# Ethnic Variation in Adiponectin and Leptin Levels and Their Association With Adiposity and Insulin Resistance

ANDREW MENTE, PHD[1,2]
FAHAD RAZAK, BENG, MSC[1]
STEFAN BLANKENBERG, MD[3]
VLAD VUKSAN, PHD[4]
A. DARLENE DAVIS, RN[5]
RUBY MILLER, RN[5]
KOON TEO, MBBCH, PHD[1,2]
HERTZEL GERSTEIN, MD, MSC[1,2]
ARYA M. SHARMA, MD, PHD[1,2]
SALIM YUSUF, MBBS, DPHIL[1,2]
SONIA S. ANAND, MD, PHD[1,2]
FOR THE STUDY OF HEALTH ASSESSMENT AND RISK EVALUATION (SHARE) AND SHARE IN ABORIGINAL PEOPLES (SHARE-AP) INVESTIGATORS

**OBJECTIVE** — To investigate ethnic differences in adiponectin and leptin concentration and to determine whether these adipokines and a high–glycemic index diet account for ethnic variation in insulin resistance.

**RESEARCH DESIGN AND METHODS** — In 1,176 South Asian, Chinese, Aboriginal, and European Canadians, fasting blood samples were drawn, and clinical history and dietary habits including glycemic index/glycemic load were recorded using standardized questionnaires. Insulin resistance was defined using homeostasis model assessment–insulin resistance (HOMA-IR).

**RESULTS** — Adiponectin concentrations were significantly higher in Europeans (adjusted mean 12.94 [95% CI 2.27–13.64]) and Aboriginal people (11.87 [11.19–12.59]) than in South Asians (9.35 [8.82–9.92]) and Chinese (8.52 [8.03–9.03]) (overall $P < 0.001$). Serum leptin was significantly higher in South Asians (11.82 [10.72–13.04]) and Aboriginal people (11.13 [10.13–12.23]) than in Europeans (9.21 [8.38–10.12]) and Chinese (8.25 [7.48–9.10]). BMI and waist circumference were inversely associated with adiponectin in every group except the South Asians ($P < 0.001$ for interaction). Adiponectin was inversely and leptin was positively associated with HOMA-IR ($P < 0.001$). The increase in HOMA-IR for each given decrease in adiponectin was larger among South Asians ($P = 0.01$) and Aboriginal people ($P < 0.001$) than among Europeans. A high glycemic index was associated with a larger decrease in adiponectin among South Asians ($P = 0.03$) and Aboriginal people ($P < 0.001$) and a larger increase in HOMA-IR among South Asians ($P < 0.05$) relative to that in other groups.

**CONCLUSIONS** — South Asians have the least favorable adipokine profile and, like the Aboriginal people, display a greater increase in insulin resistance with decreasing levels of adiponectin. Differences in adipokines and responses to glycemic foods parallel the ethnic differences in insulin resistance.

*Diabetes Care* **33:1629–1634, 2010**

Epidemiologic studies have consistently shown that compared with nonwhite ethnic populations, people of European origin have a relatively low prevalence of insulin resistance and type 2 diabetes despite having comparable or greater body weight (1,2). Although there is controversy regarding the definition and use of the term "metabolic syndrome," which is often applied to the clustering of risk factors such as abdominal obesity, elevated glucose, abnormal lipids, and elevated blood pressure, insights into the pathophysiology of adipose tissue and the presence of these insulin resistance–related factors may be gained from studies of high- and low-risk populations.

Insulin resistance is closely associated with abdominal adiposity, a surrogate measure of visceral adiposity (3). Adipocytes secrete a variety of bioactive substances known as adipokines, including two proteins, adiponectin and leptin. Adiponectin, a plasma protein secreted from visceral adipose tissue, increases insulin sensitivity and tissue fat oxidation, resulting in reduced circulating fatty acid levels (4). Leptin, a protein that circulates in proportion with body fat mass, provides information about nutritional status and subcutaneous fat mass to neural centers that regulate feeding behavior, appetite, and energy expenditure (5). It is therefore plausible that differences in adiponectin and leptin levels correlate with ethnic variations in insulin resistance and metabolic syndrome–related factors.

Dietary factors may also potentially influence adipokine levels and insulin sensitivity. There is a growing body of literature showing that higher consumption of foods with high glycemic index/glycemic load values is associated with lower adiponectin levels in both healthy and diabetic individuals (6) and higher leptin levels (7). Glycemic foods are known to induce both hyperglycemia and hyperinsulinemia (8,9). Conversely, high intake of fiber may attenuate the glycemic effect of a full meal, and cereal fiber intake is positively associated with adiponectin (6). Previous studies have shown that ethnic populations at higher risk for metabolic syndrome–related conditions largely consume a diet consisting of foods with a high glycemic index (10). It is not known whether a higher consumption of glycemic foods influences adipokine levels and insulin resistance in these populations.

Using a multiethnic population–based sample in which adiposity, adipokines, and insulin resistance–related

From the [1]Population Health Research Institute, Hamilton Health Sciences, McMaster University, Hamilton, Ontario, Canada; the [2]Departments of Medicine and Clinical Epidemiology and Biostatistics, McMaster University, Hamilton, Ontario, Canada; the [3]Department of Medicine, University of Mainz, Mainz, Germany; the [4]Departments of Nutritional Sciences and Endocrinology, University of Toronto, Toronto, Ontario, Canada; and [5]Six Nations Health Services, Ohsweken, Ontario, Canada.
Corresponding author: Sonia S. Anand, anands@mcmaster.ca.
Received 28 July 2009 and accepted 29 March 2010. Published ahead of print at http://care.diabetesjournals.org on 22 April 2010. DOI: 10.2337/dc09-1932.

factors were measured in a standardized manner, we investigated *1*) ethnic variation in levels of adipokines, *2*) whether higher intake of glycemic foods differentially affects adipokine levels across ethnic populations, and *3*) whether levels of adipokines and high glycemic index/glycemic load account for ethnic variation in insulin resistance.

## RESEARCH DESIGN AND METHODS

— The study population comprised Canadians of South Asian, Chinese, Aboriginal, or European origin who participated in the Study of Health Assessment and Risk in Ethnic Groups (SHARE), a cross-sectional prevalence study of cardiovascular disease risk factors conducted between 1996 and 1998 (2). Individuals were randomly selected from three cities (Toronto, Hamilton, and Edmonton) and from the Six Nations Reservation (Ohsweken, ON) as described previously (1,2). Subjects' ages were 35–75 years, and they had lived in Canada for ≥5 years. Individuals with treated diabetes or chronic debilitating illnesses such as terminal cancer and renal failure were excluded. Ethics approval was obtained from the McMaster University Research Ethics Board and all participating institutions. Informed consent was obtained from each subject. The investigation conforms with the principles outlined in the Declaration of Helsinki.

### Assessment of adipokines and insulin resistance–related factors

Participants completed lifestyle questionnaires, which recorded information on physical activity, smoking patterns, and dietary intake as described previously (1,2). Physical measurements included height, weight, and waist and hip circumference using a standardized protocol. The physical activity index involved summing ordinal categories of intensity of physical exertion estimated from reported type of work, time spent playing sports, and type of leisure-time activities. Occupation, sports, and leisure-time activities were classified according to exertion as 1 = low, 2 = moderate, and 3 = high based on the published literature (10).

Fasting blood samples were collected in the morning from all participants. Blood samples were collected and processed according to a standard protocol and were shipped to the core laboratory in Hamilton for analysis. All subjects underwent a 12-h fast before blood was drawn, and nondiabetic participants underwent an oral glucose tolerance test with measurement of glucose, insulin, triglyceride, and free fatty acid levels at baseline and at 2 h after the glucose load. Glucose was measured using enzymatic methods, and insulin was determined by manual radioimmunoassay assay (Diagnostic Products Corporation, Los Angeles, CA). Analysis of adiponectin and leptin was performed in the laboratory of Dr. Stephan Blankenberg at the University of Mainz (Mainz, Germany) using a commercially available human adiponectin ELISA (RD195023100) and human leptin ELISA (RD191001100), produced by Biovendor Research Products. For adiponectin, the intra-assay imprecision is 6.4–7.0% and the interassay imprecision is 7.3–8.2%. For leptin, the intra-assay imprecision is 3.0–7.5% and the interassay imprecision is 3.2–9.2%. Basal insulin resistance was calculated using the previously validated homeostasis model assessment of insulin resistance (HOMA-IR).

### Dietary assessment

On the food-frequency questionnaire, participants reported how often, on average, they consumed selected foods in the previous year. We calculated nutrient intakes by multiplying the average nutrient content of a particular food portion by the number of times it was consumed. Glycemic index and glycemic load were estimated based on the International Table of Glycemic Index for specific foods. Briefly, we assigned glycemic index values to each of the individual food frequency questionnaire food items by manual review of the glycemic index table. We then computed sex- and serving size–specific glycemic loads for each of the food items using the weighted mean methods as described by Subar et al. (11). Each unit of glycemic load represents both the quality and the quantity of carbohydrate intake (or the equivalent of 1 g of carbohydrate from white bread). The overall glycemic index for each participant was calculated by dividing the participant's glycemic load by the total grams of carbohydrate consumed, which represents the overall quality of carbohydrate intake.

### Statistical analysis

All analyses were computed using SAS (version 9.1; SAS Institute, Cary, NC). Distributions of adiponectin, leptin, and HOMA-IR were highly skewed, so geometric means and natural logs are presented, adjusting for age, sex, and adiposity measures where appropriate. Post hoc pairwise comparisons were performed using Tukey tests to adjust for multiple comparisons. Pearson correlation coefficients and linear regression were used to assess the association among continuous variables, with age, sex, and markers of adiposity used as covariates where indicated. The independent predictive value of logarithmically transformed adiponectin and leptin on insulin resistance was examined in a linear regression model with log-transformed HOMA-IR as the dependent variable along with other known determinants of insulin resistance as independent variables (age, ethnicity, smoking, BMI, waist-to-hip ratio [WHR], glycemic load, energy intake, C-reactive protein, and physical activity). Linear regression modeling was also used to assess effects of glycemic index or load on adipokine concentration and insulin resistance by ethnic origin. Differences with $P < 0.05$ were considered statistically significant.

## RESULTS

— Complete data were available for 1,258 people from the SHARE population. From this sample, 18 individuals who had implausible levels of adiponectin (>100 μg/ml) or leptin (>180 ng/ml) and 64 with treated diabetes were excluded, leaving a final sample of 1,176 participants.

The characteristics of participants are displayed in Table 1. The mean age of the overall population was 50.3 years, and men and women were equally represented. Significant differences in age and lifestyle factors including current smoking, physical activity, measures of adiposity, plasma lipids, diastolic blood pressure, and insulin resistance were present among ethnic groups (Table 1). For example, Aboriginal people had substantially higher BMI and abdominal obesity relative to those of other ethnic groups. Conversely, people of Chinese origin had the lowest BMI and abdominal adiposity. Europeans have a higher BMI, yet less abdominal obesity compared with South Asians. Despite these differences, South Asians and Aboriginal people were more insulin resistant than the Europeans (all $P < 0.05$).

### Adipokines and ethnicity

Adiponectin levels were significantly higher in Europeans and Aboriginal people than in those of the other ethnic groups (age- and waist-adjusted mean [95% CI] in Europeans 12.96 μg/ml [12.27–13.64] and Aboriginal people

**Table 1—*Distribution of risk factors among Europeans, Chinese, South Asians, and Aboriginal people living in Canada***

| | European | Chinese | South Asian | Aboriginal | Overall *P* value* |
|---|---|---|---|---|---|
| *n* | 312 | 303 | 317 | 244 | |
| Age (years) | 51.3 ± 0.6 | 47.8 ± 0.6 | 49.4 ± 0.6 | 52.9 ± 0.6 | <0.001[1–6] |
| Female sex | 160 (51.3) | 150 (49.5) | 142 (44.8) | 141 (57.8) | 0.169 |
| Current smoker | 52 (16.7) | 17 (5.6) | 32 (10.1) | 97 (39.8) | <0.05[1–6] |
| BMI (kg/m$^2$)† | 27.5 ± 0.3 | 23.8 ± 0.3 | 26.1 ± 0.3 | 31.9 ± 0.3 | <0.001[1–6] |
| WHR† | 0.87 ± 0.004 | 0.86 ± 0.004 | 0.88 ± 0.004 | 0.93 ± 0.004 | <0.001[1–6] |
| LDL cholesterol (mmol/l)† | 3.17 ± 0.05 | 3.14 ± 0.05 | 3.29 ± 0.04 | 3.16 ± 0.05 | 0.085[4] |
| HDL cholesterol (mmol/l)† | 1.19 ± 0.02 | 1.19 ± 0.02 | 1.05 ± 0.02 | 1.07 ± 0.02 | <0.001[2–5] |
| Triglycerides (mmol/l)† | 1.39 ± 0.07 | 1.46 ± 0.08 | 1.70 ± 0.07 | 1.68 ± 0.07 | <0.001[2–5] |
| C-reactive protein (mg/l)† | 1.25 ± 0.17 | 0.70 ± 0.14 | 1.79 ± 0.20 | 3.32 ± 0.28 | <0.001[1–6] |
| Free fatty acids (mEq/ml)† | 512 ± 13 | 523 ± 13 | 535 ± 13 | 544 ± 14 | 0.35 |
| Fasting glucose (mmol/l) | 5.10 ± 0.06 | 5.18 ± 0.06 | 5.45 ± 0.06 | 5.56 ± 0.07 | <0.001[2–5] |
| 2-h glucose (mmol/l) | 6.08 ± 0.17 | 6.79 ± 0.17 | 7.14 ± 0.16 | 6.41 ± 0.19 | <0.001[1,2,6] |
| A1C (%) | 5.32 ± 0.04 | 5.59 ± 0.04 | 5.76 ± 0.04 | 5.95 ± 0.05 | <0.001[1–6] |
| Impaired fasting glucose | 8 (2.6) | 1 (0.3) | 7 (2.2) | 11 (4.5) | 0.16 |
| Impaired glucose tolerance | 35 (11.2) | 46 (15.2) | 56 (17.7) | 30 (12.3) | 0.11 |
| Newly diagnosed type 2 diabetes‡ | 16 ± 5.1 | 12 ± 4.0 | 27 ± 8.5 | 21 ± 8.7 | 0.02 |
| HOMA-IR† | 2.12 ± 0.14 | 2.23 ± 0.11 | 3.03 ± 0.23 | 4.85 ± 0.35 | <0.001[2–6] |
| Systolic blood pressure (mmHg)† | 119 ± 0.9 | 119 ± 0.9 | 120 ± 0.9 | 118 ± 1.0 | 0.625 |
| Diastolic blood pressure (mmHg)† | 73 ± 0.6 | 75 ± 0.6 | 76 ± 0.6 | 67 ± 0.6 | <0.001[1–3,5,6] |
| Energy intake (kcal)† | 1,994 ± 41 | 1,850 ± 44 | 1,754 ± 42 | 2,218 ± 49 | <0.001[1–3,5,6] |
| High physical activity | 90 ± 31.6 | 44 ± 16.7 | 43 ± 15.5 | 41 ± 22.9 | <0.001[1,2,5,6] |
| Physical activity (h/week)† | 8.05 ± 0.09 | 7.11 ± 0.09 | 7.25 ± 0.09 | 7.74 ± 0.10 | <0.001[1–3,5,6] |
| Adiponectin (μg/ml)§ | | | | | |
| Men | 10.89 ± 0.86 | 7.53 ± 0.88 | 8.26 ± 0.45 | 9.63 ± 0.39 | <0.0001[1,2,5,6] |
| Women | 15.01 ± 1.13 | 10.28 ± 0.71 | 11.02 ± 0.76 | 13.15 ± 0.63 | <0.0001[1–3,5,6] |
| Total | 12.96 ± 0.73 | 8.52 ± 0.57 | 9.35 ± 0.43 | 11.87 ± 0.41 | <0.0001[1,2,4–6] |
| Leptin (ng/ml)‖ | | | | | |
| Men | 5.93 ± 0.86 | 5.37 ± 0.88 | 7.24 ± 0.60 | 5.37 ± 1.39 | <0.0001[2,4,6] |
| Women | 13.60 ± 1.36 | 14.01 ± 1.21 | 22.87 ± 1.54 | 16.95 ± 2.07 | <0.0001[2–6] |
| Total | 9.21 ± 0.85 | 8.25 ± 0.77 | 11.82 ± 0.94 | 11.13 ± 1.21 | <0.0001[2–5] |

Data are means ± SEM or *n* (%). *$P$ values: [1]$P < 0.05$, European vs. Chinese; [2]$P < 0.05$, European vs. South Asian; [3]$P < 0.05$, European vs. Aboriginal people; [4]$P < 0.05$, Chinese vs. South Asian; [5]$P < 0.05$, Chinese vs. Aboriginal people; [6]$P < 0.05$, South Asian vs. Aboriginal people. †Means are adjusted for age and sex. ‡New clinically diagnosed type 2 diabetes as determined by 2-h oral glucose tolerance test. §Geometric means for adiponectin are adjusted for age and waist circumference (because adiponectin is highly correlated with waist girth). ‖Geometric means for leptin are adjusted for age and BMI (because leptin is highly correlated with BMI).

11.87 μg/ml [11.19–12.59] vs. South Asians 9.35 μg/ml [8.82–9.92] and Chinese 8.52 μg/ml [8.03–9.03]; overall $P < 0.001$) (Table 1). Serum leptin levels were significantly higher in South Asians (11.82 [10.72–13.04]) and Aboriginal people (11.13 [10.13–12.23]) than in Europeans (9.21 [8.38–10.12]) and Chinese (8.25 [7.48–9.10]) (Table 1).

South Asian women and men had significantly higher leptin concentrations than all other groups (Table 1). A significant sex by ethnic origin interaction with serum leptin was present ($P = 0.01$), as Aboriginal women had significantly higher leptin concentrations than European women for the same BMI, whereas no significant differences were observed among men. No significant sex by ethnic origin interaction was observed for adiponectin.

## Adipokines and ethnicity by adiposity

Adiponectin was strongly and inversely associated with all adiposity measures, whereas leptin was positively associated with the adiposity measures (all *P* values <0.001). As shown in Table 1 (available in an online appendix at http://care.diabetesjournals.org/cgi/content/full/dc09-1392/DC1), adiponectin was negatively associated with BMI, waist circumference, waist adjusted for hip circumference, and WHR in Europeans, Chinese, and Aboriginal people, but not in South Asians. Leptin was significantly associated with BMI and central adiposity measures across all ethnic groups.

## Adipokines and insulin resistance

To determine whether adiponectin and leptin were independently associated with insulin resistance over and above factors known to be associated with insulin resistance (i.e., adiposity, glycemic diet, smoking, and physical activity), a multivariate linear regression analysis was performed. As shown in Table 2 (available in the online appendix), factors that were positively associated with insulin resistance included South Asian ($P < 0.001$), Chinese ($P < 0.001$), and Aboriginal ($P < 0.001$) ancestry, serum leptin ($P < 0.001$), age ($P = 0.01$), BMI ($P < 0.001$), and WHR ($P < 0.001$). Factors that were negatively associated with insulin resistance included serum adiponectin ($P < 0.001$), female sex ($P = 0.02$), and physical activity ($P = 0.003$). C-reactive protein, glycemic index/glycemic load, current smoking, and energy intake were not independently associated with insulin resistance after accounting for the above

factors (Table 2). These factors accounted for 0.3% ($P = 0.11$) of the variance in HOMA-IR scores beyond the contribution of other factors. The individual factor contributions to the model are shown in Table 3 (available in the online appendix).

In a linear regression analysis of HOMA-IR scores as a function of log adiponectin by ethnic categories, the association between adiponectin and insulin resistance varied significantly across ethnic groups ($P = 0.009$ for interaction), as the increase in HOMA-IR for each given decrease in adiponectin was larger among South Asians ($P = 0.024$) and Aboriginal people ($P = 0.010$) than among Europeans. No significant effect modification by ethnicity was found for leptin. There was no significant three-way effect modification among ethnicity, adiponectin and leptin, and BMI and WHR.

### Adipokines, insulin resistance, and glycemic index/glycemic load

No significant association of glycemic index or glycemic load with adipokine concentration was observed overall. In an assessment of effect modification by BMI and ethnicity, there was a significant three-way effect modification among ethnicity, glycemic index, and BMI in predicting adiponectin ($P = 0.006$). Among study participants with BMI $\geq$30 kg/m$^2$, there was a larger decrease in adiponectin levels for each given increase in glycemic index in South Asians ($P = 0.03$) and Aboriginal people ($P < 0.001$) than in Europeans. However, among nonobese participants, the degree of change in adiponectin with greater glycemic index was similar across ethnic groups. No significant two-way or three-way effect modification among ethnicity, glycemic index/glycemic load, and BMI was found for leptin.

In a linear regression analysis for HOMA-IR scores as a function of glycemic index by ethnic categories, the association between glycemic index and insulin resistance varied significantly across ethnic groups ($P = 0.01$ for interaction), as a positive association was found only among South Asians ($P < 0.001$). The increase in HOMA-IR for each given increase in glycemic index was significantly larger among South Asians than among Europeans ($P = 0.03$), Chinese ($P = 0.008$), and Aboriginal people ($P = 0.006$). No significant effect modification by ethnicity was found for glycemic load ($P = 0.31$). There was no significant three-way effect modification among ethnicity, glycemic index/glycemic load, and adiponectin or leptin.

**CONCLUSIONS** — To our knowledge, this investigation is the first to compare adiponectin, leptin, and other insulin resistance–related factors in a large randomly assembled multiethnic population. Our study demonstrates that South Asians have an unfavorable adipokine profile, which is characterized by lower adiponectin and higher leptin for the same degree of adiposity as Europeans. Furthermore, we have shown that a greater consumption of foods with a high glycemic index is associated with a significantly larger decrease in adiponectin among South Asians and Aboriginal people compared with Chinese and Europeans. In addition, these groups display a greater increase in insulin resistance with decreasing levels of adiponectin, and a high glycemic diet predicts higher insulin resistance predominantly in South Asians. To date, few studies have assessed dietary effects on adipokine concentrations, and we know of no previous study to assess dietary glycemic index in relation to adipokine profile and insulin resistance in multiple ethnic groups including populations at high risk. Our findings suggest that modifying intake of glycemic foods could especially improve adipokine concentrations and insulin sensitivity in South Asians and Aboriginal people, two populations at increased risk for insulin resistance.

The large size and ethnic variation of our cohort allowed us to examine adiponectin and leptin over a wide range of body weights and abdominal fat distribution. Overall, adiponectin decreased and leptin increased with higher adiposity, which is consistent with recent evidence showing that the amount of intra-abdominal fat modulates serum adipokine levels (12). Cultured adipocytes derived from visceral fat are known to secrete an increased amount of adiponectin in response to insulin or rosiglitazone treatment, whereas adipocytes derived from subcutaneous fat are unaffected (13). In subgroup analyses, we found that there is no association between adiponectin and BMI, waist circumference, waist adjusted for hip circumference, or WHR in South Asians. Similar results have been observed in a recent study of healthy South Asians (14), yet another investigation showed an inverse association between adiponectin and BMI in South Asian women with gestational diabetes (15). Our findings may reflect the lack of specificity of measuring adiposity using BMI and waist circumference or may reflect different pathogenic pathways including the type, amount, and distribution of adipose tissue accumulation as well as dietary and genetic influences that may exist among South Asians.

Few previous studies have assessed adipokine levels in South Asians compared with those in individuals of European origin. Several reports showed that adiponectin is significantly lower in South Asians than in Europeans (16,17). These studies, however, were generally small and often did not include a comparison group (15,18). In our study, South Asians displayed the least favorable adipokine profile (lower adiponectin and higher leptin) of all ethnic groups, despite having BMI comparable to that of Europeans. The metabolic disturbance behind these differences is not known but may be related to differences in adipocyte properties (e.g., hypertrophic versus hyperplastic adipocytes) or distribution of adipose tissue between the groups (19). We also found strikingly high levels of leptin among South Asian women compared with those among European women, a finding that was noted previously in young adults (20). Higher leptin in women than in men is attributed to their greater subcutaneous fat mass. However, prior studies did not assess whether these relationships vary by ethnic origin. Our subgroup analysis showed a significantly stronger correlation between serum insulin and leptin among European women than among South Asian women, whereas WHR and leptin levels showed similar correlation values across groups. Therefore, distribution of fat (i.e., truncal obesity) and hyperinsulinism do not appear to explain the higher leptin levels among South Asian women. There is, however, evidence that hypertrophic adipocytes secret more leptin than do hyperplastic adipocytes (21), and subcutaneous adipocyte hypertrophy could account for the higher leptin levels in South Asians (19).

Our study showed that Aboriginal people have higher leptin levels than Europeans but similar adiponectin levels. The findings of previous studies comparing adiponectin levels in Aboriginal people with those in Europeans have been inconclusive, with some studies reporting lower levels in Aboriginal people (22) and other recent studies showing no significant differences (12). These discrepant

findings may also be attributable to the smaller sample of participants and different sample selection. Our finding that Chinese have lower adiponectin levels than do Europeans is consistent with previous results in East Asian populations (23) and with our past observations showing that, despite having relatively low BMI, the Chinese are at increased risk of developing impaired fasting glucose, impaired glucose tolerance, and type 2 diabetes with central fat accumulation (2).

Our finding that lower adiponectin and elevated leptin levels are independent determinants of increased insulin resistance over and above lifestyle factors, anthropometric indexes, and inflammatory markers is consistent with previous cohort studies (24). In addition, when we assessed the effect of serum adipokines on insulin resistance by ethnic group, South Asians and Aboriginal people showed a significantly greater increase in insulin resistance per unit decrease in adiponectin than Chinese and Europeans. Several smaller studies (18), but not all (16,17), have also related low adiponectin to HOMA-IR values in young healthy South Asians. In Alaskan and Canadian Native populations, adiponectin was also inversely associated with HOMA-IR scores (12,25). Our findings suggest that pharmacologic agents that work by altering adiponectin levels may be more effective in these groups. Adiponectin levels increase with reduction in fat mass, administration of peroxisome proliferator–activated receptor-γ agonists such as the thiazolidinediones, and renin-angiotensin system blockade, and individuals with the lowest baseline levels may benefit the most from these therapeutic strategies (4).

Our finding that glycemic index predicts greater increases in insulin resistance in South Asians compared with that in other ethnic groups has not been reported previously. There is some evidence that carbohydrate intake is associated with poorer glycemic control in South Asians (8) and Aboriginal people (9), but total glycemic index/glycemic load or long-term intake were not evaluated. In this study, we did not find evidence that glycemic index is associated with insulin resistance. Nevertheless, our findings show that South Asians and Aboriginal people display a greater decrease in adiponectin with increasing glycemic index, particularly at higher BMI. We are not aware of any previous study that assessed dietary effects on adipokine concentrations in ethnic populations. Our findings suggest that modifying intake of glycemic foods could improve adipokine concentrations and insulin sensitivity most profoundly in South Asians and Aboriginal people.

Although the underlying mechanism linking glycemic index to adipokine levels is not clear, the findings are compatible with the overall poor adipokine profile observed in these groups. Glycemic foods are known to induce both hyperglycemia and hyperinsulinemia. There is evidence that adipose tissue expression of adiponectin is inversely correlated with fasting glucose concentration and that a glucose-rich diet reduces adiponectin expression in adipose tissue (26). South Asian and Aboriginal populations largely consume a diet consisting of foods with a high glycemic index (10). It has been recognized that glycemic foods influence body fat (27), which suggests that the effects could be at least partly mediated by adipose tissue–related pathways. Conversely, fiber intake is positively associated with adiponectin (6) and may promote the clearance of lipids and thus reduce free fatty acids available for storage in adipose tissue (28).

Our study has some limitations. We used surrogate measures of adiposity (e.g., BMI and waist circumference), which are merely proxy indicators of visceral fat, a stronger predictor of insulin resistance. Although these measures show considerable variation among different ethnic groups (1,2), the findings relating central fat with adiponectin in our study should be viewed with caution. Our study used total adiponectin as opposed to the active high–molecular-weight multimer, which may be the critical determinant of insulin sensitivity. Nevertheless, total adiponectin was significantly associated with insulin resistance, and ethnic comparisons in our study probably reflect similar relative differences in high–molecular-weight adiponectin. Drug treatment for diabetes can alter adipokine concentration, and thus, we excluded these patients from our analyses. In a sensitivity analysis that further excluded patients with new clinically diagnosed diabetes ($n = 76$), the ethnic differences in adipokine levels and accompanying associations between adipokines and insulin resistance were unaltered. Exclusion of individuals with impaired fasting glucose ($n = 27$), impaired glucose tolerance ($n = 167$), and newly diagnosed diabetes preserved the trends that we observed, although the significance was diminished because of a loss of statistical power.

In summary, South Asians have an unfavorable adipokine profile compared with that of other ethnic groups and, like the Aboriginal people, display a greater increase in insulin resistance with decreasing levels of adiponectin. Differences in adipokines and responses to glycemic foods parallel the increased propensity of certain ethnic groups to develop insulin resistance.

**Acknowledgments**— This study was supported by the Canadian Institutes of Health Research (grant MT-12790), the Heart and Stroke Foundation of Canada, and Merck Frosst Canada. A.M. holds a Heart and Stroke Foundation of Canada Postdoctoral Research Fellowship. S.Y. holds a Heart and Stroke Foundation of Ontario Chair in Cardiovascular Research. S.S.A. holds the Eli Lilly May Cohen endowed chair in Women's Health Research and the Michael G. DeGroote Heart and Stroke Foundation of Ontario Research Chair in Population Health Research.

No potential conflicts of interest relevant to this article were reported.

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