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ORIGINAL ARTICLE

Characteristics and prevalence of the metabolic syndrome among three ethnic groups in Canada

J Liu[1], AJG Hanley[1,2,3], TK Young[1], SB Harris[4] and B Zinman[2,3]

[1]*Department of Public Health Science, University of Toronto, Toronto, Ontario, Canada;* [2]*Leadership Sinai Centre for Diabetes, Mt Sinai Hospital, Toronto, Ontario, Canada;* [3]*Department of Medicine, University of Toronto, Toronto, Ontario, Canada and* [4]*Centre for Studies in Family Medicine, University of Western Ontario, London, Ontario, Canada*

Objective: To compare the characteristics and prevalence of the metabolic syndrome (MetS) among Native Indians, Inuit, and non-Aboriginal Canadians.
Methods: The study was based on four cross-sectional studies conducted in the late 1980s and early 1990s involving three ethnic groups living in contiguous regions in central Canada: Oji-Cree Indians from several reserves in northern Ontario and Manitoba, Inuit from the Keewatin region of the Northwest Territories, and non-Aboriginal Canadians (predominantly of European heritage) in the province of Manitoba. The MetS was identified among adult subjects according to the National Cholesterol Education Program (NCEP) definition. Prevalence rates were standardized to the 1991 Canadian national population.
Results: The age-standardized prevalence of the MetS varied by ethnic group, ranging from as high as 45% among Native Indian women to as low as 8% among Inuit men. Compared with Canadians of European origin, Indians had a worse metabolic profile, while Inuit had a better metabolic profile except for a high rate of abdominal obesity. The NCEP criteria in identifying individuals with the MetS were compared to those of the World Health Organization (WHO) in a subset of subjects with the requisite laboratory data. There was moderate agreement between the NCEP and WHO definitions, with a kappa value of 0.63 (95% confidence interval 0.56–0.70).
Conclusions: The results indicate that the MetS is prevalent in diverse ethnic groups in Canada but varies in the pattern of phenotypic expression. Given the diverse nature of these populations, careful consideration should be given to developing culturally appropriate community-based prevention strategies aimed at reducing the frequency of this syndrome.
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Keywords: metabolic syndrome; North American Indians; Inuit; lipids; diabetes; hypertension

Introduction

A cluster of cardiovascular risk factors including central obesity, hypertension, glucose intolerance, and dyslipidemia, currently referred to as the metabolic syndrome (MetS), has been extensively investigated for several decades.[1–6] Whether risk factor clusters collectively indicate a discrete unifying disorder is unclear, and mechanisms underlying the link between cardiovascular disease (CVD) risk factors remain uncertain. However, many investigators have suggested that obesity-associated insulin resistance and/or compensatory hyperinsulinemia is a common pathogenic factor for the associations among individual components of the MetS.[1–5] It has been found that the MetS is associated with a risk of coronary heart disease, stroke, and CVD mortality greater than that of its individual components,[7] and is a significant predictor of incident type 2 diabetes.[8]

CVD and type 2 diabetes are considered relatively new diseases in Aboriginal communities in Canada.[9–11] Low CVD mortality rates were found among Aboriginal populations in Canada compared with general populations prior to 1980s.[10,11] However, with significant social, economic, and cultural changes over the past several decades, Aboriginal people have been undergoing a health transition, with declining rates of communicable diseases and increasing chronic illnesses. Obesity and type 2 diabetes have reached an epidemic stage in some Canadian Aboriginal communities.[9,12] Among the Native Indian population of the province of Ontario, the rate of

Correspondence: Professor TK Young, Department of Public Health Sciences, Banting Institute, University of Toronto, 100 College Street, Suite 207B, Toronto, Ontario, Canada M5G 1L5.
E-mail: kue.young@utoronto.ca
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hospitalizations for ischemic heart disease has doubled between 1981 and 1997, despite declining rates in the general population.[13]

Both the World Health Organization (WHO) and the National Cholesterol Education Program (NCEP) Adult Treatment Panel III have published working definitions of the MetS,[14–17] and the prevalence rates of the MetS have been assessed in various populations around the world.[18–31] However, little is known about the distribution of MetS among Aboriginal Canadians. The aim of this study was to estimate the prevalence of MetS and describe its characteristics between two Canadian Aboriginal groups (Native Indians and Inuit) compared to non-Aboriginal Canadians of predominantly European origin.

Methods

Data source

This report was based on data from four cross-sectional, population-based surveys: Northern Indians Chronic Disease Study (NICDS 1986–1987), Manitoba Heart Health Survey (MHHS 1989–1990), Keewatin Health Assessment Study (KHAS 1990–1991), and Sandy Lake Health and Diabetes Project (SLHDP 1993–1995) (Figure 1). The NICDS, MHHS, and KHAS were approved by the Committee on the Use of Human Subjects in Research of the Faculty of Medicine, University of Manitoba. The University of Toronto Research Ethics Committee approved the SLHDP. Signed informed consent was obtained from all individual participants and the studies were approved by all community councils. The samples were representative of the noninstitutionalized adult population in their respective catchment areas. These surveys employed comparable methods in anthropometric measurement, blood pressure, and laboratory analyses for fasting plasma glucose and serum lipids. A complete description of the methods used has been given in detail in previous publications.[9,12,32–38] Waist circumference was measured as the abdomen midway between the lower rib margin and the iliac crest in NICDS, as the narrowest circumference below the lower rib in MHHS and KHAS, and the minimal circumference between the umbilicus and xiphoid process in SLHDP. This slight difference in measurement is unlikely to contribute to substantial variation of the prevalence of the MetS between the surveys. The protocol for other anthropometric measurements and blood pressure was similar for all studies. In KHAS, capillary plasma glucose was determined in most participants, with slightly more than half also having a current venous sample. The capillary plasma glucose values in individuals without venous samples were converted by a linear regression equation adjusting for age and sex. In total, the number of subjects who were $\geqslant 18$ years of age at the time of surveys and had all the relevant data available included 666 Oji-Cree Indians from NICDS, 514 Oji-Cree Indians from SLHDP, 238 Inuit from KHAS, and 2058 non-Aboriginal Canadians from MHHS.

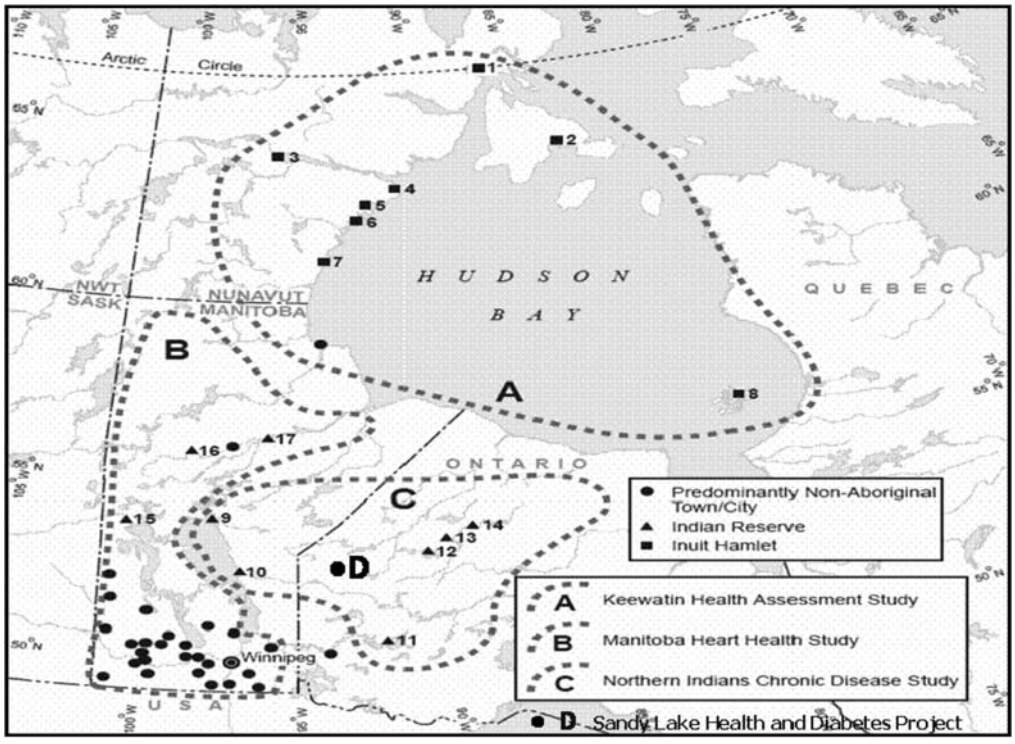

Figure 1 Map of the study areas of the SLHDP, NICDS, KHAS, and MHHS.

Definition of the MetS

According to the NCEP criteria,[16,17] subjects were considered to have MetS if they had any three or more of the following five disorders: (1) abdominal obesity: waist circumference >102 cm in men and >88 cm in women; (2) hypertriglyceridemia: ⩾1.69 mmol/l (⩾150 mg/dl); (3) low high-density lipoprotein cholesterol (HDL-C): <1.04 mmol/l (<40 mg/dl) in men and <1.29 mmol/l (<50 mg/dl) in women; (4) high blood pressure: ⩾130/85 mmHg; (5) high fasting glucose: ⩾6.1 mmol/l (⩾110 mg/dl). In addition, subjects who had previous diagnosis or treatment for hypertension or diabetes were considered as having high blood pressure or high fasting glucose, respectively.

The prevalence of the MetS defined by a modification of the WHO definition[16] was also calculated among subjects from SLHDP. A participant had WHO-defined MetS if he or she had diabetes, impaired glucose tolerance, impaired fasting glucose or insulin resistance, plus two or more of the following abnormalities: (1) high blood pressure: ⩾140/90 mmHg or previous diagnosed hypertension with treatment; (2) dyslipidemia: triglyceride concentration ⩾1.69 mmol/l (⩾150 mg/dl) and/or HDL cholesterol <0.9 mmol/l (<35 mg/dl) in men and <1.0 mmol/l (<39 mg/dl) in women; (3) central obesity: waist-to-hip ratio of >0.90 in men or >0.85 in women and/or body mass index (BMI) >30 kg/m^2. Insulin resistance was defined as the highest quartile (>4.18 units) of the distribution of the homeostasis model assessment (HOMA) index of insulin resistance among subjects with normal glucose tolerance, calculated from the following equation: $HOMA_{IR}$ = fasting insulin (μU/ml) × fasting plasma glucose (mmol/l)/22.5.[39] Fasting plasma insulin was determined by radioimmunoassay (Pharmacia) with an interassay coefficient of variation (CV) of 7.2–8.8%. Lastly, microalbuminuria data were not available for SLHDP participants; so this component was not considered to calculate the prevalence of the WHO-defined MetS.

Additionally, a subject was considered to be obese if his or her BMI was equal to, or greater than, 30 kg/m^2 according to the WHO classification.[40]

Statistical analysis

Analyses were performed using the Statistical Analysis System (SAS, version 8.2, SAS Institute). Data from Native Indians from SLHDP and NICDS were pooled for analyses, as participants were very closely affiliated linguistically and culturally. Means and standard deviations, medians and interquartile ranges (for skewed variables), or proportions were presented by ethnicity. The study populations were divided into six age groups, with ⩾55 years as the oldest age group due to the young age structure of Aboriginal populations in Canada.[41] The crude and age-specific prevalence of metabolic abnormalities was calculated by gender and ethnicity. Prevalence rates were standardized to the 1991 Canadian national population[42] and presented as percentages with 95% confidence intervals (CI). To examine an independent effect of ethnicity on the MetS as defined by NCEP, a logistic regression model, including the potential confounders age, sex, and BMI, was performed. (Note that BMI is not part of the criteria for MetS.) A Kappa value was used to assess the agreement between the WHO and NCEP definitions among SLHDP subjects.

Results

The characteristics and crude prevalence of metabolic components among the three ethnic groups are presented in Tables 1 and 2. Among Native groups, the mean age was 37 years and 56% were women. Among non-Aboriginal subjects, the mean age was 50 years and 49% were women (Table 1). The overall crude rate of the NCEP-defined MetS was 33.3% among Native Indians, 13.5% among Inuit, and 30% among non-Aboriginal subjects. The crude rates of the

Table 1 Characteristics of the study populations: Oji-Cree Indians, Inuit, and non-Aboriginal Canadians, aged 18 years or older

Variable	*Oji-Cree Indians* (n = *1180*)	*Inuit* (n = *238*)	*Non-Aboriginal* (n = *2058*)
Age (year)[a]	36.6±13.5	36.7±15.2	49.7±18.0
Gender (% women)	56	56	49
Waist circumference (cm) [a]	96.6±13.1	88.3±12.4	88.6±13.9
BMI (kg/m^2)[a]	27.9±5.4	26.6±4.46	26.5±4.84
Systolic BP (mmHg)[a]	125.2±17.2	120.3±17.3	129.5±18.9
Diastolic BP (mmHg)[a]	74.7±12.7	75.8±10.2	78.2±9.47
FPG (mmol/l)[b]	5.10 (4.50–5.80)	5.10 (4.77–5.45)	5.34 (4.99–5.77)
Triglycerides (mmol/l)[b]	1.3 (0.94–1.84)	0.93 (0.69–1.27)	1.33 (0.94–1.94)
HDL-C (mmol/l)[a]	1.25±0.32	1.44±0.40	1.25±0.33
Current smoking (%)[c]	51.7 (47.5–55.9)	59.9 (49.5–70.3)	25.6 (23.2–28.1)
Obesity (%, BMI ⩾30)[c]	36.3 (32.4–40.3)	28.7 (21.1–36.2)	18.3 (16.4–20.3)
Previous diabetes (%)[c]	13.6 (11.0–16.2)	1.4 (0.0–2.9)	5.2 (4.2–6.1)
Hypertension under treatment (%)[c]	16.2 (13.2–19.1)	7.3 (3.4–11.2)	24.6 (22.4–26.7)

Data presented are: [a]Means±s.d. [b]Medians (interquartile range). [c]Percentage (95% CI), directly standardized to the 1991 Canadian population; BMI, body mass index; BP, blood pressure; FPG, fasting plasma glucose; HDL-C, high-density lipoprotein cholesterol.

Table 2 Crude prevalence of NCEP-defined metabolic syndrome and components among Oji-Cree Indians, Inuit, and non-Aboriginal Canadians, aged 18 years or older

	Oji-Cree Indians			*Inuit*			*Non-Aboriginal*		
	Total (n = *1180*)	*Men* (n = *51*)	*Women* (n = *666*)	*Total* (n = *238*)	*Men* (n = *105*)	*Women* (n = *133*)	*Total* (n = *2058*)	*Men* (n = *1055*)	*Women* (n = *1003*)
Abdominal obesity	56.2	35.4	72.2	29.0	11.4	42.9	26.0	22.8	29.2
Hypertriglyceridemia	30.2	32.7	28.2	8.8	5.7	11.3	33.5	37.3	29.5
Low HDL Cholesterol	45.9	33.1	55.9	26.5	17.1	33.8	42.5	39.1	46.2
High blood pressure[a]	42.0	51.2	34.8	36.6	43.8	30.8	54.0	57.3	50.6
High fasting glucose[b]	21.9	22.0	21.8	6.3	3.8	8.3	17.8	20.3	15.3
$\geqslant$1 component	84.2	79.0	88.3	60.9	53.3	66.9	76.1	76.2	76.1
$\geqslant$2 components	59.4	50.2	66.5	30.6	20.9	38.3	50.6	53.3	48.0
$\geqslant$3 components[c]	33.3	28.2	37.2[d]	13.5	6.7	18.8[d]	29.9	30.6	29.2
$\geqslant$4 components	15.7	13.4	17.4	2.1	1.0	3.0	13.8	13.6	13.9
= 5 components	3.5	3.5	3.5	—	—	—	3.3	2.9	3.6

[a]High blood pressure including hypertension with treatment. [b]High fasting glucose including self-reported diabetes. [c]$\geqslant$3 components = metabolic syndrome. [d]Compared to men, $P<0.01$.

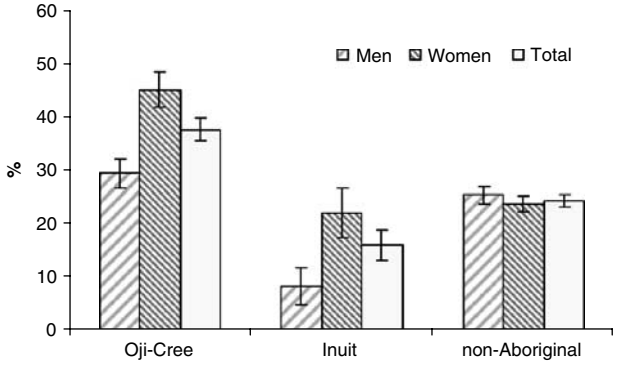

Figure 2 Age-standardized prevalence (% $\pm$s.e.) of NCEP-defined metabolic syndrome among Oji-Cree Indians, Inuit, and non-Aboriginal population.

MetS were higher in women compared with men in Native groups (Native Indians: 37.2% in women vs 28.2% in men; Inuit: 18.8% in women vs 6.7% in men; $P<0.01$). There was no gender difference among non-Aboriginal people (Table 2).

The age-standardized prevalence of the MetS varied according to ethnic group, ranging from as high as 45% among Native Indian women to as low as 8% among Inuit men (Table 3 and Figure 2). Women had markedly higher prevalence of abdominal obesity than men in the same ethnic group. Compared with non-Aboriginal people, Native Indians had significantly higher prevalence of abdominal obesity and hyperglycemia and similar rates of low HDL-cholesterol, hypertriglyceridemia, and high blood pressure. Inuit had the lowest rates of hypertriglyceridemia, low HDL cholesterol, and high fasting glucose, and intermediate rate of abdominal adiposity. The rate of high blood pressure in Inuit was similar to the rate among non-Aboriginal people.

The age-standardized prevalences of overall obesity (BMI $\geqslant 30\,kg/m^2$) were 36% (95% CI, 32–40%) among Native Indians, 29% (95% CI, 21–36%) among Inuit, and 18% (95% CI, 16–20%) among non-Aboriginal people, respectively. Logistic regression analysis showed that age and BMI were associated with a significant increased risk of the MetS. After adjustment for age, sex, and BMI, Native Indians had an increased odds of the MetS and Inuit had a decreased odds of the MetS, compared to non-Aboriginal subjects (Table 4). No interaction between ethnicity and sex or ethnicity and BMI was found (data not shown).

The prevalence of the WHO-defined MetS was also calculated among SLHDP Indians. The overall crude and age-standardized prevalence were 33 and 40.3%, respectively. No gender difference was found. There was moderate agreement between NCEP and WHO definitions, with kappa value of 0.63 (95% CI 0.56–0.70). Among all 514 participants, 26.3% ($n=135$) were diagnosed as having the MetS by both definitions, 56.6% ($n=291$) were diagnosed as not having the MetS by both definitions, 9.3% ($n=48$) had the MetS under the NCEP criteria but not the WHO criteria, and 7.2% ($n=37$) had the MetS under the WHO criteria but not the NCEP criteria. Among men, the WHO-defined MetS was slightly more frequent than the NCEP-defined MetS; the prevalence of central obesity as defined by the WHO among men was as twice as the prevalence of abdominal obesity by the NCEP, mainly because 72% of men had a waist-to-hip ratio >0.9 (Table 5). Among women, the NCEP-defined MetS was more frequent than the WHO-defined MetS, mainly due to the high rates of low HDL-cholesterol and high blood pressure as defined by the NCEP.

Discussion

In the present paper, we have confirmed that the MetS is common among the population residing in contiguous

Table 3 Age-standardized prevalence (with 95% CI) of NCEP-defined metabolic syndrome and components among Oji-Cree Indians, Inuit, and non-Aboriginal Canadians, aged 18 years or older[a]

	Oji-Cree Indians			*Inuit*			*Non-Aboriginal*		
	Total (n = *1180*)	*Men* (n = *514*)	*Women* (n = *666*)	*Total* (n = *238*)	*Men* (n = *105*)	*Women* (n = *133*)	*Total* (n = *2058*)	*Men* (n = *1055*)	*Women* (n = *1003*)
Abdominal obesity	60.3 (55.2–65.4)	38.9 (32.9–44.9)	77.4 (69.4–85.5)	34.7 (26.3–43.2)	15.8 (6.2–23.3)	47.5 (34.3–60.6)	22.5 (20.4–24.6)	19.5 (16.7–22.3)	25.6 (22.5–28.8)
Hyper-triglyceridemia	31.3 (27.8–34.8)	32.4 (27.2–37.6)	31.0 (26.0–36.1)	10.0 (5.6–14.3)	8.4(1.1–15.6)	11.6 (5.4–17.7)	29.0 (26.6–31.4)	34.0 (30.2–37.8)	24.3 (21.3–27.2)
Low HDL cholesterol	44.2 (40.1–48.2)	32.2 (27.1–37.3)	53.8 (47.5–60.1)	25.3 (18.7–31.8)	14.2 (6.6–21.9)	32.3 (22.4–42.2)	41.6 (38.6–44.7)	37.3 (33.2–41.3)	46.0 (41.5–50.4)
High blood pressure[b]	48.8 (44.0–53.4)	55.7 (48.4–62.9)	43.7 (37.2–50.2)	41.5 (32.3–50.7)	49.9 (34.0–65.8)	35.6 (23.9–47.3)	45.4 (42.5–48.4)	49.3 (44.8–53.8)	42.0 (38.0–46.0)
High fasting glucose[c]	26.4 (22.9–30.0)	24.9 (20.0–29.7)	28.3 (23.0–33.6)	8.18 (3.9–12.5)	5.1(0.0–10.4)	10.8 (4.0–17.6)	13.8 (12.2–15.3)	15.5 (13.1–17.8)	12.0 (9.9–14.0)
⩾ 1 component	87.2 (81.2–93.3)	82.3 (73.7–90.9)	90.9 (82.4–99.4)	66.0 (54.6–77.3)	58.2 (41.2–75.2)	70.7 (55.0–86.4)	71.1 (67.3–75.0)	70.7 (65.2–76.2)	71.7 (66.3–77.2)
⩾ 2 components	63.8 (58.6–69.0)	53.7 (46.7–60.7)	71.9 (64.1–79.6)	34.7 (26.4–43.0)	25.2 (13.6–36.8)	41.6 (29.4–53.8)	44.0 (41.0–47.0)	46.6 (42.2–51.0)	41.5 (37.5–45.5)
⩾ 3 components[d]	37.5 (33.4–41.6)	29.3 (24.2–34.3)	45.0 (38.6–51.5)	16.0 (10.3–21.8)	8.2 (1.4–14.9)	22.0 (12.8–31.2)	24.3 (22.2–26.5)	25.3 (22.1–28.4)	23.7 (20.8–26.6)
⩾ 4 components	18.0 (15.2–20.8)	14.7 (11.1–18.4)	21.3 (16.9–25.8)	2.7 (0.3–5.1)	1.8 (0.0–5.4)	3.4 (0.0–7.0)	10.5 (9.2–11.9)	10.38 (8.8–12.9)	10.3 (8.5–12.2)
= 5 components	4.4 (3.0–5.9)	3.9 (2.0–5.9)	5.2 (2.8–7.6)	—	—	—	2.4 (1.7–3.0)	2.1 (1.3–3.0)	2.6 (1.7–3.5)

[a]Directly standardizing to the 1991 Canadian population. [b]High blood pressure including hypertension under treatment. [c]High fasting glucose including self-reported diabetes. [d]⩾3 components = metabolic syndrome.

Table 4 Odds ratios (95% CIs) for the metabolic syndrome based on age, sex, BMI, and ethnicity[a]

Effect	*Unit*	*Odds ratio*	*95% CI*	P
Sex	Women vs men	1.060	0.889–1.265	0.5147
Ethnicity	Oji-Cree Indians vs non-Aboriginal	1.489	1.207–1.838	<0.0001
	Inuit vs non-Aboriginal	0.434	0.277–0.681	<0.0001
Age	Per 10 year increase	1.513	1.428–1.605	<0.0001
BMI	Per 5 kg/m^2 increase	3.438	3.090–3.839	<0.0001

[a]Logistic model with metabolic syndrome as dependent variable, age, sex, ethnicity, and BMI as independent variables.

regions of central Canada, with heterogeneity by sex and ethnicity. The age-standardized prevalence of the NCEP-defined MetS ranged from as high as 45% among Oji-Cree Indian women to as low as 8% among Inuit men. Our findings of ethnic difference in the prevalence rate of the MetS agree with the findings from a sample of four ethnic groups (South Asian, Chinese, European, and Native Indian) in Canada,[27] which reported Native Indians from the Six Nation reserve having the highest and people of Chinese origin having the lowest prevalence. In our study, using a unique combined data set from four contemporary large population-based studies, which employed similar methods, we investigated not only the prevalence of the MetS but also the prevalence of individual components of the syndrome with a comparison among three ethnic groups (Native Indians, Inuit, and non-Aboriginals) in Canada using direct standardization.

Consistent with studies in Native Americans[19] and Greenland Inuit,[31] Native women had a higher prevalence of NCEP-defined MetS than Native men. However, the prevalence of MetS was not significantly different between non-Aboriginal men and women, findings which are similar to those from the American whites.[18] Ethnic variability in individual components of the MetS may have an important impact on the prevalence of the MetS. In general, populations of European origin appear to be more predisposed to atherogenic dyslipidemia than other populations.[18,43] Blacks of African origin are prone to hypertension when they gain weight, they also appear to be susceptible to type 2 diabetes.[44,45] Native Americans and Hispanics are especially susceptible to type 2 diabetes, but are less likely to develop hypertension than are blacks.[23,43–45] Our analysis also revealed the ethnic variation in prevalence of the different components used to define the syndrome. Generally, compared with non-Aboriginal people, Native Indians had a worse metabolic profile with high rates of abdominal obesity and hyperglycemia, and Inuit had a better metabolic profile except for a high rate of abdominal obesity.

The obesity epidemic is considered as mainly responsible for the rising prevalence of the MetS.[6] Park *et al*.[23] reported that the prevalence of the MetS in obese subjects (BMI⩾30) was almost 12-fold higher than that in normal-weight subjects. Since the middle of the 20th century, Native

Table 5 Prevalence of metabolic syndrome and components as defined by NCEP and WHO among Oji-Cree Indians from SLHDP[a]

	NCEP			*WHO*		
	Total (n = *514*)	*Men* (n = *224*)	*Women* (n = *290*)	*Total* (n = *514*)	*Men* (n = *224*)	*Women* (n = *290*)
Metabolic syndrome	35.6	30.4	39.7	33.5	32.6	34.1
Abdominal (central) obesity[b]	57.4	37.1	73.1	77.2	75.0	79.0
BMI $\geqslant 30\,kg/m^2$	—	—	—	35.6	28.1	41.4
Waist-to-hip ratio >0.9 (M) >0.85 (W)	—	—	—	73.2	75.0	71.7
High blood pressure	27.0	32.6	22.8	15.2	17.0	13.8
Glucose abnormality	31.1	30.8	31.4	55.8	48.7	61.4
Hypertriglyceridemia	32.9	36.6	30.0	—	—	—
Low HDL Cholesterol	47.7	36.2	56.6	13.6	12.1	14.8
Dyslipidemia[c]	59.3	50.0	66.6	38.9	39.3	38.6

[a]Data presented as %. [b]Abdominal (central) obesity: for NCEP, waist circumference criteria; for WHO, combined BMI and/or WHR criteria. [c]Combined dyslipidemia: triglyceride $\geqslant$1.69 mmol/l or HDL-cholesterol <1.04 mmol/l (M), <1.29 mmol/l (W); for WHO, triglyceride $\geqslant$1.69 mmol/l or HDL-cholesterol <0.9 mmol/L (M), <1.0 mmol/l (W).

Indians and Inuit in Canada have experienced substantial changes in their lifestyle from a physically demanding hunting, trapping, and fishing nomadic life with fluctuating energy intake to a sedentary life with relatively high energy intake, leading to an epidemic of obesity in these populations.[33] In the present study, about one-third of Oji-Cree Indians and Inuit had obesity according to international criteria. Using logistic regression, the odds ratio for MetS was 3.4 with each $5\,kg/m^2$ increase in BMI. However, the ethnic disparity in the prevalence of the MetS cannot be explained entirely by ethnic discrepancy in obesity, since Inuit had more overall and abdominal obesity than non-Aboriginal subjects, yet had a much lower rate of the MetS. In addition, Native Indians had an increased odds of the syndrome and Inuit had a decreased odds of the syndrome after adjustment of age, sex, and BMI. These findings suggest that factors other than BMI (e.g. genetic predisposition, maintenance of traditional diet, or physical activity) may be playing certain roles.

Genetic research in the frequencies of putative 'deleterious' alleles from 13 candidate genes in atherosclerosis and/or diabetes among three Canadian populations[46] showed that there were significant differences in the frequencies of five of the 13 alleles between Oji-Cree and Inuit. Compared with the Inuit, the Oji-Cree has significantly higher frequencies of *AGT M174* and *MTHFR 677T* and lower frequencies of *HL-480C*, *APOE E4*, and *FABP2 T54*. However, both Oji-Cree and Inuit also had an excess of 'deleterious alleles' compared with whites.[46] The wide difference in the MetS prevalence among these groups cannot be explained entirely by differences in genetic architecture. Environmental factors, particularly the diet and level of activity, may determine disease susceptibility.

In Canada, although the ways of life of Aboriginal people have been broadly disrupted through contact with Euro-Canadians, the onset, extent, and pace of Euro-Canadian influence on Inuit and Native Indian lifestyle vary, which may explain the variation in the burden of so-called 'western diseases' such as diabetes, CVD, and hypertension.[47] The difference in the MetS rates between two Aboriginal groups may be due to a greater maintenance of traditional lifestyle and diet in Inuit people[11,48] compared to Native Indians, who have had a much longer and more extensive exposure to western influence.[47] A report from SLHDP suggested that a higher consumption of processed foods and 'fast food' was associated with an increased risk of obesity and diabetes in the Oji-Cree community.[49] It has been suggested that traditional diet rich in fish and marine mammals was associated with a favorable level of serum lipids among Aboriginal people.[50–52] A recently published study in Greenland Inuit[50] reported that serum lipids were significantly associated with the degree of westernization. HDL cholesterol decreased and triglyceride increased with westernization. Contrary to the prevailing hypothesis of the negative influence of westernization on obesity for Indigenous people, one study showed that BMI and central fat patterning decrease with Westernization among Greenland Inuit women.[53]

The prevalence of the MetS is dependent on which definition is used. Since both NCEP and WHO definitions use many of the same variables including central or abdominal obesity, dyslipidemia, hypertension, and hyperglycemia, moderate agreement between the two definitions in diverse populations[22,29,32] including the SLHDP Native Indians from our study was expected. However, substantial differences exist. Only fasting plasma glucose was used to assess glycemic status in the NCEP definition,[16,17] which misdiagnosed 50 subjects with IGT and diabetes based on 2-h plasma glucose values. The high rate of overall and central obesity as defined by the WHO accounts for much of the higher prevalence of the WHO-defined MetS in men. As expected, the prevalences of high blood pressure and combined dyslipidemia were higher with the NCEP definition because of the lower thresholds used. Thus, estimates of

the prevalence of the MetS are influenced by the selection of the diagnostic criteria and the variability of evaluated populations.

In conclusion, our research has shown that the MetS is prevalent in diverse ethnic groups but varies in the pattern of phenotypic expression. These observed prevalence rates may underestimate the current prevalence of the syndrome in Native Canadian populations, as over the past decade the prevalence of obesity and diabetes in Aboriginal population has been steadily increasing.[9,54] Given the diverse nature of these populations, careful consideration should be given to developing culturally appropriate community-based prevention strategies aimed at reducing the frequency of this syndrome.[55,56]

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References

1 Reaven GM. Role of insulin resistance in human disease. *Diabetes* 1988; **37**: 1595–1607.
2 Defronzo RA, Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care* 1991; **14**: 173–193.
3 Ferrannini E, Haffner SM, Mitchell BD, Stern MP. Hyperinsulinaemia: the key feature of a cardiovascular and metabolic syndrome. *Diabetologia* 1991; **34**: 416–422.
4 Stern MP. Diabetes and cardiovascular disease: the common soil hypothesis. *Diabetes* 1995; **44**: 369–374.
5 Liese AD, Mayer-Davis EJ, Haffner SM. Development of the multiple metabolic syndrome: an epidemiologic perspective. *Epidemiol Rev* 1998; **20**: 157–172.
6 Meigs JB. Invited commentary: insulin resistance syndrome? Syndrome X? Multiple metabolic syndrome? A syndrome at all? Factor analysis reveals patterns in the fabric of correlated metabolic risk factors (review). *Am J Epidemiol* 2000; **152**: 908–911.
7 Isomaa B, Almgren P, Tuomi T, Forsen B, Lahti K, Nissen M *et al*. Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes Care* 2001; **24**: 683–689.
8 Lorenzo C, Okoloise SEM, Williams K, Stern MP, Haffner SM. The metabolic syndrome as predictor of type 2 diabetes. *Diabetes Care* 2003; **26**: 3151–3159.
9 Young TK, Reading J, Elias B, O'Neil JD. Type 2 diabetes mellitus in Canada's first nations: status of an epidemic in progress. *CMAJ* 2000; **163**: 561–566.
10 Trovato F. Mortality differentials in Canada, 1951–1971: French, British, and Indians. *Cult Med Psychiatry* 1988; **12**: 459–477.
11 Young TK, Michael E, Moffatt K, O'Neil JD. Cardiovascular diseases in a Canadian arctic population. *Am J Public Health* 1993; **83**: 881–887.
12 Harris SB, Gittelsohn J, Hanley A, Barnie A, Wolever TM, Gao J *et al*. The prevalence of NIDDM and associated risk factors in Native Canadians. *Diabetes Care* 1997; **20**: 185–187.
13 Shah BR, Hux JE, Zinman B. Increasing rates of ischemic heart disease in the native population of Ontario, Canada, Arch. *Intern Med* 2000; **160**: 1862–1866.
14 Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabetes Med* 1998; **15**: 539–553.
15 WHO. Definition, diagnosis and classification of diabetes mellitus and its complications, part 1: diagnosis and classification of diabetes mellitus. *Report of a WHO Consultation* 1999.
16 National Cholesterol Education Program. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *JAMA* 2001; **285**: 2486–2497.
17 Grundy SM, Brewer HB, Cleeman JI, Snith SC, Lenfant C. Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation* 2004; **109**: 433–438.
18 Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA* 2002; **287**: 356–360.
19 Resnick HE. Metabolic syndrome in American Indians. *Diabetes Care* 2002; **25**: 1246–1247.
20 Meigs JB, Wilson PWF, Nathan DM, D'Agostino RB, Williams K, Haffner SM. Prevalence and characteristics of the metabolic syndrome in the San Antonio Heart and Framingham Offspring Studies. *Diabetes* 2003; **52**: 2160–2167.
21 Aguilar-Salinas CA, Rojas R, Gomez-Perez FG, Valles V, Rios-Torres JM, Franco A *et al*. Analysis of the agreement between the World Health Organization criteria and the National Cholesterol Education Program-III definition of the metabolic syndrome: results from a population-based survey. *Diabetes Care* 2003; **26**: 1635.
22 Ford ES, Giles WH. A comparison of the prevalence of the metabolic syndrome using two proposed definitions. *Diabetes Care* 2003; **26**: 575–581.
23 Park YW, Zhu SK, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The metabolic syndrome prevalence and associated risk factor findings in the US population from the third national health and nutrition examination survey, 1988–1994. *Arch Intern Med* 2003; **163**: 427–436.
24 Al-Lawati JA, Mohammed AJ, Al-Hinai HQ, Jousilahti P. Prevalence of the metabolic syndrome among Omani adults. *Diabetes Care* 2003; **26**: 1781–1785.
25 Azizi F, Salehi P, Etemadi A, Zahedi-Asl S. Prevalence of metabolic syndrome in an urban population: Tehran Lipid and Glucose Study. *Diabetes Res Clin Pract* 2003; **61**: 29–37.
26 Ardern CI, Katzmarzyk PT, Janssen I, Ross R. Discrimination of health risk by combined body mass index and waist circumference. *Obes Res* 2003; **11**: 135–142.
27 Anand SS, Yi Q, Gerstein H, Lonn E, Jacobs R, Vuksan V *et al*. Relationship of metabolic syndrome and fibrinolystic dysfunction to cardiovascular disease. *Circulation* 2003; **108**: 420–425.
28 Jaber LA, Brown MB, Hammad A, Zhu Q, Herman WH. The prevalence of the metabolic syndrome among Arab Americans. *Diab Care* 2004; **27**: 234–238.
29 Lee WY, Park JS, Noh SY, Rhee EJ, Kim SW, Zimmet PZ. Prevalence of the metabolic syndrome among 40,698 Korean metropolitan subjects. *Diab Res Clin Pract* 2004; **65**: 143–149.

30 Panagiotakos DB, Pitsavos C, Chrysohoou C, Skoumas J, Tousoulis D, Toutouza M *et al*. Impact of lifestyle habits on the prevalence of the metabolic syndrome among Greek adults from the ATTICA study. *Am Heart J* 2004; **147**: 106–112.
31 Jørgensen ME, Bjerregaard P, Gyntelberg F, Borch-Johnsen K, the Greenland Population Study. Prevalence of the metabolic syndrome among the Inuit in Greenland. A comparison between two proposed definitions. *Diab Med* 2004; **21**: 1237–1242.
32 Young TK, Chateau D, Zhang M. Factor analysis of ethnic variation in the multiple metabolic (Insulin Resistance) syndrome in three Canadian populations. *Am J Hum Biol* 2002; **14**: 649–658.
33 Young TK, Sevenhuysen G. Obesity among northern Indians: patterns, determinants and consequences. *Am J Clin Nutr* 1989; **49**: 786–793.
34 Young TK. Prevalence and correlates of hypertension in a subarctic Indian population. *Prev Med* 1991; **20**: 474–485.
35 Gelskey DE, Young TK, Macdonald SM. Screening with total cholesterol: determining sensitivity and specificity of the National Cholesterol Education Program's guidelines from a population survey. *J Clin Epidemiol* 1994; **47**: 547–553.
36 Young TK, Gelskey DE. Is non-central obesity metabolically benign? Implications for prevention from a population survey of Canadians. *JAMA* 1995; **274**: 1939–1941.
37 Young TK. Sociocultural and behavioural determinants of obesity among Inuit in the central Canadian Arctic. *Soc Sci Med* 1996; **43**: 1665–1671.
38 Hanley AJG, Harris SB, Barnie A, Gittelsohn J, Wolever TM, Logan A *et al*. The Sandy Lake Health and Diabetes Project: design, methods and lessons learned. *Chronic Dis Canda* 1995; **16**: 149–156.
39 Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985; **28**: 412–419.
40 World Health Organization. *Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation on Obesity*. WHO: Geneva, 2000.
41 Statistics Canada. 2001 Census Aboriginal peoples of Canada: a demographic profile. Cat 96F0030XIE2001007.
42 Statistics Canada. Profile of census metropolitan areas and census agglomerations (1991 census). Part A and Part B. Ottawa, 1992 and 1993; Cat 93-337 and 93-338.
43 Grundy SM. Obesity, metabolic syndrome, and coronary atherosclerosis. *Circulation* 2002; **105**: 2696–2698.
44 Okosun IS, Liao Y, Rotimi CN, Prewitt TE, Cooper RS. Abdominal adiposity and clustering of multiple metabolic syndrome in White, Black and Hispanic Americans. *Ann Epidemiol* 2000; **10**: 263–270.
45 Haffner SM, D'Agostino Jr R, Saad MF, Rewers M, Mykkanen L, Selby J *et al*. Increased insulin resistance and insulin secretion in nondiabetic African-Americans and Hispanics compared with non-Hispanic whites: the insulin resistance atherosclerosis study. *Diabetes* 1996; **45**: 742–748.
46 Hegele RA. Lessons from genetic studies in Native Canadian populations. *Nutr Rev* 1999; **57**: S43–S50.
47 Young TK, Szathmary E, Evers S, Wheatley B. Geographical distribution of diabetes among the native population of Canada: a national survey. *Soc Sci Med* 1990; **31**: 129–139.
48 Young TK. Obesity, central fat patterning and their metabolic correlated among the Inuit of the central arctic. *Hum Biol* 1996; **68**: 245–263.
49 Gittelsohn J, Wolever TM, Harris SB, Harris-Giraldo R, Hanley AJ, Zinman B. Specific patterns of food consumption and preparation are associated with diabetes and obesity in a native Canadian community. *J Nutr* 1998; **128**: 541–547.
50 Bjerregaard P, Jørgensen ME, Borch-Johnsen K. Serum lipids of Greenland Inuit in relation to Inuit genetic heritage, westernization and migration. *Atherosclerosis* 2004; **174**: 391–398.
51 Dewailly E, Blanchet C, Gingras S, Lemieux S, Holub BJ. Cardiovascular disease risk factors and n-3 fatty acid status in the adult population of James Bay Cree. *Am J Clin Nutri* 2002; **76**: 85–92.
52 Dewailly E, Blanchet C, Lemieux S, Sauvé L, Gingras S, Ayotte P *et al*. n_3 Fatty acids and cardiovascular disease risk factors among the Inuit of Nunavik. *Am J Clin Nutr* 2001; **74**: 464–473.
53 Bjerregaard P, Jørgensen ME, Andersen S, Mulvad G, Borch-Johnsen K, The Greenland Population Study. Decreasing overweight and central fat patterning with Westernization among the Inuit in Greenland and Inuit migrants. *Int J Obes Relat Metab Disord* 2002; **26**: 1503–1510.
54 Green C, Blanchard J, Young TK, Griffith J. The epidemiology of diabetes in the Manitoba-registered First Nation population: current patterns and comparative trends. *Diabetes Care* 2003; **26**: 1993–1998.
55 Gittelsohn J, Harris SB, Whitehehad S, Wolever TM, Hanley A, Barnie A *et al*. Developing diabetes interventions in an Ojibwa-Cree community in northern Ontario: linking qualitative and quantitative data. *Chronic Dis Can* 1995; **16**: 157–164.
56 Macaulay AC, Harris SB, Lévesque L, Cargo M, Ford E, Salsberg J *et al*. Primary prevention of type 2 diabetes: experience of 2 Aboriginal communities in Canada. *Can J Diabetes* 2003; **27**: 464–475.