Effectiveness of a community-directed 'healthy lifestyle' program in a remote Australian Aboriginal community

Abstract

Objective: To assess the sustainability and effectiveness of a community-directed program for primary and secondary prevention of obesity, diabetes and cardiovascular disease in an Aboriginal community in north-west Western Australia.
Method: Evaluation of health outcomes (body mass index, glucose tolerance, and plasma insulin and triglyceride concentrations) in a cohort of high-risk individuals (n=49, followed over two years) and cross-sectional community samples (n=200 at baseline, 185 at two-year and 132 at four-year follow-ups), process (interventions and their implementation) and impact (diet and exercise behaviour).
Results: For the high-risk cohort, involvement in diet and/or exercise strategies was associated with protection from increases in plasma glucose and triglycerides seen in a comparison group; however, sustained weight loss was not achieved. At the community level, significant reductions were observed in fasting insulin concentration but no change in prevalence of diabetes, overweight or obesity. Weight gain remained a problem among younger people. Sustainable improvements were observed for dietary intake and level of physical activity. These changes were related to supportive policies implemented by the community council and store management.
Conclusions: Community control and ownership enabled embedding and sustainability of program, in association with social environmental policy changes and long-term improvements in important risk factors for chronic disease.
Implications: Developmental initiatives facilitating planning, implementation and ownership of interventions by community members and organisations can be a feasible and effective way to achieve sustainable improvements in health behaviours and selected health outcomes among Aboriginal people.

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Mortality due to diabetes is much greater for Aboriginal men and women in Western Australia than for their non-Aboriginal counterparts.[1] Similarly, and despite a trend to decreased mortality from cardiovascular diseases (CVD) in Australia generally,[2] premature mortality from coronary heart disease (CHD) and stroke among Aboriginal people remains far greater than for the general Australian population.[3,4]

There are numerous social and biological factors associated with epidemic rates of 'lifestyle' diseases. Adverse social circumstances are a risk factor for CVD and diabetes, both in and of themselves and through their association with other risk factors such as obesity and insulin resistance.[5-11] Weight loss has unequivocal benefits for diabetic persons[12-14] and the acute and long-term benefits of moderate exercise for the treatment and prevention of diabetes and CVD are equally clear.[15-17] Diet is also important in the aetiology of diabetes and CVD, with a high intake of saturated fat and low intakes of fresh fruit and vegetables increasing the risk of these conditions.[18] In a group of diabetic Australian Aboriginal people, temporary reversion to a traditional hunter-gatherer lifestyle resulted in marked improvements in all of the metabolic abnormalities of diabetes and CHD risk.[19] These changes were associated with weight loss in the context of increased physical activity and the consumption of a low-fat diet.

Translating knowledge of risk factor relations into effective primary and secondary prevention strategies in high-risk communities remains a challenge. Health promotion campaigns aimed at the general Australian population may be less than effective in Indigenous communities due to limited penetration (particularly in remote areas with limited communications) and/or inappropriate language and messages. Public health initiatives arising from and directed by local communities offer a potentially more effective means of reducing disease risk

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within the limitations imposed by wider economic and social conditions.[20-22]

This report examines trends in diet and physical activity behaviours, body weight, glucose tolerance and fasting insulin and triglyceride concentration for a remote Australian Aboriginal community responsible for implementing and directing a lifestyle improvement program. We consider a cohort of overweight and diabetic people tracked over two years from the outset of the intervention program. Trends in the broader community were assessed over four years by way of cross-sectional community surveys. We also report evaluation results for community-level process and impact assessment.

Methodology

Setting

Looma Aboriginal community, in the remote Kimberley region of north-west Australia, was established in 1973. By 1997 the population had grown to more than 500 with facilities including a store, school, health centre, council offices and a Home and Community Care centre. Many of the residents came from surrounding cattle stations in the Derby and Fitzroy Crossing areas and some had grown up living a traditional hunter-gatherer lifestyle in the desert. Concerned about the apparently high morbidity and mortality from diabetes, the community initiated a lifestyle intervention program in late 1993.

Program context

The Looma Diabetes Programme, later renamed Looma Healthy Lifestyle, began in late 1993 after two of the authors (GAW and KOD) were approached by community members aware of an intervention program in another Kimberley community.[23] A first step was to secure funds to employ a Diabetes Nurse Educator (with skills appropriate to community-based work) to assist Looma community members in the design and implementation of appropriate and potentially sustainable physical activity and dietary modifications. Later on, several Aboriginal Health Workers were employed, with two working part-time and eventually becoming responsible for the day-to-day running of the program.

The community identified several problems likely to increase people's risk of diabetes:

- lack of knowledge about diabetes and how it can be prevented and managed by diet and physical activity;
- lack of affordable, healthy food choices; and
- a lack of infrastructure to encourage regular physical activity.

Program features

(i) Intervention among high-risk overweight and diabetic people: Interventions included formal and informal education sessions, regular physical activity groups and dietary changes such as cutting fat from meat before cooking, reducing intake of refined carbohydrate (sugar *per se* and carbonated beverages) and increasing consumption of fresh vegetables and fruit. Cooking classes and store tours were organised to help people identify healthy food choices. Physical activity was promoted with regular hunting trips, participation in sport (basketball or football, 2-3 sessions per week) and regular walking groups (3-4 times per week, one hour per session). Several family groups began regular walking independently of the program. Informal education sessions about diabetes were an important component of the program. Weekly body weight and blood glucose checks were available for those who requested them.[24]

(ii) Intervention in the wider community: After two years, a growing awareness in the wider community of the importance of diet and physical activity for preventing chronic disease led the community to rename the program Looma Healthy Lifestyle.[25] There was a strong emphasis on dissemination of messages about diet and physical activity to family members by those persons taking part in the high-risk intervention program. Health promotion activities, arising from the initial intervention and in which all community members were invited to participate, were undertaken to initiate normative change and enabling conditions.[23,24] Details of initiatives are given below.

Throughout the course of the intervention, technical advice, data analysis and feedback of results and advocacy to health and funding bodies was provided by researchers.

Program evaluation

(i) Intervention among high-risk overweight and diabetic people: Ninety-six volunteers were screened at two or more six-month intervals over 24 months, starting in late 1993. Of these volunteers, full sets of follow-up data were obtained for 49 persons. Measures included body weight and height, 75 g oral glucose tolerance test (OGTT) (exempting those with unequivocal fasting hyperglycaemia), fasting plasma triglyceride and insulin concentrations, and diet and physical activity questionnaires. Of persons for whom follow-up was incomplete (n=47), reasons for non-participation in screening were: absent from community (n=27), mourning (n=1), illness (n=1), death (n=2) or declining to attend for unspecified reasons (n=11). Missing data accounted for five further persons. Only persons followed-up completely were included in longitudinal analyses. Relative to persons with complete datasets, non-participants and persons with missing data were leaner at baseline [(mean ± SD) BMI 26.6 ± 0.7 kg/m^2 versus 28.6 ± 0.6 kg/m^2; $p<0.05$] but similar in terms of age, gender, baseline biochemical variables and diet and physical activity habits.

The primary evaluation question in cohort analyses was change in outcomes over 24 months. A secondary research question was differential change between persons classified according to diet and physical activity habits at the first follow-up. Not all persons followed-up were active participants in interventions; persons participating in diet or physical activity interventions (the 'intervention group') were compared with persons not participating in diet or physical activity interventions (the 'non-intervention group'). The latter is not a true 'control' group in the sense that the groups were self-selected. However, in the absence of comparable longitudinal data from another community not involved in an intervention program, this classification was used to determine the efficacy of intervention strategies in the high-risk cohort.

(ii) Intervention in the wider community: Community trends in risk factors following program implementation were assessed for three cross-sectional, community surveys conducted in (1) November 1993-January 1994 (baseline), (2) February 1996 (two-year follow-up) and (3) November 1997 (four-year follow-up). Participation in these surveys was voluntary. Variables examined were as for longitudinal cohorts. Evaluation of process (that is, intervention initiatives, council policy supporting these initiatives, etc) and impact (self-reported diet and physical activity changes) at the community level was also undertaken. We did not collect comparison data from a community not implementing a healthy lifestyle intervention program.

Methods and diagnostic criteria

Body weight was measured to 0.1 kg and height to 1 cm. Blood samples were collected in the morning after an overnight fast, before administration of a 75 g glucose load, for analysis of plasma glucose, insulin and triglyceride concentrations. Glucose was assayed at a regional hospital laboratory (PathCentre Derby; coefficient of variation [CV]=2%) and other samples transported to Melbourne for analysis. The inter-assay CV for triglycerides was 6%. Insulin was determined by radio-immunoassay with a specific antibody (Linco Research Inc., St Louis, US; inter-assay CV <10% at lower concentrations, and <14% at the upper end of the working range). Participants were classified using World Health Organization criteria as either normoglycaemic or as having impaired glucose tolerance (IGT) or diabetes. Physical activity and dietary habits were assessed by questionnaire. Physical activity was coded as minimal, moderate (e.g. regular walking for 1 hour 3-4 times per week) or vigorous (e.g. regular sport or work on cattle stations). Dietary questions addressed reductions in fat (yes/no) and sugar intake (yes/no).

The project was approved by Deakin University Ethics Committee, the institution responsible at the time of study. Participants gave written, informed consent to screening procedures.

Statistical analyses

For longitudinal analyses, change was assessed using repeated measures ANOVA (SPSS Inc.). Univariate F statistics for changes in outcomes and outcome by intervention/non-intervention group interaction terms are reported. The Greenhouse-Geisser epsilon adjustment was made when the sphericity assumption was not upheld. Given low numbers and limited statistical power, interactions were considered statistically significant at $p<0.1$.

For cross-sectional analyses of community surveys, trends in continuous variables were tested by ANOVA. As response rates differed for persons aged 15–34 years and those aged 35+ years, separate analyses were conducted for the two age groups. Time (year of survey) and gender were taken as fixed effects and age as a covariate. The survey samples are not truly cross-sectional as later surveys include some persons screened on more than one occasion (61% of the survey sample on average). This is unavoidable for studies of small, highly motivated communities over a short period of time. The data were nevertheless treated as cross-sectional observations, a conservative assumption given that greater power would be obtained by taking into account the inter-correlation for repeated measures. For persons in the older age group, response rates were high for all surveys and the sample was considered representative of the older population of the community. Among younger persons, response rates fell in later surveys, raising the possibility of selection bias in that those persons returning for repeat screenings may have been more, or less, healthy than the younger population as a whole. For this reason, a dummy variable was coded for each subject to indicate whether that individual was screened only once or had returned for one or more repeat screenings. Second order interactions were included in models, but omitted if non-significant. Changes in proportions with time were tested using Cochran's Q test or the Mantel-Haenszel age-weighted chi-square test. Triglyceride and insulin data were log-transformed prior to statistical analyses.

Results

Longitudinal evaluation of initial program, 0–24 months

Baseline characteristics of the 49 persons screened at each six-month interval are shown in Table 1. The majority (82%) of persons were overweight or obese by standard criteria (BMI >25 and ≤30 kg/m^2 or BMI >30 kg/m^2 respectively). The intervention and non-intervention groups (see above) were similar with respect to age, gender, BMI, prevalence of abnormal glucose tolerance and baseline physical activity and dietary improvement habits (see Table 1).

Table 1: Baseline characteristics of participants screened at baseline, 6, 12, 18 and 24 months.

	Intervention group	Non-intervention group	*p*
n	32	17	
Men:women	25:75	41:59	0.242
BMI (kg/m^2)	28.5 ± 0.8[a]	28.9 ± 1.1[a]	0.781
Age (years)	49 ± 3[a]	43 ± 4[a]	0.197
IGT (%)	31	24	0.569
Diabetes (%)	44	47	0.825
Attempting dietary change (%)			
None	76	100	0.053[b]
Sugar reduction	21	0	
Fat reduction	3	0	
Sugar & fat reduction	0	0	
Physical activity (%)			
Sedentary	72	76	0.862*
Moderate	24	12	
Vigorous	4	12	

Notes:
(a) Continuous data are shown as mean ± sem (t-test), categorical data as percentages (chi-square test).
(b) none vs. any dietary/physical activity strategy.

Body mass index: For the cohort as a whole, mean BMI underwent a significant change over the first 24 months of intervention (Figure 1A). The change reflected primarily decreases at six months (p<0.001) and 12 months (p=0.060) relative to baseline. The interaction between change in BMI and intervention/non-intervention group approached significance (p=0.101). The change in BMI from baseline to six months was larger for the intervention group than for the non-intervention group (p=0.012). This difference was no longer apparent by 12 months.

Metabolic control: Fasting plasma glucose concentration changed significantly across groups over the first 24 months of the program (Figure 1B) with a mean decrease of 0.9 mmol/L at six months compared with baseline (p=0.021). By 12 months, fasting glucose returned to baseline values. The interaction between change in plasma glucose and intervention/non-intervention group was not statistically significant (p=0.132). In those persons who received an OGTT at zero, six and 12 months, there were significant changes in 2-h plasma glucose (Figure 1C) with a mean fall of 1.6 mmol/L at six months compared with baseline (p=0.010) and a return to baseline values by 12 months. The interaction between changes in 2-h glucose and intervention/non-intervention group was not statistically significant (p=0.154).

Fasting insulin concentration varied significantly across groups over the first 24 months of the program (Figure 1D), with a lower mean concentration at 18 months compared with baseline (p=0.004). The interaction between change in fasting insulin and

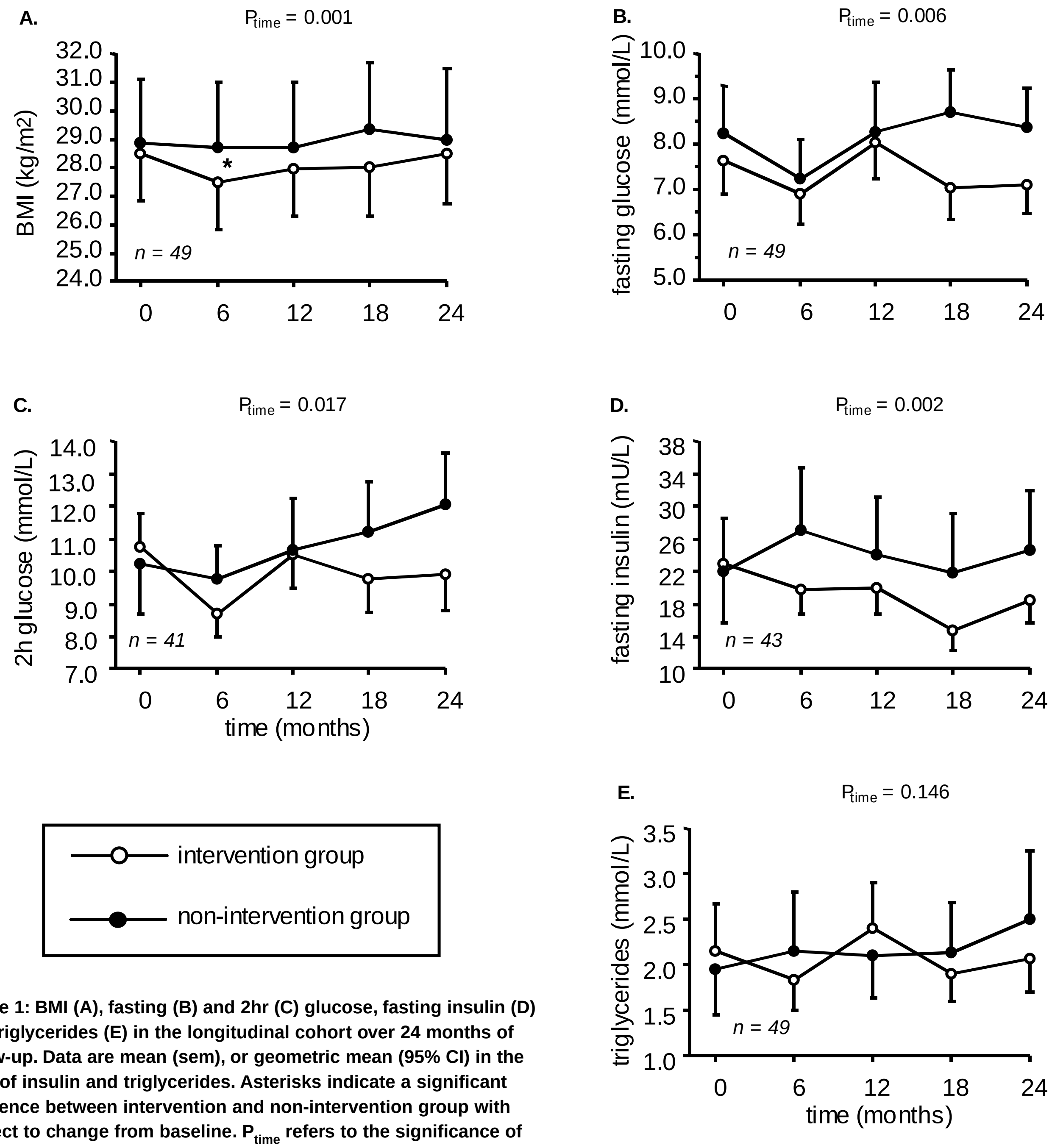

Figure 1: BMI (A), fasting (B) and 2hr (C) glucose, fasting insulin (D) and triglycerides (E) in the longitudinal cohort over 24 months of follow-up. Data are mean (sem), or geometric mean (95% CI) in the case of insulin and triglycerides. Asterisks indicate a significant difference between intervention and non-intervention group with respect to change from baseline. P_{time} refers to the significance of change over time for the whole cohort.

intervention/non-intervention group approached statistical significance (p=0.103), reflecting differential change in insulin between the groups from baseline to 18 months (p=0.058) and 24 months (p=0.061). Fasting plasma triglyceride concentration did not vary with time (Figure 1E) nor was there a differential change over time between the two groups (p for interaction=0.158); however, the data suggest a seasonal variation in the intervention group consistent with the trends in insulin.

Further analysis was conducted for a contrast of the baseline and 24-month time points. Change in 2-h plasma glucose from baseline to 24 months was significantly different between groups (mean change [95% CI]=-0.9 [-1.9–0.2] mmol/L for the intervention group, 1.5 [-0.8–3.7] mmol/L for the non-intervention group; p=0.030). Similarly, change in triglyceride from baseline to 24 months was significantly different between two groups (mean change [95% CI]=-0.27 [-0.73–0.20] mmol/L for the intervention group, 0.85 [-0.22–1.91] mmol/L for the non-intervention group; p=0.016). Changes in other variables from baseline to 24 months were not statistically different between intervention and non-intervention groups.

Community-wide intervention initiatives and trends in risk factors, 0-4 years

Process evaluation

Table 2 lists community initiatives undertaken as part of Looma Healthy Lifestyle. In 1995, a community member was appointed to manage the community store with a mandate to increase sales of fresh vegetables and fruit and to reduce sales of high-fat and high-sugar items. Art competitions and sporting festivals based on the theme 'Fitness Fights Diabetes' were conducted several times each year and attracted wide community participation. Several sporting teams were reactivated and included a number of older community members, and the council appointed a sport and recreation officer. The council also provided support in the form of an office for program workers to use as a base, and allowed the use of council facilities for screening procedures. Health education classes conducted by Aboriginal Health Workers were held in the community school.

Impact evaluation

Figure 2 shows proportions of persons undertaking dietary and physical activity changes. The data pertain only to the 35+ year age group, as baseline data were sparse for younger persons. Prior

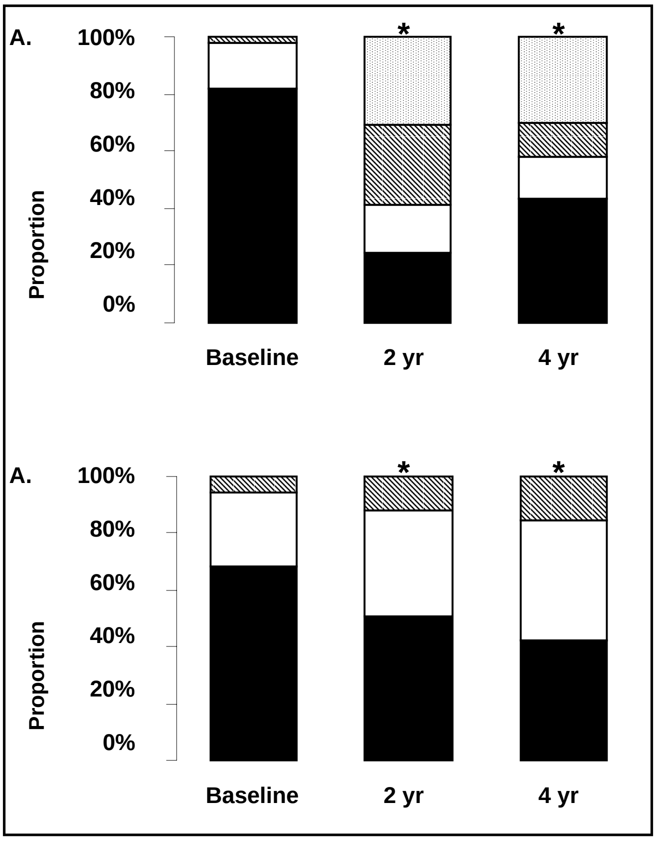

Figure 2: Self-reported dietary and physical activity habits among older persons (35+ years) in the three surveys. (A) Proportions of persons reducing their intake of sugar (white portion of columns), fat (hatched), both sugar and fat (stippled) or neither (black). (B) Proportions of persons engaging in vigorous (hatched portion of columns), moderate (white) or no (black) physical activity. Asterisks indicate a significant trend to changing proportions (none vs. any intervention strategy).

Table 2: Community initiatives activated following commencement of Looma Healthy Lifestyle.

Level	Initiative
Looma Diabetes Program/ Looma Healthy Lifestyle	Appoint community members as diabetes workers. Regular hunting trips for program participants. Nutrition education for diabetic people. Healthy cooking classes. Store tours to identify healthy food choices.
Community Council	Appointment of community member as store manager with mandate to improve food quality. Non-smoking policy in public buildings. Provision of vehicle for Looma Diabetes Program. Appointment of sport and recreation officer. Health promotion (sporting/art competitions).
Community store	Improved quality and quantity of fresh produce. Availability of wholemeal bread and flour. Replacement of butter with margarine.
Community members	Regular family walking groups. Activation of sporting teams.

to intervention, few persons reported attempting dietary strategies to reduce their intake of sugar and/or fat (Figure 2A). At the two-year follow-up, there were significantly fewer persons reporting no attempts to lower their intake of fat and/or sugar (χ^2=42.0, p<0.001) and this trend was still apparent at four years (χ^2=17.1, p<0.001). Before intervention, more than 60% of persons reported that they undertook no form of regular physical activity (Figure 2B). The proportion of sedentary individuals was significantly lower at two years (χ^2=4.0, p=0.046) and four years (χ^2=8.1, p=0.004) compared with baseline.

Outcome evaluation

Demographics and response rates: Anthropometric and biochemical outcome measures were assessed in three sequential cross-sectional community risk factor surveys. Response rates among younger people (15-34 years) for the baseline, two-year and four-year surveys were 74%, 61% and 43% respectively. Response rates among older people (35+ years) for the baseline, two-year and four-year surveys were 100%, 90% and 80% respectively. Reasons for non-participation at the two-year follow-up were absence from the community at the time of screening (28%), old age/frailty (7%) or declining to attend for unspecified reasons (65%). Reasons for non-participation at the four-year follow-up were absence from the community at the time of screening (45%), old age/frailty (2%) or declining to attend for unspecified reasons (53%).

While response rates for older community members were consistently high, response rates fell for younger people in later surveys. Nevertheless, survey samples for the 15-34 year age group are arguably representative of this age range since, for the final survey, the proportion of the sample previously screened (54%) was similar to the proportion of non-responders previously screened (55%). This suggests no apparent selection bias for or against people screened previously. Furthermore, results for persons screened multiple times were not significantly different from those for persons screened only once for any of the outcome variables, except for mean BMI among young women (see below).

Body mass index: Among young persons 15-34 years, mean BMI increased by 1.6 kg/m^2 over the four-year follow-up period (p<0.05; Table 3). This is equivalent to an approximate weight gain of 1 kg per person per year. The magnitude of this gain did not differ between genders (p=0.631). Young women had higher BMI than young men (p<0.001). Among young women, mean BMI was higher for those screened multiple times compared with those screened only once (p<0.05) but the change in BMI with time was similar (p=0.560). Mean BMI did not change among persons 35 years and older (see Table 3).

Metabolic control: Mean fasting plasma glucose concentration did not vary with time among age groups (see Table 3). Mean glucose was greater for young women than younger men (p=0.015). The prevalence (age-adjusted using the baseline survey sample as the reference population) of IGT was 17.7%, 21.1% and 14.8% at baseline, two years and four years respectively (χ^2 for linear trend=0.265, p=0.607). The age-adjusted prevalence of diabetes was 24.1%, 21.8% and 24.7% at baseline, two years and four years respectively (χ^2 for linear trend = 0.003, p=0.955).

Among persons aged 15-34 years, mean fasting insulin concentration tended to decrease with time but this effect was not statistically significant (see Table 3). Younger women had higher fasting insulin concentrations than younger men (p<0.001). Among older persons, mean fasting insulin concentration decreased with time (p<0.05; Table 3); gender differences were not apparent.

Table 3: Trends in BMI and plasma glucose, insulin and triglycerides over time: cross-sectional community surveys.

	15–34 years		P_{year}	35 year+		P_{year}
n	men	women		men	women	
baseline	51	51		47	50	
2 year	42	52		40	47	
4 year	28	25		29	43	
BMI, kg/m^2						
Baseline	22.6 (21.7-23.6)		0.028	26.4 (25.5-27.4)		0.909
2 year	24.3 (23.3-25.3)			26.1 (25.1-27.1)		
4 year	24.8 (23.6-26.1)			26.5 (25.4-27.6)		
Fasting glucose (mmol/L)						
Baseline	4.9 (4.6-5.3)		0.272	7.5 (6.7-8.3)		0.469
2 year	5.3 (4.9-5.7)			7.8 (7.0-8.6)		
4 year	5.2 (4.7-5.7)			7.2 (6.3-8.1)		
Fasting insulin (μU/mL)						
Baseline	17 (15-20)		0.174	21 (18-23)		0.018
2 year	17 (15-19)			17 (15-19)		
4 year	13 (11-16)			16 (15-19)		
Fasting triglycerides (mmol/L)						
Baseline	1.4 (1.3-1.6)		0.929	2.0 (1.8-2.3)		0.941
2 year	1.5 (1.3-1.7)			2.1 (1.9-2.3)		
4 year	1.4 (1.3-1.6)			2.1 (1.8-2.3)		

Note:
Estimated marginal means (95% confidence interval), adjusted for age and gender.

Fasting plasma triglyceride concentration did not change during the intervention period in either age group (see Table 3).

Discussion

Despite clear evidence that healthy diet and regular physical activity are associated with reductions in the risk and severity of diabetes and CVD, examples of community-based programs to bring about these changes are rare. The present report describes a community-directed healthy lifestyle program and associated changes in body weight and biochemical measures of glucose metabolism in a remote setting. The program developed, over time and at the direction of the community, from one focusing on body weight and metabolic control in overweight and diabetic people to a more holistic, community-wide approach to management and prevention of chronic disease.

Initially, a group of overweight and diabetic persons began a diet and physical activity program aimed at achieving sustained weight loss and improved metabolic control. Although there was an initial reduction in BMI among persons implementing diet and/or physical activity strategies, sustained weight loss was not achieved. Furthermore, at the level of the wider community, mean BMI increased among younger persons over the four-year follow-up period. This demonstrates the difficulty of achieving and maintaining weight loss, particularly in already-overweight individuals. By way of comparison, the changes observed in the longitudinal cohort were more modest than those reported for the first year of the Zuni Diabetes Project, where participants lost 4 kg on average.[26] The Zuni Diabetes Project was a community-based physical activity program among native Americans that led to significant reductions in body weight in diabetic persons and reductions in the number of persons requiring oral hypoglycaemic medication. We are unaware of any community-based programs that have achieved reductions in the prevalence of obesity.

In terms of metabolic control, there were significant falls in fasting and two-hour plasma glucose concentrations after six months in the longitudinal cohort but these decreases were of similar magnitude in both the intervention and the non-intervention groups. Thus, the change in mean glucose concentrations may be a seasonal effect, with lower values occurring in the cooler, less humid, dry season. Seasonality of health outcomes in the Kimberley region has been reported previously with regard to a higher incidence of low birthweight during the wet season.[27] In our longitudinal cohort, there was evidence that the diet/physical activity intervention group was protected from increases in plasma glucose and triglycerides compared with the non-intervention group over a two-year follow-up period, suggesting improved metabolic control. However, at the level of the wider community, there was no significant change in the prevalence of diabetes or IGT over four years of follow-up. Like obesity, the high prevalence of diabetes appears to be an intractable problem, at least over the relatively short period of follow-up examined in this and similar community-based studies.[28,29] It is likely that the only means of reducing the prevalence of diabetes is to prevent new cases among younger people and wait for this cohort to move into the older age groups.

In contrast to the lack of success in reversing obesity and diabetes, important improvements were observed for several coronary risk factors. In the longitudinal cohort, fasting insulin concentration fell significantly over 24 months of follow-up, particularly in the intervention group. This change, accompanied by a fall in plasma glucose and the prevention of increases in plasma triglyceride concentration relative to the non-intervention group, suggests improved insulin sensitivity. Increased physical activity presumably contributed to this apparent increase in insulin sensitivity[15-17] as physical activity is far easier in the cooler, dry season than in the hot and humid summer months. A lower intake of saturated fat may also have contributed to greater insulin sensitivity.[30] At the wider community level, although there was no significant change in average insulin, glucose or triglyceride levels among younger people over four years of follow-up, this needs to be interpreted in the context of a significant weight gain over the same period. Since insulin sensitivity is inversely related to body fatness,[31] higher fasting insulin levels might have been expected among younger people. Among older community members, where there was no net change in BMI, fasting insulin concentration fell, suggesting improved insulin sensitivity consistent with the reported increase in physical activity. For the longitudinal cohort, those attempting diet and/or exercise changes were protected from the increase in plasma triglyceride concentration observed in the comparison group over two years of follow-up. Qualitatively at least, there appeared to be a seasonal trend in the intervention group consistent with the changes in insulin sensitivity. However, at the community level, there was no change in mean triglyceride concentration over four years of follow-up.

A study in a Native American community (of a sample younger than that for Looma, and all non-diabetic) did not demonstrate improvements in plasma glucose, lipids or insulin, while body weight increased over a one-year follow-up.[32] It did, however, identify improvements in self-reported dietary habits and physical activity levels. At Looma there was a marked and sustained increase in the proportion of older community members reporting regular physical activity and attempts to reduce their intake of fat and sugar, and this was consistent with the apparent improvement in insulin sensitivity. Although we have not formally tested individuals' knowledge of diabetes, the increase in the proportion of persons reporting dietary and physical activity strategies suggests greater knowledge of the importance of diet and physical activity in prevention and self-management of diabetes. This, along with testimonials from health workers, suggests that program participants now have a greater understanding of diabetes, its likely causes and the adverse impacts of its complications. Such knowledge is a starting point for intervention but is not of itself sufficient to promote sustainable change: environmental characteristics promoting sustainable change are also required.[33] At Looma, in addition to specific education and intervention strategies undertaken within the longitudinal cohort, this type of environmental change was addressed in a number of ways. For example, physical activity was facilitated by council policies, including the decision to appoint a sport and recreation officer to organise sporting competitions, while store management policies assisted in allowing healthy food choices. Thus changes in the social environment were perhaps at least partly responsible for enabling the improvements observed here. Broader issues of poverty, unemployment and food supply require attention before optimal results can be achieved in remote areas generally.[34,35]

That Looma Healthy Lifestyle continues to operate at the time of writing (six years after its commencement) is, in our opinion, a measure of success in itself. We believe this longevity is due to several factors:

- the program has widespread community support and is now run entirely by community members;
- the ongoing commitment from Aboriginal Health Workers, the community council, store management and other community groups; and
- analysis of data by academic staff associated with the program and prompt feedback of results to the community, particularly in the early stages.

The role of participants involved in the initial intervention processes as disseminators of health messages was an important aspect. Although not a randomised, clinical trial in the style of that recently reported from a native American population,[32] the greater participation in and longevity of the Looma program is likely due to greater community control over both its design and implementation. Narayan et al. noted the difficulty of achieving sustained adherence to intervention strategies.[32] A truly 'randomised' design is unlikely to be a useful model for community-based interventions, since communities and individuals *choose* whether or not to undertake such programs. Furthermore, a rigorous adherence to specific intervention protocols defined by researchers can limit community control of a program: there is no apparent reason to expect that interventions imposed from outside the community should necessarily receive support from community members. A recent Canadian study identified flexibility to allow community-directed changes in focus of an intervention program as a factor associated with sustainability of community-based interventions.[36] In this context, evaluation of intervention process and impact becomes essential to assist in ultimately ascribing cause and effect relationships between intervention strategies and outcomes. Although our evaluation of Looma Healthy Lifestyle has demonstrated it to be effective in many respects, it nevertheless remains essentially a 'case study' and concurrent interventions in a number of communities with systematic process evaluation would be a more scientifically robust means of identifying generalisable intervention strategies.

The *process* by which a community initiates, develops and implements an intervention program can itself contribute to improved health outcomes. A sense of control over events is associated with positive health outcomes, an effect at least partly mediated through mechanisms related to insulin sensitivity.[5] Insulin resistance underlies numerous CVD risk factors including glucose intolerance, dyslipidaemia and abnormalities of haemostatic function. These psychosocial aspects may have relevance to the results observed in the longitudinal cohort. The self-selected 'intervention group', although similar to the comparison group with respect to biochemical, anthropometric and behavioural variables at baseline, may have achieved a greater sense of control and been more highly motivated to undertake and sustain intervention strategies, resulting in metabolic improvements relative to the comparison group.

The Looma Healthy Lifestyle program was associated with sustained, if modest, improvements in several important biochemical and behavioural risk factors for diabetes and CVD. A detailed examination of diet-related markers of CVD risk (hyperhomocysteinaemia, hypercholesterolaemia, antioxidant status) was beyond the scope of this report but major improvements have been achieved in this area (Rowley et al., unpublished data). The most notable feature of Looma Healthy Lifestyle is its sustainability and the degree to which it is embedded in and directed by the community. These features may give the program the best chance of achieving success in long-term improvements of health outcomes.

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References

1. Gracey M, Veroni M. Comparative hospitalisation and mortality rates of Aboriginal and non-Aboriginal Western Australians in their sixth and seventh decades. *Aust N Z Med J* 1995; 25: 27-31.
2. Beaglehole R, Stewart AW, Jackson R, et al. Declining rates of coronary heart disease in New Zealand and Australia, 1983-1993. *Am J Epidemiol* 1997; 145: 707-13.
3. Thomson NJ. Recent trends in Aboriginal mortality. *Med J Aust* 1991; 154: 235-9.
4. Veroni M, Gracey M, Rouse I. Patterns of mortality in Western Australian Aboriginals, 1983-1989. *Int J Epidemiol* 1994; 23: 73-81.
5. Brunner EJ, Marmot MG, Nanachahal K, et al. Social inequality in coronary risk: central obesity and the metabolic syndrome. Evidence from the Whitehall II study. *Diabetologia* 1997; 40: 1341-49.
6. Lantz PM, House JS, Lepkowski JM, et al. Socioeconomic factors, health behaviours and mortality. Results from a nationally representative prospective study of US adults. *J Am Med Assoc* 1998; 279: 1703-8.
7. Manson JE, Rimm EB, Stampfer MJ, et al. Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet* 1991; 338: 774-8.
8. Manson JE, Nathan DM, Krolewski AS, et al. A prospective study of exercise and incidence of diabetes among US male physicians. *J Am Med Assoc* 1992; 268: 63-7.
9. Pan X, Li G, Hu Y-H, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. *Diabetes Care* 1997; 20: 537-44.

10. Colditz GA, Willett WC, Stampfer MJ, et al. Weight as a risk factor for clinical diabetes in women. *Am J Epidemiol* 1990; 132: 501-13.
11. DeFronzo RA. Insulin resistance, hyperinsulinemia and coronary artery disease: A complex metabolic web. *J Cardiovasc Pharmacol* 1992; 30(11 Suppl): S1-16.
12. Paisey RB, Harvey P, Rice S, et al. An intensive weight loss programme in established Type 2 diabetes and controls: Effects on weight and atherosclerosis risk factors at 1 year. *Diabet Med* 1998; 15: 73-9.
13. Bosello O, Armellini F, Zamboni M, Fitchet M. The benefits of modest weight loss in type II diabetes. *Int J Obesity* 1997; 21(1 Suppl): S10-13.
14. Agurs-Collins TD, Kumanyika SK, Ten Have TR, Adams-Campbell LL. A randomised controlled trial of weight reduction and exercise for diabetes management in older African-American subjects. *Diabetes Care* 1997; 20: 1503-11.
15. Fluckey JD, Hickey MS, Brambrink JK, et al. Effects of resistance exercise on glucose tolerance in normal and glucose-intolerant subjects. *J Appl Physiol* 1994; 77: 1087-92.
16. Pan X-R, Li G-W, Hu Y-H, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and diabetes study. *Diabetes Care* 1997; 20: 537-44.
17. Eriksson K-F, Lindgärde F. No excess 12-year mortality in men with impaired glucose tolerance who participated in the Malmö Preventive Trial with diet and exercise. *Diabetologia* 1998; 41: 1010-6.
18. O'Dea K, Walker KZ. Diet in the aetiology of diabetes. In: Flatt PR, Ioannides C, editors. *Drugs, diet and disease. Vol. 2: Mechanistic approaches to diabetes*. Herts(UK): Prentice Hall, 1995: 121-52.
19. O'Dea K. Marked improvement in carbohydrate and lipid metabolism in diabetic Australian Aborigines after temporary reversion to traditional lifestyle. *Diabetes* 1984; 33: 596-603.
20. Simmons D, Voyle J, Swinburn B, O'Dea K. Community-based approaches for the primary prevention of non-insulin-dependent diabetes mellitus. *Diabet Med* 1997; 14: 519-26.
21. Bracht N, Finnegan JR, Rissel C, et al. Community ownership and program continuation following a health demonstration project. *Health Educ Res* 1994; 9: 243-55.
22. Anderson I. *Koorie health in Koorie hands*. Melbourne: Koorie Health Unit, Health Department of Victoria, 1988: 108-10.
23. Spinks M, White G. Looma, Western Australia: Diabetes Program. In: Bear-Wingfield R, editor. *Sharing Good Tucker Stories. A Guide for Aboriginal and Torres Strait Islander Communities.* Canberra: Commonwealth Dept. of Health and Family Services, 1996: 63-9.
24. White GA, Skinner K, Skinner M, et al. A community-based intervention program for the prevention and treatment of obesity and diabetes in Aboriginal people – the Looma Diabetes Program. In: Rural and remote Australia: health for all by the Year 2000. *Proceedings of the 4th National Rural Health Conference; 1997 Feb 9-1: Perth (WA).* Canning Bridge (WA): Promaco Conventions (for the National Rural Health Alliance), 1997: 653-7.
25. Skinner M, Skinner K, Skinner B, et al. Looma Healthy Lifestyle. In: Nutrition Networks 97 Conference. *Proceedings of the 5th National Nutrition Networks Conference; 1997 Oct 15-17: Adelaide (SA)*. Adelaide (SA): Nutrition Networks '97 Organising Committee, 1997: 18-23.
26. Heath GW, Leonard BE, Wilson RH, et al. Community-based exercise intervention: Zuni Diabetes Project. *Diabetes Care* 1987; 10: 579-83.
27. Rousham EK, Gracey M. Seasonality of low birthweight in indigenous Australians: an increase in pre-term birth or intrauterine growth retardation? *Aust N Z J Public Health* 1998; 22: 669-72.
28. Lee AJ, Bailey APV, Yarmirr D, et al. Survival tucker: improved diet and health indicators in an Aboriginal community. *Aust J Public Health* 1994; 18: 277-85.
29. Dowse GK, Gareeboo H, Alberti KGMM, et al. Changes in population cholesterol concentrations and other cardiovascular risk factor levels after five years of the non-communicable disease intervention programme in Mauritius. *Brit Med J* 1995; 311: 1255-9.
30. Borkman M, Storlien LH, Pan DA, et al. The relation between insulin sensitivity and the fatty acid composition of skeletal-muscle phospholipids. *N Engl J Med* 1993; 328: 238-44.
31. Golay A, Felber JP. Evolution from obesity to diabetes. *Diabetes Metab* 1994; 20: 3-14.
32. Narayan KMV, Hoskin M, Kozak D, et al. Randomized clinical trial of lifestyle interventions in Pima Indians: a pilot study. *Diabet Med* 1998; 15: 66-72.
33. Green LW, Kreuter MW. *Health Promotion planning: An educational and environmental approach.* Mayfield: Mountain View, 1991.
34. Ring IT, Firman D. Reducing indigenous mortality in Australia: Lessons from other countries. *Med J Aust* 1998; 169: 528-33.
35. Leonard D, Beilin R, Moran M. Which way kaikai blo umi? Food and nutrition in the Torres Strait. *Aust J Public Health* 1995; 19: 589-95.
36. O'Loughlin J, Renaud L, Richard L, et al. Correlates of the sustainability of community-based heart health promotion interventions. *Prev Med* 1998; 27: 702-12.