

# Relationships between glycaemic abnormalities, obesity and insulin resistance in nondiabetic Polynesians of New Caledonia.

Roselyne Defay, Isabelle Jaussent, Annie Lacroux, Annick Fontbonne

► **To cite this version:**

Roselyne Defay, Isabelle Jaussent, Annie Lacroux, Annick Fontbonne. Relationships between glycaemic abnormalities, obesity and insulin resistance in nondiabetic Polynesians of New Caledonia.. Int J Obes (Lond), 2007, 31 (1), pp.109-113. 10.1038/sj.ijo.0803384 . inserm-00000102

**HAL Id: inserm-00000102**

**<https://www.hal.inserm.fr/inserm-00000102>**

Submitted on 7 Nov 2006

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

**Relationships between glycaemic abnormalities, obesity, and insulin resistance in non diabetic Polynesians of New Caledonia.**

Running title: Diabetes risk profile in Polynesians

5

R Defay<sup>1</sup>, I Jaussent<sup>2</sup>, A Lacroux<sup>1</sup>, A Fontbonne<sup>1</sup>, and the CALDIA Study Group

<sup>1</sup>INSERM, UR024 Epiprev, IRD, F-34394 Montpellier, France; and <sup>2</sup>INSERM E0361, F-34093 Montpellier, France.

10 Correspondence:

Dr A Fontbonne

Current postal address:

IRD–UR 024, 911 avenue Agropolis, BP64501, F-34394 Montpellier Cedex 5, France

E-mail: [fontbon@montp.inserm.fr](mailto:fontbon@montp.inserm.fr)

15 Tel: +(33)467416158

Fax: +(33)467416330

## Abstract

**OBJECTIVE:** Polynesians in New Caledonia have an increased risk for developing diabetes, compared to Melanesians or Europeans. They are also more prone to obesity. The aim of this study was to analyse differences in the pre-diabetic state that may explain the varying susceptibility to diabetes between these three ethnic groups, focusing on the balance between insulin resistance and capacity of pancreatic cells to secrete insulin.

**DESIGN AND SUBJECTS:** The CALDIA Study is a population-based cross-sectional survey of diabetes prevalence conducted in New Caledonia. All participants who did not have diabetes, according to the results of a 0-2h oral glucose tolerance test (n=392), were selected for analysis.

**RESULTS:** Compared to Europeans, Polynesians and Melanesians had significantly higher body mass indices (BMI) and waist-to-hip ratios (WHR). Polynesians had higher fasting plasma glucose values than Europeans or Melanesians (6.03 mmol/l, vs 5.78 and 5.46, respectively;  $P<0.0001$ ). Fasting plasma insulin level and the estimate of insulin resistance by homeostasis model assessment (HOMA) were not significantly different between the three ethnic groups. HOMA estimate of  $\beta$ -cell secretory capacity was lower in Polynesians compared to the two other ethnic groups (83.1 mU/mmol, vs 119.3 and 125.2, respectively;  $P<0.02$ ).

**CONCLUSION:** Despite a high prevalence of central obesity, as judged by high BMI and WHR, in Polynesians of New Caledonia, their high risk of diabetes may be more strongly related to a defect in insulin secretion capacity than to insulin resistance.

---

**Keywords:** epidemiology; Polynesians; insulin resistance; insulin secretion; type 2 diabetes

## Introduction

Type 2 diabetes is considered to be the result of a two-step process.<sup>1</sup> In the first “prediabetic” step characterised by insulin resistance, compensatory hyperinsulinaemia helps maintain normal glucose levels. In the second step there is a decline in  $\beta$ -cell secretory capacity together with a progressive elevation of glucose levels which define diabetes.<sup>2</sup> This two-step mechanism has been shown to apply to many populations around the world (e.g., Pima Indians, Micronesians, Mexican Americans, South Asians).<sup>3</sup> However, the balance between insulin resistance and deficient insulin secretion in the pathogenesis of type 2 diabetes appears to vary between ethnic groups.<sup>4</sup> Regarding Polynesians, a population with a high prevalence of obesity and diabetes, one study in New Zealand reported that, after adjustment for body mass index, Polynesians were not more insulin-resistant than Europeans, who are much less susceptible to diabetes.<sup>5</sup>

Previous publications from the CALDIA Study, a large diabetes screening survey conducted in the multi-ethnic population of New Caledonia, have shown that Polynesians indeed had the highest degree of central obesity, and the highest prevalence rate of diabetes compared to the other two major ethnic groups living in the archipelago (15.3%, vs 8.4% in Melanesians or Europeans).<sup>6,7</sup> In order to further examine the abnormalities that could explain their higher risk of diabetes, we selected from the CALDIA database non diabetic subjects with the purpose of comparing the degree of insulin resistance and of  $\beta$ -cell secretory capacity (using the homeostasis model assessment, or HOMA) between ethnic groups.

## Population and methods

The CALDIA Study was conducted from 1992 to 1994 to determine the prevalence of diabetes in New Caledonia. The rationale, design and methods of the study were previously described in detail.<sup>6</sup> To summarise, the target population for the CALDIA Study were subjects

aged 30-59, resident in New Caledonia for more than 10 years. Subjects were recruited all over the territory (North province, Noumea, and Loyalty Islands). In the North province, 12 small towns and 101 villages out of 199 were randomly selected. In Noumea, six suburbs were chosen because they included all the ethnic groups. In the Loyalty Islands, all 85 villages participated. As a whole, 9390 subjects (representing a response rate of 78%) were visited at home for screening, where they all had a capillary blood glucose (CBG) measurement with a reflectance meter (One Touch<sup>®</sup>, LifeScan). All previously known diabetic subjects and subjects having a CBG value  $\geq 6.1$  mmol/l when fasting, or CBG  $\geq 7.8$  mmol/l when non fasting (n=643), were invited to come to the health centre for a more detailed examination. The response rate for this examination was 91.5% (588 subjects). At the same time, a selection of 517 subjects with CBG  $< 6.1$  mmol/l, matched by ethnic group, gender, age, and location, also underwent the examination.

At the health centre, participants answered a standardised questionnaire and underwent anthropometric measurements. Body mass index ( $\text{BMI} = \text{weight (kg)} / \text{height (m)}^2$ ) was used to report general obesity. According to the World Health Organization (WHO) criteria,<sup>8</sup> subjects with BMI 25-29.9 were considered overweight and subjects with BMI  $\geq 30$  were classified as obese. Waist and hip circumferences were measured in the standing position to the nearest cm, the first at the umbilicus, the second at the iliac crest. The waist-to-hip ratio (WHR) was calculated as an index of upper-body adiposity.

A two-hour oral glucose tolerance test (OGTT) with a 75-g glucose load was performed according to the WHO recommendations.<sup>9</sup> Blood samples were locally centrifuged, frozen and sent to Noumea central laboratory. Fasting plasma glucose (FPG) and 2-hour plasma glucose (2h-PG) levels were assessed by the glucose oxidase method in Noumea. Fasting plasma insulin (FPI) was measured by radioimmunoassay (RIAgnost, Behring, Los Angeles, CA) at the Henri Mondor Hospital, Créteil, France.

The study protocol and procedures were approved by all local medical commissions involved, and all participants gave informed consent for their participation.

Among the 1105 subjects having a complete examination, we excluded diabetic subjects, known or newly diagnosed, that is having FPG  $\geq 7$  mmol/l or 2h-PG  $\geq 11.1$  mmol/l at the OGTT.<sup>9</sup> One unclassified subject was also excluded. Of the remaining subjects, only those of European, Melanesian, or Polynesian origin were retained. Subjects were classified as normoglycaemic (FPG  $< 6.1$  mmol/l and 2h-PG  $< 7.8$  mmol/l), having impaired fasting glucose (IFG) ( $6.1 \text{ mmol/l} \leq \text{FPG} < 7.0 \text{ mmol/l}$  and 2h-PG  $< 7.8$  mmol/l), or impaired glucose tolerance (IGT) ( $7.8 \text{ mmol/l} \leq 2\text{h-PG} < 11.1 \text{ mmol/l}$ , irrespective of FPG values).<sup>9</sup> To assess insulin resistance and  $\beta$ -cell function, the homeostasis model assessment (HOMA),<sup>10</sup> was applied to fasting plasma glucose and insulin values as follows: insulin resistance (HOMA-IR) = FPI (mU/l)  $\times$  FPG (mmol/l) / 22.5;  $\beta$ -cell function (HOMA-BC) = [20  $\times$  FPI (mU/l)] / [FPG (mmol/l) - 3.5].

Age, sex, anthropometric variables and variables characterising glucose-insulin regulation were compared between the three ethnic groups using  $\chi^2$  test for categorical variables and analysis of variance for continuous variables. Glucose homeostasis variables were also compared after multiple adjustment for age, sex and BMI, by analysis of covariance. When comparisons were statistically significant, two-by-two comparisons were carried out, using the Bonferroni correction. Continuous variables with a log-normal distribution were log-transformed before testing, and back-transformed into natural values for presentation. All statistical analyses were performed with SAS statistical package software version 9.1 (SAS Institute, Cary, NC).

## Results

After exclusion of subjects with missing values for the variables of interest, analysis

was performed on 392 subjects (57 Europeans, 287 Melanesians, 48 Polynesians). Their general characteristics are shown in Table 1. Age and sex distribution were not significantly different between the groups. Mean BMI and WHR were highest in Polynesians and lowest in Europeans. Overweight and obesity were significantly more prevalent ( $P<0.0001$ ) in Polynesians or Melanesians than in Europeans.

Non-adjusted and age, sex and BMI-adjusted comparisons of variables characterising glucose-insulin regulation are shown in Table 2. Fasting plasma glucose values were significantly different between the three groups, with the highest mean value observed in Polynesians, who also had the highest prevalence rate of glycaemic anomalies (IFG or IGT: 43.7%, vs 35.1% in Europeans and 22.6% in Melanesians,  $P<0.01$ ). Non-adjusted two-hour plasma glucose was not significantly different between the three groups, but the differences became significant when adjusted for age, sex and BMI. Despite the marked differences in morphotype between the groups, the geometric means of fasting insulin and HOMA-IR were not significantly different. By contrast, there was a significant difference ( $P<0.02$ ) for the marker of insulin secretion capacity, HOMA-BC, with the lowest value observed in Polynesians. When the analysis was performed after adjustment for BMI, the differences became significant for FPI ( $P<0.004$ ) and HOMA-IR ( $P<0.002$ ). The two-by-two comparisons with Bonferroni adjustment (Table 3) indicated that for these markers of insulin resistance, the differences were between Europeans and the two other ethnic groups, with Europeans having significantly higher indices of insulin resistance. By contrast, insulin secretory capacity was significantly lower in Polynesians compared to the two other ethnic groups.

To better understand this finding, the three ethnic groups were divided according to BMI (normal, overweight or obese). At any given body mass, Polynesians had the highest fasting glucose values together with the lowest fasting insulin values, denoting a clear lack of insulin, both absolute and relative to glucose values (Figure 1, panels A and B). This was

confirmed by the fact that the curve for HOMA-BC in Polynesians was consistently and largely lower, at any given body mass, compared to the two other groups (Figure 1, panel C).

## Discussion

5 Polynesians are known to be a population at high risk for type 2 diabetes. This was confirmed in the CALDIA study, where diabetes prevalence rate was twice that observed in Europeans or Melanesians.<sup>6</sup> Moreover, in our analysis restricted to non-diabetic subjects, Polynesians were markedly overweight with upper-body obesity, and they already exhibited abnormalities of glucose homeostasis (higher FPG values and lower prevalence of  
10 normoglycaemic subjects), which are signs of susceptibility to diabetes.<sup>11</sup> While obesity is commonly associated with insulin resistance and hyperinsulinaemia, and the classical view is that these anomalies are markers of the prediabetic state,<sup>12-14</sup> the only accompanying anomaly we found in Polynesians was a relative defect in insulin secretion. The fact that our radioimmunoassay was not specific for insulin does not challenge the finding, since, if there  
15 are differences of proinsulin proportion between the study groups, it is probably higher in Polynesians given their “worse” glucose homeostasis. After adjustment for BMI, insulin sensitivity in both Polynesians and Melanesians was actually greater than in Europeans. Although we used an indirect method (HOMA) for estimation of insulin secretion and insulin resistance, the HOMA indices have been validated against the glucose clamp, for various  
20 degrees of glucose tolerance and plasma insulin levels,<sup>15</sup> and have been largely used in epidemiological studies.<sup>5,16</sup>

Our results are consistent with those of Simmons et al., who found that Polynesians of New-Zealand were not “intrinsically insulin resistant” despite a high prevalence rate of diabetes and a greater central and overall obesity compared to Europeans of the same area.<sup>5</sup> In  
25 their study, although Polynesians had significantly higher markers of insulin resistance in



univariate comparisons, there were no differences after adjustment for BMI (in our study, this was the case even before adjustment for BMI).

Altogether, these two studies suggest that in Polynesians, the progression from normal glucose tolerance to type 2 diabetes may not include an insulin resistance state characterised by compensatory hyperinsulinaemia. It is already known that the pathogenesis of type 2 diabetes is a combination of insulin resistance and reduced insulin secretion,<sup>2</sup> and the predominant mechanism is possibly different in various ethnic groups.<sup>4</sup> Our study points out to pancreatic deficiency being the main anomaly taking part in the Polynesians' high susceptibility to diabetes mellitus. However, the absence of a frank defect of insulin sensitivity is unexpected, given their general morphotype, since excess adiposity is normally accompanied by insulin resistance<sup>17</sup> and decrease in insulin sensitivity is even considered a regulatory response to weight gain, which it serves to limit.<sup>18</sup> Of course, we cannot exclude that HOMA-IR is not a correct estimate of insulin resistance in this ethnic group, although it has been validated in subjects with normal to low insulin concentrations at least in one study.<sup>15</sup> Besides, fasting plasma insulin levels and insulin sensitivity are reciprocally related,<sup>19</sup> making it unlikely that Polynesians would actually be insulin resistant while they have normal insulin levels. If we consider that Polynesians are truly not insulin resistant, one explanation may be that BMI is not a good estimate of adiposity in this particular ethnic group. Indeed, the universal BMI standards for defining "overweight" and "obesity" in adults are based on the risk of adiposity-related diseases in Caucasians,<sup>20</sup> and this may not apply to all populations. For instance, in a meta-analysis about ethnic differences in the associations between BMI and percent body fat, Polynesians had the highest mean BMI whereas their degree of body fatness was below that of some ethnic groups such as Indonesians, Thais or Ethiopians.<sup>21</sup> Another study showed that, at higher BMI levels, Polynesians had significantly more lean mass than Europeans.<sup>22</sup> Another possible explanation

is that, since Polynesians in New Caledonia are generally employed in heavy, energy-consuming work, this may significantly enhance their insulin sensitivity.<sup>4,23</sup> Also, it cannot be excluded that Polynesians have an insulin-deficient form of diabetes, different from type 2.<sup>2,24,25</sup>

5           Whatever the explanation, if it is confirmed that insulin resistance in the prediabetic state is not the prominent anomaly in this ethnic group (and maybe others), a new approach to the prevention of type 2 diabetes should be designed.

## Acknowledgements

10       We thank each member of the CALDIA Study Group who permitted the design and/or conduction of the CALDIA Study. Coordination, INSERM - in France: L Papoz (principal coordinator), D Simon, J Cubeau, A Lacroux, A Forhan and A Ponton; in New Caledonia: S Barny (local coordinator), D Juranville, H Lmahdi and R Manuohalalo. Health and Social Services in New Caledonia - Medical and Research Commission: F Ledoux and C Merger;

15       Territory: M Germain; North Province: P Calen and P Genty; Loyalty Islands: J Coscoquela and P Buffet; South Province: JG Lambert and P Baqué; and Biochemistry (Territorial Hospital): S Solar and Y Barguil.

We thank V Wirquin of the Henri Mondor Hospital Laboratory (Créteil, France) for the measures of fasting plasma insulin.

20       This research was financed by grant 492015 from the INSERM (Institut National de la Santé et de la Recherche Médicale), grant 607006 from the French Ministry of Cooperation, with additional support from the Medical Commission and the Provinces of New Caledonia, the Rotary and Lions Clubs of Noumea, and Behring Diagnostic Laboratory.

## Title and legend to figure

**Figure 1.** Levels of fasting plasma glucose (FPG, panel A), fasting plasma insulin (FPI, panel B) and HOMA estimate of  $\beta$ -cell function (HOMA-BC, panel C) by degree of overweight in  
5 non diabetic Europeans, Melanesians, and Polynesians in New Caledonia.

## References

1. Saad MF, Knowler WC, Pettitt DJ, Nelson RG, Charles MA, Bennett PH. A two-step model for development of non-insulin-dependent diabetes. *Am J Med* 1991; **90**: 229-235.
2. Expert Committee. Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care* 1997; **20**: 1183-1197.
3. Zimmet PZ, McCarty DJ, de Courten MP. The global epidemiology of non-insulin-dependent diabetes mellitus and the metabolic syndrome. *J Diabetes Complications* 1997; **11**: 60-68.
4. Abate N, Chandalia M. The impact of ethnicity on type 2 diabetes. *J Diabetes Complications* 2003; **17**: 39-58.
5. Simmons D, Thompson CF, Volklander D. Polynesians: prone to obesity and Type 2 diabetes mellitus but not hyperinsulinaemia. *Diabet Med* 2001; **18**: 193-198.
6. Papoz L, Barny S, Simon D. Prevalence of diabetes mellitus in New Caledonia: ethnic and urban-rural differences. CALDIA Study Group. CALedonia DIAbetes Mellitus Study. *Am J Epidemiol* 1996; **143**: 1018-1024.
7. Tassie JM, Papoz L, Barny S, Simon D. Nutritional status in adults in the pluri-ethnic population of New Caledonia. The CALDIA Study Group. *Int J Obes Relat Metab Disord* 1997; **21**: 61-66.
8. World Health Organization: *Report of a WHO consultation on obesity: preventing and managing the global epidemic*. Geneva: WHO, 1998.
9. World Health Organization: *Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus*. Geneva: WHO, 1999.

10. 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.
11. Shaw JA, Zimmet PZ, Decourten M, Dowse GK, Chitson P, Gareeboo H, Hemraj F, 5 Fareed D, Tuomilehto J, Alberti KGMM. Impaired fasting glucose or impaired glucose tolerance - What best predicts future diabetes in Mauritius? *Diabetes Care* 1999; **22**: 399-402.
12. Lillioja S, Mott DM, Spraul M, Ferraro R, Foley JE, Ravussin E, Knowler WC, 10 Bennett PH, Bogardus C. Insulin resistance and insulin secretory dysfunction as precursors of non-insulin-dependent diabetes mellitus. Prospective studies of Pima Indians. *N Engl J Med* 1993; **329**: 1988-1992.
13. Dowse GK. Incidence of NIDDM and the natural history of IGT in Pacific and Indian Ocean populations. *Diabetes Res Clin Pract* 1996; **34(Suppl)**: S45-S50.
14. Haffner SM, Stern MP, Mitchell BD, Hazuda HP, Patterson JK. Incidence of type II 15 diabetes in Mexican Americans predicted by fasting insulin and glucose levels, obesity, and body-fat distribution. *Diabetes* 1990; **39**: 283-288.
15. Bonora E, Targher G, Alberiche M, Bonadonna RC, Saggiani F, Zenere MB, Monauni T, Muggeo M. Homeostasis model assessment closely mirrors the glucose clamp technique in the assessment of insulin sensitivity: studies in subjects with various 20 degrees of glucose tolerance and insulin sensitivity. *Diabetes Care* 2000; **23**: 57-63.
16. Haffner SM, Miettinen H, Stern MP. The homeostasis model in the San Antonio Heart Study. *Diabetes Care* 1997; **20**: 1087-1092.
17. Segal KR, Landt M, Klein S. Relationship between insulin sensitivity and plasma leptin concentration in lean and obese men. *Diabetes* 1996; **45**: 988-991.
- 25 18. Ravussin E, Swinburn BA. Pathophysiology of obesity. *Lancet* 1992; **340**: 404-408.

19. Ferrannini E, Balkau B. Insulin: in search of a syndrome. *Diabet Med* 2002; **19**: 724-729.
20. Nakagami T, Qiao Q, Carstensen B, Nhr Hansen C, Hu G, Tuomilehto J, Balkau B, Borch Johnsen K, The DECODE-DECODA Study Group. Age, body mass index and Type 2 diabetes-associations modified by ethnicity. *Diabetologia* 2003; **46**: 1063-1070.
21. Deurenberg P, Yap M, Van Staveren WA. Body mass index and percent body fat: a meta analysis among different ethnic groups. *Int J Obes Relat Metab Disord* 1998; **22**: 1164-1171.
22. Swinburn BA, Ley SJ, Carmichael HE, Plank LD. Body size and composition in Polynesians. *Int J Obes Relat Metab Disord* 1999; **23**: 1178-1183.
23. Gippini A, Mato A, Pazos R, Suarez B, Vila B, Gayoso P, Lage M, Casanueva FF. Effect of long-term strength training on glucose metabolism. Implications for individual impact of high lean mass and high fat mass on relationship between BMI and insulin sensitivity. *J Endocrinol Invest* 2002: 520-525.
24. Velho G, Froguel P. Genetic, metabolic and clinical characteristics of maturity onset diabetes of the young. *Eur J Endocrinol* 1998; **138**: 233-239.
25. Zimmet P. Antibodies to glutamic acid decarboxylase in the prediction of insulin dependency. *Diabetes Res Clin Pract* 1996; **34 Suppl**: S125-S131.

**Table 1.** General characteristics of the participants, by ethnic group. Values are means (95% confidence intervals) for continuous variables, or numbers (percentages) for categorical variables.

	<i>Ethnic group</i>			p
	<i>Europeans</i>	<i>Melanesians</i>	<i>Polynesians</i>	
	<i>n = 57</i>	<i>n = 287</i>	<i>n = 48</i>	
Age (years)	47.1 (45.2-49.0)	47.0 (46.1-47.8)	47.9 (45.6-50.1)	NS
Men	23 (40.4)	96 (33.5)	16 (33.3)	NS
BMI (kg/m <sup>2</sup> ):				
<25	27 (47.4)	57 (19.9)	13 (27.1)	<0.0001
25-29	22 (38.6)	120 (41.8)	15 (31.2)	
≥30	8 (14.0)	110 (38.3)	20 (41.7)	
Mean <sup>a</sup>	25.5 (24.6-26.5)	28.7 (28.1-29.2)	29.0 (27.2-31.0)	<0.0001
WHR	0.89 (0.86-0.91)	0.92 (0.91-0.93)	0.94 (0.92-0.96)	<0.001

<sup>a</sup>Log-transform applied. BMI, body mass index. WHR, waist-to-hip ratio.

**Table 2.** Markers of glucose-insulin regulation by ethnic groups. Values are means (95% confidence intervals) for continuous variables, or numbers (percentages) for categorical variables

	<i>Ethnic group</i>			<i>p</i> <sup>b</sup>	<i>p</i> <sup>c</sup>
	<i>Europeans</i>	<i>Melanesians</i>	<i>Polynesians</i>		
	<i>n = 57</i>	<i>n = 287</i>	<i>n = 48</i>		
FPG (mmol/l)	5.78 (5.62-5.93)	5.46 (5.38-5.54)	6.03 (5.89-6.18)	<0.0001	<0.0001
2h-PG (mmol/l) <sup>a</sup>	5.99 (5.59-6.42)	5.68 (5.50-5.87)	6.11 (5.72-6.53)	NS	<0.03
Glucose tolerance:					
Normal	37 (64.9)	222 (77.4)	27 (56.3)	<0.01	<0.002
IFG	12 (21.1)	40 (13.9)	17 (35.4)		
IGT	8 (14.0)	25 (8.7)	4 (8.3)		
FPI (pmol/l) <sup>a</sup>	78.3 (63.0-97.3)	67.0 (60.6-74.1)	61.7 (49.7-76.6)	NS	<0.004
HOMA-IR <sup>a</sup>	3.33 (2.67-4.15)	2.68 (2.42-2.98)	2.75 (2.19-3.45)	NS	<0.002
HOMA-BC <sup>a</sup>	119.3 (94.7-150.3)	124.8 (112.2-138.9)	83.1 (68.0-101.5)	<0.02	<0.002

<sup>a</sup>Log-transform applied. <sup>b</sup>crude. <sup>c</sup>covariance analysis with age, sex and BMI as adjusted covariables. FPG, fasting plasma glucose. 2h-PG, 2 hour-plasma glucose. IFG, impaired fasting glucose. IGT, impaired glucose tolerance. FPI, fasting plasma insulin. HOMA-IR and HOMA-BC, homeostasis model assessment of insulin resistance and  $\beta$ -cell secretory capacity. NS, not significant.



**Table 3.** Two-by-two comparisons between the ethnic groups for markers of glucose-insulin regulation. Levels of significance are corrected using the method of Bonferroni.

	<i>Ethnic group</i>		
	<i>Europeans vs Melanians</i>	<i>Europeans vs Polynesians</i>	<i>Melanians vs Polynesians</i>
FPG	<0.0003	NS	<0.0001
2h-PG	<0.05	NS	NS
FPI	<0.007	<0.007	NS
HOMA-IR	<0.001	<0.02	NS
HOMA-BC	NS	<0.004	<0.003

