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Changes in cardiovascular disease risk factors over 30 years in Polynesians in the French Pacific Territory of Wallis Island

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STRUCTURED ABSTRACT

Background: Wallis Island is part of a French Territory in the South Pacific. In 1980 the prevalence of hypertension (HT) and type 2 diabetes mellitus (T2DM) was low, consistent with a subsistence economy. Considerable social and economic changes have occurred over the last 30 years.

Methods: Survey data from 1980 and 2009 were analysed by sex in ten-year age groups, and 25-64 years age-standardised to the 2008 Census. Means and prevalences were calculated for blood pressure (BP), fasting plasma glucose (FPG), body mass index (BMI), blood cholesterol and triglycerides as risk factors contributing to cardiovascular disease (CVD).

Results: During 1980-2009 there were significant increases ($p<0.05$) in age-standardised means and prevalences of BP and HT, FPG and T2DM, BMI and obesity, blood cholesterol (men) and triglycerides; and non-significant increases in mean diastolic BP and FPG in women. Mean cholesterol and the prevalence of elevated cholesterol declined in women. HT prevalence increased from 12% to 43% in men and from 15% to 30% in women, with 42% of the increase in men and 33% of the increase in women statistically explained by increases in BMI. T2DM increased from 2.3% to 12.2% in men and from 4.0% to 15.8% in women, with 35% of the increase in men and 26% of the increase in women statistically explained by increases in BMI.

Conclusions: Risk factors for CVD have increased considerably in Wallis Island over the past 30 years, consistent with modernisation in way of life.

KEYWORDS

Cardiovascular Diseases; Diabetes Mellitus; Obesity; Polynesia; Hypertension; Risk Factors; Cholesterol; Triglycerides

INTRODUCTION

Cardiovascular disease (CVD), type 2 diabetes mellitus (T2DM) and their risk factors, including hypertension (HT) and obesity, have reached considerable proportions among adults in many developing nations, particularly in some Pacific Island populations [1-3]. Wallis and Futuna is a Polynesian French Territory in the South Pacific between Fiji and Samoa. It is relatively isolated with limited natural resources and a population of 13,445 (2008 Census) [4]. Population growth has been moderated by considerable out-migration to New Caledonia (also a French Territory), with 21,300 people designating Wallisian or Futunan ethnicity in the 2009 New Caledonia Census [5]. In view of changes in availability of goods and services, and food consumption habits [6,7], it is anticipated that CVD and its risk factors would have increased in recent decades.

In 1980 a survey of T2DM, HT and other risk factors for CVD was conducted on samples of Polynesian adults on Wallis Island [8], and in 2009 a survey with similar methodology was undertaken involving samples of Polynesian adults from the islands of both Wallis and Futuna [9]. Due to greater isolation and differences in culture, Futunans from the 2009 survey were not included in the present analyses to ensure comparability between the two surveys. Using these population-based cross-sectional surveys of adults 25-64 years the present study examines over 30 years on Wallis Island: (1) changes in CVD risk factors in the population, and the trajectory and magnitude of any change; (2) variation in changes by age and sex; and (3) the extent to which changes in body mass index (BMI) statistically explain changes in the prevalence of HT and T2DM. There are no other studies of changes in CVD risk factors in a Pacific Island population over such a long period, and from a baseline of such relatively low HT and T2DM

prevalence. A similar study in the Indian Ocean island of Mauritius found secular increases from 1987 to 2009 in T2DM and fasting plasma glucose (FPG) [10]. The ethnic composition of the surveyed Mauritian population were predominantly of South Asian and African (Creole) descent [10,11], and the prevalence of T2DM was 12.8% in 1987, consistent with an already modernised population.

METHODS

Study populations

In the 1980 survey a village was randomly selected from each of the three main districts of Wallis Island, and the entire population aged ≥20 years (on the 1979 electoral roll) was invited to participate. The response rate was 97% of those present in the villages at the time of the survey. Due to a larger than expected number of people temporarily absent from their village, the sample was augmented by randomly selecting a further 76 participants from two additional villages (response rate 100%). The age structure of the survey sample, the 1976 Wallis Census, and the electoral roll of each village were generally similar. Analysis in the present study is based on the 213 men and 228 women aged 25-64 years [8]; a sampling fraction of 25% (based on the 1976 Census).

The 2009 survey was conducted on the islands of Wallis and Futuna, however, for the purposes of the present study only participants from Wallis Island were included in analysis to facilitate comparisons with the 1980 survey. In 2009 a population list by village and by household was available, and a random selection of households by district was followed by random selection of one person from each household aged ≥18 years and >2 years residence. The quota method was used to fill required strata by sex, district and age group to ensure representativeness with the 2008 Census, with a response rate was 86%. Analysis in the present study is based on the 116 men and 154 women aged 25-64 years from the Wallis Island sample [9]; a sampling fraction of 7% (based on the 2008 Census).

Both surveys aimed to include only Wallis Polynesians. The population of Wallis Island is over 97% Polynesian. Inclusion of non-Polynesians is considered to be minimal.

Data collection

In both surveys questionnaires were administered through interview by the survey team. In 1980 blood pressure (BP) was measured with random zero muddler mercury sphygmomanometers (which resets baselines between successive readings), and in 2009 OMRON M3 electronic BP monitors were employed. The mean of the first and second BP measurements (unaffected by observer bias) were used for analysis in both surveys. Measurements of height (by measuring stick with a movable perpendicular head baton) and weight (by scale) were taken once in both surveys, and used to determine BMI: weight (kilograms) / height (metres)^2. In 1980 T2DM hyperglycaemia was based on venous plasma glucose 2 hours after a 75g oral glucose load. However, a fasting blood specimen was also taken and the plasma frozen and analysed a week later in Melbourne (Australia); the latter data are used in the present study to ensure comparability. In 2009, a fasting venous blood specimen was taken and the frozen plasma analysed in Noumea (New Caledonia) [8,9].

Definitions of morbidity

HT is defined as systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg and/or currently taking medication for HT [12]. T2DM is defined as FPG ≥7.0 mmol/L and/or currently taking medication for T2DM [12]. Obesity is defined as; BMI ≥30 according to the standard definition of the World Health Organisation (WHO) [12]; and

BMI >32 as suggested by Swinburn et al. [13] since Polynesians have a significantly higher ratio of lean mass: fat mass compared with Europeans.

Statistical analysis

Data were analysed by sex by ten-year age groups; and as one age group (25-64 years) directly age-standardised to the 2008 Census population of Wallis Island (a locally relevant standard) because of alteration in the age structure of the Wallis population between the 1976 and 2008 Censuses from changes in fertility, mortality and out-migration. The effect of BMI on increases in HT and T2DM is assessed using logistic regression by comparing the odds ratio (OR) for period after adjusting for age and BMI, compared with adjusting for age alone. Statistical significance was assessed at $p<0.05$. Changes in the frequency distributions of BMI between 1980 and 2009 were assessed using histograms, mean and standard deviation (SD), and median and inter-quartile range (IQR). Data were analysed using SAS version 9.4 [SAS Institute Inc., Cary, NC, USA].

Biological analysis

Biological parameters in 1980 were measured by a SMAC Technicon 20 autoanalyser (Technicon Instrument Corp., Tarrytown, N.Y. 10591) in Melbourne, and in 2009 by an Architect autoanalyser (Abbott Laboratories, Abbott Park, Illinois, USA) in Noumea. In 1980 FPG was measured by glucose oxidase/peroxidasewhereas in 2009 the hexokinase/glucose-6-phosphate dehydrogenase reaction was employed Cholesterol measurement was with the same enzymatic method (esterase/oxidase/peroxidase with phenolic chromophore) in 1980 [14] and 2009. For plasma triglycerides, in 1980 a glycerol-3-phosphate dehydrogenase reaction on

glycerol released after the action of the lipase was used, whereas in 2009 the reaction was based on glycerol phosphate oxidase on total glycerol. Internal and external standards for chemical pathologies laboratories have been in use in Australia from the 1960s, and were in use in Noumea during biochemical analysis of the 2009 survey measurements.

RESULTS

Between 1980 and 2009 in Wallisian men and women aged 25-64 years there was a statistically significant increase in the means and prevalences (age-standardised) of BP and HT, FPG and T2DM, BMI and obesity, and triglycerides; except for non-significant increases in mean DBP and FPG in women (Table 1-4).

BMI increased in men from 1980 (mean 27.4, SD 4.8; median 26.5, IQR 24.0-30.0) to 2009 (mean 32.5, SD 6.0; median 31.3, IQR 28.7-35.4); and in women from 1980 (mean 29.9, SD 5.3; median 29.8, IQR 25.9-33.6) to 2009 (mean 34.7, SD 6.8; median 34.6, IQR 30.7-39.0) Frequency distributions broadened and shifted to the right (Figure 1).

Means and prevalences of cholesterol (age-standardised) significantly increased in men and decreased in women during 1980-2009, and by 2009 cholesterol had become significantly higher in men (Table 4). Sex-specific differences in BP emerged during 1980-2009 with significantly higher means and prevalences in men in 2009 (Table 1). Mean BMI and obesity prevalence, using BMI ≥30 [12] or BMI >32 [13], remained significantly higher in women than men in 1980 and 2009 (Table 3).

In 1980 there was no significant increase in mean SBP with age in men, and no significant increase in HT prevalence with age for either sex; whilst in 2009 both sexes demonstrated significant increases in mean SBP and DBP, and HT prevalence, with increasing age (Table 1).

For HT, the OR for the period effect in men in 2009, compared to 1980 (referent), after adjusting for age, was 5.9 (95%CIs 3.3 to 10.5), and after adjusting for age and BMI was 3.4 (1.8 to 6.4) – a 42% change. For HT in women, the OR for period, after adjusting for age, was 3.3 (1.9 to 5.8), and after adjusting for age and BMI was 2.2 (1.2 to 4.1) - a 33% change. For T2DM the OR for the period effect in men for 2009, compared to 1980 (referent), after adjusting for age, was 6.5 (2.1 to 20.3), and after adjusting for age and BMI was 4.3 (1.3 to 14.0) – a 35% change. For T2DM in women the OR after adjusting for age was 7.4 (3.0 to 18.7), and after adjusting for age and BMI was 5.5 (2.1 to 14.3) - a 26% change.

DISCUSSION

Between 1980 and 2009 the prevalence of HT increased more than three-fold in men and two-fold in women, with 42% of the increase in men and 33% of the increase in women statistically explained by increases in BMI. In 1980 the prevalence of HT in both sexes, and mean SPB in males, did not rise with age, whilst in 2009 there was a significant rise with age in BP means and prevalences in both sexes. Previous studies have found a greater rise in BP with age in urbanised populations compared to rural populations with a mainly subsistence economy [15,16].

Sodium intake is a known risk factor for HT [17]. Although not an accurate measure of sodium intake and excretion, urinary sodium concentrations in the 1980 suggests that sodium consumption in Wallis was low (men: 1.8; women: 2.1 g/L) in comparison to Wallis Polynesians living in the more modernised Noumea (men: 3.8; women: 2.9 g/L) [8]. This is consistent with higher BP in populations in modernised societies, which has occurred in Wallis over the past 30 years. The 2009 survey did not collect data on sodium consumption.

In 2009, alcohol consumption on Wallis Island, also a known risk factor for HT [18,19], was 22% for drinking alcohol at least once a week, and 14% were classified as heavy drinkers - at least three drinks at a time at least once a week [9]. The 1980 survey did not collect data on alcohol consumption.

In 1980 the prevalence of T2DM in Wallis was relatively low, and comparable to other rural and traditional Polynesian populations at that time, including rural Western Samoa [1] and

Tuvalu [20]. It was suggested that in light of the prevalence of obesity in Wallis, this relatively low prevalence of T2DM may indicate that physical activity and/or the traditional diet were protective in these circumstances [21], although the duration of obesity may yet have been insufficient to produce a corresponding increase in T2DM. Between 1980 and 2009 the prevalence of T2DM increased more than five-fold in men and almost four-fold in women, with 35% of the increase in men and 26% of the increase in women statistically explained by increases in BMI. This is similar to previous comparable studies using repeated cross-sectional samples of individuals in Mauritius [10], the United States of America [22] and Europe [23], which found that increases in FPG and T2DM were partly or largely explicable by increases in BMI.

Using the WHO standard definition for obesity (BMI ≥30) or the cut-point suggested by Swinburn et al. [13] for Polynesian populations (BMI >32), obesity was already an issue in 1980 in Wallisian women of all age groups and in middle aged men. This indicates that modernisation of way of life was occurring in Wallis in the early 1980's, and as observed in other Polynesian populations, such as Tuvalu [20], women were affected by obesity earlier than men. BMI frequency distributions indicate that the population distribution of BMI in both sexes between 1980 and 2009 has shifted to the right (higher values) with broadening of the shape of the distribution, but no change in the difference between the mean and median, suggesting a total population phenomenon, rather than development of obesity in a minority.

Although no dietary survey data is available for Wallis to examine changes in consumption of saturated fats during 1980-2009, a possible explanation for the decline in blood cholesterol in

women during this period could be a substantial decline in the consumption of coconut products, an important component of calories (energy intake) in some Pacific populations. Dietary studies of other Pacific Island populations around the 1980's, including Tokelau, Pukapuka and Kiribati, identified women as having higher dietary intake of saturated fats [24] and higher serum cholesterol levels [24-26] in comparison to men, which was considered to be related to greater consumption of coconut products [25,26]. In Pacific Island populations where coconut was traditionally a staple dietary component, consumption decreased when more convenient cooked foods (especially rice and flour) become readily available [27,28], and a similar trend may have occurred in Wallis. Further investigation is needed to identify sex-specific explanations of the observed changes in serum cholesterol.

Tobacco consumption is a known risk factor for CVD. In the 2009 survey nearly 50% of participants reported smoking tobacco over the previous 12 months, and 43% reported daily smoking. The 1980 survey did not collect data on smoking habits.

In 2009, 25% of men and 47% of women reported low levels of physical activity (less than 600 MET-minutes of physical activity per week), also a risk factor for CVD. These data were not collected in 1980, although with less subsistence activity it is likely that physical activity has decreased over the past 30 years.

The 1980 and 2009 surveys of CVD risk factors in Wallis are both based on randomly selected samples from a homogenous population on one island, used similar methods for data collection, and are analysed according to the same definitions for HT, T2DM and obesity. Potential

limitations of the present study are that random sample selection was employed at the village level in 1980 and at the household level in 2009. However, both surveys were representative of the population at the nearest previous Census. The 2009 sample in the present study is smaller than the 1980 sample because of exclusion of Futunans from the 2009 survey to ensure comparability of participants from Wallis Island only. However, statistical testing is used to assess differences between samples. Modifications over time have occurred in biological analysis of FPG and plasma cholesterol and triglycerides, mainly resulting in the reduction of interferences [29,30]. There are larger coefficients of variation for some 1980 measures compared to 2009, with the 2009 readings likely to be more precise, especially for cholesterol and triglycerides.

Comparison of the 1980 and 2009 surveys show that in the formerly traditional island society of Wallis, considerable increases in risk factors for CVD have occurred over the past 30 years consistent with changes in way of life. The decline in mean cholesterol in women may be due to lower coconut intake, however this requires further investigation. Intensive preventive interventions are indicated to lower risk factors for CVD. These increases have implications for future premature morbidity and mortality, and costs to the health care system.

Figure 1. Body Mass Index Wallis Polynesians 1980 and 2009

FOOTNOTES: Normal (Gaussian) distributions derived from frequency distributions of body mass index expressed as proportions of participants by sex and survey; participants: 1980 men= 213, women= 201; 2009 men= 116 women = 154.

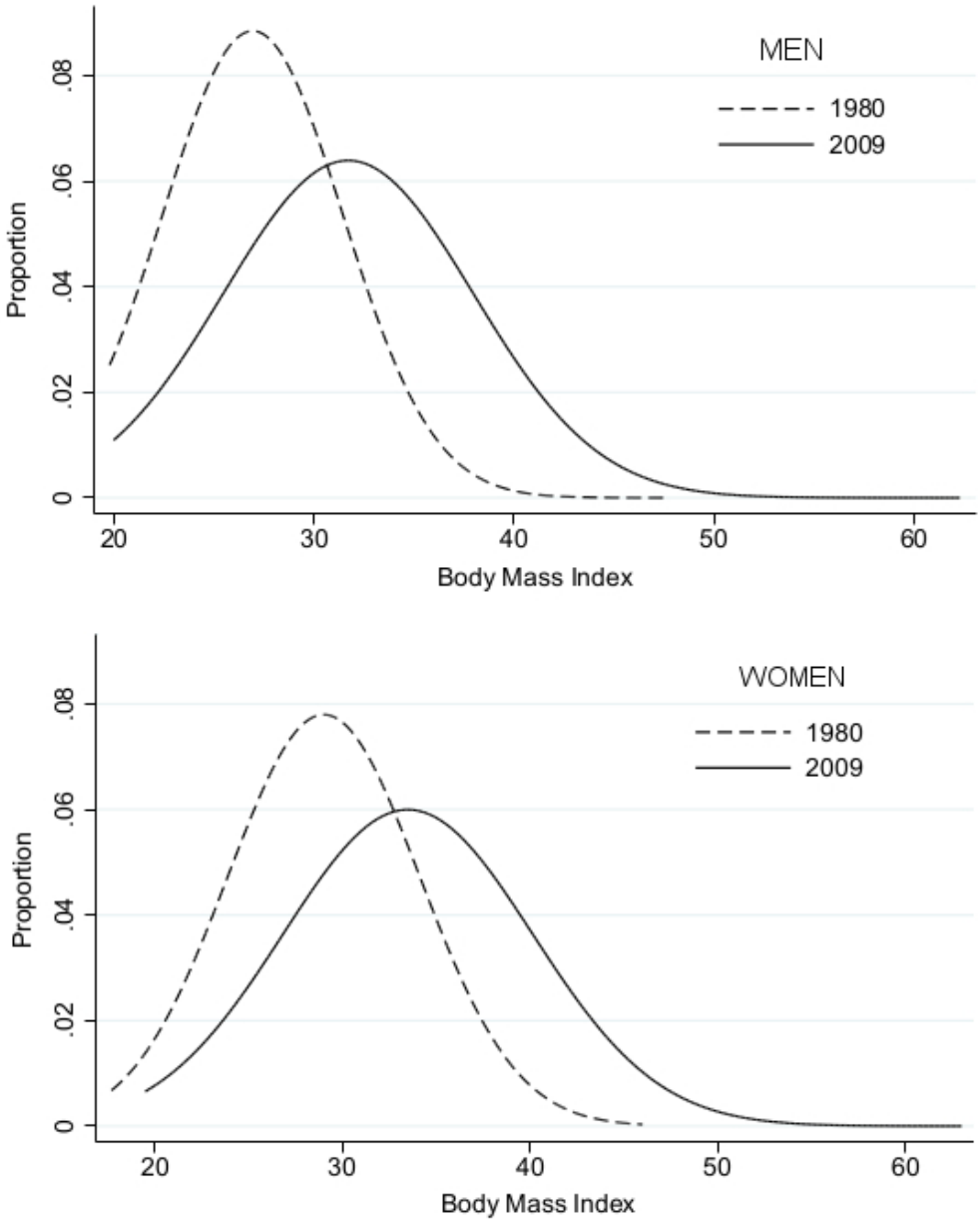

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Table 1 Mean systolic/diastolic blood pressure and hypertension prevalence, Wallis Polynesians, 1980 and 2009

Age	Sample		Mean SBP		Mean DBP		% Hypertension	
Group	1980	2009	1980	2009	1980	2009	1980	2009
				MALE				
25-34	80	23	118.8	131.0 ***	71.6	74.5	10.0	17.4
			(115.9-121.7)	(126.6-135.3)	(69.0-74.2)	(71.1-77.8)	(4.4-18.8)	(5.0-38.8)
35-44	56	23	115.6	130.0 ***	72.4	78.3 **	7.1	30.4 *
			(111.9-119.3)	(124.5-135.6)	(70.0-74.7)	(74.4-82.2)	(2.0-17.3)	(13.2-52.9)
45-54	41	32	117.3	141.0 ***	75.6	83.1 **	12.2	57.6 ***
			(112.7-121.8)	(134.5-147.5)	(71.9-79.4)	(78.9-87.2)	(4.1-26.2)	(39.2-74.5)
55-64	36	37	119.9	141.5 ***	75.1	82.1 *	19.4	64.9 ***
			(113.9-125.9)	(133.8-149.2)	(70.4-79.9)	(77.7-86.6)	(8.2-36.0)	(47.5-79.8)
Linear age trend p-value			ns	<0.05	<0.05	<0.05	ns	<0.05
25-64^	213	115	117.8	136.2 ***	73.5	79.6 ***	11.7	43.0 ***
			(115.8-119.8)	(133.0-139.5)	(71.9-75.1)	(77.5-81.6)	(7.4-16.1)	(34.0-52.3)
				FEMALE				
25-34	81	34	110.2	121.1 ***	71.6	72.3	7.4	15.2
			(107.2-113.2)	(117.5-124.8)	(69.3-73.8)	(68.6-76.0)	(2.7-15.4)	(5.1-31.9)
35-44	61	51	114.2	123.2 **	74.7	75.0	19.7	9.8
			(109.7-118.7)	(118.9-127.4)	(71.4-78.0)	(72.2-77.9)	(10.6-31.8)	(3.3-21.4)
45-54	52	40	120.7	141.3 ***	76.3	81.6*	11.5	58.5 ***
			(116.1-125.4)	(135.5-147.1)	(73.4-79.2)	(78.3-84.8)	(4.4-23.4)	(42.1-73.7)
55-64	34	28	127.7	137.7	75.6	75.7	23.5	57.1 **
			(120.7-134.7)	(129.3-146.1)	(71.4-79.7)	(70.9-80.5)	(10.7-41.1)	(37.2-75.5)
Linear age trend p-value			<0.05	<0.05	<0.05	<0.05	ns	<0.05
25-64^	228	153	117.4	129.0 ***	74.5	75.9	15.1	29.6 ***
			(115.1-119.8)	(126.1-131.9)	(73.0-76.0)	(74.1-77.6)	(10.4-19.7)	(22.4-36.9)

FOOTNOTES: SBP, systolic blood pressure; DBP, diastolic blood pressure; % Hypertension, prevalence of hypertension SBP ≥140 and/or DBP ≥90 mmHg and/or self-report taking medication for hypertension; 95% confidence intervals in brackets; ^ age-standardised to the 2008 Census of Wallis Island; *p<0.05, ** p<0.01, *** p<0.001, ns (not significant p≥0.05).

Table 2 Mean fasting plasma glucose and the prevalence of diabetes, Wallis Polynesians, 1980 and 2009

Age	Sample		Mean FPG		% Impaired FPG		% Diabetes Mellitus	
Group	1980	2009	1980	2009	1980	2009	1980	2009
MALE								
25-34	80	23	4.7	5.8	1.3	13.0 *	0.0	8.7 *
			(4.5-4.8)	(4.6-7.1)	(0.0-6.7)	(2.8-33.6)	(0.0-5.0)	(1.1-28.0)
35-44	56	23	5.0	5.0	3.6	17.4	1.8	0.0
			(4.5-5.4)	(4.7-5.3)	(0.0-12.3)	(5.0-38.8)	(0.0-9.6)	(0.0-16.0)
45-54	40	33	5.1	5.8 *	5.0	9.1	0.0	18.2 **
			(4.9-5.3)	(5.1-6.5)	(0.0-16.9)	(1.9-24.3)	(0.0-9.0)	(7.0-35.5)
55-64	36	37	5.4	5.3	5.6	5.4	8.3	18.9
			(4.6-6.2)	(5.0-5.6)	(0.0-18.7)	(0.6-18.2)	(1.8-22.5)	(8.0-35.1)
Linear age trend p-value			<0.05	ns	ns	ns	<0.05	<0.05
25-64^	212	116	5.0	5.5 **	3.7	10.8 **	2.3	12.2 ***
			(4.8-5.2)	(5.2-5.8)	(1.1.-6.2)	(5.1-16.5)	(0.3-4.4)	(6.2-18.2)
FEMALE								
25-34	81	33	4.4	4.8	0.0	6.1	0.0	3.0
			(4.2-4.6)	(4.4-5.1)	(0.0-5.0)	(0.7-20.2)	(0.0-0.5)	(0.1-15.8)
35-44	61	51	5.0	4.9	4.9	2.0	4.9	7.8
			(4.7-5.4)	(4.7-5.2)	(1.0-13.7)	(0.1-10.5)	(1.0-13.7)	(2.2-18.9)
45-54	52	41	5.2	6.1	7.7	12.2	5.8	26.8 **
			(4.8-5.6)	(5.3-6.8)	(2.1-18.5)	(4.1-26.2)	(1.2-16.0)	(14.2-42.9)
55-64	34	28	5.7	6.2	5.9	7.1	5.9	39.3 **
			(4.6-6.8)	(5.5-6.9)	(0.7-19.7)	(0.9-23.5)	(0.7-19.7)	(21.5-59.4)
Linear age trend p-value			<0.05	<0.05	<0.05	ns	ns	<0.05
25-64^	228	153	5.0	5.3	4.5	6.3	4.0	15.8 ***
			(4.8-5.3)	(5.1-5.6)	(1.9-7.2)	(2.5-10.1)	(1.5-6.6)	(10.1-21.6)

FOOTNOTES: FPG, fasting plasma glucose; % Impaired FPG, prevalence of FPG ≥6.1 and <7.0 mmol/L and not taking medication for type 2 diabetes mellitus (T2DM); % Diabetes Mellitus, prevalence of FPG ≥7.0 mmol/L and/or taking medication for T2DM; 95% confidence intervals in brackets; ^ age-standardised to the 2008 Census of Wallis Island; *p<0.05, ** p<0.01, *** p<0.001, ns (not significant p≥0.05).

Table 3 Mean body mass index and obesity prevalence, Wallis Polynesians, 1980 and 2009

Age	Sample		Mean BMI		% Obese BMI≥30		%Obese BMI>32	
Group	1980	2009	1980	2009	1980	2009	1980	2009
Male								
25-34	80	23	26.6	32.3 ***	11.3	52.2 ***	6.3	47.8 ***
			(25.9-27.3)	(29.7-35.0)	(5.3-20.3)	(30.6-73.2)	(2.1-14.0)	(26.8-69.4)
35-44	56	23	27.8	33.5 ***	26.8	69.6 ***	16.1	52.2 ***
			(26.5-29.2)	(30.7-36.3)	(15.8-40.3)	(47.1-86.8)	(7.6-28.3)	(30.6-73.2)
45-54	41	33	28.1	32.7 **	31.7	72.7 ***	24.4	45.5
			(26.3-30.0)	(30.3-35.0)	(18.1-48.1)	(54.5-86.7)	(12.4-40.0)	28.1-63.7)
55-64	36	37	27.3	31.7 ***	33.3	54.1	11.1	37.8 *
			(25.5-29.1)	(30.0-33.4)	(18.6-51.0)	(36.9-70.5)	(3.1-26.1)	(22.5-55.2)
Linear age trend p-value			ns	ns	<0.05	ns	ns	ns
25-64^	213	116	27.4	32.5 ***	25.0	61.7 ***	14.1	45.4 ***
			(26.8-28.1)	(31.4-33.6)	(19.2-30.8)	(52.8-70.6)	(9.4-18.8)	(36.2-54.5)
Female								
25-34	74	34	29.0	36.5 ***	37.8	73.5 ***	29.7	73.5 ***
			(27.9-30.0)	(33.5-39.5)	(26.8-49.9)	(55.6-87.1)	(19.6-41.5)	(55.6-87.1)
35-44	52	51	30.9	33.9 **	53.8	80.4 **	40.4	70.6 **
			(29.4-32.4)	(32.3-35.5)	(39.5-67.7)	(66.9-90.2)	(27.0-54.9)	(56.2-82.5)
45-54	45	41	29.5	35.3 ***	53.3	85.4 ***	37.8	73.2 ***
			(27.8-31.3)	(33.4-37.2)	(37.9-68.3)	(70.8-94.4)	(23.8-53.5)	(57.1-85.8)
55-64	30	28	30.3	32.5	50.0	64.3	36.7	39.3
			(28.3-32.3)	(30.1-34.8)	(31.3-68.7)	(44.1-81.4)	(19.9-56.1)	(21.5-59.4)
Linear age trend p-value			ns	ns	ns	ns	ns	<0.05
25-64^	201	154	29.9	34.7 ***	48.6	77.2 ***	36.1	67.3 ***
			(29.2-30.6)	(33.6-35.8)	(41.7-55.5)	(70.7-83.8)	(29.5-42.7)	(59.9-74.7)

FOOTNOTES: BMI, body mass index; % Obese BMI ≥30, prevalence obesity according to the standard definition of the World Health Organisation [12]; % Obese BMI>32, prevalence of obesity as suggested by Swinburn et al. [13] for Polynesian populations; 95% confidence intervals in brackets; Pregnant women excluded; ^ age-standardised to the 2008 Census of Wallis Island.*p<0.05, ** p<0.01, *** p<0.001, ns (not significant p≥0.05).

Table 4 Mean cholesterol and elevated cholesterol and triglyceride prevalence, Wallis Polynesians, 1980 and 2009

Age	Sample		Mean Cholesterol		% Cholesterol ≥5.2		%Triglycerides ≥1.7	
Group	1980	2009	1980	2009	1980	2009	1980	2009
MALE								
25-34	78	23	3.7	4.5 **	6.4	30.4 **	1.3	26.1 ***
			(3.5-3.9)	(4.0-5.0)	(2.1-14.3)	(13.2-52.9)	(0.0-6.9)	(10.2-48.4)
35-44	78	23	3.8	4.3 **	3.6	13.0	1.8	43.5 ***
			(3.6-4.0)	(3.9-4.7)	(0.4-12.3)	(2.8-33.6)	(0.0-9.5)	(23.2-65.5)
45-54	78	23	4.0	4.5 *	12.8	21.9	2.5	34.4 ***
			(3.7-4.3)	(4.2-4.9)	(4.3-27.4)	(9.3-40.0)	(0.0-13.2)	(18.6-53.2)
55-64	78	23	4.0	4.3	5.7	16.2	2.9	27.0 **
			(3.7-4.3)	(4.1-4.6)	(0.7-19.2)	(6.2-32.0)	(0.0-14.9)	(13.8-44.1)
Linear age trend p-value			<0.05	ns	ns	ns	ns	ns
25-64^	208	115	3.9	4.4 ***	6.8	20.7 ***	2.0	32.1 ***
			(3.8-4.0)	(4.2-4.6)	(3.4-10.3)	(13.2-28.2)	(0.1-4.0)	(23.5-40.7)
FEMALES								
25-34	71	34	4.2	3.8	15.5	2.9	9.7	20.6
			(3.9-4.5)	(3.5-4.1)	(8.0-26.0)	(0.0-15.3)	(4.0-19.0)	(8.7-37.9)
35-44	53	51	4.2	3.8 *	17.0	9.8	1.9	15.7 *
			(3.9-4.4)	(3.6-4.0)	(8.0-29.8)	(3.3-21.4)	(0.0-10.0)	(7.0-28.6)
45-54	50	41	4.3	4.0	20.0	7.3	6.0	26.8 **
			(4.0-4.6)	(3.8-4.3)	(10.0-33.7)	(1.5-19.9)	(1.3-16.6)	(14.2-42.9)
55-64	32	28	4.6	4.0 *	15.6	10.7	3.1	10.7
			(4.2-4.9)	(3.7-4.4)	(5..3-32.8)	(2.3-28.2)	(0.0-16.2)	(2.3-28.2)
Linear age trend p-value			ns	ns	ns	ns	ns	ns
25-64^	206	154	4.3	3.9 **	17.2	7.5**	5.3	18.9 ***
			(4.2-4.4)	(3.8-4.0)	(12.1-22.3)	(3.3-11.6)	(2.2-8.3)	(12.8-25.1)

FOOTNOTES: % Cholesterol ≥5.2, prevalence of serum cholesterol ≥5.2 mmol/L; % Triglycerides ≥1.7 prevalence of triglycerides ≥1.7 mmol/L; 95% confidence intervals in brackets; ^ age-standardised to the 2008 Census of Wallis Island *p<0.05, ** p<0.01, *** p<0.001, ns (not significant p≥0.05).