Variation by body mass index and age in waist-to-hip ratio associations with glycemic status in an aboriginal population at risk for type 2 diabetes in British Columbia, Canada[1–3]

Mark Daniel, Stephen A Marion, Samuel B Sheps, Clyde Hertzman, and Diane Gamble

ABSTRACT

Background: It is unclear whether obesity and age modify or confound relations between abdominal adiposity and metabolic risk factors for type 2 diabetes.

Objective: Our objective was assess the consistency of relations between abdominal adiposity and glycemic variables across discrete categories of obesity and age.

Design: We performed a stratified analysis of prevalence data from a rural screening initiative in British Columbia, Canada. Subjects were Salishan Indians, all healthy relatives of individuals with type 2 diabetes [*n* = 151; age: 18–80 y; body mass index (BMI, in kg/m^2): 17.0–48.2]. We measured waist-to-hip ratio (WHR) (2 categories); insulin, glycated hemoglobin (Hb A_{1c}), and 2-h glucose concentrations (2 categories); and BMI (4 categories). BMI and age-specific odds ratios (ORs) and 95% CIs were calculated.

Results: WHR-glycemic variable relations were not consistent across BMI and age strata. Risks associated with high WHR were: for persons with BMIs from 25 to 29, elevated insulin (OR: 6.71; 95% CI: 1.41, 34.11) and Hb A_{1c} (OR: 16.23; 95% CI: 2.04, 101.73) concentrations; for persons aged 18–34 y, elevated insulin concentrations [OR: indeterminate ($^+\infty$); 95% CI: 1.89, $^+\infty$]; and, for persons aged 35–49 y, elevated Hb A_{1c} (OR: $^+\infty$; 95% CI: 3.17, $^+\infty$) and 2-h glucose (OR: 9.15; 95% CI: 1.74, 59.91) concentrations.

Conclusions: WHR discriminates risk of type 2 diabetes in overweight but not obese individuals. Abdominal adiposity is associated with elevated insulin concentrations in younger age groups and with impaired glucose control in middle-aged groups, suggesting metabolic staging by age on a continuum from insulin resistance to impaired glucose tolerance. *Am J Clin Nutr* 1999;69:455–60.

KEY WORDS Type 2 diabetes, North American Indians, body fat distribution, obesity, abdominal adiposity, waist-to-hip ratio, body mass index

INTRODUCTION

Obesity is considered the strongest risk factor for type 2 diabetes (1), and body mass index (BMI; in kg/m^2) is a standard predictor of diabetic status and plasma glucose and glycated hemoglobin (Hb A_{1c}) concentrations in aboriginal Canadian (2–4) and other populations (5–7) at high risk for type 2 diabetes. Similar associations have also been reported for abdominal adiposity (8–10). Most studies have used the anthropometric indicator waist-to-hip ratio (WHR) to assess adipose tissue distribution. The validity of WHR as an indicator was determined by in vivo methods (11, 12).

It is unclear whether relations between adipose tissue distribution and glycemic variables are a function of obesity. Furthermore, the influence of age on these relations has been overlooked. Studies analyzing relations between obesity, regional adiposity, and chronic disease have often used multivariate analytic techniques (13–17). Age and obesity are typically treated as confounding "nuisance" variables, and effect modification has not been adequately assessed.

This report responded to the limitations of multivariate analyses of regional adiposity and glycemic status. We examined the consistency of relations between abdominal adiposity and glycemic variables across discrete levels of obesity and age.

SUBJECTS AND METHODS

Data analyzed in this report were derived from a type 2 diabetes screening initiative in registered Indians (Interior Salishan) on reserves in the Okanagan region (18) of British Columbia. The population and setting were described previously (19). The Okanagan is in the South Mainland Zone quadrant defined by the Medical Services Branch, Pacific Region, Health Canada. For on-reserve registered Indians aged ≥18 y in the Okanagan, we estimated a crude diabetes prevalence rate of 36.1/1000 (*n* = 1276) (19).

[1] From the Department of Epidemiology and Preventive Medicine, Monash University, Melbourne; the Department of Health Care and Epidemiology, University of British Columbia, Vancouver, Canada; the Division of Health and Social Programs, Okanagan University College, Kelowna, Canada.

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[3] Reprints not available. Address correspondence to M Daniel, Department of Epidemiology and Preventive Medicine, Monash Medical Centre, Level 5, Block E, 246 Clayton Road, Clayton, Melbourne 3168, Victoria, Australia. E-mail: mark.daniel@med.monash.edu.au.

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Participant selection and measurement protocol

Persons with established (physician-diagnosed) diabetes were identified before screening by using records maintained by the local Medical Services Branch unit. Diabetic individuals and their first- and second-degree relatives were asked to participate in a diabetes diagnostic and risk factor screening initiative. Relatives were considered to be at risk both genetically and behaviorally. Health education efforts in this population have been few and could not be expected to have reduced risk of disease in relatives of diabetic persons. Incentives to participate were not offered; however, participants were told they would receive detailed explanations of results.

Tests were conducted in community meeting halls between 0730 and 1200. Ethical approval was provided by the University of British Columbia Behavioral Sciences Screening Committee. All participants provided informed, written consent. Pregnant women and minors aged <18 y were excluded from testing. Persons with established diabetes participated in testing but were excluded from the analyses reported here. Persons taking medications known to influence blood glucose concentrations were omitted from analysis (20).

Of 194 participants in screening initiatives, 31 were previously diagnosed diabetics. A further 12 persons were ineligible for the analyses reported here because they used medication that affects glucose concentrations. The resultant sample of 151 persons includes 6 persons subsequently diagnosed (after screening) with diabetes (4 women and 2 men), using World Health Organization criteria (21). Besides the measures reported here, the screening included assessment of psychosocial and behavioral variables (reported elsewhere;19).

Blood samples and analytic methods for glycemic variables

For screening purposes, participants provided a venous blood sample after a 12-h fast. From these samples, Hb A_{1c} and insulin concentrations were determined. Two-hour glucose concentrations were determined from blood samples drawn 2 h after subjects consumed a 75-g carbohydrate load, ingested for diagnostic purposes after fasting blood samples were drawn. The results of 2-h oral glucose tolerance tests for diabetes screening have been reported elsewhere (19).

Whole blood specimens collected in EDTA anticoagulant were used to determine Hb A_{1c}. Analyses for insulin and 2-h glucose were performed on serum specimens. Serum was obtained on location by low-speed centrifugation at 1800 × *g* for 10 min at room temperature. Whole blood and serum samples were stored at 4°C for transport for analysis at a regional hospital laboratory. Analyses were performed on the day of specimen collection. Blood samples stood at room temperature ≤15 min before refrigeration or centrifugation.

Serum insulin concentrations were determined by using microparticle enzyme immunoassay kits (IMx; Abbott Laboratories, Abbott Park, IL). Percentage Hb A_{1c} was measured by using ion capture assay kits (IMx). Two-hour blood glucose concentrations were assessed by using enzymatically linked assay kits (Kodak Ektachem; Eastman Kodak Co, Rochester, NY). Intra- and interassay CVs were, respectively, insulin, 3.8% and 4.2%; Hb A_{1c}, 4.4% and 4.6%; and 2-h glucose, 1.2% and 1.8%.

Anthropometric measurements

All measures were performed by nurses trained in anthropometric measurement. Participants wore light clothing with footwear removed. Weight, height, and waist and hip girths were assessed, taking the median of 3 measures as the true value (22). A calibrated beam balance and a stadiometer were used to measure weight and height. Minimum waist girth was taken where the waist was best defined, halfway between the costal border and the iliac crest. Maximum hip girth was obtained at the level of the greatest posterior protuberance. BMI and WHR were calculated.

Data analysis

Values for WHR were dichotomized into high and low categories according to age- and sex-specific norms (23) from the Canada Fitness Survey (24). Cutoffs corresponded to the 75th percentiles for given combinations of sex and age (**Table 1**). This approach accounts for sexual dimorphism in pelvic structure as well as normal increases in WHR with increasing age. The 75th percentile was chosen as the cutoff point because it corresponded with the geometric mean for the distribution of WHR in the study population; it is also the point beyond which risk of all-cause mortality increases substantially in both men and women (25).

Values for fasting plasma insulin, Hb A_{1c}, and 2-h glucose concentrations were dichotomized into high and low categories by using median sample values that approximated geometric means and tended toward the midpoints of normal clinical ranges. Cutoff points and normal ranges were as follows: insulin, 76.0 pmol/L (14–180 pmol/L); Hb A_{1c}, 5.4% (4.4–6.4%); and 2-h glucose, 5.2 mmol/L (<7.8 mmol/L).

Values for BMI were grouped into 4 categories: <25, 25–29, 30–34, and ≥35. These categories recognize that the range of BMI associated with minimum mortality is 20–25 in both men and women, with progressive increases in all-cause mortality at 30 and 35 (26). The categories are consistent with the US National Heart, Lung, and Blood Institute's evidence-based clinical guidelines (27), which define overweight as BMI = 25–29 and obesity as BMI ≥ 30.

Age was grouped into 3 categories: 18–34, 35–49, and ≥50 y. Preliminary analyses showed that age group was associated with classifications of Hb A_{1c} ($P = 0.0001$) and 2-h glucose ($P = 0.002$) concentrations, but not with classifications of insulin, WHR, or BMI.

Sex was not significantly associated in pooled or age-specific analyses with the dichotomized outcome variables or with categories of age or BMI. For all variables except WHR (continuous versions, before categorization), mean differences between sexes were minor and not significant. For these reasons sex was omitted as a separate variable. Sex was accounted for in use of sex-specific cutoffs for categorizing WHRs.

Lifestyle factors potentially related to fatness, fat distribution measures, and glycemic variables were assessed in separate analyses as possible confounders of WHR-glycemic outcome

TABLE 1
Age- and sex-specific cutoff points for classification of high compared with low waist-to-hip ratios[1]

	Waist-to-hip ratio	
Age(y)	Men	Women
18–34	0.89	0.79
35–49	0.95	0.82
50–64	0.98	0.84
≥65	0.99	0.86

[1]Based on age- and sex-specific norms (23) from the Canada Fitness Survey (24).

relations. Diet and physical activity were related in crude analyses to WHR and glycemic outcomes. Controlling by stratification for age and BMI nullified the influence of diet and physical activity. Our results were unlikely to be confounded by lifestyle factors associated with age and BMI.

Statistical analysis

Two separate stratified analyses were performed to determine the relation between high WHR and high concentrations of insulin, Hb A_{1c}, and 2-h glucose. The strength of the relation between WHR and glycemic variables was based on a maximum likelihood estimation of the odds ratios (ORs), two-tailed exact 95% confidence limits for ORs, and exact mid-*P* values.

The first analysis assessed the age-adjusted relation between WHR and the 3 dichotomized outcomes separately for each BMI category. Likelihood ratio tests of uniformity were conducted for each BMI category to evaluate effect modification by age. A Cochran-Mantel-Haenszel trend test, stratified by age group, was then performed across all BMI categories. The test evaluated for each outcome the hypothesis that poor glycemic status increased proportionately with increasing BMI, ignoring WHR.

The second analysis assessed the BMI-adjusted relation between WHR and the 3 outcomes, separately for each age group (ie, the roles of age and BMI were reversed). Age-specific crude and summary BMI-adjusted ORs were calculated. Likelihood ratio tests of uniformity were conducted to evaluate effect modification by BMI.

Maximum likelihood estimation of parameters for stratified analyses was conducted by using MLEPID software (28); α was set at 0.05. The algorithm used by MLEPID is based on equations 12–25 in chapter 12 of Rothman (29). The routine MOWAN was used to calculate Miettinen's mid-*P* exact CIs for crude (unstratified) analyses (30). The Statcalc module of EPI INFO was used to test linear trends in proportions (31).

RESULTS

Characteristics of the population sampled are presented in **Table 2**. Women comprised 65% of the sample, men 35%. There were no significant differences between men and women in mean age, BMI, or concentrations of insulin, Hb A_{1c}, or 2-h glucose. Mean WHR was greater in men than in women.

Age-adjusted, BMI-specific, summary ORs describing relations between WHR and glycemic status are shown in **Table 3**. Likelihood ratio tests of uniformity across age categories were not significant at any BMI category for any glycemic variable. Therefore, age seems to confound, rather than modify, BMI-specific associations between WHR and glycemic status. Effect modification by BMI, however, was evidenced by differences in estimated ORs across BMI strata. Poor glycemic status associated with high WHR, adjusted for age, was significant for the overweight category (BMI 25–29) for insulin (OR: 6.71) and Hb A_{1c} (OR: 16.23). Insulin and Hb A_{1c} were not significantly related to WHR for other BMI categories. When adjusted for age, WHR was not associated with 2-h glucose concentration for any BMI category.

Ignoring WHR, there were significant linear age-adjusted trends in poor glycemic status across increasing BMI categories for insulin and 2-h glucose concentrations (**Table 4**). The trend test result for Hb A_{1c} was not significant.

Age-specific crude and BMI-adjusted summary ORs for relations between WHR and glycemic status are shown in **Table 5**. Tests of uniformity across BMI categories were not significant at any age category for any glycemic variable, indicating that BMI did not modify age-specific associations between WHR and glycemic status. Rather, as evidenced by differences between estimated crude and BMI-adjusted summary ORs, BMI confounded age-specific relations between WHR and glycemic status. This was most apparent for the association between WHR and insulin in the group aged ≥50 y. For the group aged 35–49 y, adjusted for BMI, high WHR was significantly associated with elevated Hb A_{1c} [OR: indeterminate (+∞)] and 2-h glucose (OR: 9.15) concentrations. The BMI-adjusted risk of elevated insulin concentrations associated with high WHR was significant in the group aged 18–34 y (OR: +∞). Other relations were not significant.

Indeterminate point estimates and upper confidence limits in Tables 3 and 5 are a consequence of one or more strata tables with a common zero cell; the lower exact confidence limit, however, and the two-tailed exact maximum likelihood mid-*P* values provide a basis for evaluating strength of effect.

TABLE 2
Characteristics of 151 participants at familial risk for type 2 diabetes[1]

	Men (n = 53)	Women (n = 98)
Age (y)	42.2 ± 13.2	42.0 ± 14.5
Body mass index (kg/m^2)	28.1 ± 5.7	28.9 ± 5.7
Waist-to-hip ratio[2]	0.945 ± 0.077	0.869 ± 0.081
Fasting insulin (pmol/L)	98.4 ± 149.6	100.4 ± 92.5
Hemoglobin A_{1c} (%)	5.7 ± 1.7	5.4 ± 0.6
2-h Glucose (mmol/L)	5.52 ± 3.83	5.67 ± 1.82

[1] $\bar{x}$ ± SD.
[2] Significantly different between sexes (t = 5.60 with 149 df; P = 0.0001).

DISCUSSION

In our study population, relations between abdominal adiposity and glycemic status were not consistent across age or BMI

TABLE 3
Age-adjusted relations between waist–to–hip ratio (WHR) and poor glycemic status by BMI[1]

Outcome and BMI (in kg/m^2)	WHR-outcome association
Insulin	
<25 (n = 40)	2.41 (0.28, 22.24)
25–29 (n = 56)	6.71 (1.41, 34.11)[2]
30–34 (n = 34)	0.62 (0.01, 13.77)
≥35 (n = 21)	7.91 (0.40, $^+\infty$)[3]
Hb A_{1c}	
<25 (n = 40)	4.42 (0.63, 33.18)
25–29 (n = 56)	16.23 (2.04, 101.73)[4]
30–34 (n = 34)	2.24 (0.14, 67.86)
≥35 (n = 21)	1.57 (0.09, 55.13)
2-h Glucose	
<25 (n = 40)	5.42 (0.57, $^+\infty$)[3]
25–29 (n = 56)	2.59 (0.72, 9.21)
30–34 (n = 34)	$^+\infty$ (0.30, $^+\infty$)[3]
≥35 (n = 21)	0.50 (0.01, 6.92)

[1] Odds ratio; exact mid-*P* 95% CI in parentheses.
[2] WHR-outcome association was significant: [2]P = 0.012, [4]P = 0.003.
[3] Indeterminate ($^+\infty$) point estimates and exact upper CI values are consequences of one or more strata tables with a common zero cell.

TABLE 4
Age-adjusted associations between poor glycemic status and BMI, relative to the lowest BMI category[1]

Outcome and BMI (in kg/m^2)	Poor glycemic status outcome association
Insulin[2]	
<25 (n = 40)	1.00
25–29 (n = 56)	3.87 (1.21, 14.56)
30–34 (n = 34)	8.99 (3.32, 49.61)
≥35 (n = 21)	14.61 (3.42, 69.20)
Hemoglobin A_{1c}[3]	
<25 (n = 40)	1.00
25–29 (n = 56)	1.63 (0.58, 4.62)
30–34 (n = 34)	2.71 (0.87, 8.25)
≥35 (n = 21)	2.42 (0.61, 8.92)
2-h Glucose[4]	
<25 (n = 40)	1.00
25–29 (n = 56)	1.92 (0.72, 5.21)
30–34 (n = 34)	5.02 (1.46, 19.44)
≥35 (n = 21)	4.22 (1.04, 17.39)

[1]Odds ratio; exact mid-P 95% CI in parentheses.
[2,4]Significant linear trend in proportions: [2](χ^2 = 27.7 with 1 df, P <0.00001; [4]χ^2 = 8.0 with 1 df, P = 0.005.
[3]Nonsignificant trend test (χ^2 = 3.8 with 1 df, P = 0.052).

categories. Our stratified analysis showed that age and BMI can both confound and modify associations between WHR and glycemic status. High WHR was a significant predictor of risk in overweight subjects (BMI 25–29) for high insulin (OR: 6.71) and Hb A_{1c} (OR: 16.23) concentrations, adjusted for age. Although the age-adjusted prevalence of high insulin concentrations increased monotonically (P <0.00001) from the lowest BMI category (<25) across the overweight (25–29) and 2 obese categories (30–34 and ≥35), after fixing BMI we observed no consistent WHR-insulin relation. For BMIs beyond the overweight range, the discriminatory ability of WHR was not apparent.

In overweight but not in clinically obese individuals, a predominance of abdominal adiposity is a strong indication of risk for poor glycemic status. At higher BMIs, however, overall adiposity is better related than abdominal adiposity to poor glycemic status. Intraabdominal fat is accepted as the physiologic cause of conditions associated with abdominal adiposity indexed by WHR (32). The importance of WHR lies in its ability to discriminate risk among overweight individuals at low risk on the basis of BMI. Our results support the use of waist and hip girths for screening people whose overall adiposity is not pronounced. Moreover, our results contradict the perception that a slight degree of overweight might actually be healthy (25). Fat distribution must be evaluated.

WHR was not related in this study to poor glycemic status at BMIs ≥30. This is not to suggest that a high BMI is not related to health risk, but that WHR is a poor indicator of the risks associated with a high BMI. We observed strong associations between increasing BMI and worsening glycemic status. Although the abdominal region is most responsive to changes in weight (33), high levels of overall adiposity may obfuscate the interpretation of WHR as selectively indexing intraabdominal adiposity. High BMI is associated with elevated abdominal and peripheral adiposity (32). Therefore, for persons with BMIs <30, the relative amount of intraabdominal fat indexed by WHR may be more important than the absolute amount of intraabdominal fat. At BMIs ≥30, the situation is reversed, presumably because a critical mass of intraabdominal fat has been achieved in the development of obesity.

Obesity per se is not a requirement for increased risk of diabetes (34), yet little attention has been given to regional adiposity as a risk factor in the absence of general obesity. Adipose tissue distribution may explain, in part, why many nonobese persons develop type 2 diabetes, and why many obese persons never develop it. Genetic determinants regulating adipose tissue distribution (35, 36) may reflect different pathways by which genotype is related to type 2 diabetes and metabolic abnormalities in aboriginal populations (37, 38).

Disease phenotypes may become manifest only as the result of a highly variant series of complex interactions with environmental challenges to metabolism (39, 40). Dramatic changes in diet and physical activity among aboriginal populations over the past several decades most likely interact with genetic determinants of obesity and fat patterning in influencing risk of diabetes (8, 41). Whereas dietary factors and physical activity levels are reflected by obesity and fat distribution measures (42), their unique contribution to the risk of developing diabetes has been

TABLE 5
Age-specific associations for waist–to–hip ratio (WHR) and glycemic outcomes, crude and adjusted for BMI[1]

Outcome and age group (y)	WHR-outcome association
Insulin	
18–34 (n = 53)	
Crude	17.29 (2.53, 385.07)[2]
BMI-adjusted	$^{+}\infty$ (1.89, $^{+}\infty$)[3,4]
35–49 (n = 62)	
Crude	1.94 (0.65, 6.03)
BMI-adjusted	1.39 (0.35, 5.49)
≥50 (n = 36)	
Crude	16.25 (2.49, 121.79)[5]
BMI-adjusted	11.70 (0.96,$^{+}\infty$)[3]
Hemoglobin A_{1c}	
18–34 (n = 53)	
Crude	2.67 (0.55, 19.62)
BMI-adjusted	1.01 (0.20, 5.96)
35–49 (n = 62)	
Crude	10.00 (2.22, 68.37)[2]
BMI-adjusted	$^{+}\infty$ (3.17, $^{+}\infty$)[3,6]
≥50 (n = 36)	
Crude	3.00 (0.73, 12.38)
BMI-adjusted	3.06 (0.53, 16.46)
2–h Glucose	
18–34 (n = 53)	
Crude	2.05 (0.50, 10.38)
BMI-adjusted	0.91 (0.16, 5.44)
35–49 (n = 62)	
Crude	5.67 (1.72, 19.80)[7]
BMI-adjusted	9.15 (1.74, 59.91)[8]
≥50 (n = 36)	
Crude	4.00 (0.77, 22.74)
BMI-adjusted	1.29 (0.21, 8.61)

[1]Odds ratio; exact mid-P 95% CI in parentheses.
[2,4–8]Significant association: [2]P = 0.001, [4]P = 0.009, [5]P = 0.0007, [6]P = 0.0008, [7]P = 0.003, [8]P = 0.0005.
[3]Indeterminate ($^{+}\infty$) point estimates and exact upper CI values are consequences of one or more strata tables with a common zero cell.

difficult to assess. Some studies have shown links between the prevalence of type 2 diabetes and intake of particular dietary constituents (43, 44). At the population level, however, the overall nature of dietary change is a stronger etiologic factor for type 2 diabetes than associations with consumption of certain foods (8).

Our second analysis, in which we controlled for BMI, shows variation by age in associations between WHR and glycemic status. These results suggest metabolic staging (45) by age from insulin resistance to impaired glucose tolerance and, possibly, type 2 diabetes. The finding that abdominal adiposity is associated with elevated insulin in young persons (aged 18–34 y) and with high Hb A_{1c} and 2-h glucose concentrations in middle-aged persons (aged 35–49 y) is aligned with a 2-step model for development of type 2 diabetes. In this model, insulin resistance is a consequence of abdominal obesity, leading to impaired glucose tolerance (step 1) and, with β-cell failure, progression to type 2 diabetes (step 2) (46, 47).

Age-related differences in personal preference for body size may covary with dietary patterns in aboriginal populations (48). Controlling for BMI and age by stratification prevents such confounding, however. Therefore, it is possible that younger persons, in whom WHR predicts high insulin concentrations, may proceed to develop the fasting hyperglycemia observed in middle-aged persons, and that some of these individuals will develop type 2 diabetes at a later age. Early detection and intervention to reduce abdominal adiposity in individuals <35 y of age with family histories of diabetes may be of major importance in preventing type 2 diabetes (49). In such actions, an understanding of body size preference, its variation with age and sex, and links to dietary behavior, may be particularly important (50).

Cutoff points used in this study to dichotomize WHR reflected normative age-associated increases in abdominal adiposity. That such changes are normative does not mean they are healthy. Use of population-based 75th percentile values as cutoffs for sex and age combinations prevents confounding in WHR classifications and is necessary for pooled analysis of the data. Nevertheless, this strategy may underestimate WHR-outcome associations as might be determined by using sex- but not age-dependent cutoff points to define risk associated with low compared with high WHR (eg, 0.90 for men and 0.80 for women).

Limitations of this study include the possibility of bias in blood test results if participants failed to fast before blood samples were drawn. Such behavior would influence blood test results, but it is unlikely that any such bias might vary systematically with classifications of WHR. Measurement error in blood and anthropometric measures is possible, and would bias ORs toward 1.0. Categorizing variables before analyzing associations reduces but does not eliminate this possibility. That more women than men participated in community screening is unlikely to have biased our results, given the lack of association in any analysis of sex with glycemic variables, age, or BMI.

The generalizability of these results to populations other than Interior Salishan people is a matter of degree and is not properly assessed as a dichotomy. The indigenous populations of Canada and the United States are genetically heterogeneous (40) and it may be that studies in other peoples would produce similar findings. At least some relations between body composition and diabetes, however, may be specific to particular ethnic groups (51). Replication of our analyses in other populations, both aboriginal and nonaboriginal, may further clarify the influence of BMI and age on relations between WHR and glycemic status.

We conclude that relations between abdominal adiposity and glycemic variables are not consistent across discrete levels of obesity and age in aboriginal men and women at familial risk for type 2 diabetes. Abdominal adiposity is a critical measure of risk in persons at low risk on the basis of BMI, independent of age. The age-specific patterns of association of WHR with hyperinsulinemia and fasting hyperglycemia suggest that abdominal adiposity relates to the development of insulin resistance and hyperglycemia in individuals at familial risk for type 2 diabetes more strongly than to obesity per se. The notion that a slight degree of overweight might actually be healthy should be reevaluated. Strong linear trends exist between increasing BMI and worsening glycemic status. WHR is a poor indicator, however, of the risks associated with a BMI ≥30.

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