Original Article

Implementation of a successful lifestyle intervention programme for New Zealand Maori to reduce the risk of type 2 diabetes and cardiovascular disease

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Lifestyle programmes have been shown to reduce the risk of type 2 diabetes in European populations. The participation of Maori in many mainstream health programmes is poor. This study evaluates a lifestyle intervention programme which is acceptable to Maori and which has objective outcome measures to determine the effectiveness of the programme. Thirty six Maori men and women were recruited for a 4 month programme involving modification of diet and exercise. Insulin sensitivity was measured using a euglycaemic insulin clamp, body composition using dual-energy-absorptiometry and fitness using a submaximal exercise test. Secondary outcome measures included anthropometry, blood pressure, fasting glucose and insulin levels, and lipid profiles. There was a 24% improvement in insulin sensitivity (from 5.1 to 6.3 G/mIU/L, *P*=0.03, *N*=29). This was associated with a reduction of 3.1 kg in weight (95%CI -4 to -2) and a reduction of 7mmHg in systolic blood pressure (95%CI -13, -1). This approach successfully reduced risk for type 2 diabetes and cardiovascular disease in New Zealand Maori in the short term.

Key Words: diabetes prevention, lifestyle intervention, 2 diabetes, New Zealand Maori, prevention programmes .

Introduction

The frequency of type 2 diabetes is believed to be increasing at an alarming rate in New Zealand and other countries. This is especially so in Maori and Pacific Island groups in New Zealand and amongst other indigenous people throughout the world. By the year 2006, a 13% increase in the total population of New Zealand is predicted, but a 30% increase in the total number of people with diabetes is expected, (47% increase for Maori and a 70% increase for Pacific Islanders).[1] The consequences of developing diabetes are enormous with vascular complications accounting for much of the morbidity and premature mortality associated with the disease. Diabetes is the leading cause of blindness, kidney failure and lower limb amputations in adult New Zealanders. It is a major risk factor for myocardial infarction, heart failure, strokes, peripheral neuropathy and early death. Maori suffer disproportionately from the complications of diabetes for several reasons including a longer duration of diabetes and reduced access to appropriate health care.

Lifestyle modification reduces the risk of progression from impaired glucose tolerance (IGT) to type 2 diabetes and significantly reduces cardiovascular risk factors.[2,3] The American Diabetes Prevention Programme included several minority groups amongst them a Samoan population living in California. The lifestyle programme involving intensive support was associated with risk reduction similar to that seen amongst those of European descent.[2] The challenge in New Zealand is to develop a lifestyle intervention programme which is acceptable to Maori and which has been convincingly proven to reduce the risk of developing diabetes. This study is the first of its kind to address both these issues in the indigenous people of New Zealand. The primary outcome measure is insulin sensitivity, as resistance to the action of insulin is a major risk factor for the development of type 2 diabetes and cardiovascular disease.

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Methods

Our original intention was to carry out a randomised study with participants allocated to modest or intensive lifestyle modification programmes or to a control group. However, this approach proved impractical, since information was shared between the two intervention groups. Furthermore, a control group was not acceptable as participants knew they were at risk of diabetes and believed they should be offered appropriate advice. There was therefore no alternative but to study participants before and after intervention. Information was available from a parallel study carried out in New Zealanders of European extraction which provided an indication of extent of change in variables of interest over time.[4]

Instead of the conventional approach of health professionals recruiting participants, a novel 'snow balling' technique was used for recruitment, which is described in the preceding paper. Those with diagnosed diabetes were excluded, but it was deemed culturally inappropriate to exclude participants found to have impaired glucose metabolism after they had volunteered. The programme was implemented over a 4 month period, since it was expected that the variables of interest including body composition, measures of insulin sensitivity and cardiovascular risk factors would have changed appreciably over this time frame if the programme was successful.

Dietary intervention

The details of the dietary programme are described in the preceding paper. The diets were individually prescribed and based on each participant's usual intake or an energy level designed to lead to gradual and sustained weight reduction. Compliance was assessed by a 4-day diet record at baseline prior to the intervention and after at 4 months, and a daily record sheet, where type and amount of recommended foods was recorded. Diet records were analysed using Diet Cruncher for Macintosh (Marshall-Seeley, Dunedin, New Zealnd, 1999, version 1.1.0) which used food composition data from the New Zealand Institute for Crop and Food Research Ltd.[5] Some foods were supplied free of charge (cereals, low fat spread and canola oil), and all dietary information, including lists of suitable foods, sample eating plans, foods to be avoided, cooking and preparation advice, and recipes were provided.

Exercise intervention

The programme details are outlined in the preceding paper. At baseline, assessment of recent participation in physical activity was based on the Life in New Zealand (LINZ) validated questionnaire,[6] and during the study the type and duration of activity was recorded on a daily record sheet.

Outcome measures

The procedures for measuring weight, waist and hip circumference, blood pressure, fasting insulin, glucose, and lipids have been described previously.[7] Methods for measurement of body composition, insulin sensitivity and the submaximal exercise test have been described for a parallel study performed in non Maori.[4] For the measures of insulin sensitivity we have expressed the glucose infused (G, units in mg/kg/min) for total body weight (Gbw) and for fat free mass (Gffm) using Humes formula.[8] Gbw and Gffm are divided by the average insulin level during the final 60 minutes of the test (Gbw/i, Gffm/i), in G/mU/L.

A one mile walk test was conducted at baseline and monthly during the 4 month intervention to provide feedback and motivation to participants. Prior to the test, participants were instructed to complete four laps of a standard athletics track walking as quickly as possible. The total time taken to complete the mile and an average heart rate, measured at the quarter, half, three-quarter and mile marks during the test, were recorded.

Clinical visits

At the time of the euglycaemic insulin clamp, anthropometry and blood pressure measurements were made and fasting blood samples taken for lipid measurements. Within one week participants had a DXA scan and an exercise test. All these measures constituted baseline data. Intervention was commenced as soon as all baseline measures were collected. Participants were seen by the researchers weekly for a weight measurement and a brief dietary and exercise assessment. If participants did not attend, they were contacted by phone and progress was discussed and a further appointment was made. At one monthly intervals, participants had the following measurements: weight, waist and hip measurements, blood pressure, a fasting blood test for glucose, insulin and lipid profile and a one mile walk test. Those smokers who wished to stop were encouraged to do so either before starting the programme or after the 4 month period, given the difficulty of addressing smoking cessation, weight, diet and exercise issues all at once.

Statistical methods

Pre and post intervention measures were compared with a paired t test. Differences and 95% confidence intervals are presented.

Results

Thirty six participants were recruited, 28 female and 8 male (mean age = 41.3 yr; range 24-60). Of the 36 participants none were known to have diabetes, but 5 (13%) were found to meet the criteria for diabetes (fasting glucose >7.0 mmol/L) and 4 (11%) had impaired fasting glucose (fasting glucose 6.1-6.9 mmol/L). The majority had at least one member of their family with diagnosed type 2 diabetes. Fourteen were smokers (3 male, 11 female), and continued to smoke over the 4 month intervention. Five female participants dropped out due to other commitments, health or family issues or they were not yet ready to make a lifestyle change. This group of 31 Maori probably represents those at higher risk of type 2 diabetes compared to the general Maori community given that they were heavier (mean BMI 34, SD 6.1) than those of a similar age range measured in the 1997 National Nutrition Survey (mean BMI 29 or 31 depending on age).[9]

Not all of the 31, who completed the intervention were able to have all of the outcome measures performed. This was due to a variety of technical reasons, for example poor venous access for blood measures during the clamp or weight exceeding the 120kg limit for the DXA scanner. Complete measures both before and after intervention were obtained for the following number of participants: 29 (81%) for the euglycaemic insulin clamp, 26 (72%) for DXA, 31 (86%) for fasting blood tests, 30 (83%) for weight and BMI, 29 (81%) for waist circumference and blood pressure, 19 (53%) diet records, 23 (64%) for predicted VO_2max, and 28 (78%) for stage and heart rate measurements during the exercise test.

Insulin sensitivity improved significantly (Table 1). This occurred in parallel with reductions in weight, waist circumference, BMI, and systolic blood pressure (Table 2), total and truncal fat (Table 1), fasting glucose and insulin (Table 3). Reported intake of carbohydrate and dietary fibre increased at the expense of total fat, saturated fat, monounsaturated fat and cholesterol (Table 4). Participants managed a higher work load for a given heart rate post intervention, but measures of aerobic fitness were unchanged (Table 5).

Discussion

Our initial intention to carry out a randomised study proved impossible, but it has been possible to obtain helpful information regarding the extent to which lifestyle modification can influence insulin sensitivity and selected indicators of cardiovascular risk in Maori. Data from the control group of a randomised trial carried out in a predominantly European group suggested that there is very little regression to the mean in the variables of interest without intervention.[4] For example, the insulin sensitivity index for the control group at baseline and four months was 2.6 and 3.1 G/mIU/L respectively. A 20% improvement in insulin sensitivity undoubtedly represents a significant risk reduction for the development of type 2 diabetes.[10] It is encouraging that this occurred in an intervention programme acceptable to this group of high risk people in association with a modest weight reduction of 3.1kilograms over the four month period. It is noteworthy that the weight loss was accounted for entirely by a reduction in fat mass with no loss in lean body mass. The reduction in systolic blood pressure is substantial, similar to that observed in the formal trial undertaken in non Maori and of an order of magnitude comparable with that achieved by some antihypertensive medication, and likely to be of considerable clinical relevance. Not surprisingly insulin and glucose levels fell, although the reduction in triglyceride levels as well as total and low density lipoprotein cholesterol levels were less marked than expected. Disappointingly, no significant change in aerobic fitness was observed. However, the number of participants who could have this measured was reduced and perhaps too small for this analysis to be meaningful. It is likely an increase in aerobic and muscular fitness was achieved based on attendance and participation in supervised exercise sessions. Overall the improvement in risk factors for type 2 diabetes and cardiovascular disease is encouraging in this pilot programme, at least in the short term. However, a high level of support was required to achieve this benefit. The level of support for maintenance of these lifestyle issues is also likely to be high and requires evaluation now that the intervention programme has been shown to be successful over a four month period. A maintenance programme is now in place.

Table 1. Body composition (DXA) and insulin sensitivity (insulin clamp) at baseline and after 4 months intervention

Variable	*N*	Baseline Mean (±SD)	Final Mean (±SD)	Difference (95% CI of difference)	*P* value for difference
Gbw/i	29	3.1 (1.9)	3.9 (2.3)	0.8 (0.1, 1.4)	0.02
Gffm/i	29	5.1 (2.9)	6.3 (3.5)	1.2 (0.2, 2.2)	0.03
Total fat(kg)	26	37.8 (11.5)	35.8 (11.4)	-2.0 (-2.8, -1.2)	0.000
Truncal fat(kg)	26	20.5 (6.9)	19.4 (7.0)	-1.1 (-1.7, -0.5)	0.001
Lean mass(kg)	26	50.9 (10.5)	50.8 (10.4)	-0.1 (-0.6, 0.4)	0.64

SD = standard deviation Gbw/i = glucose infused for body weight/average plasma insulin (G/mIU/L) Gffm/i = glucose infused for fat free mass/average plasma insulin (G/mIU/L)

Table 2. Clinical measurements at baseline and after 4 months intervention (*N*=31)

Variable	Baseline Mean (±SD)	Final Mean (±SD)	Difference (95% CI of difference)	*P* value for difference
Weight(kg)	97.8 (21.6)	94.8 (20.9)	-3.1 (-4.0, -2.1)	0.000
Waist(cm)	108.0 (16)	101.0 (15)	-7 (-9, -5)	0.000
BMI(kg/m^2)	34.2 (6.1)	33.1 (6.0)	-1.1 (-1.4, -0.7)	0.000
SBP(mm/Hg)	137.0 (21)	131.0 (17)	-7 (-13, -1)	0.04
DBP(mm/Hg)	85.0 (10)	84.0 (80)	-1 (-4, -2)	0.50

BMI = body mass index, SBP = systolic blood pressure, DBP = diastolic blood pressure, SD = standard deviation.

Table 3. Fasting glucose, insulin and lipids at baseline and after 4 months intervention (*N*= 31)

Variable	Baseline Mean (±SD)	Final Mean (±SD)	Difference (95% CI of difference)	*P* value for difference
Glucose (mmol/L)	5.7 (1.2)	5.2 (0.9)	-0.5 (-0.8, -0.2)	0.001
Insulin (mIU/L))	24.4 (15.4)	19.3 (13.9)	-5.1 (-8.4, -1.7)	0.004
Chol (mmol/L)	5.8 (1.4)	5.4 (1.0)	-0.4 (-0.8, 0.1)	0.09
HDL (mmol/L)	1.05 (0.28)	1.01 (0.25)	-0.04 (-0.14, 0.06)	0.46
TAG (mmol/L)	1.71 (0.69)	1.49 (0.68)	-0.22 (-0.45, 0.02)	0.07
LDL (mmol/L)	3.9 (1.2)	3.6 (1.0)	-0.3 (-0.7, 0.0)	0.08

SD = standard deviation, Chol = total cholesterol, HDL = high density lipoprotein, TAG = triglyceride, LDL = low density lipoprotein.

Table 4. Energy and macronutrient composition of diets at baseline and after 4 months intervention (*N*=19)

Variable	Dietary Targets	Baseline Mean (±SD)	Final Mean (±SD)	Difference (95% CI of difference)	*P* value for difference
Energy(kJ)	-	7965 (1739)	7000 (1610)	-965 (-1915, -15)	0.05
Total fat (% E)[#]	32	39 (5)	28 (8)	-11 (-15, -6)	0.0001
SFA (% E)	11	17 (2)	10 (4)	-7 (-9, -4)	0.0001
PUFA (% E)	7	5 (2)	5 (2)	0 (-1, 1)	0.96
MUFA (% E)	14	14 (3)	10 (3)	-3 (-5, -1)	0.004
Carbohydrate (% E)	50	40 (7)	50 (8)	10 (5, 14)	0.000
Protein (% E)	18	16 (2)	17 (3)	1 (-0.5, 2)	0.17
Alcohol (% E)	-	2 (3)	2 (5)	0 (-2, 2)	0.94
Cholesterol (mg/24h)	200	293 (81)	216 (110)	-76 (-142, -11)	0.03
Fibre (g/24h)	25	17(5)	25(4)	8 (5,10)	0.000

SFA = saturated fats, MUFA = monounsaturated fat, PUFA = polyunsaturated fat, Chol = cholesterol, SD = standard deviation. [#]%E = % of total energy

Table 5. Measures of aerobic fitness at baseline and after 4 months intervention

Variable	*N*	Baseline Mean (±SD)	Final Mean (±SD)	Difference (95% CI of difference)	*P* value for difference
Predicted VO_2max[#]	23	32.2 (7.8)	33.1(7.7)	0.9 (-0.7, 2.6)	0.25
†Stage	28	3.7 (0.9)	4.1 (0.8)	0.4 (0.2, 0.7)	0.001
‡Heart rate(bpm)	28	138 (12)	140 (10)	3 (-2, 7)	0.24

SD = standard deviation; [#](ml/min/kg); †stage using the modified Bruce protocol; ‡heart rate given for same stage pre and post intervention

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