

# Long-Term (5-Year) Effects of a Reduced-Fat Diet Intervention in Individuals With Glucose Intolerance

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**OBJECTIVE** — To determine whether reducing dietary fat would reduce body weight and improve long-term glycemia in people with glucose intolerance.

**RESEARCH DESIGN AND METHODS** — A 5-year follow-up of a 1-year randomized controlled trial of a reduced-fat ad libitum diet versus a usual diet. Participants with glucose intolerance (2-h blood glucose 7.0–11.0 mmol/l) were recruited from a Workforce Diabetes Survey. The group that was randomized to a reduced-fat diet participated in monthly small-group education sessions on reduced-fat eating for 1 year. Body weight and glucose tolerance were measured in 136 participants at baseline, 6 months, and 1 year (end of intervention), with follow-up at 2 years ( $n = 104$ ), 3 years ( $n = 99$ ), and 5 years ( $n = 103$ ).

**RESULTS** — Compared with the control group, weight decreased in the reduced-fat-diet group ( $P < 0.0001$ ); the greatest difference was noted at 1 year ( $-3.3$  kg), diminished at subsequent follow-up ( $-3.2$  kg at 2 years and  $-1.6$  kg at 3 years), and was no longer present by 5 years (1.1 kg). Glucose tolerance also improved in patients on the reduced-fat diet; a lower proportion had type 2 diabetes or impaired glucose tolerance at 1 year (47 vs. 67%,  $P < 0.05$ ), but in subsequent years, there were no differences between groups. However, the more compliant 50% of the intervention group maintained lower fasting and 2-h glucose at 5 years ( $P = 0.041$  and  $P = 0.026$ , respectively) compared with control subjects.

**CONCLUSIONS** — The natural history for people at high risk of developing type 2 diabetes is weight gain and deterioration in glucose tolerance. This process may be ameliorated through adherence to a reduced fat intake.

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Impaired glucose tolerance (IGT) is a major risk factor in the development of type 2 diabetes (1) with conversion rates ranging from 3.6–8.7% per year (2). Any measures that can reduce the progression to diabetes in such high-risk populations will have major health benefits. Weight gain is a critical factor in this progression, increasing the risk ~2- to 12-fold, depending on the amount of weight gained (3). Long-term intake of fat

and/or saturated fat also seems to have an important effect on the risk of developing type 2 diabetes, independent of weight gain (4,5). Combined diet and physical activity interventions have been found to be successful in reducing the rate of conversion from IGT to type 2 diabetes (6–8).

Decreasing fat intake is a central part of the dietary approach to weight reduction. Because fat is very energy dense, it

has minor effects on suppressing appetite and stimulating fat oxidation, and it is readily stored by the body as fat (9). A high-fat diet results in passive overconsumption of total energy (10), and simple reduction of dietary fat alone has been shown to cause weight loss (11,12). However, there has been much debate about the long-term impact of reducing total fat in the diet. Some suggest that it should be a central strategy; others believe that the effect of reducing total fat is weak and that a diet high in monounsaturated fatty acid should be recommended (13,14).

We previously reported a 1-year randomized controlled trial of a reduced-dietary-fat intervention (15). The reduced-fat-diet group significantly decreased its mean daily fat intake and total energy intake compared with the control group without affecting micronutrient intake. We now report on the 5-year follow-up of that study to examine the long-term impact of the reduced-fat-diet program on body weight, glucose tolerance, and conversion to type 2 diabetes.

## RESEARCH DESIGN AND METHODS

Participants were selected from a previous Workforce Diabetes Survey (16) in which oral glucose tolerance tests (OGTT) were performed in 4,833 workers, aged 40 years and older, from 41 work sites around Auckland between 1988 and 1990. Of those participants surveyed, 162 (2.8%) were classified as having IGT by 1985 World Health Organization criteria (2-h blood glucose 7.8–11.0 mmol/l) (17), and another 114 workers (2%) had high normal blood glucose concentrations (7.0–7.8 mmol/l). In this combined “glucose intolerant” group (i.e., 2-h blood glucose 7.0–11.0 mmol/l,  $n = 276$ ), 237 participants could be contacted (~2 years after the original survey); 176 participants entered the intervention, and 136 (77%) completed the 1-year intervention period. Of these 136 participants, we were able to reassess 104 (76%) at 2 years, 99 (73%) at 3 years, and 103 (76%) at 5 years.

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**Abbreviations:** CD, control diet; IGT, impaired glucose tolerance; OGTT, oral glucose tolerance test; RF, reduced fat.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Before randomization, participants underwent repeat OGTT for redefinition of glucose tolerance status, which, in many cases, had changed since the original testing in the Workforce Diabetes Survey ~2 years prior. The participants were then individually assigned by simple randomization using an unmarked envelope system to an intervention group, the members of which were asked to consume a reduced-fat (RF) but otherwise ad-libitum diet, or to a control-diet (CD) group, the members of which continued with their usual diet. Six participants from one work site, all of whom were women from the Pacific Islands, were assigned to the RF group, because individual randomization may have caused some contamination of the CD-group participants. All participants found to have diabetes on re-testing were referred to their general practitioners for management but were still randomized for the study. All participants provided informed consent, and the study was approved by the Auckland Hospital Research Ethics Committee.

### **Dietary intervention**

Participants in the RF group entered a 1-year structured program aimed solely at reducing the total amount of fat in their usual diet. The program involved education on reducing dietary fat intake, personal goal-setting, and self-monitoring (food diaries) through a series of monthly small-group sessions as previously described (15). Briefly, the education sessions detailed the reasons for reducing dietary fat, showed participants how to identify high-fat foods (including reading labels), and identified a variety of strategies for reducing fat intake. The food diaries counted dietary fat intake for 2 days each week on a rotating schedule of days to capture weekdays and weekends. Participants used a simple fat-counter booklet to estimate the fat content of foods. Those in the CD group received only general dietary advice about healthy food choices upon entering the trial.

Compliance with the program was assessed by attendance at the monthly meetings and completion of the diet diaries (compliance score = mean of the percentage of meetings attended and the percentage of diaries filled). Those with compliance scores above the median for the group were classified as compliers for the analysis.

### **Measurements**

Height and weight were recorded with participants wearing lightweight clothing and no shoes using a Seca 2000 scale with attached stadiometer (Seca, Hamburg, Germany). Participants in both groups completed a comprehensive 3-day food diary before randomization and after 1 year. Foods were measured using standard measuring cups and spoons and weight-approximation diagrams. Diaries were analyzed for nutrients using Nutritionist III software (Version 6; N-Squared Computing, Silverton, OR), to which the nutrient values of ~600 New Zealand foods had been added.

A standard 75-g OGTT was performed at each review. Diabetes, IGT, and impaired fasting glycemia were defined using the revised World Health Organization criteria and classification of diabetes mellitus (18). Measurements were taken at randomization and at 0.5, 1, 2, 3, and 5 years. Insulin was not measured at years 2, 3, and 5.

### **Statistical analyses**

Mean (SE) values, adjusted for age, sex, ethnicity, and baseline measurement, were calculated using SAS PROC MIXED (SAS Institute, Cary, NC) (19). The repeated-measures model included time since baseline for each of the time points, treatment group, and the time by treatment interaction and used an unstructured variance-covariance matrix to model the correlations between time points. The Satterthwaite option was used to approximate the correct denominator degrees of freedom for the tests of fixed effects. A linear model was assumed from time 0–5 years. Analyses were performed using random coefficients models, which allow the slopes and intercepts to vary randomly for each participant.

Categorical variables were compared using the  $\chi^2$  test, and Student's *t* test was used for continuous variables. All statistical analyses were performed using SAS (19). Participants who dropped out before the end of the 12-month intervention period were not included in the analysis because if their last observation had been carried forward, the differences between the two groups would have been artificially maintained over the follow-up period. Carrying the last observation forward assumes that assigning a no-change status is a conservative analysis, which is

not the case, because weight change over time is nonlinear.

## **RESULTS**

### **Participants**

The baseline characteristics of participants who completed the 1-year intervention are presented in Table 1. There were no significant differences between the groups. The 40 participants who did not complete the 1-year intervention (4 died, 1 became pregnant, 7 developed serious illnesses, 4 moved out of Auckland, and 24 dropped out of the study) were of similar age to those who did complete the 1-year intervention but had a significantly ( $P < 0.05$ ) higher BMI ( $30.7 \pm 0.7$  vs.  $29.1 \pm 0.4$  kg/m<sup>2</sup>) and included a higher proportion of women (42 vs. 27%) and individuals of Maori or Pacific descent (40 vs. 24%). The baseline characteristics of the participants who were lost to 5-year follow-up ( $n = 33$ ) were not significantly different from the rest of the participants, except that they were older (53.5 vs. 49.9 years,  $P = 0.029$ ).

There were no differences in sex or glucose tolerance status between those participants in the RF group classified as compliers or noncompliers, but Europeans were more likely to be compliers (66 vs. 18%,  $P < 0.001$ ). Interestingly, at the end of year 1, there was no difference in fat intake between compliers and noncompliers.

### **Lifestyle changes**

The dietary changes over the intervention year are shown in Table 2. The total energy intake and the absolute intake (gram/day) of fat decreased in the RF group, whereas changes in the other macronutrients and fiber were not different between the groups. As a percentage of total energy, fat decreased, whereas carbohydrate and protein increased in the RF group compared with the CD group.

At the end of year 1, 20% of the RF group reported an increase in recreational exercise over the previous year compared with 9% in the CD group ( $P = 0.002$ ). At the end of year 2, 21% of the RF group reported that they were exercising as a means to lose weight compared with 12% of the control group ( $P = 0.087$ ), but by the end of year 3, these percentages had reversed (13 vs. 21%,  $P = 0.13$ ). By the end of year 1, four participants in the RF

**Table 1—Baseline characteristics of participants who completed the 1-year intervention**

	RF group	CD group
<i>n</i>	66	70
Age (years)	52.5 ± 0.8	52.0 ± 0.8
Sex (M:F)	45:21	56:14
Ethnicity		
European	44 (66.7)	53 (75.7)
Maori	7 (10.6)	5 (7.1)
Pacific Islander	13 (19.7)	9 (12.9)
Other	2 (3.0)	3 (4.3)
Anthropometry		
Weight (kg)	85.46 ± 1.80	84.33 ± 1.55
BMI (kg/m <sup>2</sup> )	29.08 ± 0.55	29.17 ± 0.48
Waist circumference (cm)	100.48 ± 1.42	101.60 ± 1.28
Waist to hip ratio	0.944 ± 0.008	0.954 ± 0.008
Glucose tolerance test		
Fasting glucose (mmol/l)	6.7 ± 0.2	6.6 ± 0.2
2-h glucose (mmol/l)	7.5 ± 0.3	7.9 ± 0.3
Fasting insulin (mIU/l)	16.0 ± 1.1	15.8 ± 0.9
2-h insulin (mIU/l)	50.2 ± 4.2	47.0 ± 3.8
Glucose tolerance status		
Normal	24 (36.4)	23 (32.9)
Impaired fasting glucose	11 (16.6)	15 (21.4)
Impaired glucose tolerance	10 (15.1)	9 (12.9)
Diabetes	21 (31.8)	23 (32.9)

Data are means ± SEM or *n* (%) unless otherwise indicated.

group had quit smoking compared with two participants in the CD group.

### Changes in weight and glucose tolerance

The mean changes in weight, BMI, and glucose tolerance at 0.5, 1, 2, 3, and 5 years (Table 3) were adjusted for age, sex, ethnicity, and baseline measurements. The RF group lost weight and achieved a lower BMI during the intervention pe-

riod, and this was sustained for 3 years. By 5 years, however, the mean weight of the RF group was not significantly different from that measured before the intervention (1.1 kg) or from the CD group.

There was no overall effect of diet on fasting glucose, but there was a significant effect on 2-h glucose over the period ( $P < 0.0001$ ) (Table 3). There was a significantly lower 2-h insulin at 1 year. Changes in glucose tolerance status are shown in

Fig. 1. There were no significant differences between the RF and CD groups at baseline and 0.5 years, but the intervention showed a significant effect on glucose tolerance status ( $P = 0.015$ ) at 1 year. A smaller proportion of participants had type 2 diabetes or IGT in the RF group (47 compared with 67%). No intervention effect was present at 2, 3, or 5 years.

### Effects with greater compliance

The RF group was subdivided in half according to compliance (as described in RESEARCH DESIGN AND METHODS). The compliers showed a significantly lower blood glucose level (after adjustment for age, sex, and ethnicity) at fasting ( $P = 0.041$ ) and 2 h ( $P = 0.024$ ) at 5 years compared with the CD group (Fig. 2). In contrast, noncompliers showed no significant changes versus the CD group. The compliers showed a significantly lower weight in the first 3 years, but at 5 years, there were no significant differences between the groups (Fig. 2). Compliers also showed significantly lower fasting and 2-h insulin levels at 1 year ( $P = 0.023$  and 0.018, respectively) compared with the CD group (data not shown) but were not tested subsequently.

**CONCLUSIONS**— What are the long-term effects of promoting reductions in total dietary fat? That question is currently under debate, and we have examined the effects of a 1-year intervention to reduce dietary fat on body weight and glucose tolerance. Participants were not expecting to lose weight during the intervention, because they were told that the

**Table 2—Daily energy, macronutrient, and fiber intakes determined by 3-day diet diaries**

	RF group ( <i>n</i> = 49)			CD group ( <i>n</i> = 61)			<i>P</i> *
	Baseline Mean ± SD	1 year Mean ± SD	Change Mean ± SEM	Baseline Mean ± SD	1 year Mean ± SD	Change Mean ± SEM	
Energy (kcal)	2,195 (610)	1,832 (481)	−362 (92)	2,366 (693)	2,307 (856)	−59 (93)	0.016
Fat (g)	86.1 (32.6)	52.1 (24.5)	−34.0 (4.8)	96.5 (35.7)	90.0 (43.6)	−6.6 (4.5)	<0.0001
% Energy	34.6 (6.5)	25.9 (8.8)	−8.7 (1.3)	36.1 (6.6)	33.8 (7.2)	−2.3 (1.0)	<0.0001
Carbohydrate (g)	250 (74)	251 (90)	1.0 (14)	261 (80)	250 (73)	−11 (9)	0.49
% Energy	46.2 (8.3)	54.5 (11.7)	8.3 (1.5)	45.0 (9.1)	45.6 (10.1)	0.6 (1.2)	<0.0001
Protein (g)	91.3 (27.3)	83.6 (25.6)	−7.7 (3.7)	96.1 (31.1)	95.1 (45.3)	−1.0 (4.3)	0.25
% Energy	16.9 (3.3)	18.6 (4.1)	1.7 (0.6)	16.7 (4.1)	16.5 (3.8)	−0.2 (0.6)	0.025
Alcohol† (g)	14.6 (21.1)	10.0 (14.4)	−4.6 (20.5)	17.5 (29.2)	22.1 (34.0)	4.6 (25.8)	0.21
% Energy	4.4 (6.2)	3.5 (4.7)	−0.9 (0.8)	4.5 (6.7)	5.8 (7.9)	1.3 (0.7)	0.19
Fiber (g)	20.9 (8.1)	20.5 (10.1)	−0.4 (1.3)	19.4 (7.8)	18.4 (6.6)	−1.0 (1.2)	0.73
g/1,000 kcal	9.9 (3.9)	11.2 (4.8)	1.3 (0.6)	8.7 (3.7)	8.8 (3.2)	0.1 (0.5)	0.061

\*Difference in changes between the groups; Student's *t* test; †distribution not normal, Wilcoxon rank-sum test.

Table 3—Mean changes in anthropometrical and biochemical measurements (SEM) from baseline over 5 years in the RF and CD groups adjusted for baseline measurements

	Time (years)					Overall effect of diet (P)*
	Intervention period		2.0	3.0	5.0	
	0.5	1.0				
n (RF/CD)	66/70	66/70	47/57	48/51	51/52	—
Weight (kg)						
RF	-2.97 ± 0.54†	-3.32 ± 0.68†	-3.15 ± 0.78†	-1.60 ± 0.78†	1.06 ± 0.64	<0.0001
CD	-0.08 ± 0.43	0.59 ± 1.61	1.06 ± 0.46	2.13 ± 0.70	1.26 ± 0.68	
BMI (kg/m <sup>2</sup> )						
RF	-0.99 ± 0.18†	-1.09 ± 0.24†	-1.01 ± 0.28‡	-0.46 ± 0.28‡	0.72 ± 0.28	<0.0001
CD	-0.01 ± 0.15	0.22 ± 0.15	0.38 ± 0.15	0.75 ± 0.24	0.59 ± 0.27	
Fasting glucose (mmol/l)						
RF	0.04 ± 0.17	0.08 ± 0.16	-0.17 ± 0.26	-0.04 ± 0.18	0.02 ± 0.18	NS
CD	0.11 ± 0.16	0.17 ± 0.13	0.05 ± 0.24	0.09 ± 0.22	0.29 ± 0.30	
2-h glucose (mmol/l)						
RF	-0.36 ± 0.36	0.01 ± 0.33	-0.76 ± 0.42	0.20 ± 0.37	1.02 ± 0.40	<0.0001
CD	0.13 ± 0.37	0.74 ± 0.35	0.01 ± 0.49	0.48 ± 0.45	2.30 ± 0.54	
Fasting insulin (mIU/l)						
RF	-3.71 ± 1.58	-4.87 ± 1.09	—	—	—	<0.0001
CD	-4.31 ± 0.86	-3.80 ± 0.83	—	—	—	
2-h insulin (mIU/l)						
RF	-12.01 ± 6.16	-14.94 ± 4.20‡	—	—	—	0.0103
CD	-6.20 ± 3.64	-1.90 ± 3.75	—	—	—	

Significant difference between RF and CD groups at each time point. \*Effect of diet assessed by the interaction term group time (i.e., does the time course of the changes over the 5 years differ with time); †P < 0.01; ‡0.01 = P < 0.05.

study was testing the effect of fat intake on blood glucose levels. In the medium term, up to 3 years, significant weight loss occurred that was not sustained at 5 years. Glucose tolerance also improved initially with the diet but was not significantly different at 5 years. However, the participants who were most compliant with the program did show lower fasting and 2-h blood glucose levels at 5 years.

The amount of weight loss in our study (3.3 kg at 1 year) was similar to other reduced-fat ad libitum intervention trials (12,20,21), and also like most interventions for weight loss, weight was regained in the long term (22). Greater compliance to the intervention resulted in greater weight loss and more lasting effects on glucose tolerance, consistent with other studies showing that compliance with the intervention, long-term maintenance of low-fat eating patterns, and weight loss were all strongly interrelated (23). Similarly, more intensive, longer-term interventions that combined exercise with diet modification in individuals with IGT showed significant health gains of sustained weight reduction and improvements in glucose tolerance over 5

(6) and 6 years (7), suggesting that a higher level of compliance was reached during that length of intervention. Sustained therapeutic contact was considered an important factor in maintaining

the effectiveness of lifestyle modifications (24). These studies, together with our findings, suggest that to be successful, interventions should probably be multifactorial and longer in duration. Otherwise,

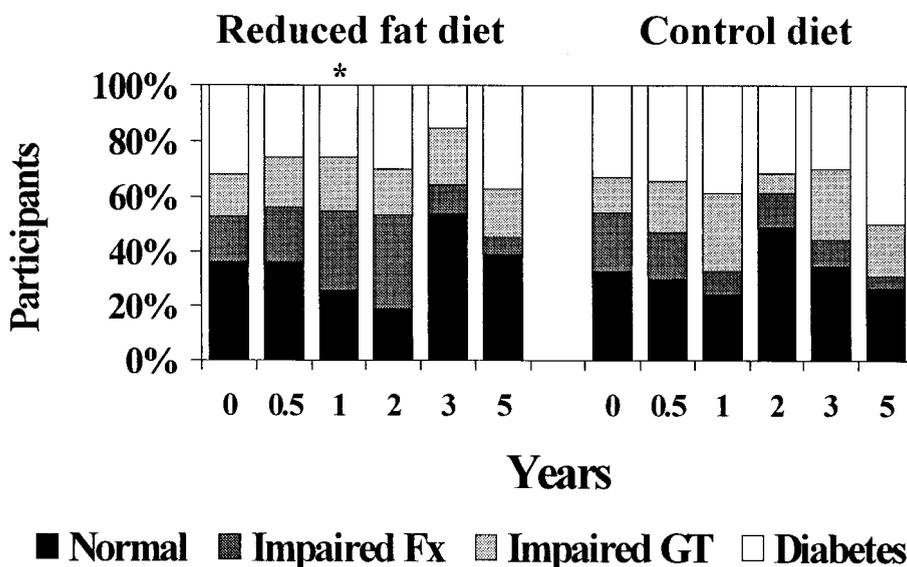
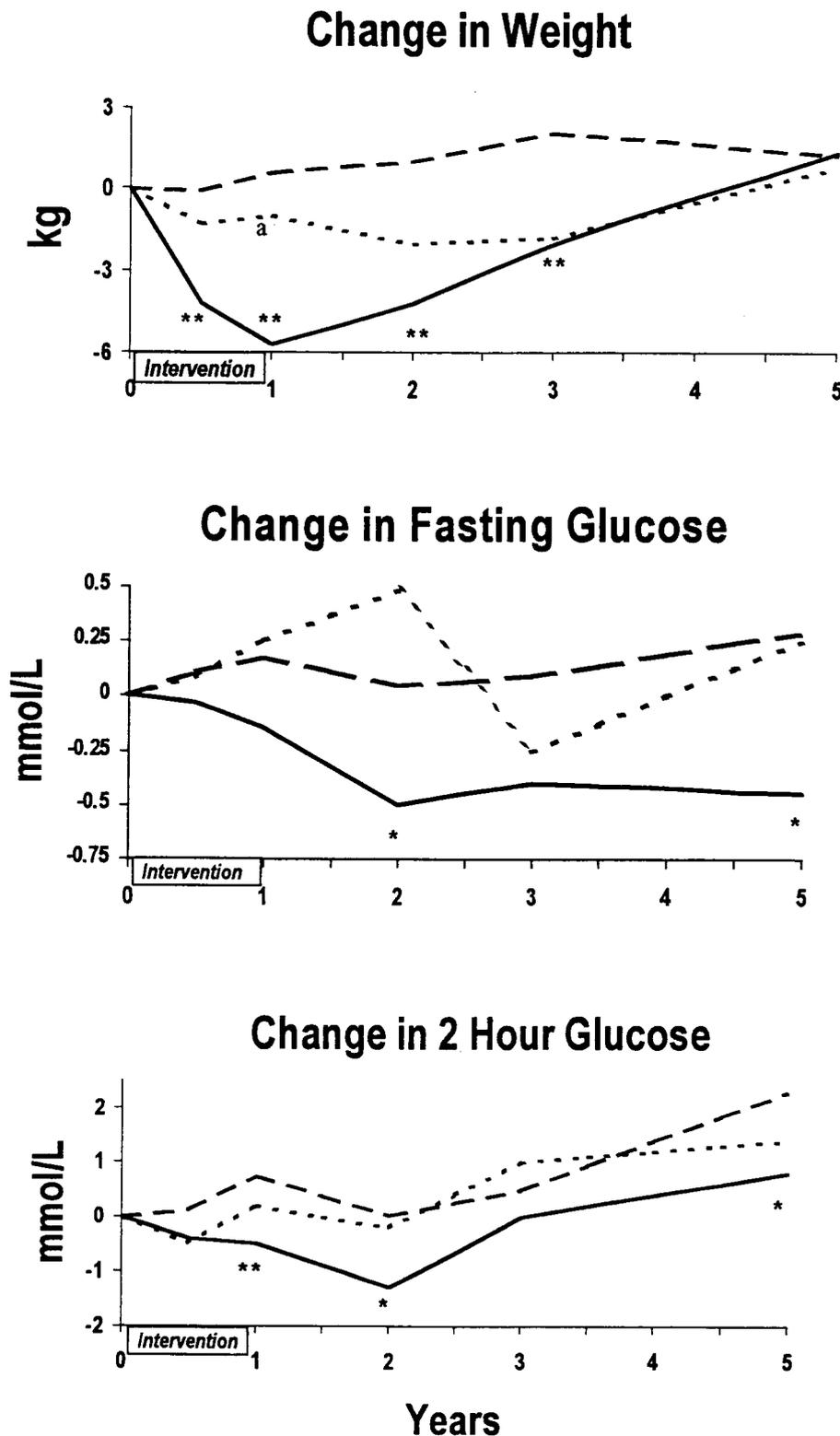


Figure 1—Changes in glucose tolerance status over time in the RF and CD groups. A significant difference is shown between the RF and CD participants at 1 year. \*P < 0.015.



**Figure 2**—Long-term changes in weight, fasting glucose, and 2-h blood glucose concentrations in the CD group (---), the least compliant RF group (-.-.-), and the most compliant RF group (—). Significant differences are shown between the most compliant RF and CD groups (\* $P < 0.05$ , \*\* $P < 0.01$ ) and between the least compliant RF and CD groups (<sup>a</sup> $P < 0.05$ ).

the initial efforts and enthusiasm for healthier lifestyles become eroded by the external obesogenic environmental forces (25). If, on the other hand, the lifestyles can be maintained, they will probably reduce the amount of weight gain and the risk of diabetes.

Our results may have implications for other studies of diabetes prevention in high-risk groups. The dietary interventions in the large Diabetes Prevention Program (26) will be sustained over the 5-year period of the trial, and the “dose” is high in the intensive lifestyle group. Physical activity is also included, but no environmental interventions are planned. Interestingly, in our study, the participants randomized to the RF group also seemed to increase their levels of physical activity; therefore, promoting healthy behaviors through dietary changes may have stimulated the participants to exercise more.

Our study was limited by the number of people, particularly of Maori and Pacific Island ethnicity, who were either lost to follow-up ( $n = 24$ , 17.6%) or who had low compliance to the intervention. These ethnic groups are at higher risk of developing diabetes than Europeans. The manner of recruitment into the study may also have limited participation. People who learn from a work-site survey that their blood glucose level is somewhat elevated may not be as ready or willing to change their lifestyle behaviors as people who already have known health problems such as diabetes.

In conclusion, an intervention aimed at reducing dietary fat alone improved body weight and glucose tolerance over a 2- to 3-year period but was only sustained in more compliant participants.

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