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ORIGINAL ARTICLE

South Pacific Islanders resist type 2 diabetes: comparison of aerobic and resistance training

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Abstract The purpose of this study was to evaluate the effectiveness of two exercise modalities for improving glycosylated hemoglobin (HbA1c) and associated clinical outcomes in Polynesian adults diagnosed with type 2 diabetes and visceral obesity. Twenty-six adults were randomized to receive resistance training or aerobic training, 3×/week, for 16 weeks. Dependent variables collected before and after intervention included: diabetes markers including HbA1c, blood lipids, relevant cytokines (C-reactive protein, adiponectin), and anthropometric and hemodynamic indices. Eighteen participants (72% female; age: 49.3 ± 5.3 years; waist circumference: 128.7 ± 18.7 cm) completed the intervention and follow-up assessments. Body mass index in the whole cohort at baseline indicated Class III (morbid) obesity (43.8 ± 9.5 kg/m^2). Compliance to training was 73 ± 19 and 67 ± 18% in the aerobic and resistance training groups, respectively. HbA1c remained elevated in both groups after training. Aerobic training reduced systolic and diastolic blood pressure and increased serum triglycerides (all $P < 0.05$). No other exercise-induced adaptations were noted within or between groups. Post hoc analysis using pooled data indicated that higher adherence to training (≥75% attendance, $n = 8$) significantly reduced waist circumference ($P < 0.001$) and tended to reduce body weight and fasting insulin (all $P \leq 0.11$) versus lower adherence (<75% attendance, $n = 10$). In conclusion, this study did not demonstrate an improvement in HbA1c with exercise in morbidly obese Polynesian people. Future investigations involving exercise regimens that are more practicable and which involve greater frequency and duration of training may be required to induce significant and clinically meaningful adaptations in this unique diabetes population.

Keywords Aerobic training · Resistance training · Glycemic control · Ethnicity · Polynesian · Morbid obesity

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Introduction

The Māori and Pacific Island people living in New Zealand are particularly vulnerable to type 2 diabetes, with an incidence rate nearly threefold higher than their Caucasian counterparts (8.2 vs. 3.1%) (Diabetes New Zealand 2008). This disparity has been driven by unprecedented rates and

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extreme levels of obesity in the Polynesian people (Simmons et al. 1994) and has contributed to significant inequalities in life expectancy (Bramley et al. 2004). By 2020, an estimated 18% of Polynesian people will be diagnosed with type 2 diabetes as compared to only 4% of New Zealanders of European origin (Diabetes New Zealand, 2008). Clearly, efforts must be directed toward closing this gap.

Exercise prescription involving resistance training, aerobic training, or a combination thereof, has been shown to significantly reduce glycosylated hemoglobin (HbA1c) and is therefore advocated for diabetes management and potential remission (Albright et al. 2000). However, the majority of trials used to formulate current exercise guidelines have involved primarily Caucasian patients, or the ethnicity has not been reported. This is notable given that certain cohorts are more severely affected by this disease than others (Abate and Chandalia 2003).

The cause for the rift in health status between ethnic groups remains unclear, but is likely due to a combination of environmental and biological factors. Historical evidence suggests that obesity and diabetes were rare and increased sharply in Polynesian people after exposure to an inactive lifestyle, low quality diet, and socioeconomic marginalization, the known consequences of colonization and globalization. Moreover, studies involving Polynesian people (McAuley et al. 2002) and other ethnic cohorts have revealed that diabetes markers such as fasting insulin, insulin sensitivity, pancreatic β-cell function, and glucoregulation differ by ethnicity even after controlling for confounding variables such as age and adiposity (Abate and Chandalia 2003). Causative mechanisms, such as fat distribution patterns and quantity of fat-free mass, have also been shown to vary between Polynesians and other ethnicities (Rush et al. 2009).

Given these interethnic differences, there is a clear rationale for investigating chronic exercise responses in these cohorts both independently and comparatively. Accordingly, a number of exercise trials have recently been conducted in ethnic populations who suffer disproportionately from type 2 diabetes, including African (Van Rooijen et al. 2004; Winnick and Schuster 2008), Indian (Misra et al. 2008), Hispanic (Castaneda et al. 2002), and Chinese (Sykes et al. 2004) cohorts. Several of these trials have shown that HbA1c can be reduced with $\geq$12 weeks of training, while others have shown no effect (Sykes et al. 2004; Van Rooijen et al. 2004).

This is the first study to prescribe exercise training in Polynesian adults diagnosed with type 2 diabetes and visceral obesity. We hypothesized that participants randomized to receive either resistance or aerobic training for 16 weeks would improve HbA1c and related metabolic outcome measures. The research was considered preliminary and exploratory, for although these exercise modalities have been shown to improve glycemic control in other cohorts, the exercise response of this ethnically distinct and highly obese population had never been systematically investigated to date.

Methods

Cultural consultation and study design

All methods were reviewed and approved by Polynesian cultural consultants and the Central Regional Ethics Committee, New Zealand (CEN/07/08/054; ACTRN #12609001085268). Cultural consultation revealed that potential participants considered randomization to a non-exercise control group unethical, as previously noted in a lifestyle intervention trial (McAuley et al. 2003). The study design was therefore modified from a randomized controlled trial (RCT) comparing resistance training to usual care (no exercise) to a trial evaluating and comparing resistance and aerobic training.

Participants and randomization

The participants were recruited through direct referrals by local health-care professionals and evaluated for eligibility via medical screening process between February and August of 2008. Eligibility criteria included: (1) self-identified Polynesian descent; (2) a clinical diagnosis of type 2 diabetes; (3) visceral obesity defined as a waist circumference $\geq$88 cm in women and $\geq$102 in men; (4) no regular exercise participation for the previous 6 months; (5) no change in diabetes medications for the previous 2 months; and (6) no acute or chronic medical conditions for which exercise would be contraindicated. Medical screening procedures were developed in consultation with the study endocrinologist (J.K.) and were in accordance with established guidelines for exercise prescription.

The participants were randomly assigned following baseline testing via computer-generated randomization list (http://www.randomization.com), stratified by gender in blocks of four, to receive either resistance training or aerobic training for 16 weeks.

Interventions

All participants attended supervised exercise sessions three times per week on non-consecutive days at a health and fitness facility (City Fitness©) located in the predominantly Polynesian suburb of Porirua, New Zealand. Given that lack of time is the most frequently cited barrier to exercise adoption, the duration and frequency of training were

equated. Groups exercised in parallel, three sessions per week, and the duration of each session ranged from 40 to 60 min, increasing progressively over time.

The exercise regimens were developed in accordance with guidelines published by the American College of Sports Medicine (Albright et al. 2000). Pre- and post-exercise heart rate, blood pressure, and blood glucose were monitored and recorded each session. Participants continued to receive their usual medical care and were instructed to maintain their dietary and physical activity habits during the trial.

The resistance training group performed two to three sets of eight major exercises using machine weights (Cybex International, Medway, MA, USA) targeting all the major muscle groups of the body for six to eight repetitions to neural fatigue. Exercises included: seated leg press, knee extension, knee flexion, chest press, lat pulldown, overhead press, biceps curl, and triceps extension. Approximately, 1 min of rest was provided between sets and exercises, and loads were increased by 5% when participants could perform ten repetitions. The aerobic training included exercise on a cycle ergometer (Life Fitness, Schiller Park, IL, USA). The program gradually progressed from 65 to 85% of their heart rate reserve (HRR) (i.e., $HRR = (HR_{max} - HR_{rest}) + HR_{rest}$) during the first 2 weeks of training, where it was maintained for the remainder of the study. Heart rate and blood pressure were monitored and recorded at peak steady state workloads. Watts and duration at peak workload were increased to accommodate improved fitness levels over time. Compliance to training was defined as the number of training sessions attempted divided by the number offered multiplied by 100%.

Outcome measures

Assessments at baseline and following the 16-week intervention period were completed at the Kenepuru Community Hospital, Porirua, New Zealand. Participants were evaluated following a 12-h overnight fast. Follow-up testing was standardized to 72 h after completion of the final exercise session to alleviate the confounding effect of the last training session.

All blood samples were sent to the medical laboratories of the Capital & Coast District Health Board and the Canterbury District Health Board for measurement of HbA1c, fasting glucose and insulin, C-peptide, total cholesterol, triglycerides, HDL cholesterol, free fatty acids, high-sensitivity C-reactive protein (CRP), and adiponectin with coefficients of variation ranging from 3.0 to 8.8%.

Insulin resistance was estimated by two methods that have been validated against the euglycemic insulin clamp: homeostasis model assessment (HOMA2-IR software, version 2.2.2, Oxford University) (Diabetes Trials Unit 2007), and the McAuley Index (McAuley et al. 2001).

Resting systolic and diastolic blood pressures (SBP and DBP) were measured in duplicate from the left arm after 5 min of seated rest on a standard hospital sphygmomanometer, with the lowest blood pressure being recorded.

Height and weight were measured using standard protocols on a calibrated stadiometer and scale, respectively. Body mass index (BMI) in kg/m^2 was calculated from these measures. Percent body fat was collected via bioelectrical impedance analysis (Tanita TBF-310 analyzer, Tanita Corporation, Arlington Heights, IL, USA). Waist circumference was measured at the end of normal expiration at the midpoint between the lower costal margin and the iliac crest.

Statistical analyses

Statistical analyses were performed using StatViewTM statistical software package (Version 5.0 SAS Institute, Cary, NC, USA). Data from participants who were unavailable for follow-up assessment were excluded, per protocol analysis. All data were visually inspected and statistically evaluated for normality (skewness and kurtosis between -1 and $+1$). Normally distributed data were described as mean $\pm$ SD. Baseline differences between groups were evaluated using an independent t test or Chi-square as appropriate. Within and between group changes from weeks 0 to 16 were analyzed by repeated measures ANOVA. A P value of <0.05 was accepted as statistically significant.

Results

Participant recruitment

A flow diagram detailing recruitment and attrition is presented in Fig. 1. Fifty potential participants expressed interest in the study. Thirteen (26%) were excluded because they did not meet entry criteria. Of 37 eligible subjects, 11 (30%) declined to participate. A total of 26 participants were randomized; of these, 18 (69%) completed the training program and follow-up assessments and were included in the final analyses.

Baseline characteristics

Baseline characteristics for the resistance training group, aerobic training group, and total cohort are presented in Table 1. With the exception of higher systolic blood pressure in the aerobic training group ($P = 0.01$) and higher use of diuretics in the resistance training group ($P = 0.02$), no statistically significant differences were noted between groups. There was a trend for higher HbA1c

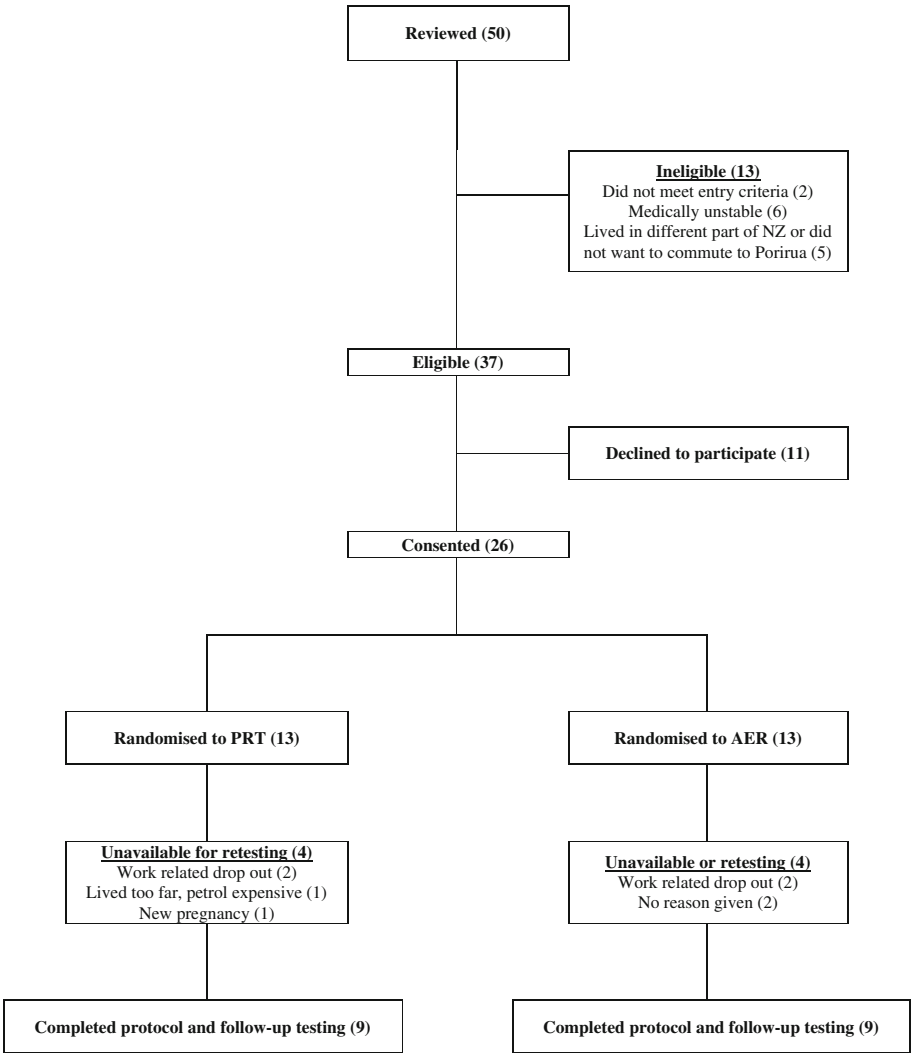

Fig. 1 Recruitment flowchart

($P = 0.07$) and use of ACE inhibitors in the resistance training group ($P = 0.14$).

All participants met the Adult Treatment Panel III definition for metabolic syndrome (Grundy et al. 2004). BMI of the total cohort indicated Class III (morbid) obesity (43.8 ± 9.5 kg/m^2), while duration of known diabetes ranged from 0.5 to 13 years. The majority of participants were women (72%), fully employed (89%), married (83%), and had completed a minimum of high school education (96%). Age ranged from 39 to 59 years, and the cohort was young compared to previous studies of exercise in type 2 diabetes (Castaneda et al. 2002; Dunstan et al. 2002). As much as 56% noted New Zealand Māori ancestry and only two indicated mixed ancestry (Samoan/Cook Islands Māori/Tokelauan/European, and Fijian/New Zealand Māori).

A history of tobacco use was common ($n = 10$), and five participants were current smokers. Two participants had a history of myocardial infarction, while none noted prior cerebrovascular accident or cardiac surgery. Use of medications was high; metformin and ACE inhibitors were commonly prescribed. Weekly status checks on any medication changes occurred throughout the 16-week intervention period. No changes in medication were noted.

Compliance

Compliance to training was 73 ± 19 and 67 ± 18% in the aerobic and resistance training groups, respectively. In the total cohort, attendance ranged from 20 to 47 of 48 possible sessions (i.e., 42–98%). Only eight participants, five from the aerobic training group and three from the resistance training group, attended at least 36/48 (75%) available sessions. The most common reasons for missed sessions included work, family, flu-like illness, and funerals. Completion of resistance training and aerobic training protocols was 100% when participants were present for exercise sessions, ensured via direct supervision of all participants.

Table 1 Baseline subject characteristics for subjects completing the protocol ($n = 18$)

Characteristic	Resistance training	Aerobic training	Total cohort
n	9	9	18
Age (years)	48 ± 6	51 ± 4	49 ± 5
Sex (female/male)	6/3	7/2	13/5
Height (cm)	166.2 ± 8.2	167.9 ± 5	167.1 ± 6.7
Weight (kg)	118.6 ± 38.5	126.8 ± 18.6	122.7 ± 29.6
Body mass index (kg/m^2)	42.7 ± 12.1	45.0 ± 6.5	43.8 ± 9.5
Waist circumference (cm)	125.4 ± 23.2	131.9 ± 13.5	128.7 ± 18.7
Systolic blood pressure (mmHg)	123.2 ± 19.4	147.3 ± 16.1*	135.3 ± 21.3
Diastolic blood pressure (mmHg)	85.7 ± 13.8	90.4 ± 5.7	88.1 ± 10.6
Self-identified ethnicity			
New Zealand Māori	6	4	10
Cook Islands Māori	1	2	3
Samoan	1	1	2
Fijian	–	1	1
Tokelauan	1	–	1
Tongan	–	1	1
Diabetes duration (years) [range]	2.6 ± 1.8 [0.5 – 5]	3.9 ± 4.3 [0.5 – 13]	3.3 ± 3.3 [0.5 – 13]
Glycosylated hemoglobin (HbA1c) (%)	10.7 ± 2.1	8.9 ± 1.9	9.8 ± 2.1
Diabetes management regimen			
Diet only (*n*)	1	2	3
Oral hypoglycemics (*n*)	7	6	13
Oral hypoglycemics and insulin (*n*)	1	1	2
Blood pressure lowering medications (*n*)			
ACE inhibitors (*n*)	7	4	11
Diuretics (*n*)	4*	0	4
β–Blockers (*n*)	2	1	3
Angiotensin II receptor antagonist (*n*)	1	0	1
Lipid lowering medication medications (*n*)	5	3	8
Current smoker (*n*)	3	2	5

Data expressed as mean ± SD. Baseline comparisons determined by independent sample *t* test or Chi-square

ACE angiotensin converting enzyme

* Statistically significant difference observed between groups at baseline ($P \leq 0.05$)

Adverse events

One male participant experienced syncope during the performance of a resistance training exercise. The episode resolved upon placing him in the supine position. An ambulance was contacted and evaluation at a nearby hospital ruled out myocardial infarction. The participant resumed and completed the training program after clearance from his general practitioner. No other adverse events were noted.

Primary outcome

All outcome measures are presented in Table 2. HbA1c was elevated (≥7%) in all participants at baseline, with eight participants in the resistance training group and two in the aerobic training group presenting with values greater than 10%. No change in HbA1c was noted within or between groups after the 16-week intervention period.

Secondary outcomes

All participants were insulin resistant at baseline according to HOMA2-IR and the McAuley Index. Diabetes markers, including measure of insulin resistance, fasting glucose, fasting insulin, C-peptide, and free fatty acids did not change significantly within or between groups over time. However, there was a trend toward reduced fasting insulin ($P = 0.09$) in the aerobic training group.

Baseline CRP ranged from 1.0 to 35.7 mg/L in the total cohort and was elevated above the 90th percentile for normal adults (≥3.0 mg/L) in 12 participants (67%), while

Table 2 Primary and secondary outcomes

Outcome measure	Resistance training				Aerobic training				*P* (between groups)
	Week 0	Week 16	Change	*P*	Week 0	Week 16	Change	*P*	
Primary outcome									
HbA1c (%)	10.7 ± 2.1	10.6 ± 2.4	−0.1 ± 1.1	0.86	8.9 ± 1.9	8.8 ± 2.1	−0.1 ± 0.6	0.60	0.92
Secondary outcomes									
Diabetes markers									
HOMA2-IR index	2.9 ± 2	2.9 ± 1.9	0.0 ± 0.5	1.00	3.9 ± 1.9	2.9 ± 1.3	−0.9 ± 1.6	0.13	0.17
McAuley index	5.2 ± 1.5	5.2 ± 1.3	0.0 ± 0.5	0.85	5.2 ± 0.8	5.3 ± 0.8	+0.1 ± 0.7	0.59	0.95
Glucose (mmol/L)	9.5 ± 3.5	11.4 ± 4	+1.9 ± 3.2	0.17	10.2 ± 3.3	10.4 ± 2.9	+0.2 ± 1.6	0.72	0.19
Insulin (pmol/L)	140.7 ± 100.1	134.1 ± 103.1	−6.6 ± 24	0.50	177.4 ± 82.5	134.8 ± 64.1	−42.7 ± 65.8	0.09	0.19
C-peptide (nmol/L)	1.6 ± 1.1	1.6 ± 1	+0.1 ± 0.5	0.69	1.4 ± 0.3	1.5 ± 0.7	+0.1 ± 0.5	0.52	0.85
Free fatty acids (mEq/L)	0.5 ± 0.3	0.6 ± 0.3	+0.1 ± 0.2	0.40	0.7 ± 0.2	0.7 ± 0.2	0.0 ± 0.2	0.86	0.69
Cytokines									
Log C-reactive protein	0.6 ± 0.5	0.5 ± 0.5	−0.2 (±0.5)	0.41	0.8 ± 0.4	0.6 ± 0.4	−0.2 (±0.4)	0.22	0.90
Adiponectin (μg/ml)	5.6 ± 1.9	5.6 ± 2.2	0.0 ± 1.4	0.96	6.7 ± 3.3	6.7 ± 3.2	+0.1 ± 2.2	0.94	0.93
Blood lipids									
Total cholesterol (mmol/L)	4.9 ± 1.5	4.5 ± 1	−0.4 ± 0.9	0.21	4.5 ± 0.4	4.7 ± 0.4	+0.3 ± 0.6	0.22	0.08
HDL cholesterol (mmol/L)	1.3 ± 0.4	1.3 ± 0.5	0.0 ± 0.1	0.37	1.1 ± 0.2	1.1 ± 0.2	0.0 ± 0.1	0.80	0.64
LDL cholesterol (mmol/L)	2.7 ± 1.4	2.4 ± 0.7	−0.3 ± 0.8	0.27	2.6 ± 0.6	2.7 ± 0.4	+0.1 ± 0.5	0.71	0.23
Triglycerides (mmol/L)	2.2 ± 1.2	2.0 ± 1	−0.2 ± 0.6	0.35	1.6 ± 0.5	1.9 ± 0.6*	+0.3 ± 0.2	0.004	0.03
Anthropometric markers									
Body weight (kg)	118.6 ± 38.5	118.9 ± 37.5	+0.2 ± 3.4	0.85	126.8 ± 18.6	125.5 ± 19.7	−1.3 ± 3.6	0.30	0.35
BMI (kg/m^2)	42.7 ± 12.1	42.7 ± 11.7	0.0 ± 1.1	0.91	45 ± 6.5	44.5 ± 6.9	−0.5 ± 1.3	0.32	0.37
Waist circumference (cm)	125.4 ± 23.2	124.3 ± 23.2	−1.1 ± 2.8	0.30	131.9 ± 13.5	131.1 ± 14.7	−0.8 ± 5.1	0.64	0.91
Body fat (%)	50.2 ± 7.6	49.8 ± 6.1	−0.4 ± 2.5	0.68	49.6 ± 5.2	48.8 ± 6	−0.7 ± 2.6	0.43	0.76
Hemodynamic markers (mmHg)									
SBP	123.2 ± 19.4	125.6 ± 17	+2.3 ± 16.9	0.69	147.3 ± 16.1	131.1 ± 9.1*	−16.2 ± 13.0	0.006	0.02
DBP	85.7 ± 13.8	83.1 ± 8	−2.6 ± 11.2	0.51	90.4 ± 5.7	85.8 ± 4.6*	−4.7 ± 4.9	0.02	0.61

Data expressed as mean ± SD. C-reactive protein was non-normally distributed and log transformed

HbA1c Hemoglobin A1c, *BMI* body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HOMA2-IR* homeostasis modeling assessment for insulin resistance, *LDL* low density lipoprotein, *HDL* high density lipoprotein

* Statistically different from baseline value within group

mean adiponectin levels in both groups were within the desired 5–25 μg/ml range. CRP and adiponectin did not change significantly in either group or between groups over time.

Lipid lowering medications were prescribed in eight participants (Table 1). Mean total and LDL cholesterol were only slightly elevated (>4 and >2 mmol/L, respectively) in both groups at baseline. Triglycerides were elevated (>1.7 mmol/L) in the resistance training group only, while HDL cholesterol was normal (>1 mmol/L) in both groups. No significant within or between group changes were observed in total cholesterol, HDL cholesterol, or LDL cholesterol within or between groups. However, serum triglycerides increased significantly secondary to aerobic training ($P = 0.004$), and this change was significant between groups ($P = 0.03$).

Anthropometric measures, including BMI, waist circumference and percent body fat indicated extreme obesity at baseline. None of these markers changed significantly within or between groups post-intervention.

The use of ACE inhibitors ($P = 0.14$) and diuretics ($P = 0.02$) was higher in the resistance training group versus at baseline (Table 1). Mean SBP and DBP in the resistance training indicated normotension, while these markers were elevated in the aerobic training group. SBP and DBP significantly decreased after 16 weeks of aerobic training ($P = 0.006$ and $P = 0.02$, respectively), and the change in SBP was significantly different between groups over time ($P = 0.02$).

Post hoc analyses

Post hoc analyses were conducted to evaluate the effect of adherence on adaptation to exercise. First, a repeated measure ANOVA was conducted using pooled data between participants who attended ≥75% of the exercise

sessions versus those who attended <75% of sessions. Higher adherence significantly reduced waist circumference ($P < 0.001$), and tended to improve body weight ($P = 0.11$) and plasma insulin ($P = 0.11$) versus lower adherence. Second, simple regression analysis using pooled data for the whole cohort revealed that higher attendance was associated with the largest reductions in waist circumference ($r = -0.664$; $P = 0.003$) and CRP ($r = -0.492$; $P = 0.04$). No other associations between attendance and change in outcome measures were found.

Discussion

This is the first study to investigate prescribed exercise training in Polynesian adults with type 2 diabetes and morbid obesity. Our findings suggest that resistance or aerobic training prescribed 3×/week for 16 weeks is insufficient to improve HbA1c. Analyses of secondary outcomes revealed that aerobic training reduced SBP and DBP while increasing triglycerides. No other effects were noted within or between groups. However, post hoc analyses revealed that higher compliance resulted in several significant and clinically meaningful adaptations. The training regimens were well tolerated with only one adverse event reported.

We acknowledge these findings as preliminary and the need for large-scale RCTs, given our sample size and potentially low statistical power. However, several studies enrolling a comparable number of participants ($n = 18$–30) have documented reductions in HbA1c (Baldi and Snowling 2003; Misra et al. 2008; Shenoy et al. 2009 and related indices (i.e., fasting insulin (Baldi and Snowling 2003), blood lipids, waist circumference, subcutaneous fat (Misra et al. 2008), and blood pressure and heart rate (Shenoy et al. 2009) following 10–16 weeks of exercise training prescribed at a similar dose and intensity. There are several reasons for the lack of adaptation noted in many of our primary and secondary end points. In general, these explanations relate to the extreme level of obesity in our cohort, compliance to training, and the doses of exercise prescribed.

Excess adiposity interrupts the insulin-signaling cascade and can ultimately lead to type 2 diabetes (Stannard and Johnson 2003). Interestingly, insulin sensitivity in Polynesian people has been found to be lower at an equivalent level of adiposity (BMI and total and truncal fat) versus Caucasians (McAuley et al. 2002). The mechanisms underlying this interethnic disparity remain unclear, but may be related to the concept of a thrifty phenotype, an evolved propensity to more efficiently store fat (i.e., triglyceride) as an energy source (Lindsay and Bennett 2001). Several authors assert that insulin resistance is a homeostatic mechanism that up-regulates fatty acid oxidation when triglyceride stores are excessive (Stannard and Johnson 2006). A low threshold for insulin resistance, as noted in Polynesian people, would enhance survival in an environment of perpetual physical activity and seasonal food availability (McAuley et al. 2002) (Stannard and Johnson 2006). However, in a modern environment of food overabundance and chronic inactivity, this trait would predispose to obesity, advanced metabolic diseases and early mortality (Stannard and Johnson 2006).

The level of obesity documented in our Polynesian participants (Table 2) was considerably higher versus other cohorts reported in the exercise in diabetes literature (BMI $\leq$ 36 kg/m^2) (Castaneda et al. 2002; Sigal et al. 2007). Castaneda et al. (2002) enrolled participants with a mean BMI of approximately 31 kg/m^2 and noted significant improvements in HbA1c secondary to 16 weeks of resistance training prescribed three times per week. Further, Sigal et al. (2007) prescribed thrice-weekly training in participants with a mean BMI of approximately 35 kg/m^2 and noted significant improvements in HbA1c with 22 weeks of isolated aerobic or resistance training. Importantly, both of these trials documented improvements in body composition in the exercising patients (Castaneda et al. 2002; Sigal et al. 2007).

It is possible that such an extreme level of obesity in our cohort may have delayed the effect of the interventions prescribed. Hence, greater frequency and duration of training may have reduced fat mass and/or increased fat-free mass and improved HbA1c. Exercise has been shown to improve diabetes markers via insulin-independent and insulin-dependent mechanisms of glucose uptake through the increased expression and activity of key regulatory intracellular proteins, and this effect is mediated by the augmentation of fat and fat-free mass (Rice et al. 1999). In the present study, no change in body weight, BMI, percent fat, or visceral adiposity were noted in either exercise group (Table 2). Accordingly, the diabetes markers remained unchanged.

However, despite these findings, our post hoc analyses revealed that participants who attended ≥75% of training sessions had significantly reduced waist circumference ($P < 0.001$), and tended to improve body weight and plasma insulin concentrations versus low adherers. Higher adherence was also associated with reduced CRP ($r = -0.492$; $P = 0.04$) and hence reduced the risk of cardiovascular morbidity and mortality (Pearson et al. 2003). These findings are important as they suggest that significant and clinically meaningful adaptations are possible in this cohort with higher doses of exercise.

The metabolic adaptations noted in our high adherers would likely precede other responses. Higher adherence, greater frequency of training, and longer training duration

may have reduced HbA1c. Future studies must direct greater attention toward fostering exercise adherence in this cohort by making interventions more practicable. Work and family obligations were cited as common reasons for missed sessions, perhaps due to the relatively younger age of our participants. Active interventions integrated into work hours and involving family members may increase exercise adherence and hence contribute to greater adaptation.

The microevolution of Polynesian people has been influenced by extreme physical activity for daily living, food procurement, dance and rituals, and inter-island travel, among other reasons (Price 1939). Polynesian people, particularly Māori, have historically been revered for their high levels of physical endurance and fitness (Price 1939). It could be hypothesized that Polynesian people, in general, are able to tolerate and respond more favorably to higher doses of exercise than other ethnic populations. RCTs stratifying by ethnicity, prescribing and comparing various exercise interventions, are required to investigate this hypothesis. Sigal et al. (2007) have shown that multimodal exercise interventions are more effective than isolated modalities. Such interventions remain to be investigated in Polynesian people.

Circulating adiponectin is inversely proportional to atherogenic and inflammatory markers, insulin resistance, and obesity (Kriketos et al. 2004), whereas CRP is directly proportional to these markers (Pearson et al. 2003). Exercise-induced increases in adiponectin and reductions in CRP are likely mediated via visceral, intramyocellular, and whole body fat loss. The lack of improvement of these cytokines is consistent with the lack of improvement of anthropometric markers. Further, the lack of change of adiponectin may have been influenced by the fact that this cytokine was within the desired range in both groups at baseline.

Resting SBP and DBP were significantly reduced in participants prescribed aerobic training. This is a clinically important finding as hypertension is the most common metabolic abnormality in Māori and Pacific Island people living in New Zealand (Simmons and Thompson 2004). Exercise-induced enhancement of blood pressure has been noted in a trial enrolling Indian patients with type 2 diabetes (Shenoy et al. 2009). The lack of change of hemodynamic indices in the resistance training group was expected as this group was normotensive at baseline, perhaps due to effective pharmacologic management.

Mean triglyceride levels in the aerobic training group at baseline were within normal limits ($\leq$1.7 mmol/L) at baseline, but significantly increased after 16 weeks of exercise training (Table 2). The reason for this increase is not clear, but may be attributed to dietary or medication changes and/or possible regression toward the mean. Other blood lipid measures tended to be within normal limits or only slightly elevated and did not change in response to intervention.

The main limitation of our study was the lack of a non-treatment control group. Appropriate cultural consultation may possibly enable the inclusion of a non-exercising control group within an RCT, provided that the overall objectives of the research are outlined a priori. Additional limitations of our study included the lack of documentation of key confounding variables including diet and physical activity. An attempt to document these measures via participant-administered questionnaires proved unsuccessful due to low participant compliance. Future trials may require the use of more objective measures, such as global positioning satellite and accelerometers, and researcher-administered dietary questionnaires.

In summary, resistance or aerobic training prescribed three sessions/week for 16 weeks was well tolerated, but did not improve HbA1c in Māori and Pacific Island people with type 2 diabetes and morbid obesity. Future investigations involving exercise regimens that are more practicable and which involve greater frequency and duration of training may be required to induce significant and clinically meaningful adaptations in this unique diabetes population.

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Conflicts of interest None.

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