Paradoxical Relationships Between Anthropometric Variables and Phenotypic Expression of the Metabolic Syndrome in Nondiabetic Polynesians of New Caledonia

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In the multiethnic population of New Caledonia, the CALDIA (CALedonia DIAbetes Mellitus) Study showed that the prevalence of type 2 diabetes was much higher (15.3%) in Polynesians (mostly Wallisians) than in Melanesians or Europeans (8.4%) (1). Polynesians also exhibit high rates of obesity (2,3), known to be a risk factor for insulin resistance and type 2 diabetes (4,5). However, recent analyses of the CALDIA Study showed that despite high indexes of abdominal obesity, Polynesians had low fasting plasma insulin levels and homeostasis model assessment of insulin resistance (HOMA-IR) (6). Since abdominal obesity is usually associated with impaired fasting glucose, dyslipidemia, and/or hypertension, forming the metabolic syndrome (7,8), we investigated whether these features were present in this group, in comparison with Melanesians and Europeans.

RESEARCH DESIGN AND METHODS

— The CALDIA Study is a large population-based study of diabetes prevalence in New Caledonia. The protocol has been previously described in detail (1). A population-based sample of 9,390 subjects, aged 30–59 years, residing in New Caledonia >10 years were visited at home for capillary blood glucose measurement. Among them, 643 subjects were known to have diabetes or had capillary blood glucose values ≥6.1 mmol/l when fasting or ≥7.8 mmol/l when not fasting (positive screenees). A total of 588 subjects (response rate 91.5%), together with 517 negative screenees matched by ethnic group, sex, age, and location, underwent a more detailed examination, including a 2-h oral glucose tolerance test and anthropometric and biochemical measurements. Of these, we selected for the analysis 58 Europeans, 298 Melanesians, and 63 Polynesians with no known or newly diagnosed diabetes (i.e., fasting plasma glucose [FPG] <7 mmol/l and 2-h plasma glucose <11.1 mmol/l at the oral glucose tolerance test), no antihypertensive treatment, and no missing data for variables of interest.

We compared metabolic syndrome parameters across the ethnic groups and studied the association between metabolic variables and waist circumference. All statistical analyses were performed using SAS software.

RESULTS

— Means of metabolic syndrome parameters for each ethnic group are shown in Fig. 1. Despite having the highest means of waist circumference, FPG, and a higher prevalence of glycemic abnormalities, Polynesians had lower triglycerides and systolic blood pressure (SBP) than Melanesians. Levels of triglycerides, SBP, and HOMA-IR were lower in Polynesians compared with Europeans after adjustment for age, sex, and BMI, but the differences were not statistically significant. Levels of HDL cholesterol were similar between the three groups with or without adjustment.

To assess the contribution of abdominal obesity to the variability of metabolic syndrome parameters, regression analyses were performed separately for each ethnic group, after adjustment for age and sex. In all ethnic groups, waist circumference was positively associated with SBP. As expected, we also found a positive correlation between waist circumference and triglycerides in Europeans ($P < 0.02$) and Melanesians ($P < 0.0001$). Surprisingly, there was no such correlation in Polynesians. FPG was significantly associated with waist circumference only in Melanesians ($P < 0.0004$). HDL cholesterol levels were not associated with waist circumference in any group.

CONCLUSIONS

— Polynesians did not exhibit the expected cluster of abnormalities usually observed in the metabolic syndrome, given their obesity indexes. They displayed the lowest triglyceride levels, together with relatively low mean values for SBP. Moreover, triglycerides and FPG levels were independent of waist circumference. This lack of association might, however, be a consequence of a lack of power and should be confirmed on larger samples.

Our study is another observation that the relation between obesity and the cluster of metabolic syndrome abnormalities differs according to ethnicity (9,10), which raises discussions about the validity of metabolic syndrome definitions (11). For example, Canadian or Greenland Inuits have rather low prevalence of dyslipidemia and hypertension despite high occurrence of abdominal obesity (12,13). Conversely, South Asian populations

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Abbreviations: FPG, fasting plasma glucose; HOMA-IR, homeostasis model assessment of insulin resistance; SBP, systolic blood pressure.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

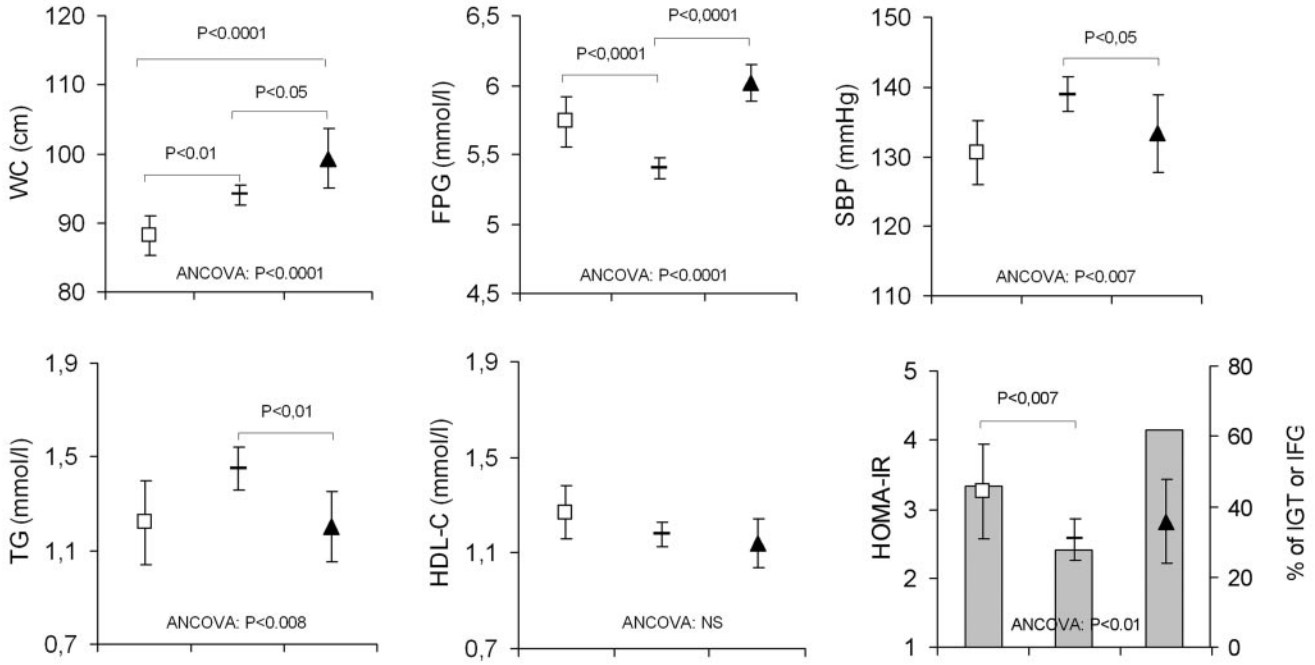

Figure 1—*Mean values and 95% CIs of metabolic syndrome parameters, HOMA-IR, and prevalence of impaired fasting glucose (IFG) or impaired glucose tolerance (IGT). ANCOVA* P *values for ethnic group effect are adjusted for age and sex (for waist circumference [WC]) and for age, sex, and BMI (for FPG, SBP, triglycerides [TG], HDL cholesterol [HDL-C], and HOMA-IR).* P *values for two-by-two comparisons using Bonferroni correction are indicated when significacnt. Triglycerides and HDL cholesterol were log transformed for calculations.* □*, Europeans;* –, Melanesians; ▲, Polynesians.

often exhibit the cluster of metabolic syndrome abnormalities, although they have low obesity rates compared with Caucasian populations; however, they generally have an excess of visceral fat (14).

One explanation for these discrepancies may be that amount of overall or visceral fat is poorly assessed by usual anthropometric indexes (15). We had to rely on these because no direct estimation of fat mass or body composition was available in the CALDIA Study, and some studies have shown that Polynesians had higher percentage of lean mass and higher bone mineral density compared with Caucasians (16–18). Moreover, it was observed that most Polynesians in the CALDIA Study were employed in heavy work, which may enhance their muscular mass.

The particular metabolic profile we observed in Polynesians may also be related to a low degree of insulin resistance, considered as the leading factor predisposing to the metabolic syndrome (19–21). In a previous study, we have shown in accordance with Simmons et al. (3) that Polynesians had low fasting insulin levels and HOMA-IR values (6). This may be due to their high physical activity or other environmental factors, such as diet, or to genetic factors.

In conclusion, despite their large body mass, nondiabetic Polynesians do not seem to display the expected profile of disorders described under conventional definitions of the metabolic syndrome. This suggests that either anthropometric indexes do not properly reflect body fat mass or that pathogenetic mechanisms leading to the syndrome are not sufficiently understood. This may have implications for the diagnosis and characterization of metabolic syndrome across populations, as well as for the assessment of its related disease risks.

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