

Obesity, Central Fat Patterning, and Their Metabolic Correlates among the Inuit of the Central Canadian Arctic

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Abstract I report on the occurrence and metabolic correlates of obesity among the Inuit (Eskimos) of the central Canadian Arctic using data from the Keewatin Health Assessment Study conducted during 1990 and 1991 in eight Inuit communities in the Northwest Territories ($n = 434$, adults aged 18 years and older). Data from the 1990 Manitoba Heart Health Survey among 2200 predominantly white residents of the province of Manitoba were used for comparison. Judging by body mass index and two skinfold thicknesses, obesity among the Inuit 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 lifestyle. Obesity is more prevalent among women, among whom there is also a higher degree of central fat patterning based on the waist-to-hip ratio. When different categories of obesity are compared, blood pressure and one or more of the lipids show an increasing trend but glucose or insulin level shows no significant change. This observation distinguishes the Inuit from other populations. Even where a relationship exists, as with triglyceride and HDL cholesterol levels, the magnitude of the response is lower among the Inuit. The differential effect of obesity on glucose, blood pressure, and lipid levels in the Inuit compared with non-Inuit suggests a type of selective insulin resistance, the underlying mechanism of obesity and several chronic diseases. Inuit metabolism reflects their almost exclusive diet of fat and proteins traditionally. From the public health perspective it is important to monitor and ameliorate the impact of changing diet and physical activity on the prevalence of obesity and associated health effects.

Considerable evidence indicates that obesity is now prevalent among many Native American populations (Broussard et al. 1991; Harrison and Ritenbaugh 1992). Previous studies have generally concluded that obesity has not been prevalent among the Inuit (or Eskimo) population, which has occupied

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Human Biology, April 1996, v. 68, no. 2, pp. 245–263.

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KEY WORDS: OBESITY, CENTRAL FAT, BODY MASS INDEX, WAIST-TO-HIP RATIO, INUIT, ARCTIC, PLASMA LIPIDS, BLOOD PRESSURE, GLUCOSE

the Arctic regions of North America for at least 5000 years (Schaefer 1977; Mann et al. 1962; Shephard et al. 1973), even though deposition of subcutaneous fat as insulation is a major adaptation to the cold environment of the Arctic among warm-blooded animals in general. So (1980) suggested that cultural innovations, such as efficient clothing and shelter, vigorous physical activity, and a diet high in protein and fat, obviate the need for subcutaneous fat to conserve body heat. Historically, the Inuit have also been spared the deleterious health effects of obesity, such as diabetes, coronary heart disease, and hypertension (Schaefer 1981). That the Inuit are atypical among Native Americans with regard to obesity and its health effects could be the result of genetic predisposition and/or a later and less intense exposure to the acculturative influences of modern Western society.

Over the past two decades substantial social and cultural changes have occurred among the Inuit, such as settlement in towns and villages, reduction in traditional hunting activities, and declining dependence on a land- and marine-mammal-based diet. Given such a background, three questions can be posed: (1) Is obesity prevalent among the Inuit today? (2) Is the obesity that occurs metabolically harmful? (3) What are the health implications of the emergence of obesity in this Arctic population?

These questions can be addressed using data obtained from the Keewatin Health Assessment Study (KHAS), a comprehensive community health survey conducted during 1990 and 1991 in seven Inuit communities in the Keewatin region of the Northwest Territories, Canada (60°–69° N and 80°–102° W), and the community of Sanikiluaq on the Belcher Islands near the eastern shore of Hudson Bay (Figure 1). Comparative data from a predominantly white population were available from 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.

Methods

Details of the study design and survey methods have been published by Moffatt et al. (1993) and Young et al. (1995). In the Keewatin region a complete census conducted for the study revealed a total population of 2975 individuals aged 18–74 years, 20% of whom were randomly selected ($n = 599$) and invited to participate in the survey. Of those invited, 72.5% participated, yielding a final sample of 434 individuals (199 males, 235 females). For the study reported here 47 individuals who did not report full or part Inuit ancestry were excluded from analysis, resulting in a final sample of 387 individuals.

The survey consisted of an interviewer-administered questionnaire, clinical examination, and laboratory tests. The questionnaire seeks sociodemographic information and data on personal health and lifestyle habits. It was

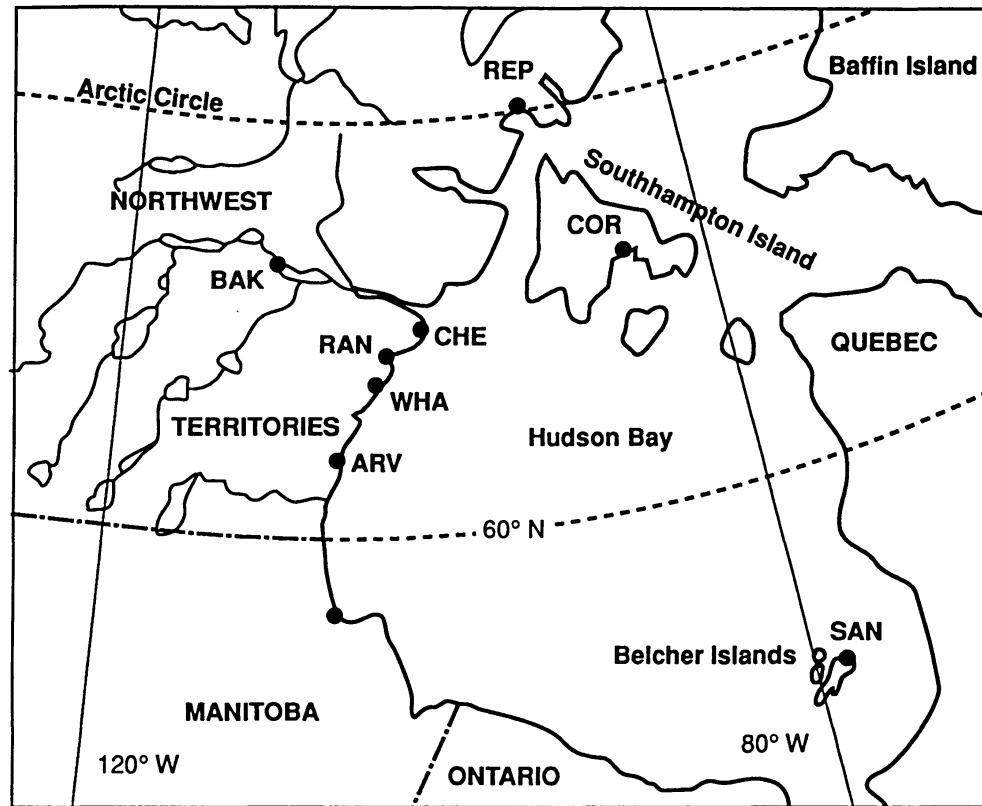


Figure 1. Location of study communities in the Keewatin region, Northwest Territories, Canada. REP, Repulse Bay; COR, Coral Harbour; BAK, Baker Lake; CHE, Chesterfield Inlet; RAN, Rankin Inlet; WHA, Whale Cove; ARV, Arviat; SAN, Sanikiluaq.

adapted from existing health survey protocols, such as the WHO MONICA study of cardiovascular risk factors (World Health Organization 1989) and the Canada Heart Health Survey (MacLean et al. 1992). Standardized procedures were used in performing blood pressure and anthropometric measurements. Two blood pressure measurements were taken at least 10 min apart after resting, using an appropriate cuff size of a standard mercury sphygmomanometer. Anthropometry consisted of measurements of the subscapular and triceps skinfold thicknesses, waist and hip girths, and height and weight. Field staff were trained by instructors from the Canada Heart Health Survey. 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.

Venipuncture was performed on participants after overnight fasting. Specimens were analyzed by the National Lipid Reference Laboratory at the University of Toronto. Details of the methods used in determining total cho-

lesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglyceride (TG) levels have been described by Connelly et al. (1992) and Young et al. (1995). Fasting plasma insulin levels were measured at the Endocrinology/Metabolism Laboratory of the University of Manitoba Health Sciences Center using the Insulin RIA 100 radioimmunoassay kit (Pharmacia Diagnostics AB, Uppsala, Sweden).

Glucose levels after fasting and after a 2-hr glucose challenge with a 75-g glucose load were determined on capillary blood obtained by fingerprick using an Ames Glucometer II (Miles Canada, Etobicoke, Ontario, Canada), a reflectance photometer. A certified laboratory technologist performed the glucometer readings. We submitted fasting plasma samples obtained by venipuncture from 118 individuals for glucose determination by an autoanalyzer using a hexokinase method in the laboratory. The correlation in glucose values between these laboratory-tested plasma samples and the glucometer-tested capillary samples was 0.44. A pairwise comparison showed that the capillary samples exceeded the plasma samples by 0.89 mmol/L on the average, with a range from -2.7 to 3.5 mmol/L. Although the use of glucometer readings to diagnose diabetes in individuals is unreliable, such data can be used to compare group means across categories of obesity. To ensure that similar conclusions were reached, I repeated the analyses using the glucose values from the smaller subset of plasma samples.

The software packages Statpac-Gold (Walonick Associates, Minneapolis, Minnesota) and SAS (SAS Institute, Cary, North Carolina) were used for all data management and statistical analyses. Because most of the anthropometric, physiological, and biochemical variables were not normally distributed (i.e., with skewness near 0 and kurtosis about 3), log-transformed data were used in the analyses.

For comparison with a southern, predominantly non-Native American population data from the MHHS were used. This survey was part of a standardized national survey of cardiovascular risk factors in all provinces of Canada. Details of both the national study (MacLean et al. 1992) and the Manitoba portion (Gelskey et al. 1994) of the survey have been published. The methods and procedures of the KHAS were comparable to those of the MHHS. The nurse-researcher in the KHAS who performed the interviews and clinical examinations was recruited from among those who took part in the MHHS. The same laboratory performed the lipid measurements. However, 2-hr postchallenge glucose level, fasting insulin level, and skinfold thicknesses were measured in the KHAS but not in the MHHS.

Results

Various indexes were constructed from the raw data on measured heights, weights, triceps and subscapular skinfold thicknesses, and waist and

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hip circumferences. These included body mass index (BMI), defined as weight (in kilograms) divided by height (in meters squared), sum of skinfold thicknesses (SUM), ratio of subscapular to triceps skinfold thickness (STR), and ratio of waist to hip circumference (WHR). The intercorrelations among the various anthropometric measures are shown in matrix form in Table 1. Table 2 shows the variation in the means of different anthropometric measures in six age-sex groups.

Using commonly used categories of BMI suggested by some authors [e.g., Bray (1989)], namely, <26 , $26-30$, and >30 , with the last two labeled as "overweight" and "obese," respectively, we see that, compared with the Manitoba population, a greater proportion of Keewatin Inuit women have a BMI exceeding 26 ($p = 0.012$ for age group <35 years; $p = 0.134$ for age group $35-49$ years; $p = 0.095$ for age group $50+$ years), whereas among men the difference is not statistically significant (Figure 2).

For men the two populations have similar age-sex-specific mean BMIs, whereas for women the Keewatin values are generally higher, with the greatest difference seen in women age $35-49$ years, where the mean BMI among the Inuit is 28.5 [95% confidence interval (CI): 27.1, 29.9], compared with a Manitoba mean of 26.5 (95% CI: 25.8, 27.2). Inuit women have significantly higher age-specific mean WHRs than Manitobans in all three age groups, whereas Inuit men have generally lower WHRs (Figure 3). Among Inuit men under 35 years of age the mean WHR is 0.858 (95% CI: 0.846, 0.870), compared with a Manitoba mean of 0.894 (95% CI: 0.887, 0.901).

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

Table 3 compares the age-adjusted means of various metabolic variables for each sex separately between quartiles of BMI, SUM, and WHR. For WHR both age and BMI are adjusted to determine whether centrality has an effect on metabolic variables independent of overall obesity represented by BMI.

It can be seen that higher blood pressure levels and an unfavorable lipid profile (high TC, TG, LDL, and/or low HDL) are present at higher quartiles of BMI and SUM. No significant differences in glucose and insulin levels between the BMI and SUM categories were detected. As a centrality index, WHR independent of BMI has little effect on any of the metabolic indexes. The absence of variation in fasting glucose level across BMI or SUM quartiles is observed whether it is based on capillary samples read by glucometer or on plasma samples determined by hexokinase methods in the laboratory.

Even though blood pressure, TC, LDL, and TG levels tend to increase and HDL level tends to decrease with increasing levels of an obesity indicator in both populations, the pattern of response is not identical. Figure 4 shows the relationship between blood pressure and the three levels of BMI in the

Table 1. Correlation Matrices for Anthropometric Variables, Keewatin Inuit

	Weight (kg)	Height (cm)	BMI	Waist Circumference	Hip Circumference	WHR	Triceps Skinfold Thickness (mm)	Subscapular Skinfold Thickness (mm)	STR
Males									
Height	0.56	—							
BMI	0.87	0.09	—						
Waist circumference	0.89	0.29	0.91	—					
Hip circumference	0.89	0.43	0.82	0.87	—				
WHR	0.68	0.10	0.76	0.88	0.54	—			
Triceps skinfold thickness	0.35	0.10	0.38	0.37	0.37	0.27	—		
Subscapular skinfold thickness	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
Females									
Height	0.36	—							
BMI	0.90	—0.07	—						
Waist circumference	0.85	0.01	0.90	—					
Hip circumference	0.92	0.21	0.89	0.85	—				
WHR	0.46	—0.21	0.58	0.80	0.37	—			
Triceps skinfold thickness	0.74	0.07	0.76	0.72	0.75	0.42	—		
Subscapular skinfold thickness	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

Numbers in table refer to Pearson correlation coefficients. See text for definitions of abbreviations.

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Table 2. Means (and Standard Errors) of Anthropometric Measures by Age and Sex among Keewatin Inuit

Anthropometric Measure	Male Age (Years)			Female Age (Years)		
	<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 circumference (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)
Hip circumference (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)
Tricep skinfold thickness (mm)	13.9 (0.88)	16.3 (1.97)	13.5 (1.29)	21.1 (0.73)	25.7 (1.43)	25.4 (1.51)
Subscapular skinfold thickness (mm)	15.2 (1.02)	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)

two populations. For blood pressure the rise-with-BMI curves are close together and not significantly different. The divergence between the Keewatin Inuit and the Manitoba sample is more clearly evident in the lipid-BMI curves, as shown in Figure 5. For each category of BMI the Keewatin sample tends to have lower TG levels ($p < 0.05$ for all BMI categories among men and above 25 in women) and higher HDL levels ($p < 0.05$ for BMI category 26-30 among men and under 30 among women).

BMI and WHR are further analyzed as continuous variables in stepwise multiple regression analyses to predict blood pressure, lipid, and glucose levels (Table 4) with this general model:

$$\log DV = \text{intercept} + b_1(\text{age}) + b_2(\log \text{BMI}) + b_3(\log \text{WHR}), \quad (1)$$

where DV is the dependent variable, for example, blood pressure, glucose, insulin, or lipid level.

Several observations can be made from Table 4. Among the Keewatin Inuit obesity is again shown to have little effect on fasting or 2-hr glucose level. A significant effect on fasting blood pressure, TG, and HDL levels can be seen in both men and women and on TC and fasting insulin levels among men only. In the presence of BMI, WHR rarely emerges as an independent predictor of metabolic effects. In the Manitoba sample both BMI and WHR are significant independent predictors of most metabolic variables, with the

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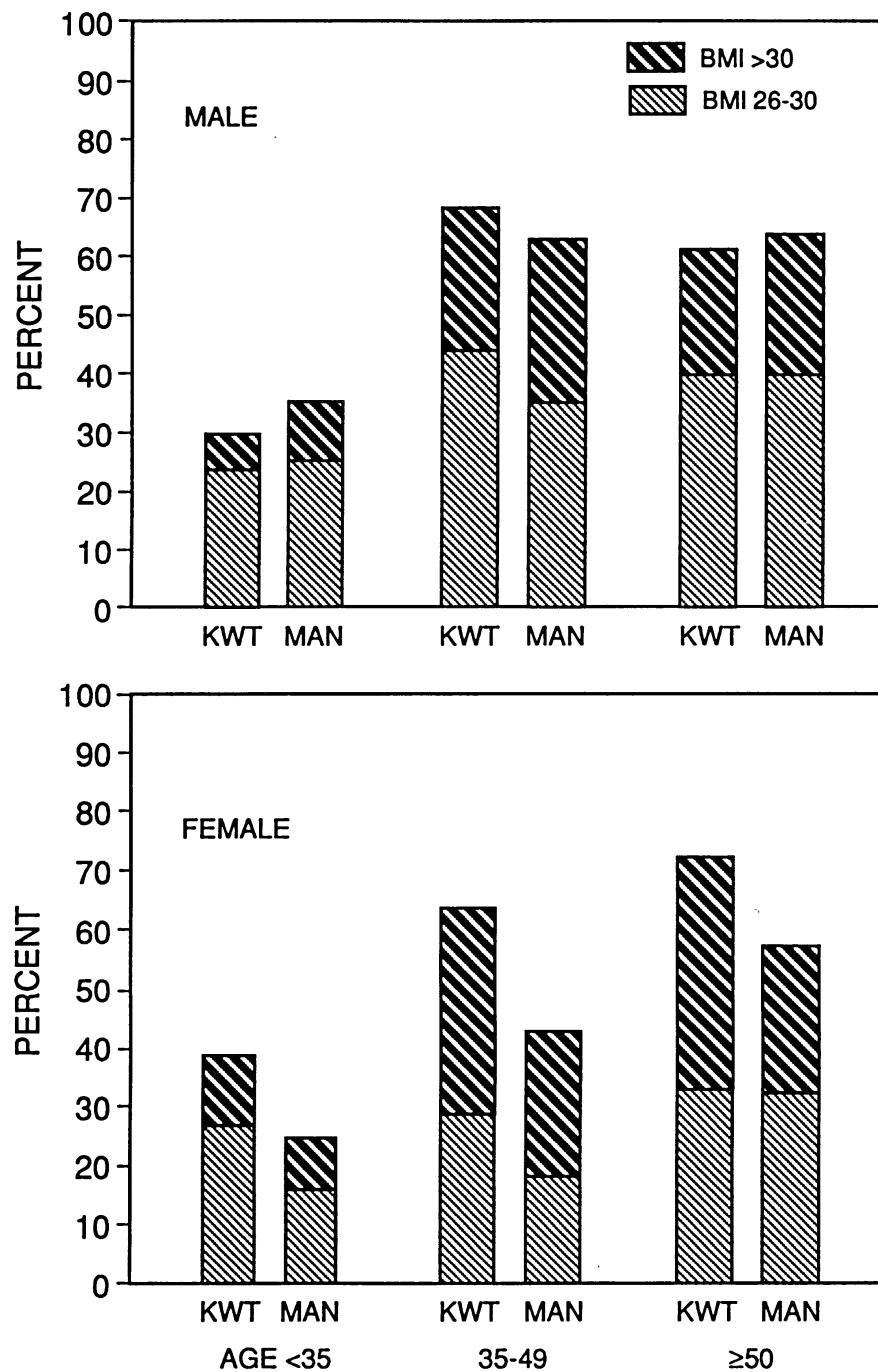


Figure 2. Prevalence of obesity (BMI = 26–30 and BMI > 30) by age and sex for Keewatin Inuit (KWT) and Province of Manitoba (MAN).

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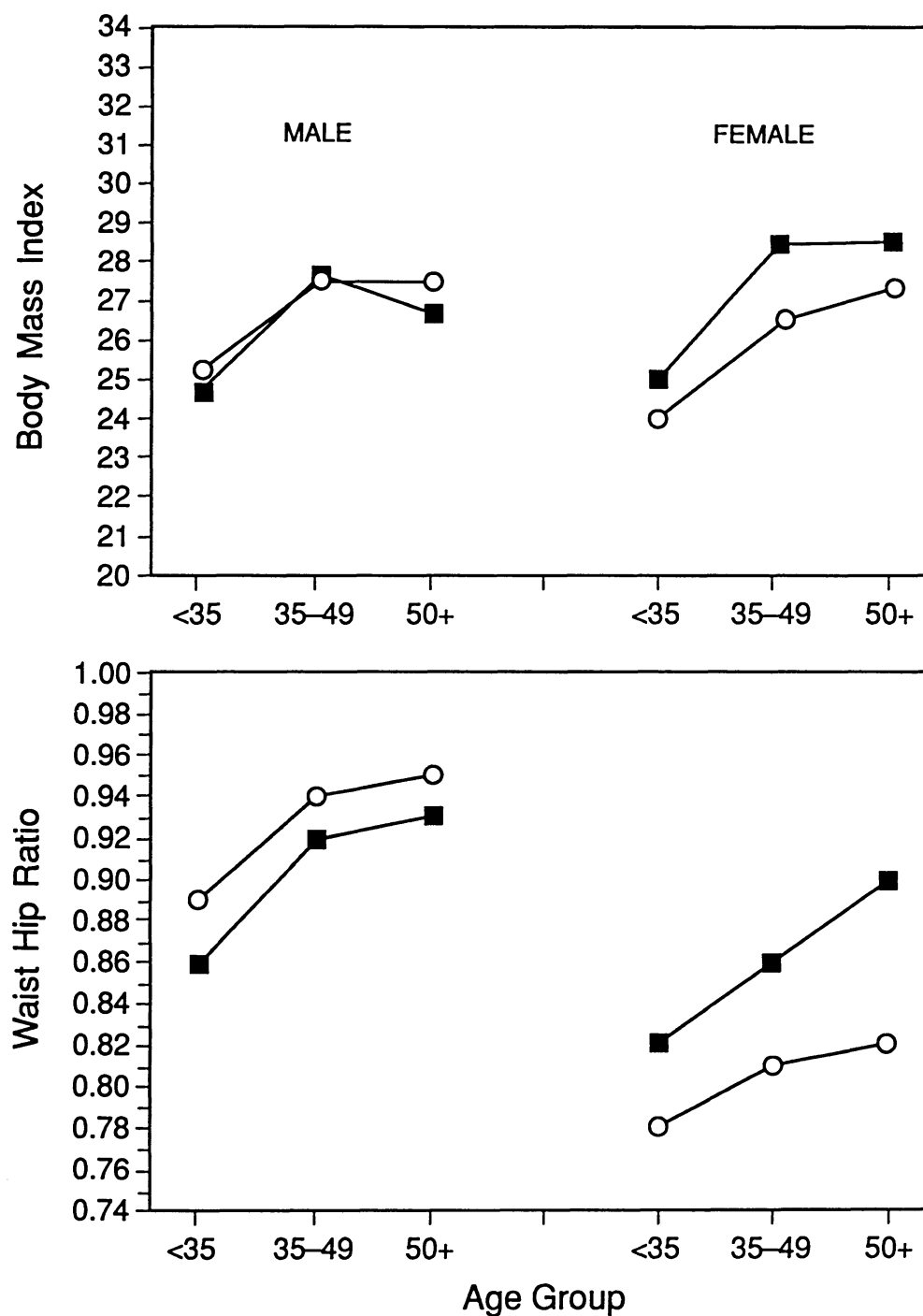


Figure 3. Mean body mass index (BMI) and waist-to-hip ratio (WHR) by age and sex for Keewatin Inuit (squares) and Province of Manitoba (circles).

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Table 3. Comparison of Sex-Specific and Age-Adjusted Means of Selected Metabolic Variables According to Quartiles (Q) of Body Mass Index (BMI), Sum of Skinfold Thicknesses (SUM), and Waist-to-Hip Ratio (WHR): Keewatin Inuit

<i>Metabolic Variable</i>	<i>Males</i>					<i>Females</i>				
	<i>Min-Q1</i>	<i>Q1-Q2</i>	<i>Q2-Q3</i>	<i>Q3-Max</i>	<i>p^a</i>	<i>Min-Q1</i>	<i>Q1-Q2</i>	<i>Q2-Q3</i>	<i>Q3-Max</i>	<i>p^a</i>
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
Sum of skinfold thicknesses										
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
Waist-to-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

Min, minimum; Q1, Q2, Q3, first, second, and third quartile, respectively; Max, maximum.

SBP, systolic blood pressure; DBP, diastolic blood pressure; FG, fasting glucose; 2hG, two-hour glucose; FIns, fasting insulin; TC, total cholesterol; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglycerides.

a. Italicized numbers mean that $p < 0.05$ (i.e., significant).

exception of diastolic blood pressure and LDL levels in women, where only BMI is predictive. In contrast to the Inuit, both BMI and WHR are predictors of fasting glucose level in the Manitoba sample. In regression models where the independent variables include age and triceps (TRI) and subscapular (SUB) skinfold thicknesses (data not shown), only SUB predicts systolic and diastolic blood pressure, HDL, and TG levels in both sexes and fasting insulin

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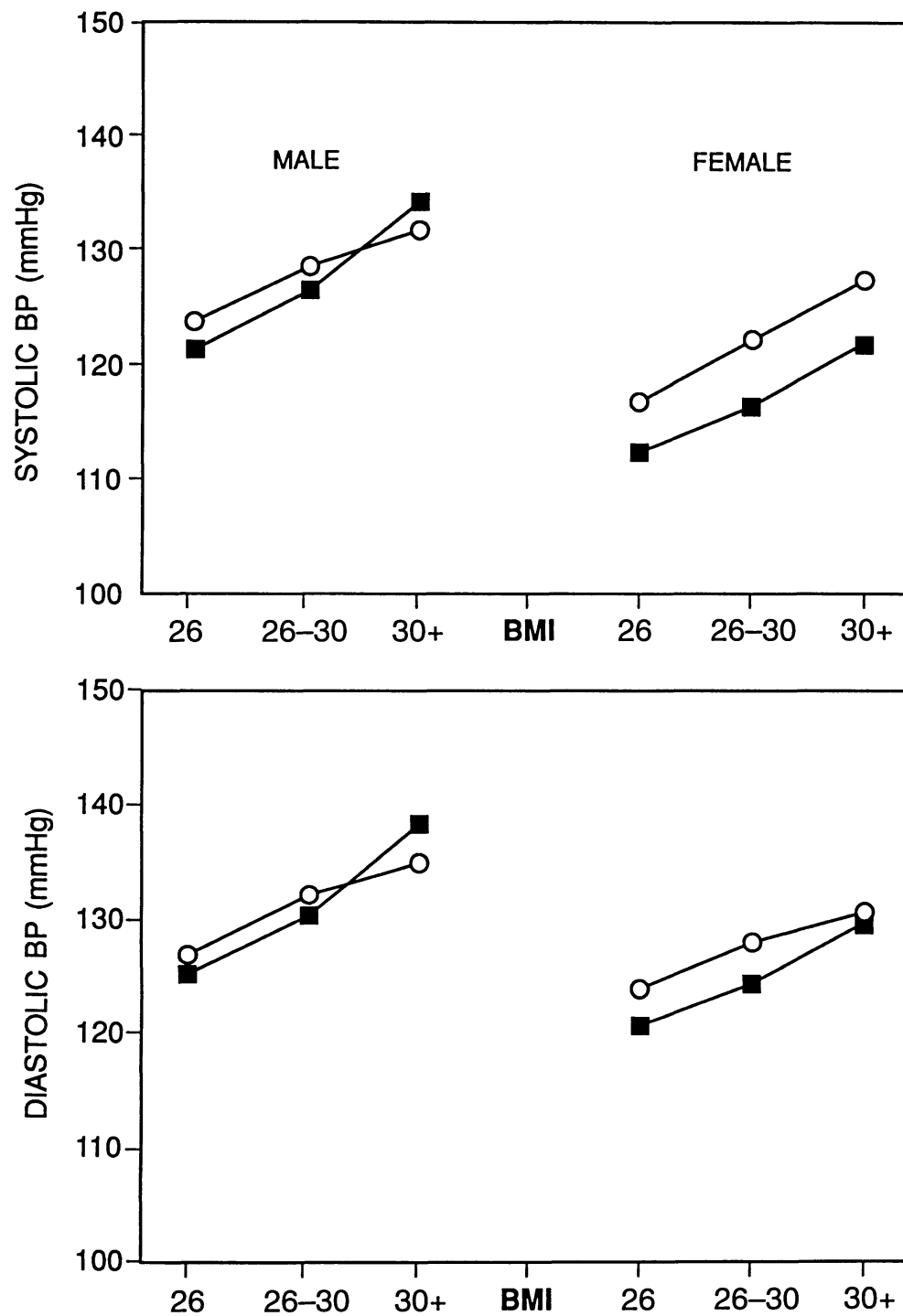


Figure 4. Age-adjusted sex-specific mean systolic and diastolic blood pressure levels by categories of body mass index (BMI) for Keewatin Inuit (squares) and Province of Manitoba (circles).

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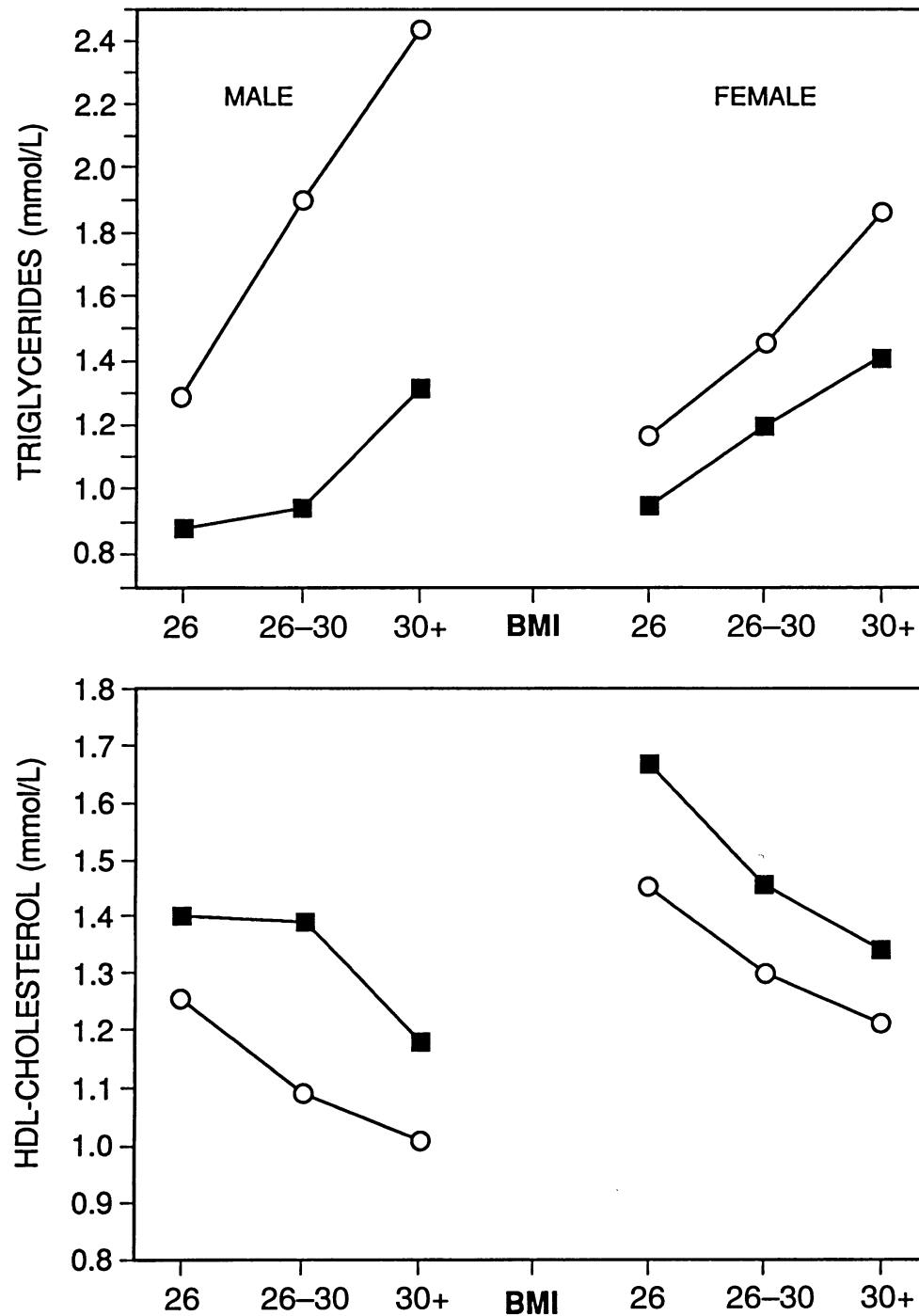


Figure 5. Age-adjusted sex-specific mean TG and HDL levels by categories of body mass index (BMI) for Keewatin Inuit (squares) and Province of Manitoba (circles).

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Table 4. Significant Independent Predictors of Selected Metabolic Variables Established by Stepwise Multiple Regression Analyses: Keewatin Inuit and Province of Manitoba

Dependent Variable	Independent Predictor (Standardized Regression Coefficient)							
	Keewatin Inuit				Province of Manitoba			
	Male	R ²	Female	R ²	Male	R ²	Female	R ²
SBP	Age (0.200)	0.137	Age (0.379)	0.303	Age (0.263)	0.208	Age (0.433)	0.352
					BMI (0.209)		BMI (0.218)	
	BMI (0.273)		BMI (0.303)		WHR (0.113)		WHR (0.114)	
DBP	Age (−0.196)	0.137	BMI (0.396)	0.157	Age (0.220)	0.254	Age(0.389)	0.304
					BMI (0.259)			
	BMI (0.355)				WHR (0.167)		BMI (0.292)	
FG	not significant	0.002	not significant	0.026	Age (0.207)	0.134	Age (0.220)	0.147
					BMI (0.173)		BMI (0.173)	
					WHR (0.092)		WHR (0.119)	
2hG	Age (0.201)	0.040	Age (0.182)	0.033	Not available		Not available	
FIns	BMI (0.617)	0.188	not significant	0.050	Not available		Not available	
	WHR (−0.369)							
TC	Age (0.413)	0.293	Age (0.442)	0.196	Age (0.362)	0.232	Age (0.420)	0.237
					BMI (0.010)		BMI (0.085)	
	BMI (0.265)				WHR (0.134)		WHR (0.089)	
HDL	Age (0.511)	0.262	Age (0.264)	0.147	BMI (−0.294)	0.134	Age (0.117)	0.115
							BMI (−0.260)	
	BMI (−0.250)		BMI (−0.389)		WHR (−0.111)		WHR (−0.480)	
LDL	Age (0.226)	0.062	Age (0.375)	0.141	Age (0.364)	0.218	Age (0.380)	0.197
					BMI (0.092)			
	WHR (0.301)				WHR (0.111)		BMI (0.139)	
TG	BMI (0.248)	0.062	BMI (0.394)	0.156	Age (0.136)	0.246	Age (0.168)	0.248
					BMI (0.264)		BMI (0.267)	
					WHR (0.223)		WHR (0.219)	

Independent variables: age, log BMI, log WHR.

SBP, systolic blood pressure; DBP, diastolic blood pressure; FG, fasting glucose level; 2hG, two-hour glucose level;

FIns, fasting insulin level; TC, total cholesterol; HDL, high-density lipoprotein; LDL, low-density lipoprotein;

TG, triglycerides.

BMI, Body mass index; WHR, waist-to-hip ratio.

and LDL levels in men, whereas TRI appears to have a negative effect on TG level among women in the presence of SUB. Neither skinfold has any effect on fasting or 2-hr glucose level.

Discussion

Both BMI and SUM were highly correlated with weight but poorly correlated with height. They were also highly correlated with each other. As measures of the centrality of fat distribution, WHR was less satisfactory than STR because WHR, particularly among men, 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 are comparable with those reported for Cree-Ojibwa Indians (Young and Sevenhuysen 1989) and Mexican Americans (Haffner et al. 1987). Garn et al. (1986) noted that BMI does not completely reflect obesity because it also correlates with relative sitting height and lean body mass. Norgan (1994) also demonstrated that across populations BMI correlates with relative sitting height. The older physical anthropology literature has shown that the Inuit tend to be short-legged [see Szathmary (1984)]. The Nutrition Canada survey of the early 1970s confirmed that relative sitting height of Inuit men and women is higher than that of Canadians nationally (Demirjian 1980).

The Keewatin skinfold data can be compared with the US white population, on which nationally representative surveys have been conducted and reported periodically. Data from the National Health and Nutrition Examination Surveys of 1971–1974 and 1976–1980 (NHANES I and II) were pooled and published in tables of anthropometric standards by Frisancho (1990). The Inuit mean triceps and subscapular skinfold thicknesses generally fall between the 50th and 75th percentile.

The Keewatin data can also 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) and the WHO MONICA Study (World Health Organization 1989). The Keewatin Inuit BMIs compare with ranges reported from many developed countries but are generally higher than those from many developing countries in Asia and Africa and unacculturated Amazonian Indians.

Since the middle of the twentieth century, the lifestyle of the Inuit has shifted substantially 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. Although Shephard et al. (1973) and Rode and Shephard (1994a,b) provided longitudinal data on changes in Inuit physique and fitness from the community of Igloolik, it would be reasonable to assume that similar changes also occurred elsewhere in the Arctic.

Based on both BMI and skinfold thicknesses, I conclude that obesity among the Inuit in the Keewatin region is as prevalent as it is in the general North American population. Obesity is more prevalent among women, among whom there is also a higher degree of central fat patterning.

Another major change that the Inuit have experienced is in their diet and nutritional status (Nobmann et al. 1992; Draper 1977; Department of National Health and Welfare 1975). Periodic starvation associated with hunting failures occurred as recently as the 1940s, and 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, particularly in the proportion of

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energy derived from carbohydrates in general and refined sugar in particular. Although the biochemical relationship between marine-mammal- and fish-based fatty acids, serum lipid levels, and obesity is complex, a decline in the diet of traditional foods may also operate in reducing the relative protection from cardiovascular diseases and diabetes that the Inuit have enjoyed until relatively recently (Young et al. 1992, 1993).

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 the cross-sectional data presented here, one can compare obese and nonobese Inuit in terms of the associated metabolic effects.

The relationship between obesity and metabolic effects among the Inuit differs in significant respects from the general population of Manitoba, among whom increasing obesity tends to be associated with higher levels of fasting glucose. Even though there is generally an increasing trend in blood pressure and lipid levels with obesity in both populations, the magnitude of response differs, particularly with respect to triglyceride and HDL cholesterol levels. At each level of BMI Manitobans have higher mean triglyceride and lower mean HDL levels than the Inuit.

The metabolic patterns associated with obesity observed in this study bear some similarity to older published reports in the western Northwest Territories and Alaska, although a direct comparison is difficult because of the different statistical approaches and the method of data presentation. A study of hospitalized Inuit in the western Arctic 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 (Schaefer et al. 1972). In a study on diabetes in Alaska Mouratoff and Scott (1973) reported that fasting blood glucose level was correlated with differential weight in men but not in women, whereas no correlation was found with the 2-hr values. "Differential weight" in Mouratoff's study was defined as the excess weight over a white American standard of the same age, sex, and height.

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 is composed predominantly of proteins and fats, with carbohydrates playing an insignificant role (Draper 1977). This means that the metabolic mechanisms in place to handle the postprandial upsurge in glucose—insulin binding, glucose uptake, glucose transport—would be much less active. The main source of glucose, needed for essential functions in the brain and erythrocytes, must be derived endogenously through gluconeogenesis in the liver. The substrates are amino acids from proteolysis and glycerol from lipolysis, the availability of which largely depends on dietary intake of proteins and fats.

DeFronzo and Ferrannini (1991) recognized that insulin resistance is the key metabolic defect in obesity and related disorders. Insulin resistance

is a constellation of metabolic processes manifested by various plasma markers, among which hyperinsulinemia is the most characteristic. Insulin resistance has two divisions, one involving glucose disposal and the other lipid oxidation. The concept of selective insulin resistance has been proposed for such groups as the Pima (Howard et al. 1991) 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. Hepatic gluconeogenesis is no longer suppressed by insulin, whereas lipogenesis continues to be stimulated by insulin. The result is excessive peripheral glucose and increased fat stores. Among the Inuit it appears that insulin resistance operates in the opposite manner. That increasing body mass has no effect on fasting glucose or 2-hr glucose level indicates that the ability to dispose of glucose is 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.

Another observation from the Keewatin study is the lower prevalence of central fat patterning in men and the higher prevalence in women compared with whites. 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. Because central fat is supposed to be more active metabolically, particularly in the mobilization of fat stores, its relative inertness in the Inuit may explain the generally lower levels of triglycerides, total cholesterol, and LDL cholesterol (and the opposite in HDL cholesterol) in 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 similarity in blood pressure increase with obesity between the Inuit and non-Inuit suggests that the mechanism may be separate from glucose and fat metabolism. The epidemiologic evidence linking hyperinsulinemia and hypertension is consistent, and Donahue et al. (1990) believed that it is mediated through an increase in circulating epinephrine and norepinephrine and increased sodium reabsorption by distal renal tubules. In this respect there may be little difference between the Inuit and other populations.

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 change through the deposition of fat is the only viable long-term adaptation for survival in the Arctic. Shephard (1991) suggested that adaptation favors the deposition of intra-abdominal fat, which stores quickly available fuel for heat production in response to cold-induced catecholamine stimulation, whereas the primary purpose of subcutaneous fat is insulation. Shephard (1991) maintained that it is intra-abdominal fat rather than subcutaneous fat that has traditionally been associated with the Inuit.

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Inuit men have lower WHRs than other populations, and 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 fat rather than intra-abdominal fat. In Bouchard's (1991) scheme Inuit obesity may be predominantly type II rather than type III.

The genes for obesity could have been selected for their value in providing insulation and fuel storage among early hominids as they penetrated the temperate and subarctic biomes. 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 been perpetuated in the population, particularly if the type of obesity was metabolically inert and its continued presence was not detrimental.

In most other populations obesity, especially central fat, can cause disease. That such obesity-related diseases have not been prominent among the Inuit (Schaefer 1981) lends support to the notion 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 promoting factors that can overcome the metabolic protection that has existed for several thousand years. Compared with many North American native populations, the Inuit are metabolically different. If this is predominantly an effect of acculturation, it would suggest that the Inuit have not been subjected to acculturation long enough or intensely enough. Alternatively, the Inuit may be more resistant to the influence of acculturation. Although genetic susceptibility may be invoked, the continuing (albeit much reduced) importance of the traditional diet and physical activity pattern may also be responsible.

Acknowledgments I wish to acknowledge the active support and participation of local governments, health agencies, and community members in the Keewatin region of the Northwest Territories, Canada. This paper was prepared while I was on sabbatical at the Institute of Biological Anthropology, University of Oxford, and benefited from the critical comments of G.A. Harrison. Acknowledgment is also extended to fellow co-investigators of the Keewatin Health Assessment Study (M.E.K. Moffatt and J.D. O'Neil) and the Manitoba Heart Health Survey (D.E. Gelskey and S.M. Macdonald) for their collaboration during the planning and data collection phases of these two studies. Funding for the Keewatin Health Assessment Study was provided by the National Health Research and Development Program (NHRDP) of the Canadian Department of National Health and Welfare through grant 6607-1550-T. The Manitoba Heart Health Project also was supported by the NHRDP through grant 6607-1553-H.

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Received 7 February 1995; revision received 17 July 1995.

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