# Heart disease and diabetes risk factors in Pacific Islands communities and associations with measures of body fat

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## Abstract

**Aims.** To describe the prevalence of obesity and other coronary heart disease and Type 2 diabetes risk factors by age and ethnic group in Pacific Island communities and to determine the associations between these risk factors and body mass index.

**Methods.** Cross-sectional data from two community-based intervention projects were combined to provide anthropometric, blood sample and blood pressure data on 1175 Pacific Islands people (467 men, 708 women) aged 20 years and over from church communities in South, Central and West Auckland. Self-reported data on diabetes status and leisure-time physical activity were also collected.

**Results.** Based on an ethnic-specific body mass index (BMI) cut-off (> 32 kg/m$^2$), 45% of men and 66% of women were obese. The age-standardised prevalence of known diabetes was 12%. Men and women aged 40 - 60 years had the highest risk factor levels and were the most sedentary. Tongans had higher risk factor levels than Samoans. In men, BMI and waist circumference were associated ($p<0.05$), in the direction of greater disease risk, with blood pressure and concentrations of total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, and blood glucose. In women, these associations were similar but less consistent.

**Conclusions.** While these data are not representative for all Pacific people living in New Zealand, they do show an extremely high prevalence of obesity and significant associations between obesity and other cardiovascular risk factors. These communities warrant a very high priority as part of public health efforts to address New Zealand's growing obesity epidemic.

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Pacific populations in New Zealand carry a heavy burden of coronary heart disease (CHD) and diabetes.[1,2] Several CHD risk factors are more prevalent amongst Pacific people than they are amongst European New Zealanders. They have higher mean blood pressures and a higher prevalence of hypertension,[3] microalbuminuria is more prevalent,[4] physical activity levels are lower and smoking rates are higher.[5] Also, Pacific people consume larger quantities of food than Europeans and their diets contain more meat and less fruit and vegetables.[6] Moreover, Pacific populations are among the most obese populations in the world and obesity is a strong independent risk factor for both CHD and Type-2 diabetes.[7,8] In contrast, some CHD risk factors are not as prevalent as might be expected in such obese populations. For example, serum cholesterol levels tend to be lower for Pacific people compared to Europeans.[9] This may be due to genetic differences and to diet, but also, weak associations between high levels of body fat and other CHD and diabetes risk factors have been described.[10]

Much of the information available on the prevalence of obesity and other CHD risk factors in Pacific populations in New Zealand is based on the Workforce Diabetes Survey[11,12] or the 1997 National Nutrition Survey (NNS97).[13] Unfortunately, both surveys have their limitations in providing the full picture of obesity in Pacific populations. The NNS97 over-sampled Pacific people but interpretation is hampered by a poor response rate (less than 50%) and low sample size (273 with anthropometric data). The workforce survey had larger numbers (n=650) and was able to examine for differences in CHD risk between Pacific ethnic groups, rather than assuming that risk is homogeneous.[11] However, the sampling frame was restricted to the older (40+ years), employed Pacific workforce in Auckland and Tokoroa.

This present study pools baseline data from two large community-based intervention projects in Auckland and describes variations in the prevalence of obesity and other CHD and diabetes risk factors by age and ethnic group. It also investigates the associations between these risk factors and measures of body fatness. While this study also has limitations of extrapolation to the New Zealand-wide Pacific communities, the overall aim is to provide information from these two large studies to build a clearer picture of these important risk patterns.

## Methods

**Participants.** Participants came from two community-based intervention programs, the South Auckland Diabetes Project (SADP) and the Samoan Ola Fa'autuata Project (SOFP). The SADP was established in 1991 as a multi-faceted program that aimed to reduce the incidence of diabetes in New Zealand through lifestyle interventions. The Samoan Ola Fa'autauta or 'Life-wise' Project was a similar community-based lifestyle program.[14] Both projects worked with Pacific Islands church groups in South, Central and West Auckland. The selection of churches for interventions was non-random but all adults from each church community were invited to take part. There was no specific selection for those who were obese or those with diabetes. The church community was defined as those people whose names were on the church roll plus their household members. From these membership lists, the baseline response rate for the SADP was 60% and for the SOFP it was 81%.

This analysis includes self-identified Pacific Islands people, aged 20 years and over who had complete anthropometric data at baseline. A total of 1175 people, 725 (287 men, 438 women) from the SADP and 450 from the SOFP (180 men, 270 women). Where our data from these Church Intervention Surveys (CIS) were compared with the National Health and Nutrition survey, we included BMI data from an additional 117 adolescent's aged 15 - 19 years. The SOFP was given ethical approval by the University of Auckland Human Subjects Ethics Committee and the SADP by the Auckland Area Health Board Ethics Committee.

**Data collection.** Both projects collected data at a series of health surveys on church premises between 1991 and 1996. Participants received information sheets, translated if necessary, and a brief presentation explaining the project. Consent was obtained from all participants (interpreters were available). Standardised techniques were used to measure weight, height, waist, and hip circumference.[15] The SOFP used Seca electronic scales (model 708) with an attached stadiometer to measure weight and height after removing heavy clothing and shoes. The SADP used the same standardised techniques, a portable stadiometer (CMS, London) and Salter spring scales. The scales were calibrated regularly. Non-stretch fibreglass tapes were used to measure waist and hip circumference. Waist circumference was measured horizontally through a point midway between the top of the iliac crest and the bottom of the ribs. Hip circumference was measured at the largest posterior extension of the buttocks.

Non-fasting venous blood samples were collected, stored and analysed for blood lipids, glucose and fructosamine levels by Medlab Ltd. Samples were measured using Roche Diagnostic protocols. Blood pressure was measured twice in the sitting position with a standard mercury sphygmomanometer using Korotkoff phase 1 and 5 sounds for systolic and diastolic blood pressure respectively. Oversize cuffs were used for large arms. Participants were considered sedentary if they did no moderate or vigorous activity during a normal week.

**Statistical Analysis.** BMI was calculated as weight (kg) divided by height (m) squared. Pacific specific BMI cutoffs were used to define overweight (26 kg/m$^2$ ≥ BMI < 32 kg/m$^2$) and obesity (BMI ≥ 32 kg/m$^2$).[16] The data were stratified by gender and analysis of variance was used to calculate adjusted age- and ethnic-specific means. Age-standardised means (Table 1) were calculated by the direct method using Segi's world population for those aged ≥ 20 years.[17] Multiple regression was used to test for linear associations between age and blood cholesterol, triglyceride, glucose and blood pressure. A second model that included a quadratic term (age-squared) was used to test for curvilinearity. With the exception of the ethnic group analysis, indicator variables were used to adjust for confounding by ethnic group and to adjust for systematic differences in risk factor levels between the two projects.

**Table 1. Association between age and anthropometric, biochemical and blood pressure measurements, mean (SEM), in Pacific Islands men. The prevalence of obesity, sedentary leisure time activity and known diabetes is also given.**

| | Age Standardised* | Age group (years) 20-29 | 30-39 | 40-49 | 50-59 | ≥ 60 | P for linear term† | P for quadratic term‡ |
|---|---|---|---|---|---|---|---|---|
| **Anthropometry**, n | 467 | 106 | 97 | 120 | 94 | 50 | | |
| Weight, kg | 97.6 (8.9) | 96.7 (l.9) | 98.3 (2.0) | 99.9 (l.8) | 99.0 (2.1) | 94.7 (2.6) | 0.69 | 0.0006 |
| Height, cm | 172.9 (3.0) | 177.1 (0.6) | 174.1 (0.7) | 171.5 (0.6) | 170.9 (0.7) | 168.9 (0.9) | <0.0001 | 0.02 |
| Body mass index, kg/m$^2$ | 32.6 (2.7) | 30.8 (0.6) | 32.3 (0.6) | 33.9 (0.5) | 33.9 (0.6) | 33.2 (0.8) | 0.0004 | <0.0001 |
| Waist, cm | 103.9 (6.9) | 96.7 (l.5) | 103.0 (1.5) | 107.2 (l.4) | 108.2 (l.6) | 108.3 (2.0) | <0.0001 | <0.0001 |
| Hip, cm | 110.7 (5.6) | 109.9 (l.2) | 109.7 (l.3) | 112.1 (1.1) | 111.2 (l.3) | 111.4 (l.6) | 0.33 | 0.13 |
| BMI > 32 kg/m$^2$, % | 45 | 32 | 37 | 59 | 56 | 47 | - | - |
| **Non-fasting lipids**, n | 352 | 67 | 69 | 94 | 79 | 43 | | |
| Total cholesterol, mmol/L | 5.80 (0.49) | 5.28 (0.13) | 5.87 (0.13) | 6.27(0.11) | 6.08(0.13) | 5.74(0.16) | 0.002 | <0.0001 |
| HDL, mmol/L | 1.10 (0.14) | 1.14 (0.04) | 1.13 (0.04) | 1.09(0.03) | 1.05(0.03) | 1.07(0.04) | 0.09 | 0.62 |
| LDL,§ mmol/L | 3.67 (0.44) | 3.17 (0.12) | 3.68 (0.13) | 4.08(0.11) | 3.94(0.12) | 3.74(0.14) | 0.0002 | <0.0001 |
| Total:HDL ratio | 5.56 (0.78) | 4.83 (0.21) | 5.67 (0.21) | 5.98(0.18) | 6.03(0.20) | 5.65(0.25) | 0.003 | 0.002 |
| Triglyceride,|| mmol/L | 2.40 (l.06) | 2.17 (0.28) | 2.65 (0.28) | 2.77(0.25) | 2.51(0.27) | 2.00(0.34) | 0.54 | 0.02 |
| **Non-fasting glucose**, n | 402 | 81 | 84 | 105 | 88 | 44 | | |
| Glucose,|| mmol/L | 6.37 (l.61) | 4.96 (0.39) | 5.36 (0.40) | 6.42 (0.35) | 7.60 (0.39) | 8.47(0.50) | <0.0001 | 0.07 |
| **Blood pressure**, n | 414 | 86 | 87 | 110 | 87 | 44 | | |
| Systolic, mmHg | 136.2 (8.4) | 126.4 (2.0) | 130.0 (2.0) | 134.1(1.8) | 145.3 (2.0) | 152.3 (2.6) | <0.0001 | 0.32 |
| Diastolic, mmHg | 86.8 (5.6) | 80.9 (l.3) | 84.2 (l.4) | 87.9 (l.2) | 92.2 (l.4) | 92.9 (l.8) | <0.0001 | <0.04 |
| **Leisure time activity**, ¶n | 342 | 94 | 75 | 75 | 66 | 32 | | |
| Sedentary, % | 22 | 14 | 19 | 31 | 24 | 28 | - | - |
| **Diabetes prevalence**, n | 490 | 114 | 104 | 121 | 98 | 53 | | |
| Known diabetes, % | 12 | 2 | 3 | 7 | 21 | 36 | - | - |

Age standardised to Segi's world population. †Model 1. Age was the independent continuous variable and variables for ethnic group and study were included in the model. ‡Model 2. Same as model 1 with the quadratric term (age squared) included. §Total n for LDL = 310 because of triglyceride levels > 4 mmol/L. ||The natural logs of triglyceride and glucose were used in the models. ¶Leisure time activity data were not collected from 1995 onwards in the South Auckland Diabetes project.

Associations between body size and lipids, glucose and blood pressure were tested using analysis of variance. Multiple regression was used where the variables were treated continuously. Triglyceride and glucose variables were log transformed to improve the normality of the distributions. Analyses were carried out in SAS version 6.10 (SAS Institute, Cary, NC, USA).

## Results

Participants in this CIS study were older (mean age 42 years for men, 41 years for women) and more likely to be female (60% of participants) than the general Pacific Islands population (1996).[18] Most were Samoan (64%) or Tongan (26%) whereas approximately 50% of the general Pacific Islands population identify themselves as Samoan and 14% as Tongan. Compared to the overall distribution of occupations in New Zealand (1991),[19] the distribution of occupations for Samoans in the SOFP project was shifted to the lower income end ($p<0.001$).

Based on our results, 81% and 86% of Pacific Islands men and women aged 15 years and over were either overweight (26 kg/m$^2$ ≥ BMI < 32 kg/m$^2$) or obese (BMI ≥ 32 kg/m$^2$), with 44% of men and 58% of women being obese (note: these are not age-standardised). We compared these percentages with results from the National Nutrition Survey in Figures 1 and 2. Compared to the NNS97, we observed a considerably lower combined prevalence of overweight and obesity for young Pacific males (87.6% vs 55.8%). On the other hand our estimates of obesity were higher (44.8% vs 27.4%) for males aged 25-44 years and overweight lower than for the NNS97. Differences were also noted for females aged 25-44 years (61.5% obese vs 34% obese; Figure 2).


Figure 1. Overweight and obesity in three age groups as reported by the Church Intervention Surveys (CIS) and the National Nutrition Survey 1997 (NNS97): Pacific males.

Tables 1 and 2 present associations between age and CHD and diabetes risk factors for men and women aged 20 years and over. Pacific men had an age-standardised mean BMI of 32.6 kg/m$^2$. Most risk factors were higher in the older age groups, although mean weight, BMI, obesity prevalence, total cholesterol, LDL, the total: HDL ratio, triglycerides and sedentary leisure time activity were lower in the 60+ age group than the younger two decade groups. Women (Table 2) had a higher aged- standardised mean BMI (34.8 kg/m$^2$) than men and a higher prevalence of obesity (60% with a BMI ≥ 32kg/m$^2$). As with men, most risk factor levels were lower in the 60+ age group than the younger age groups. Overall, 12% of men and women reported having Type-2 diabetes and approximately one-quarter did no leisure time physical activity during a normal week.

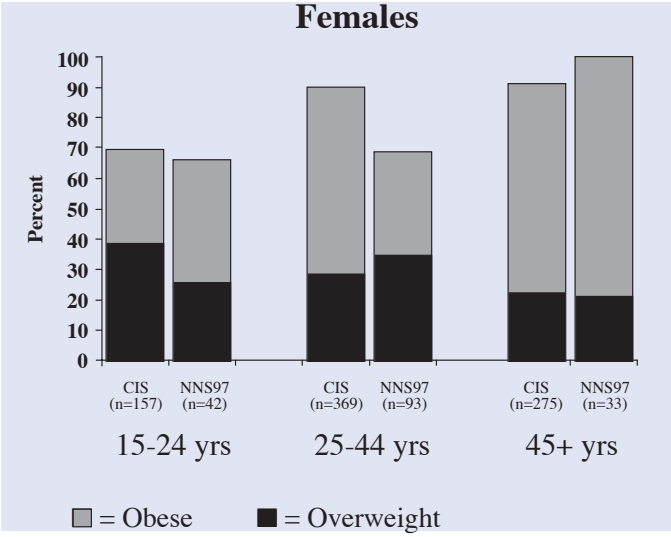


Figure 2. Overweight and obesity in three age groups as reported by the Church Intervention Surveys (CIS) and the National Nutrition Survey 1997 (NNS97): Pacific females.

A comparison of risk factors by ethnic group is given in Table 3. Tongans had the highest mean BMI and the highest prevalence of obesity. Their lipid profiles were significantly more atherogenic than Samoan profiles. Also, Tongan men had significantly higher mean glucose levels and Tongan women were significantly more likely to do no leisure time physical activity.

Table 4 shows relationship between BMI and waist circumference with other CHD and diabetes risk factors. For men, each risk factor, with the exception of HDL (where the association was inverse), was higher at higher quartiles of BMI. A similar, although less consistent pattern was observed between these risk factors and quartiles of waist circumference. Where BMI and waist circumference were treated continuously, both were positively ($p<0.05$) associated with each risk factor. There was an inverse association with HDL. These models were re-run (not shown) including squared terms for BMI or waist. None of the squared terms was significant.

For women, the variation in these risk factors with quartiles of BMI and waist was not as marked as it was for men. Mean total and LDL cholesterol and glucose levels differed little although mean systolic and diastolic blood pressure was higher with each quartile of BMI. Using continuous data, only the total:HDL cholesterol ratio, (log) triglycerides and systolic and diastolic blood pressure were positively associated with BMI. There was a significant negative association between HDL and BMI. Total cholesterol, the total:HDL cholesterol ratio, (log) triglycerides, glucose, and diastolic blood pressure were all positively associated with waist circumference in women. When the squared terms for BMI and waist were included in these models, negative associations ($p<0.05$ for BMI) were observed for cholesterol, the total:HDL cholesterol ratio, LDL and (log) triglyceride concentrations (not shown).

## Discussion

These cross-sectional analyses of CHD and diabetes risk factors came from church-based Pacific populations and are not, therefore, a representative sample. However, the sample size was large (total n = 1175) and the results are probably as characteristic of New Zealand's Pacific Islands population as

**Table 2. Association between age and anthropometric, biochemical and blood pressure measurements, mean (SEM), in Pacific Islands women. The prevalence of obesity, sedentary leisure time activity and known diabetes is also given.**

| | Age Standardised* | Age group (years) 20-29 | 30-39 | 40-49 | 50-59 | ≥60 | P for linear term† | P for quadratic term‡ |
|---|---|---|---|---|---|---|---|---|
| **Anthropometry**, n | 708 | 187 | 169 | 164 | 111 | 77 | | |
| Weight, kg | 91.2 (9.6) | 87.8 (l.6) | 93.3 (l.6) | 96.8 (l.7) | 93.7 (l.9) | 85.6 (2.3) | 0.84 | <0.01 |
| Height, cm | 161.5 (2.9) | 164.0 (0.5) | 163.0 (0.5) | 161.4 (0.5) | 160.0 (0.6) | 157.4 (0.7) | <0.01 | 0.06 |
| Body mass index, kg/m$^2$ | 34.8 (3.3) | 32.1 (0.5) | 34.9 (0.6) | 37.0 (0.6) | 36.4 (0.7) | 34.2 (0.8) | <0.01 | <0.01 |
| Waist, cm | 102.1(6.9) | 94.0 (1.1) | 101.0 (1.2) | 105.2 (1.2) | 108.4 (1.4) | 106.7 (1.7) | <0.01 | <0.01 |
| Hip, cm | 115.5 (6.9) | 111.8 (1.1) | 115.0 (1.2) | 118.9 (1.2) | 119.3 (1.4) | 114.9 (1.7) | <0.01 | <0.01 |
| BMI > 32 kg/m$^2$, % | 60 | 41 | 64 | 72 | 73 | 58 | - | - |
| **Non-fasting lipids**, n | 548 | 130 | 120 | 148 | 94 | 56 | | |
| Total cholesterol, mmol/L | 5.46 (0.46) | 4.84 (0.09) | 5.09 (0.09) | 5.44 (0.09) | 6.04 (0.10) | 6.33 (0.13) | <0.01 | 0.05 |
| HDL, mmol/L | 1.22 (0.15) | 1.26 (0.03) | 1.19 (0.03) | 1.20 (0.03) | 1.23 (0.03) | 1.19 (0.04) | 0.41 | 0.55 |
| LDL,§ mmol/L | 3.47 (0.39) | 2.96 (0.08) | 3.20 (0.08) | 3.48 (0.07) | 3.83 (0.10) | 4.21 (0.12) | <0.01 | 0.06 |
| Total:HDL ratio | 4.70 (0.62) | 4.05 (0.12) | 4.48 (0.12) | 4.70 (0.12) | 5.16 (0.14) | 5.50 (0.18) | <0.01 | 0.15 |
| Triglyceride,‖ mmol/L | 1.77 (0.53) | 1.49 (0.10) | 1.63 (0.11) | 1.79 (0.10) | 2.04 (0.12) | 2.08 (0.15) | <0.01 | 0.02 |
| **Non-fasting glucose**, n | 613 | 147 | 146 | 156 | 98 | 66 | | |
| Glucose,‖ mmol/L | 6.67 (2.25) | 5.28 (0.41) | 5.89 (0.41) | 6.62 (0.41) | 8.86 (0.49) | 7.81 (0.59) | <0.01 | 0.18 |
| **Blood pressure**, n | 631 | 152 | 150 | 156 | 103 | 70 | | |
| Systolic, mmHg | 132.0 (8.9) | 119.5 (l.6) | 123.8 (l.6) | 133.7 (l.6) | 145.9 (l.9) | 145.9 (2.3) | <0.01 | 0.11 |
| Diastolic, mmHg | 82.6 (5.8) | 76.1 (1.0) | 79.7 (l.1) | 85.6 (l.1) | 88.2 (l.2) | 87.4 (l.5) | <0.01 | <0.01 |
| **Leisure time activity**, ¶n | 478 | 149 | 109 | 99 | 70 | 51 | | |
| Sedentary, % | 27 | 25 | 29 | 29 | 26 | 29 | - | - |
| **Diabetes prevalence**, n | 732 | 194 | 175 | 167 | 118 | 78 | | |
| Known diabetes, % | 12 | 1 | 4 | 8 | 25 | 31 | - | - |

*Age standardised to Segi's world population. †Model 1. Age was the independent continuous variable and variables for ethnic group and study were included in the model. Model 2. Same as model 1 with the quadratric term (age squared) included. §Total n for LDL = 514 because of triglyceride levels > 4 mmol/L. ‖ The natural logs of triglyceride and glucose were used in the models. ¶Leisure time activity data were not collected from 1995 onwards in the South Auckland Diabetes project

**Table 3. Ethnic differences in anthropometric, biochemical and blood pressure measurements, mean (SEM). Differences in the prevalence of obesity, sedentary leisure time activity and known diabetes are also given.**

| | Men Samoan | Tongan | Other PI* | Women Samoan | Tongan | Other PI* |
|---|---|---|---|---|---|---|
| **Anthropometry**, n | 286 | 140 | 41 | 474 | 167 | 67 |
| Weight, kg | 94.5(l.1) | 101.9 (1.8)[b] | 96.7(2.8) | 89.4(0.9) | 95.9(1.7)[b] | 80.0(2.4) |
| Height, cm | 171.4(0.4) | 174.4 (0.6)[c] | 171.7(0.9) | 160.9(0.3) | 162.8(0.5)[b] | 159.8(0.7) |
| Body mass index, kg/m$^2$ | 32.1(0.3) | 33.5 (0.6)[a] | 32.7(0.8) | 34.5(0.3) | 36.1 (0.6)[a] | 34.2(0.8) |
| Waist, cm | 103.1(0.8) | 105.0(1.4) | 105.9(2.1) | 101.0(0.6) | 105.7 (1.3)[b] | 102.5(l.7) |
| Hip, cm | 108.9(0.7) | 111.5(l.1) | 112.2(l.7) | 115.0(0.6) | 117.4(l.3) | 115.6(l.7) |
| Obesity, % BMI ≥30 kg/m$^2$ | 63 | 68[a] | 51 | 73 | 75[a] | 76 |
| Obesity, % BMI >32 kg/m$^2$ | 46 | 49 | 42 | 60 | 59 | 66 |
| **Non-fasting lipids**, n | 218 | 96 | 38 | 374 | 120 | 54 |
| Total cholesterol, mmol/L | 5.58(0.07) | 5.96 (0.12)[a] | 6.00 (0.17)[a] | 5.44(0.05) | 5.61(0.10) | 5.59(0.13) |
| HDL, mmol./L | 1.16(0.02) | 1. 03 (0.03)[b] | 1.09(0.04) | 1.31(0.01) | 1. 19 (0.03)[b] | 1. 14 (0.04)[c] |
| LDL,† mmol/L | 3.52(0.07) | 3.85 (0.12)[a] | 3.80(0.16) | 3.37(0.05) | 3.62 (0.09)[a] | 3.62(0.12) |
| Cholesterol/HDL ratio | 5.03(0.11) | 6.15 (0.19)[c] | 5.72 (0.26)[a] | 4.38(0.07) | 4.89 (0.13)[b] | 5.06 (0.17)[b] |
| log Triglyceride, mmol/L | 2.16(0.15) | 2.82 (0.26)[a] | 2.28(0.35) | 1.69(0.06) | 1.82(0.11) | 1.90(0.14) |
| **Non-fasting gucose**, n | 232 | 130 | 40 | 406 | 148 | 59 |
| Glucose, mmol/L | 6.37(0.22) | 7.29 (0.3 5)[a] | 6.04(0.53) | 6.75(0.23) | 6.79(0.43) | 7.01(0.59) |
| **Blood pressure**, n | 242 | 131 | 41 | 420 | 149 | 62 |
| Systolic, mmHg | 134.6(l.1) | 137.2(l.8) | 141.0 (2.7)[a] | 131.4(0.9) | 132.4(l.7) | 137.5 (2.3)[a] |
| Diastolic, mmHg | 85.7(0.8) | 86.2(1.2) | 90.9 (1.8)[a] | 82.4(0.6) | 81.5(l.1) | 8 6.3 (1.5)[a] |
| **Leisure time acitivity**,‡ n | 231 | 96 | 15 | 347 | 106 | 25 |
| Sedentary, % | 24 | 11 | 60[b] | 25 | 36[b] | 24 |
| **Diabetes prevalence**, n | 302 | 145 | 43 | 494 | 171 | 67 |
| Known diabetes, % | 11 | 11 | 12 | 10 | 9 | 13 |

*Other Pacific Islands (PI) ethnic groups, Cook Islands Maori (n=51), Niuean (n=20), and mixed Pacific Islands ethnic group (n=39). †Total n for LDL = 824 due to triglyceride levels > 4 mmol/L. ‡Leisure time activity data were not collected from 1995 onwards in the South Auckland Diabetes Project. [a,b,c] Significantly different from Samoan ethnic group at [a]p<0.05, [b]p<0.01, [c]p<0.0001 adjusted for age group and study.

the National Nutrition Survey which was smaller and had a lower response rate. The data show that risk factors for CHD and diabetes are very high, especially in the 40-60 year age range and amongst the most obese.

The prevalence of obesity in Pacific populations has been comprehensively reviewed by Hodge et al for populations living in the Pacific Islands.[20] The review indicated that Pacific populations are amongst the most obese in the world. Western Samoans, for example, had a higher prevalence of obesity than native Hawaiians and Pima Indians.[21,22] In this study, we found that Pacific people living in New Zealand have an even higher prevalence. Our observation that women had a higher prevalence of obesity than men is consistent with findings from studies in Western Samoa and New Zealand. Schaaf et al observed no significant differences in BMI between Cook Islands Maori, Samoans,

**Table 4. Association between non-fasting blood lipids, blood glucose and blood pressure with quartiles of body mass index (BMI) and waist for Pacific Islands men and women. Multivariate regression co-efficients (β) and the variation explained by each model ($R^2$) are also included.**

| | Cholesterol mmol/L | Chol:HDL Ratio | HDL mmol/L | LDL mmol/L | Triglyceride mmol/L* | Glucose mmol/L* | Systolic BP mm Hg | Diastolic BP mm Hg |
|---|---|---|---|---|---|---|---|---|
| **Men:** BMI, n | 348 | 345 | 346 | 307 | 348 | 397 | 411 | 411 |
| Quartile (mean kg/m²) | | | | | | | | |
| 1(26.1) | 5.56 | 4.92 | 1.17 | 3.60 | 1.87 | 5.74 | 128.8 | 81.0 |
| 2(31.3) | 5.82 | 5.62[1] | 1.11 | 3.67 | 2.49 | 6.41 | 135.5[1] | 86.1[1] |
| 3(35.0) | 5.96[1] | 5.85[1] | 1.05[1] | 3.84 | 2.60 | 6.39 | 137.2[1] | 88.5[1] |
| 4(41.2) | 6. 19[1,2] | 6.38[1] | 1.02[1] | 3.95 | 3.10[1] | 7.14[1] | 142. 8[1,2] | 94.1[1,2,3] |
| Linear, β ($R^2$ %) | 0.04‡ (3.2) | 0.10‡ (5.3) | -0.01‡(1.9) | 0.02† (1.2) | 0. 04† (8.8) | 0.01‡ (2.5) | 0. 83‡ (8.3) | 0.81‡ (2.5) |
| **Men:** Waist, n | 351 | 348 | 349 | 309 | 351 | 400 | 414 | 414 |
| Quartile (mean, cm) | | | | | | | | |
| 1(84.90) | 5.51 | 4.73 | 1.21 | 3.53 | 1.44 | 5.76 | 129.9 | 81.2 |
| 2(97.10) | 5.71 | 5.31 | 1.13 | 3.70 | 2.44[1] | 5.75 | 132.1 | 84.0 |
| 3(106.3) | 6.02[1] | 5.95[1] | 1.05[1] | 3.84 | 2.67[1] | 6.75 | 138.8[1,2] | 88.4[1,2] |
| 4(120.5) | 6.11[1] | 6.19[1] | 1.04[1] | 3.83 | 3.01[1] | 6.86 | 139.5[1,2] | 91.8[1,2] |
| Linear, β ($R^2$ %) | 0.02‡ (2.1) | 0.04‡ (5.3) | -0.004‡ (2.0) | 0.01† (1.3) | 0.02‡ (5.0) | 0.005‡ (1.8) | 0.30‡ (7.0) | 0.30‡ (1.9) |
| **Women:** BMI, n | 544 | 541 | 543 | 516 | 544 | 608 | 627 | 627 |
| Quartile (mean kg/m²) | | | | | | | | |
| 1(26.2) | 5.21 | 4.21 | 1.30 | 3.28 | 1.43 | 6.39 | 127.6 | 78.3 |
| 2(31.3) | 5.48 | 4.73[1] | 1.22 | 3.50 | 1.76 | 6.62 | 128.6 | 80.5 |
| 3(35.1) | 5.47 | 4.74[1] | 1.20[1] | 3.45 | 1.85[1] | 6.66 | 130.6 | 82.2[1] |
| 4(42.5) | 5.44 | 4.80[1] | 1. 18[1] | 3.43 | 1.88[1] | 6.69 | 136.6[1, 2,3] | 86.5[1,2,3] |
| Linear, β ($R^2$ %) | 0.01(0.3) | 0.02† (1. 3) | -0.005† (1.4) | 0.005(0.1) | 0.01‡ (3.8) | 0.004(3.2) | 0.53‡ (7.7) | 0.49‡ (0.8) |
| **Women:** Waist, n | 540 | 537 | 539 | 506 | 540 | 602 | 621 | 621 |
| Quartile (mean cm) | | | | | | | | |
| 1(82.60) | 5.23 | 4.21 | 1.31 | 3.3 | 1.33 | 5.55 | 126.9 | 78.8 |
| 2(96.70) | 5.44 | 4.71[1] | 1.19[1] | 3.45 | 1.85[1] | 7.24[1] | 129.0 | 81.7 |
| 3(105.9) | 5.55[1] | 4.94[1] | 1.16[1] | 3.51 | 2.01[1] | 6.47 | 134.1[1] | 84.0[1] |
| 4(119.7) | 5.44 | 4.78[1] | 1.19[1] | 3.44 | 1.83[1] | 7.15[1] | 135.5[1] | 85.4[1] |
| Linear, β ($R^2$ %) | 0.01†(0.8) | 0.02‡ (3.4) | -0.004‡ (2.8) | 0.004 (0.4) | 0.01‡(6.0) | 0.004‡ (2.4) | 0.25‡ (4.6) | 0.18‡ (2.2) |

*The natural logs of triglyceride and glucose were used for the regression models. [1,2,3]Significantly different from quartile 1,2 or 3 respectively at p<0.01 adjusted for age, ethnic group and study. †‡Significant association with BMI or waist circumference at †p <0.05 or ‡p <0.001 adjusted for age, ethnic group and study.

Tongans and Niueans.[11] We found that Tongan men and women were bigger than their counterparts from other islands.

The lipid profiles of Pacific men and women in the current study were less atherogenic than those reported in the National Nutrition Survey,[13] and the Workforce Diabetes Survey.[11] The attenuation of risk factor levels in older Pacific people has previously been described[23,24] and probably reflects selective mortality of high risk individuals or the cohort effect of a relatively lower risk group of individuals now reaching older age.[2] The age-standardised prevalence of known diabetes (12%) was similar in these church communities to the prevalence observed in a household survey of inner urban South Auckland.[25] Systolic and diastolic blood pressure levels were comparable to those observed in Pacific members of a Seventh-Day Adventist church.[24] Also, similar increases in blood pressure with age have been observed in Pacific Islands people in the workforce.[3,26] The number of people who were sedentary during leisure-time was high in these communities. Previous New Zealand studies found that Pacific people were less involved in leisure-time activities than Maori or European and that Pacific women were less active than men.[5]

Body size was adversely associated with other CHD and diabetes risk factors. However, the associations were not as strong as those observed for European New Zealanders,[24] and other less obese populations.[27] Moreover, for Pacific women, there was evidence of attenuation between BMI and blood cholesterol and triglycerides at the upper end of the BMI distribution. Similar findings have been observed in studies of Samoans in Western Samoa and American Samoa, and Micronesian Nauruans.[28,29] The poor associations observed were attributed to extreme obesity in these populations. This suggests that these populations have reached a level of obesity above which the impact of total fat or intra-abdominal fat on CHD and diabetes risk factors becomes less apparent.

There are a number of limitations to this study. As mentioned above, neither the church communities nor the participants were randomly selected and therefore the results may not readily be generalised to New Zealand's wider Pacific population. Numbers in the studies were lower than they could have been for several variables because providing a blood sample was voluntary and up to 30% declined. Finally, combining the results of two separately conducted studies is not ideal methodology although we tried to overcome this limitation by adjusting for risk factor differences between the projects.

The SADP and the SOFP were both designed to reduce CHD and diabetes risk factors in these church communities and both had some success.[30,31] The high prevalence of obesity and other risk factors at baseline suggests that these interventions were not only warranted, but long overdue in the effort to bring the health status of these communities in line with that of other New Zealanders. Future efforts to contain the rising prevalence of obesity in New Zealand need to give priority to Pacific People.

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1. Simmons D, Gatland B, Flemming C, Scragg R. Prevalence of known diabetes in a multiethnic community. NZ Med J 1994; 107: 219-22.
2. Bell AC, Swinburn BA, Stewart AW et al. Ethnic differences and recent trends in coronary heart disease incidence in New Zealand. NZ Med J 1996; 109: 66-8.

3. Bullen C, Tipene-Leach D, Vander Hoorn S et al. Ethnic differences in blood pressure: findings from the Fletcher Challenge Auckland University Heart and Health Study. NZ Med J 1996; 109: 395-7.
4. Metcalf P, Baker JR, Scragg R et al. Microalbuminuria in a middle-aged workforce. Effect of hyperglycaemia and ethnicity. Diabetes Care 1993; 16: 1485-93.
5. Public Health Commission. The Health of Pacific Islands People in New Zealand. Wellington: Public Health Commission; 1994. p114-21.
6. Metcalf PA, Scragg RKR, Tukuitonga CF, Dryson EW. Dietary intakes of middle-aged European, Maori and Pacific Islands people living in New Zealand. NZ Med J 1998; 111: 310-3.
7. Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. Ann Intern Med. 1995; 122: 481-6.
8. Hodge AM, Zimmet PZ. The epidemiology of obesity. Bailliere's Clinical Endocrinology and Metabolism 1994; 8: 577-99.
9. Scragg R, Baker J, Metcalf P, Dryson E. Serum lipid levels in a New Zealand multicultural workforce. NZ Med J 1993; 106: 96-9.
10. Depres JP. Lipoprotein metabolism in abdominal obesity. In: Oomura Y, Tarui S, Inoue S, Shimazu T, editors. Progress in Obesity Research. London: Libbey; 1990. p285-90.
11. Schaaf D, Scragg R, Metcalf P. Cardiovacular risk factors of Pacific People in a New Zealand multicultural workforce. NZ Med J 2000; 113: 3-5.
12. Swinburn BA, Walter L, Ricketts H et al. The determinants of fat intake in a multiethnic New Zealand population. Int J Epidemiol 1998; 27: 416-21.
13. Russell DG, Parnell WR, Wilson NC et al. NZ Food: NZ People. Key results of the 1997 National Nutrition Survey. Wellington: Ministry of Health;1999.
14. Swinburn BA, Amosa H, Bell AC. The Ola Fa'autauta Project: The process of developing a church-based health promotion programme. Pacific Health Dialog 1997; 4: 20-5.
15. Lohman TG, Roche AF, Marotell R. Anthropometric standardisation reference manual. Champaign IL: Human Kinetics Press; 1988.
16. Swinburn BA, Ley SJ, Plank LD. Body size and composition in Polynesians. Int J Obes 1999; 23: 1178-83.
17. Waterhouse J, Muir CS, Correa P, Powell J, editors. Cancer incidence in five continents. Lyon: IARC Scientific Publications; 1976.
18. Census 1996. Wellington: Nerw Zealand Department of Statistics; 1996.
19. Department of Statistics. New Zealand standard classification of occupations, 1990. Wellington: Department of Statistics; 1992.
20. Hodge AM, Dowse GK, Zimmet PZ. Obesity in Pacific populations. Pacific Health Dialog 1996; 3: 77-86.
21. Aluli NE. Prevalence of obesity in a Native Hawaiian population. Am J Clin Nutr 1991; 53: 1556S-60S.
22. Knowler WC, Pettit DJ, Saad MF et al. Obesity in the Pima Indians: its magnitude and relationship with diabetes. Am J Clin Nutr 1991; 53: 1543S-51S.
23. Hodge AM, Dowse GK, Toelupe P et al. Dramatic increase in the prevalence of obesity in Western Samoa over the 13 year period 1978-1991. Int J Obesity 1994; 18: 419-28.
24. McAnulty J, Scragg R. Body mass index and cardiovascular risk factors in Pacific Islands Polynesians and Europeans in New Zealand. Ethnicity and Health 1996; 1: 187-95.
25. Simmons D, Harry T, Gatland B. Prevalence of known diabetes in different ethnic groups in inner urban South Auckland. NZ Med J 1999; 112: 316-9.
26. Scragg R, Baker J, Metcalf P, Dryson E. Hypertension and its treatment in a New Zealand multicultural workforce. NZ Med J 1993; 106: 147-50.
27. Seidell JC, Cigolini M, Charzewska J et al. Indicators of fat distribution, serum lipids, and blood pressure in European women born in 1948: The European fat distribution study. Int J Epidemiol 1989; 130: 53-65.
28. Galanis DJ, McGarvey ST, Sobal J et al. Relations of body fat and fat distribution to the serum lipid, apolipoprotein and insulin concentrations of Samoan men and women. Int J Obesity 1995; 19: 731-8.
29. Hodge AM, Dowse GK, Zimmet PZ. Association of body mass index and waist-hip circumference ratio with cardiovascular disease risk factors in Micronesian Nauruans. Int J Obesity 1993; 17: 399-407.
30. Simmons D, Flemming C, Voyle J et al. A pilot urban church-based programme to reduce risk factors for diabetes among Western Samoans in New Zealand. Diabet Med 1998; 15:136-42.
31. Bell AC, Swinburn BA, Amosa H, Scragg R. A nutrition and exercise intervention programme for controlling weight in Samoan communities in New Zealand. Int J Obesity 1999; 23(5s).