF. Tribal epidemics in the Yukon. *JAMA* 1943;123:1019-20.
- Maynard JE. Coronary heart disease risk factors in relation to urbanization in Alaskan Eskimo men. In: Shephard RJ, Itoh S, eds. *Circumpolar Health*. Toronto: University of Toronto Press; 1976:294.
- Maynard JE, Hammes LM, Kester FE. Mortality due to heart disease among Alaska Natives, 1955-65. *Public Health Rep* 1967;82:714-20.

- McAlpine PJ, Chen SH, Cox DW et al. Genetic markers in blood in a Canadian Eskimo population with a comparison of allele frequencies in circumpolar populations. *Hum Hered* 1974;24:114-42.
- McAlpine P, Simpson NE. Fertility and other demographic aspects of the Canadian Eskimo communities of Igloolik and Hall Beach. *Hum Biol* 1976;48:113-38.
- McDowell I, Newell C. *Measuring Health: A Guide to Rating Scales and Questionnaires*. New York: Oxford University Press;1987.
- McGarvey ST, Bindon JR, Crews DE, Schendel DE. Modernization and adiposity: causes and consequences. In: Little MA, Haas JD, ed. *Human Population Biology: A Transdisciplinary Science*. New York: Oxford University Press, 1969:263-79.
- Micozzi MS, Albanes D, Jones V, Chumlea WC. Correlation of body mass indices with weight, stature, and body composition in men and women in NHANES I and II. *Am J Clin Nutr* 1986;44:725-31.
- Middaugh JP. Cardiovascular deaths among Alaskan Natives, 1980-86. *Am J Public Health* 1990;80:282-5.
- Milan FA. The demography of selected circumpolar populations. In: Milan FA, ed. *The Human Biology of Circumpolar Populations*. Cambridge: Cambridge University Press; 1980: 13-35.
- Milan FA, ed. *Human Biology of Circumpolar Populations*. Cambridge: Cambridge University Press; 1980.
- Millar WJ, Stephens T. The prevalence of overweight and obesity in Britain, Canada, and United States. *Am J Public Health* 1987;77:38-41.
- Minuk GY, Ling N, Postl B, Waggoner JG et al. The changing epidemiology of hepatitis B virus infection in the Canadian North. *Am J Epidemiol* 1985;121:598-604.
- Minuk GY, Nicolle LE, Postl B, Waggoner JG, Hoofnagle JH. Hepatitis virus infection in an isolated Canadian Inuit (Eskimo) population. *J Med Virol* 1982;10:255-64.
- Minuk GY, Waggoner JG, Jernigan R, Nicolle L. E. et al. Prevalence of antibody to hepatitis A virus in a Canadian Inuit community. *Can Med Assoc J* 1982;127:850-2.
- Moran EF. Human adaptation to Arctic zones. *Annu Rev Anthropol* 1981;10:1025.
- Moran EF. *Human Adaptability: An Introduction to Ecological Anthropology*. Boulder, CO: Westview Press; 1982.

- Mouratoff GJ, Carroll NV, Scott EM. Diabetes mellitus in Eskimos. *JAMA* 1967;199:107-12.
- Mouratoff GJ, Scott EM. Diabetes mellitus in Eskimos after a decade. *JAMA* 1973;266:1345-6.
- Mowat F. *People of the Deer*. Boston: Little, Brown; 1951.
- Mueller WH, Stallones L. Anatomical distribution of subcutaneous fat: skinfold site choice and construction of indices. *Hum Biol* 1981;53:321-35.
- Murphy NJ, Boyko EJ, Schraer CD, Lanier AP, Bulkow LR. Diabetes mellitus in Alaskan Yup'ik Eskimos and Athabaskan Indians after 25 years. *Diabetes Care* 1992;15:1390-2.
- National Institutes of Health Consensus Development Panel. Health implications of obesity. *Ann Intern Med* 1985;103:1073-7.
- Nazarova AF. The genetic structure of populations of Chuckotka Peninsula Eskimos and Chukchi based on study of 13 loci of serum and erythrocyte proteins and enzymes. *Am J Phys Anthropol* 1989;79:81-8.
- Neel JV. Diabetes mellitus: a "thrifty" genotype rendered detrimental by "progress"? *Am J Hum Genet* 1962;14:353-62.
- Neel JV. The thrifty gene revisited. In: Kobberling J, Tattersall R, eds. *The Genetics of Diabetes Mellitus*. New York: Academic Press; 1982:283-93.
- Newman MT. Adaptations in the physique of American aborigines to nutritional factors. *Hum Biol* 1960;32:288-313.
- Nikitin YP. Chronic, non-communicable diseases in Native residents and newcoming populations of circumpolar areas. In: Linderholm H, Backman C, Broadbent N, Joelsson I, eds. *Circumpolar Health 87: Proceedings of the 7th International Congress on Circumpolar Health*. Oulu, Finland: Nordic Council for Arctic Medical Research; 1988:426-33.
- Nikitin YP, Klochkova E, Mamleeva FR. Comparison of diets in two Native Chukotka populations and prevalence of ischemic heart disease risk factors. *Arct Med Res* 1991; 50:67-72.
- Nobmann ED, Byers T, Lanier AP, Hankin JH, Jackson MY. The diet of Alaska Native adults: 1987-88. *Am J Clin Nutr* 1992;55:1024-32.
- Norris MJ. The demography of aboriginal people in Canada. In: Halli SS, Trovato F, Driedger L, eds. *Ethnic Demography: Canadian Immigrant, Racial and Cultural Variations*. Ottawa: Carleton University Press; 1990:33-59.

- O'Dea K. Obesity and diabetes in "the land of milk and honey". *Diab Metab Rev* 1992;8:373-88.
- Omran AR. The epidemiological transition: a theory of the epidemiology of population change. *Milbank Mem Fund Q* 1971;49:509-38.
- Ostergaard Kristensen M. Increased incidence of bleeding intracranial aneurysms in Greenland Eskimos. *Acta Neurochir (Wien)* 1983;67:37-43.
- Overfield T, Klauber MR. Prevalence of tuberculosis in Eskimos having blood group B gene. *Hum Biol* 1980;52:87-92.
- Peart AF, Nagler FP. Measles in the Canadian Arctic, 1952. *Can J Public Health* 1954;45:146-57.
- Petersen GM, Ward JI, Terasaki PI et al. Genetic polymorphisms in southwestern Alaskan Eskimos. *Hum Hered* 1991;41:236-47.
- Pettitt DJ, Knowler WC, Bennett PH, Aleck KA, Baird HR. Obesity in offspring of diabetic Pima Indian women despite normal birth weight. *Diabetes Care* 1987;10:76-80.
- Prener A, Nielsen NH, Storm HH, Hansen JP, Jensen OM. Cancer in Greenland, 1953-1985. *Acta Pathol Microbiol Immunol Scand* 1991;99(Suppl 20):7-79.
- Prineas RJ, Crow RS, Blackburn H. *The Minnesota Code Manual of Electrocardiographic Findings*. Boston: John Wright; 1982.
- Prosser CL. Perspectives of adaptation: theoretical aspects. In: Dill DB, ed. *Adaptation to the Environment*. Handbook of Physiology, Section 4. Washington, DC: American Physiological Society; 1964:11-35.
- Rabinowitch IM, Smith FC. Metabolic studies of Eskimos in the Canadian Eastern Arctic. *J Nutr* 1936;12:337-56.
- Ramirez ME. Measurement of subcutaneous adipose tissue using ultrasound images. *Am J Phys Anthropol* 1992;89:347-57.
- Rasmussen K. *Observations on the Intellectual Culture of the Caribou Eskimo*. Copenhagen: 1930. Report of the 5th Thule Expedition, 1921-24, vol 7, parts 1 and 2.
- Ravussin E, Lillioja S, Knowler WC et al. Reduced rate of energy expenditure as a risk factor for body weight gain. *N Engl J Med* 1988;318:467-72.
- Ravussin E, Swinburn BA. Pathophysiology of obesity. *Lancet* 1992;340:404-8.

- Renfrew C. Archaeology, genetics and linguistic diversity. *Man* 1992;n.s.27:445-78.
- Rennie DW, Covino BG, Blair MR, Rodahl K. Physical regulation of temperature in Eskimos. *J Appl Physiol* 1962;17:326-32.
- Revicki DA, Israel RG. Relationship between body mass indices and measures of body adiposity. *Am J Public Health* 1986;76:992-4.
- Rimoin DL, Saiki JH. Diabetes among the Navajo. II. Plasma glucose and insulin response. *Arch Intern Med* 1968;122:6-9.
- Ritenbaugh C. Obesity as a culture-bound syndrome. *Cult Med Psychiatry* 1982;6:347-61.
- Ritenbaugh C, Goodby C. Beyond the Thrifty Gene: metabolic implications of prehistoric migration into the New World. *Med Anthropol* 1989;11:227-36.
- Roberts DF. Basal metabolism, race and climate. *J Roy Anthropol Inst* 1952;82:169-83.
- Roberts DF. Body weight, race and climate. *Am J Phys Anthropol* 1953;11:533-58.
- Robitaille N, Choinière R. *An Overview of Demographic and Socio-economic Conditions of the Inuit in Canada*. Ottawa: Research Branch, Corporate Policy, Indian and Northern Affairs Canada; 1985.
- Rodahl K. Basal metabolism of the Eskimo. *J Nutr* 1952;48:359-68.
- Rode A, Shephard RJ. *Fitness and Health of an Inuit Community: 20 Years of Cultural Change*. Ottawa: Department of Indian and Northern Affairs, Circumpolar and Scientific Affairs Directorate; 1992. Publ No 92-05.
- Rode A, Shephard RJ. Ten years of "civilisation": fitness of Canadian Inuit. *J Appl Physiol* 1984;56:1472-7.
- Romaniuk A. Modernization and fertility: the case of the James Bay Indians. *Can Rev Sociol Anthropol* 1974;11:344-59.
- Rose GA, Blackburn H, Gillum RF, Prineas RJ. *Cardiovascular Survey Methods*. Geneva: World Health Organization; 1982.
- Roychoudhury AK. Genetic distance between the American Indians and the three major races of man. *Hum Hered* 1978;28:380-5.
- Roychoudhury AK, Nei M. *Human Polymorphic Genes: World Distribution*. New York: Oxford University Press; 1988.

- Rychkov YG, Sheremetyeva VA. The genetic process in the system of ancient human isolates in North Asia. In: Harrison GA, ed. *Population Structure and Human Variation*. Cambridge: Cambridge University Press; 1977:47-108.
- Rychkov YG, Sheremetyeva VA. The genetics of circumpolar populations of Eurasia related to the problem of human adaptation. In: Milan FA, ed. *The Human Biology of Circumpolar Populations*. Cambridge: Cambridge University Press; 1980:37-80.
- Sagild U, Littauer J, Jespersen SC, Andersen S. Epidemiological studies in Greenland 1962-64. I. Diabetes mellitus in Eskimos. *Acta Med Scand* 1966;179:29-39.
- Schaefer O. Glycosuria and diabetes mellitus in Canadian Eskimos. *Can Med Assoc J* 1968;99:201-6.
- Schaefer O. Glucose tolerance testing in Canadian Eskimos: a preliminary report and hypothesis. *Can Med Assoc J* 1968;99:252-62.
- Schaefer O. Pre- and post-natal growth acceleration and increased sugar consumption in Canadian Eskimos. *Can Med Assoc J* 1970;103:1059-68.
- Schaefer O. Are Eskimos more or less obese than other Canadians? A comparison of skinfold thickness and ponderal index in Canadian Eskimos. *Am J Clin Nutr* 1977;30:1623-8.
- Schaefer O. Knud Rasmussen Memorial Lecture: Ethnology, demography and medicine in the Arctic. In: Harvald B, Hansen JP, eds. *Circumpolar Health 81*. Oulu, Finland: Nordic Council for Arctic Medical Research; 1982:187-93.
- Schaefer O, Crockford PM, Romanowski B. Normalization effect of preceding protein meals on "diabetic" oral glucose tolerance in Eskimos. *Can Med Assoc J* 1972;107:733-8.
- Schaefer O, Hildes JA, Medd LM, Cameron DG. The changing pattern of neoplastic disease in Canadian Eskimos. *Can Med Assoc J* 1975;112:1399-404.
- Schaefer O, Hildes JA, Greidanus P, Leung D. Regional sweating in Eskimos compared to Caucasians. *Can J Physiol Pharmacol* 1974;52:960-5.
- Schaefer O, Steckle J. *Dietary Habits and Nutritional Base of Native Populations of the Northwest Territories*. Yellowknife, NWT: Government of the Northwest Territories; 1980.
- Schaefer O, Timmermans JF, Eaton RD, Mathews AR. General and nutritional health in two Eskimo populations at different stages of acculturation. *Can J Public Health* 1980;71:397-405.

- Schanfield MS, Crawford MH, Dossetor JB, Gershowitz H. Immunoglobulin allotypes in several North American Eskimo populations. *Hum Biol* 1990;62:773-89.
- Schraer CD, Lanier AP, Boyko EJ et al. Prevalence of diabetes mellitus in Alaskan Eskimos, Indians, and Aleuts. *Diabetes Care* 1988;11:693-700.
- Schrire C, Steiger WL. A matter of life and death: an investigation into the practice of female infanticide in the Arctic. *Man* 1974;9:161-84.
- Schurr TG, Ballinger SW, Gan YY et al. Amerindian mitochondrial DNAs have rare Asian mutations at high frequencies, suggesting they derived from four primary maternal lineages. *Am J Hum Genet* 1990;46:613-23.
- Scott EM, Duncan IW, Ekstrand V, Wright RC. Frequency of polymorphic types of red cell enzymes and serum factors in Alaskan Eskimos and Indians. *Am J Hum Genet* 1966;18:408-11.
- Scott EM, Griffith IV. Diabetes mellitus in Eskimos. *Metabolism* 1957;6:320-5.
- Scott EM, Griffith IV, Hoskins DD, Whaley RD. Serum cholesterol levels and blood pressure of Alaskan Eskimo men. *Lancet* 1958;ii:667-8.
- Seltzer CC. The anthropometry of the Western and Copper Eskimos, based on data of Vilhjalmur Stefansson. *Hum Biol* 1933;5:313-70.
- Seltzer CC. Some re-evaluations of the Build and Blood Pressure Study, 1959, as related to ponderal index, somatotype and mortality. *N Engl J Med* 1966;274:254-9.
- Shephard RJ. Work physiology and activity patterns. in: Milan FA, ed. *Human Biology of Circumpolar Populations*. Cambridge: Cambridge University Press; 1980:305-38.
- Shephard RJ. Factors associated with population variation in physiological work capacity. *Ybk Phys Anthropol* 1985;28:97-122.
- Shephard RJ. *Body Composition in Biological Anthropology*. Cambridge: Cambridge University Press; 1991.
- Shephard RJ, Hatcher J, Rode A. On the body composition of the Eskimo. *Eur J Appl Physiol* 1973;30:1-13.
- Shields GF, Hecker K, Voevoda MI, Reed JK. Absence of the Asian-specific Region V mitochondrial marker in Native Beringians. *Am J Hum Genet* 1992;50:758-65.
- Sinclair HM. The diet of Canadian Indians and Eskimos. *Proc Nutr Soc* 1953;12:69-82.

- Skinhøj P. Hepatitis and hepatitis B antigen in Greenland. II. Occurrence and interrelation of hepatitis B associated surface, core and "e" antigen-antibody systems in a highly endemic area. *Am J Epidemiol* 1977;105:99-106.
- Skinhøj P, Mikklesen F, Hollinger FB. Hepatitis A in Greenland: importance of specific antibody testing in epidemiologic surveillance. *Am J Epidemiol* 1977;105:140-7.
- Snedecor GW, Cochran WC. *Statistical Methods*. 7th ed. Ames, IO: Iowa State University Press; 1980.
- So JK. Human biological adaptation to Arctic and Subarctic zones. *Annu Rev Anthropol* 1980; 9:63-82.
- Sobal J. Obesity and social status: a framework for examining relationships between physical and social variables. *Med Anthropol* 1991;13:231-47.
- Sobal J, Stunkard AJ. Socioeconomic status and obesity: a review of the literature. *Psychol Bull* 1989;105:260-75.
- Spuhler JN. Genetic distances, trees, and maps of North American Indians. in: Laughlin WS, Harper AB, eds. *The First Americans: Origins, Affinities, and Adaptations*. New York: Gustav Fischer; 1979:135-83.
- Stager JK, McSkimming RJ. Physical environment. In: Damas D, ed. *Arctic*. Washington, DC: Smithsonian Institution; 1984:27-35.
- Statistics Canada. *Canada's Native People: 1981 Census of Canada*. Ottawa: Statistics Canada; 1984.
- Statistics Canada. *A Data Book on Canada's Aboriginal Population from the 1986 Census of Canada*. Ottawa: Statistics Canada, Aboriginal Peoples Output Program; 1989.
- Steegman AT. Human adaptation to cold. In: Damon A, ed. *Physiological Anthropology*. New York: Oxford University Press; 1975:130-66.
- Steegman AT, Hurlich MG, Winterhalder B. Coping with cold and other challenges of the boreal forest: an overview. in: Steegman TA, ed. *Boreal Forest Adaptations: The Northern Algonkians*. New York: Plenum Press; 1983:317-51.
- Stefánsson V. *The Friendly Arctic: The Story of Five Years in Polar Regions*. New York: Macmillan; 1921.

- Stepanova EG, Shubnikov EW. Diabetes, glucose tolerance and some risk factors of diabetes mellitus in Natives and newcomers of Chukotka. In: Postl BD, Gilbert P, Goodwill J et al., eds. *Circumpolar Health 90*. Winnipeg: University of Manitoba Press; 1991:413-3.
- Stern MP, Haffner SM. Body fat distribution and hyperinsulinemia as risk factors for diabetes and cardiovascular diseases. *Arteriosclerosis* 1986;6:123-30.
- Stich HF, Hornby AP. Endogenous and exogenous sources of nitrate/nitrite and nitrosamines in Canadian Inuit with a traditional or western lifestyle. in: Fortune R, ed. *Circumpolar Health 84*. Seattle, WA: University of Washington Press; 1985:92-5.
- Stinson S. Nutritional adaptation. *Annu Rev Anthropol* 1992;21:143-70.
- Stutzman CD, Nelson DM, Lanier AP. Estimates of cancer incidence in Alaskan Natives due to exposure to global radioactive fallout from atmospheric nuclear weapons testing. *Alaska Med* 1986;28:67-77.
- Sukernik RI, Lemza SV, Karaphet TM, Osipova LP. Reindeer Chukchi and Siberian Eskimos: studies on blood groups, serum proteins, and red cell enzymes with regard to genetic heterogeneity. *Am J Phys Anthropol* 1981;55:121-8.
- Sukernik RI, Osipova LP. Gm and Km immunoglobulin allotypes in Reindeer Chukchi and Siberian Eskimos. *Hum Genet* 1982;61:148-53.
- Swedlund AC, Armelagos GJ, eds. *Disease in Populations in Transition: Anthropological and Epidemiological Perspectives*. New York: Bergin and Garvey; 1990.
- Szathmary EJ. Genetic markers in Siberia and northern North American populations. *Ybk Phys Anthropol* 1981;24:37-73.
- Szathmary EJ. Human biology of the Arctic. In: Damas D, ed. *Arctic*. Washington, DC: Smithsonian Institution; 1984:64-71.
- Szathmary EJ. Peopling of northern North America: clues from genetic studies. *Act Anthropol* 1984;8:79-110.
- Szathmary EJ. The impact of low carbohydrate consumption on glucose tolerance, insulin concentration and insulin response to glucose challenge in Dogrib Indians. *Med Anthropol* 1989; 11:329-50.
- Szathmary EJ. Diabetes in Amerindian populations: the Dogrib studies. In: Armelagos G, Swedlund A, eds. *Health and Disease of Populations in Transition*. New York: Bergin and Garvey; 1990:75-103.

- Szathmary EJ, Auger F. Biological distances and genetic relationships within Algonkians. In: Steegman AT, ed. *Boreal Forest Adaptations*. New York: Plenum Press; 1983:289-315.
- Szathmary EJ, Ferrell RE. Glucose level, acculturation, and glycosylated hemoglobin: an example of biocultural interaction. *Med Anthropol Q* 1990;4:315-41.
- Szathmary EJ, Holt N. Hyperglycemia in Dogrib Indians of the NWT, Canada: association with age and a centripetal distribution of body fat. *Hum Biol* 1983;55:493-515.
- Szathmary EJ, Ossenberg NS. Are the biological differences between North American Indians and Eskimos truly profound? *Curr Anthropol* 1978;19:673-701.
- Szathmary EJ, Ritenbaugh C, Goodby CS. Dietary change and plasma glucose levels in an Amerindian population undergoing cultural transition. *Soc Sci Med* 1987;24:791-804.
- Tanner CE, Staudt M, Adamowski R et al. Seroepidemiological study for five different zoonotic parasites in northern Quebec. *Can J Public Health* 1987;78:262-6.
- Thomas WA. Health of carnivorous race: a study of the Eskimo. *JAMA* 1927;88:1559-60.
- Thorslund J. Inuit suicides in Greenland. *Arct Med Res* 1990;49:25-33.
- Thouez JP, Rannou A, Foggin P. The other face of development: Native population health status and indicators of malnutrition - the case of the Cree and Inuit of northern Quebec. *Soc Sci Med* 1989;29:965-74.
- Torrey EF, Reiff FW, Noble GR. Hypertension among Aleuts. *Am J Epidemiol* 1979;110:7-14.
- Torrioni A, Schurr TG, Yang C, Szathmary EJ et al. Native American mitochondrial DNA analysis indicates that the Amerind and the NaDene populations were founded by two independent migrations. *Genetics* 1992;130:153-62.
- Trowell HC, Burkitt DP, eds. *Western Diseases: Their Emergence and Prevention*. Cambridge, MA: Harvard University Press; 1981.
- Turner II CG. Teeth and prehistory in Asia. *Sci Am* 1989;260(2):70-77.
- U.S. Congress, Office of Technology Assessment. *Indian Health Care*. Washington, DC: US Government Printing Office; 1986; OTA-H-290.
- Usher PJ. Evaluating country food in the northern Native economy. *Arctic* 1976;21:105-20.
- Vakhtin N. *Native Peoples of the Russian Far North*. London: Minority Rights Group; 1992.

- Verdier PC, Eaton RD, Cooper B. A study of the nutritional status of an Inuit population in the Canadian high Arctic. Part 1: Biochemical evaluation. *Can J Public Health* 1987;78:229-35.
- Wallace DC, Garrison K, Knowler WC. Dramatic founder effects in Amerindian mitochondrial DNAs. *Am J Phys Anthropol* 1985;68:149-55.
- Ward JI, Lum MK, Hall DB, Silimperi DR, Bender TR. Invasive Haemophilus influenzae type b disease in Alaska: background epidemiology for a vaccine efficacy trial. *J Infect Dis* 1986;153:17-26.
- Weiss KM, Ferrell RE, Hanis CL. A New World Syndrome of metabolic diseases with a genetic and evolutionary basis. *Yearbk Phys Anthropol* 1984;27:153-78.
- Wendorf M, Goldfine ID. Archaeology of NIDDM: excavation of the "thrifty" genotype. *Diabetes* 1991;40:161-5.
- Wenzel G. *Animal Rights, Human Rights: Ecology, Economy and Ideology in the Canadian Arctic*. Toronto: University of Toronto Press; 1991.
- West KM. Diabetes in American Indians and other native populations of the New World. *Diabetes* 1974;23:841-55.
- Wilber CG, Levine VE. Fat metabolism in Alaskan Eskimos. *Exp Med Surg* 1950;8:422-5.
- Williams RC, Steinberg AG, Gershowitz H et al. Gm allotypes in Native Americans: evidence for three distinct migrations across the Bering land bridge. *Am J Phys Anthropol* 1985;66:1-19.
- World Health Organization. *Diabetes Mellitus: Report of a WHO Study Group*. Geneva: WHO; 1985. Tech Rep Ser; No 727.
- World Health Organization, MONICA Project. Risk factors. *Int J Epidemiol* 1989;18(Suppl 1):S46-S55.
- Wotton KA, Stiver HG, Hildes JA. Meningitis in the Central Arctic: a 4-year experience. *Can Med Assoc J* 1981;124:887-90.
- Young TK. Epidemiology and control of chronic diseases in circumpolar Eskimo/Inuit populations. *Arct Med Res* 1986;42:25-47.
- Young TK. Are subarctic Indians undergoing the epidemiologic transition? *Soc Sci Med* 1988;26:659-71.

- Young TK. *Health Care and Cultural Change: the Indian Experience in the Central Subarctic*. Toronto: University of Toronto Press; 1988.
- Young TK. Prevalence and correlates of hypertension in a subarctic Indian population. *Prev Med* 1991;20:474-85.
- Young TK, Moffatt MEK, O'Neil. An epidemiological perspective of injuries in the Northwest Territories. *Arct Med Res* 1993;52(Suppl 7):27-36.
- Young TK, Moffatt MEK, O'Neil JD. Cardiovascular diseases in an arctic population: an epidemiological perspective. *Am J Public Health* 1993;83:881-7.
- Young TK, Roche BA. Factors associated with clinical gallbladder disease in a Canadian Indian population. *Clin Invest Med* 1990;13:55-9.
- Young TK, Schraer CD, Shubnikoff EV, Szathmary EJ, Nikitin YP. Prevalence of diagnosed diabetes in circumpolar indigenous populations. *Int J Epidemiol* 1992;21:730-6.
- Young TK, Sevenbuysen G. Obesity in northern Canadian Indians: patterns, determinants, and consequences. *Am J Clin Nutr* 1989;49:786-93.
- Young TK, Sevenhuysen GP, Ling N, Moffatt MEK. Determinants of plasma glucose level and diabetic status in a northern Canadian Indian population. *Can Med Assoc J* 1990;142:821-30.
- Young TK, Szathmary EJ, Evers S, Wheatley B. Geographical distribution of diabetes among the Native population of Canada: a national survey. *Soc Sci Med* 1990;31:129-39.
- Young VR, Marchini JS. Mechanisms and nutritional significance of metabolic responses to altered intakes of protein and amino acids with reference to nutritional adaptation in humans. *Am J Clin Nutr* 1990;51:270-89.
- Zaadstra BM, Seidell JC, Van Noord PA et al. Fat and female fecundity: prospective study of effect of body fat distribution on conception rates. *Br Med J* 1993;306:484-7.
- Zimmerman MR, Aufderheiden AC. The frozen family of Utriagvik: the autopsy findings. *Arct Anthropol* 1984;21:53-64.
- Zimmet P, Dowse G, Finch C, Serjeantson S, King H. The epidemiology and natural history of NIDDM - lessons from the South Pacific. *Diabe Metab Rev* 1990;6:91-124.

