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A worksite programme significantly alters nutrient intakes

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Abstract

Objective: To examine whether a worksite nutrition programme using a low-fat vegan diet could significantly improve nutritional intake.

Design: At two corporate sites of the Government Employees Insurance Company, employees who were either overweight (BMI $\geq 25\,kg/m^2$) and/or had type 2 diabetes participated in a 22-week worksite-based dietary intervention study.

Setting: At the intervention site, participants were asked to follow a low-fat vegan diet and participate in weekly group meetings that included instruction and group support (intervention group). At the control site, participants received no instruction (control group). At weeks 0 and 22, participants completed 3 d dietary records to assess energy and nutrient intake.

Subjects: A total of 109 participants (sixty-five intervention and forty-four control).

Results: In the intervention group, reported intake of total fat, *trans* fat, saturated fat and cholesterol decreased significantly ($P \leq 0{\cdot}001$), as did energy and protein ($P = 0{\cdot}01$), and vitamin B_{12} ($P = 0{\cdot}002$), compared with the control group. Intake (exclusive of any use of nutritional supplements) of carbohydrate, fibre, vitamin C, magnesium and potassium increased significantly ($P \leq 0{\cdot}0001$), as did that for β-carotene ($P = 0{\cdot}0004$), total vitamin A activity ($P = 0{\cdot}004$), vitamin K ($P = 0{\cdot}01$) and sodium ($P = 0{\cdot}04$) in the intervention group, compared with the control group.

Conclusions: The present study suggests that a worksite vegan nutrition programme increases intakes of protective nutrients, such as fibre, folate and vitamin C, and decreases intakes of total fat, saturated fat and cholesterol.

Keywords
Diet
Vegetarian
Obesity
Intervention

Obesity and many health conditions to which it contributes are epidemics. According to the National Health and Nutrition Examination Survey (NHANES), one-third of Americans are obese and an additional one-third are overweight. In 2005, almost eighty-one million people in the United States had one or more forms of CVD, with over a third of all deaths related to CVD[1]. In 2006, 7 % of the US adult population had been diagnosed with cancer and 8 % had been diagnosed with diabetes[2].

Clinical trials have shown that dietary interventions can be helpful in addressing these conditions. A diet low in fat and cholesterol has been shown to prevent and reverse heart disease[3–5], improve the management of type 2 diabetes[6] and improve prognosis in some forms of cancer[7–9]. However, major diet alterations elicit a wide range of changes in macronutrient and micronutrient intake that can affect health in many ways. We implemented a low-fat vegan diet as part of a worksite nutrition programme at a major US corporation. In the course of the present study, we assessed the effects of dietary intervention on health as well as nutrient intake. Because participants were given only dietary information and encouragement and were left to implement the prescribed guidelines as they wished, it is important to assess the resulting nutrient changes.

Experimental methods

Participants

Individuals were recruited from two Government Employees Insurance Company (GEICO) corporate locations through on-site announcements, flyers and Emails. The company's headquarters in Chevy Chase, MD, USA, was designated as the intervention site, and a Fredericksburg, VA, USA, location was designated as the control site.

Individuals included in the present study were employees at one of the two study sites; had a BMI $\geq 25\,kg/m^2$ and/or a pre-existing diagnosis of type 2 diabetes mellitus (defined by a fasting plasma glucose concentration ≥126 mg/dl on two occasions or a prior physician's diagnosis of type 2 diabetes); were at least 18 years of age; and were willing to participate in either the intervention or control group, as determined by the location of the office. Exclusionary criteria included a history of alcohol abuse

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or dependence followed by any current use; current or unresolved past drug abuse; pregnancy or plans to become pregnant during the study; history of severe mental illness; inordinate fear of blood draws; current use of a low-fat vegetarian diet; unstable medical status; or, for individuals with type 2 diabetes, HbA1c >10·5 %.

Interested individuals completed a telephone screening interview with research staff. Those who appeared to satisfy participation criteria were scheduled for an in-person interview to review the study procedures and confirm eligibility. All participants provided written informed consent. Although GEICO management was supportive to the study, participation was completely voluntary. The protocol was approved by an external institutional review board.

The participants completed a practice 3 d dietary record, which was reviewed with a study dietitian for accuracy and completeness. Body weight was measured in light clothing, without shoes, using a digital scale (Befour FS-0900). Height was measured, without shoes, using a wall-mounted stadiometer. Medical histories and physical examinations were completed by a physician or a nurse practitioner. Volunteers provided blood samples for a complete blood count and chemistry panel and, for individuals with type 2 diabetes mellitus, HbA1c and fasting plasma glucose.

Dietary intervention

Participants in the intervention group were asked to follow a low-fat vegan diet (i.e. a diet excluding meat, poultry, fish, dairy products and eggs) for the duration of the 22-week study. The intervention diet included vegetables, fruit, grains and legumes and derived approximately 10 % of energy from fat, 15 % of energy from protein and 75 % of energy from carbohydrate. Participants were encouraged to favour low glycaemic index foods. No restrictions were placed on portion sizes, energy or carbohydrate intakes. Participants were asked to take a daily multiple vitamin to meet B_{12} requirements although supplements were not included in nutrient analyses. The cafeteria management at the intervention site included low-fat vegan menu options, such as oatmeal, minestrone or lentil soup, veggie burgers and portobello sandwiches among the daily offerings. Approximately one breakfast item and four lunch items (two entrées and two side dishes) that met the diet guidelines were offered daily.

Participants in the intervention group were encouraged to participate in weekly 1 h lunchtime group sessions for instruction and support. Group meetings were conducted by a physician, a registered dietitian and/or a cooking instructor following an established curriculum. Group meetings included nutrition education and cooking demonstrations.

Participants at the control site were asked not to make any changes to their diets, and no new food items were offered in the company cafeteria. Both the intervention and control groups were asked not to make any changes to their exercise patterns.

Assessment of dietary intake and adherence to dietary intervention

At baseline and 22 weeks, participants in both groups were asked to keep dietary records for three consecutive days, including two weekdays and one weekend day. Detailed instructions on dietary record keeping were provided at the beginning of the study, and food scales were provided.

Statistical methods

Dietary intake data were collected and analysed using Nutrition Data System for Research software version 2007, developed by the Nutrition Coordinating Center (NCC), University of Minnesota, Minneapolis, MN, USA. Data from the 3 d dietary records completed at baseline and at 22 weeks were analysed by one of three registered dietitians certified by the NCC.

Statistical analyses for nutrient intake included only participants who completed dietary records at baseline and at 22 weeks. Between-subject *t* tests were calculated for each nutrient to determine comparability of the intervention and control diet groups at baseline as well as whether the changes associated with the intervention diet were greater than those associated with the control diet. Within each diet group, paired comparison *t* tests were calculated to test whether the change from baseline to 22 weeks was significantly different from zero. An alpha of 0·05 was used for all statistical tests, with no adjustment for multiple comparisons. In addition to the *t* tests, we performed an analysis of covariance (ANCOVA) on the change scores to see whether differences between intervention and control groups remained while controlling for age, gender and race. We used PROC GLM in SAS statistical software package version 9·1 (SAS Institute Inc., Cary, NC, USA).

Results

Of the 170 screened individuals (seventy-six at the intervention site; ninety-four at the control site), sixty-eight at the intervention site (eighteen male; fifty female) and forty-five (two male; forty-three female) at the control site met participation criteria and were enrolled in the study. Of these, sixty-five intervention participants and forty-four control participants completed the food records at both baseline and at 22 weeks. At baseline, no significant differences were found between the groups for any clinical measure, but there were baseline differences for age ($P = 0·05$), gender ($P = 0·003$) and race ($P = 0·03$). Attendance at the intervention-site weekly meetings averaged forty-two participants (Table 1).

Clinical measures at baseline and at 22 weeks

Changes in weight, plasma lipid concentrations and blood pressure have been reported elsewhere[10]. In summary, the

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Table 1 Demographics of participants by group assignment

Characteristics	Intervention group (*n* 68)		Control group (*n* 45)		
	Mean	SD	Mean	SD	*P* value*
Age (years)	46	10·0	42	10·0	0·05
Gender, *n* (%)					0·00
Men	18	26·5	2	4·4	
Women	50	73·5	43	95·6	
Race, *n* (%)					0·03
White	30	44·1	31	68·9	
Black	27	39·7	10	22·2	
Asian	7	10·3	0		
Other	3	4·4	2	4·4	
Refused	1	1·5	2	4·4	
Ethnicity, *n* (%)					0·35
Hispanic	3	4·4	4	8·9	
Non-Hispanic	56	82·4	32	71·1	
Refused	9	13·2	9	20·0	

*P = significance of difference between groups.

intervention group experienced significant weight changes compared with the control group, including decreased weight, waist and hip circumference and waist-to-hip ratio (Table 2). BMI decreased 2·0 kg/m^2 in the intervention group and did not change in the control group. Total and LDL cholesterol concentrations decreased in the intervention group, compared with the control group, but between-group differences did not reach statistical significance (Table 2). There was no change in blood pressure for the intervention group, whereas blood pressure increased in the control group (Table 2). In addition, absenteeism due to health problems over the 22-week study period was lower among the intervention group, with a mean of 16·7 h (SE 2·5) in the intervention group, compared with 22·8 h (SE 3·6) in the control group[10].

Nutrient intake at baseline and 22 weeks

No significant differences in nutrient intake were identified between the groups at baseline except for sodium intake ($P = 0·04$). At the end of 22 weeks, the intervention group significantly reduced reported intake of energy, fat (total, saturated, monounsaturated, polyunsaturated and *trans* fats), protein, cholesterol, vitamin D, vitamin B_{12} and zinc, compared with baseline. Intervention group participants significantly increased the reported intake of carbohydrate, fibre, total vitamin A activity, β-carotene, vitamin K, vitamin C, folate, magnesium, potassium, sodium and iron. In comparison with the control group, the intervention group significantly reduced mean intake of energy, fat (total, saturated, monounsaturated and *trans*), cholesterol, protein, vitamin D and vitamin B_{12}. The intervention group significantly increased mean intake of carbohydrate, fibre, total vitamin A activity, β-carotene, folate, vitamin C, iron, sodium, magnesium and potassium, compared with the control group (Table 3).

The results of the ANCOVA were consistent with the *t* test analyses, with very minor modifications. For vitamin K, the difference between the groups was no longer significant ($P = 0·15$) because age and race were associated with vitamin K. For all other variables, there were minor changes in adjusted effect sizes and *P* values, but the differences were inconsequential.

Discussion

The intervention programme was associated with major changes in nutrient intake (exclusive of supplementation). These included significant reductions in total fat, *trans* fat, saturated fat, cholesterol, energy, protein and vitamin B_{12} and significant increases in carbohydrate, fibre, vitamin C, magnesium, potassium, β-carotene, total vitamin A activity, iron, vitamin K and sodium, compared with the control group.

The reductions in reported fat (from 37 % to 21 %), saturated fat (from 11 % to 5 %) and cholesterol (from 238 mg to 38 mg) intakes observed in the intervention group, if sustained over the long term, are of sufficient magnitude to be associated with a reduction in the risk of CVD[3,11,12] and may also be associated with reduced risk of cancer and diabetes[6,7,9].

The intervention group reported significant increases in the intake of micronutrients associated with disease prevention, including β-carotene[13,14], folate[15,16], magnesium[17] and vitamin C[18]. Increased intake of potassium is associated with a reduced risk of stroke[19,20] and, combined with a decreased intake of sodium, improves blood pressure control[21–27]. Although sodium per 4184 kJ increased on average among the intervention group, absolute sodium intake decreased by 240 mg during the study. Vitamin D and zinc intakes were below the nutrition recommendations of the Institute of Medicine at baseline and at 22 weeks for both groups.

Iron consumption in the intervention group increased significantly. Other studies using plant-based diets have shown similar increases[28,29]. Protein intake fell but remained adequate.

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Table 2 Changes in clinical variables by group assignment[10]

	Intervention (*n* 68)†						Control (*n* 45)†								
	Baseline		22 weeks		Within-group difference		Baseline		22 weeks		Within-group difference		Between-group difference (intervention − control)		
Clinical measures	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	95 % CI	*P* value
Weight (kg)	98·7	2·8	93·6	2·7	−5·1****	0·6	100·1	3·5	100·3	3·7	0·1	0·6	−5·3	−7·0, −3·5	<0·0001
Waist circumference (cm)	110·3	1·9	105·5	1·9	−4·7****	0·6	110·2	2·7	111·0	2·8	0·8	0·6	−5·5	−7·3, −3·7	<0·0001
Hip circumference (cm)	123·1	1·7	118·6	1·8	−4·5****	0·5	127·0	2·4	125·8	2·4	−1·2	0·6	−3·3	−4·8, −1·8	<0·0001
Waist-to-hip ratio	0·895	0·009	0·889	0·009	−0·006	0·003	0·866	0·011	0·880	0·011	0·014**	0·005	−0·020	−0·031, −0·009	0·0007
Cholesterol															
Total (mg/dl)	186·8	4·6	177·1	4·7	−9·8**	3·6	183·9	5·0	182·3	5·4	−1·6	3·5	−8·1	−18·6, +2·3	0·13
No medication changes‡	188·2	4·9	177·2	5·0	−11·0**	3·8	184·1	5·1	182·4	5·6	−1·7	3·6	−9·3	−20·1, +1·6	0·09
LDL (mg/dl)	103·5	3·9	99·1	4·0	−4·4	3·3	106·7	4·1	105·3	4·6	−1·4	3·2	−3·0	−12·5, +6·4	0·53
No medication changes‡	104·4	4·1	99·2	4·2	−5·2	3·5	106·8	4·2	105·4	4·7	−1·4	3·2	−3·7	−13·6, +6·1	0·45
HDL (mg/dl)	52·7	1·6	48·3	1·6	−4·3****	0·8	50·9	2·1	50·5	2·3	−0·4	0·9	−3·9	−6·4, −1·5	0·002
No medication changes‡	52·5	1·6	48·0	1·6	−4·5	0·9	50·8	2·1	50·4	2·4	−0·4	0·9	−4·1	−6·6, −1·6	0·001
TAG (mg/dl)	154·2	11·8	149·8	10·9	−4·4	6·5	131·6	9·2	135·2	13·1	3·5	10·1	−7·9	−30·5, +14·7	0·49
No medication changes‡	157·6	12·4	152·0	11·5	−5·6	6·9	132·4	9·4	136·0	13·4	3·6	10·3	−9·2	−32·8, +14·4	0·44
Blood pressure (mmHg)															
Systolic	118·8	1·7	118·8	1·7	0·0	1·4	116·4	1·6	122·1	2·5	5·7*	2·2	−5·7	−10·6, −0·9	0·03
No medication changes§	117·9	1·7	117·6	1·8	−0·3	1·3	115·6	1·7	122·3	2·9	6·6	2·3	−6·9	−11·8, −2·0	0·01
Diastolic	80·5	1·2	80·1	1·1	−0·4	1·1	78·4	1·3	83·5	1·5	5·1***	1·2	−5·6	−9·0, −2·2	0·002
No medication changes§	79·8	1·2	78·9	1·1	−0·9	1·2	77·9	1·4	83·2	1·7	5·3	1·3	−6·2	−9·8, +2·6	0·001
Haemoglobin A1c, %‖	7·4	0·3	7·1	0·5	−0·3	0·6	7·0	0·4	6·7	0·4	−0·3	0·2	0	−1·4, +1·4	0·97
No medication changes¶	7·8	0·5	6·8	0·2	−1·0	0·4	6·3	0·2	6·1	0·1	−0·2	0·1	−0·7		–

†*n* unless otherwise indicated; significantly different from baseline (within-group *t* test comparison): *$P<0·05$; **$P<0·01$; ***$P<0·001$; ****$P<0·0001$.
‡No change in antihyperlipidaemic medications between baseline and 22 weeks; intervention group, *n* 63; control group, *n* 44.
§No change in antihypertensive medications between baseline and 22 weeks; intervention group, *n* 59; control group, *n* 38.
‖Participants with diabetes; intervention group, *n* 10; control group, *n* 9.
¶Participants with diabetes with no change in diabetes medications between baseline and 22 weeks; intervention group, *n* 5; control group, *n* 6 (statistical tests were not conducted due to low numbers).

Table 3 Nutrient intakes before and after a 22-week low-fat vegan worksite programme

	Intervention group						Control group								
	Baseline		22 weeks		Difference		Baseline		22 weeks		Difference		Mean effect size†		
Nutrient§	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	95 % CI	*P* value‡
Energy (kcal)‖	1857·1	73·6	1451·5	58·3	−405·6***	68·2	1728·0	82·7	1584·9	84·2	−143·2	77·0	−262·5	−469·3, −55·7	0·01
Energy from fat (%)	36·1	1·0	20·6	0·9	−15·6	1·2	36·0	1·1	35·8	1·3	−0·2	1·3	−15·4	−18·9, −11·9	<0·0001
Energy from carbohydrate (%)	47·9	1·2	68·1	1·3	20·1	1·6	47·5	1·3	48·0	1·6	0·5	1·6	19·6	14·9, 24·3	<0·0001
Energy from protein (%)	15·7	0·4	13·8	0·4	−1·9	0·6	17·5	0·6	17·7	0·7	0·1	0·7	−2·0	−3·8, −0·2	0·03
Nutrients per 4184 kJ (1000 kcal)															
Fat (g)	40·1	1·1	23·3	1·1	−16·9***	1·3	40·5	1·2	40·1	1·4	−0·4	1·4	−16·5	−20·4, −12·5	<0·0001
n-3 (g)	1·0	0·1	0·8	0·1	−0·2**	0·1	1·0	0·1	0·9	0·1	−0·01	0·1	−0·2	−0·4, 0·02	0·1
Trans fat (g)	2·1	0·1	1·1	0·1	−1·0***	0·2	2·4	0·1	2·5	0·2	0·2	0·2	−1·2	−1·7, −0·6	<0·0001
MUFA (g)	14·96	0·5	8·0	0·5	−6·9***	0·6	14·9	0·5	14·8	0·6	0·1	0·7	−6·8	−8·7, −5·0	<0·0001
PUFA (g)	9·5	0·5	8·2	0·4	−1·3*	0·5	8·8	0·5	8·3	0·3	−0·4	0·5	−0·8	−2·3, 0·6	0·3
SFA (g)	12·4	0·5	5·1	0·4	−7·3***	0·5	13·4	0·5	13·3	0·6	−0·2	0·6	−7·2	−8·9, −5·5	<0·0001
Cholesterol (mg)	132·2	9·8	27·0	8·1	−104·6***	11·0	157·4	15·1	182·1	18·1	24·7	17·4	−129·3	−168·2, −90·4	<0·0001
Carbohydrate (g)	120·6	2·7	169·7	3·4	49·1***	4·1	117·5	3·2	118·9	4·0	1·4	4·2	47·7	35·8, 59·6	<0·0001
Fibre (g)	10·4	0·5	20·6	1·5	10·1***	0·9	8·9	0·5	10·2	1·0	1·2	1·1	8·9	6·2, 11·7	<0·0001
Protein (g)	39·8	1·0	34·5	1·1	−5·3***	1·4	43·5	1·4	44·1	1·6	0·6	1·7	−5·9	−10·2, −1·5	0·01
Total vitamin A activity (μg)	424·4	33·0	979·1	115·2	554·7***	114·3	416·7	37·1	570·4	63·0	153·7	76·0	401·0	98·8, 703·2	0·004
β-Carotene (μg)	1554·3	196·0	5201·2	673·8	3646·9***	677·0	1373·8	195·6	2030·4	402·0	656·6	453·5	2990·2	1197·5, 4782·9	0·0004
Vitamin D (μg)	1·7	0·2	1·0	0·1	−0·7**	0·2	1·9	0·3	2·4	0·3	0·5	0·3	−1·2	−1·9, −0·5	0·001
Vitamin E (mg)	6·3	0·5	7·2	0·5	0·9	0·6	5·4	0·6	6·6	1·0	1·2	0·9	−0·3	−2·3, 1·7	0·8
Vitamin K (μg)	70·0	7·9	170·7	26·9	100·8***	27·7	63·1	12·5	74·8	15·3	11·7	18·2	89·1	15·9, 162·3	0·01
Vitamin B_6 (μg)	1·0	0·1	1·1	0·1	0·1	0·1	1·0	0·1	1·1	0·1	0·1	0·1	−0·02	−0·2, 0·2	0·8
Vitamin B_{12} (μg)	2·7	0·3	1·3	0·2	−1·4***	0·3	2·9	0·3	2·8	0·3	−0·1	0·3	−1·4	−2·2, −0·5	0·002
Folate (μg)	230·5	12·9	375·9	15·9	145·4***	15·6	199·9	11·7	227·2	16·6	27·3	16·1	118·1	72·2, 163·9	<0·0001
Vitamin C (mg)	40·1	3·3	72·1	5·9	32·0***	5·8	32·1	2·9	35·1	3·7	3·0	3·7	29·0	13·8, 44·1	<0·0001
Calcium (mg)	365·4	19·7	378·2	16·0	12·9	21·2	417·1	26·0	420·6	24·4	3·5	27·2	9·4	−58·2, 77·0	0·8
Iron (mg)	8·0	0·4	11·4	0·4	3·4***	0·5	7·2	0·3	8·0	0·5	0·9	0·5	2·5	1·1, 3·9	<0·0001
Magnesium (mg)	147·5	5·8	215·8	7·0	68·4***	8·0	141·4	7·3	147·7	7·5	6·3	7·9	62·1	38·9, 85·3	<0·0001
Potassium (mg)	1291·9	51·8	1758·2	61·4	466·3***	61·3	1309·3	52·6	1283·9	54·2	−25·4	54·2	491·7	319·5, 663·9	<0·0001
Selenium (μg)	103·2	30·0	51·4	1·9	−51·8	30·2	58·3	2·1	64·1	2·8	5·8	3·0	−57·7	−130·6, 15·3	0·1
Sodium (mg)	1646·7	44·4	1991·9	66·3	345·2***	67·4	1797·1	61·3	1940·4	70·8	143·3	63·5*	201·8	9·2, 394·4	0·04
Zinc (mg)	5·5	0·3	5·0	0·2	−0·5*	0·3	5·8	0·2	6·1	0·4	0·3	0·4	−0·9	−1·8, 0·1	0·1

†From lower to upper confidence level.
‡*P* values are for *t* tests of comparisons of between-group (vegan *v.* control diet) changes (baseline to 22 weeks).
§Dietary data were reported from 3 d food records.
‖1 kcal = 4·184 kJ.
Significantly different from baseline (within-group *t* test comparison): *$P < 0·05$; **$P < 0·01$; ***$P < 0·001$.

Although dietary intake of vitamin B_{12} decreased significantly from baseline to 22 weeks for the intervention group, this calculation did not include the recommended supplementation.

The present study shows that with education on the use of low-fat vegan diets and modest worksite support, employees can implement changes in their diets that, if sustained, may reduce the risk of common and costly diseases such as heart disease, cancer and diabetes[3–9].

A strength of the present study is its translational design, showing the effect of a dietary programme in the work environment, whereas the limitations include the fact that neither the sites nor the volunteers were randomised and nutrient intake was self-reported. The trial was limited to 22 weeks.

Conclusions

The intervention group reported dramatic changes in the intake of nutrients linked to decreased risk of obesity, heart disease and other common chronic diseases. A worksite wellness programme using a low-fat vegan diet is an effective way to encourage employees to choose healthful options.

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References

1. American Heart Association (2008) Cardiovascular disease statistics. http://www.americanheart.org/presenter.jhtml?identifier=4478 (accessed December 2008).
2. Pleis JR & Lethbridge-Çejku M (2007) Summary health statistics for US adults: National Health Interview Survey, 2006. National Center for Health Statistics. *Vital Health Stat* **10**, 1–153.
3. Ornish D, Brown SE, Scherwitz LW *et al.* (1990) Can lifestyle changes reverse coronary heart disease? The Lifestyle Heart Trial. *Lancet* **336**, 129–133.
4. Ornish D, Brown SE, Scherwitz LW *et al.* (1998) Intensive lifestyle changes for reversal of coronary heart disease. *JAMA* **280**, 2001–2007.
5. Dewell A & Ornish D (2007) Plant-based dietary patterns in the control of obesity and cardiovascular risk. *Curr Cardiovasc Rep* **1**, 9–15.
6. Barnard ND, Cohen J, Jenkins DJ *et al.* (2006) A low-fat vegan diet improves glycemic control and cardiovascular risk factors in a randomized clinical trial in individuals with type 2 diabetes. *Diabetes Care* **29**, 1777–1783.
7. Ornish D, Magbanua MJ, Weidner G *et al.* (2008) Changes in prostate gene expression in men undergoing an intensive nutrition and lifestyle intervention. *Proc Natl Acad Sci USA* **105**, 8369–8374.
8. Ornish D, Weidner G, Fair WR *et al.* (2005) Intensive lifestyle changes may affect the progression of prostate cancer (discussion 1069–1070). *J Urol* **174**, 1065–1069.
9. Berkow SE, Barnard ND, Saxe GA *et al.* (2007) Diet and survival after prostate cancer diagnosis. *Nutr Rev* **65**, 391–403.
10. Ferdowsian HR, Barnard ND, Hoover VJ *et al.* (2009) A multi-component intervention reduces body weight and cardiovascular risk at a GEICO corporate site. *Am J Health Promot* (In the Press).
11. Frattaroli J, Weidner G, Merritt-Worden TA *et al.* (2008) Angina pectoris and atherosclerotic risk factors in the multisite cardiac lifestyle intervention program. *Am J Cardiol* **101**, 911–918.
12. Esselstyn CB Jr (2001) Resolving the coronary artery disease epidemic through plant-based nutrition. *Prev Cardiol* **4**, 171–177.
13. Watson RR, Prabhala RH, Plezia PM *et al.* (1991) Effect of beta-carotene on lymphocyte subpopulations in elderly humans: evidence for a dose–response relationship. *Am J Clin Nutr* **53**, 90–94.
14. Makinodan T, Lubinski J & Fong TC (1987) Cellular, biochemical, and molecular basis of T-cell senescence. *Arch Pathol Lab Med* **111**, 910–914.
15. Bazzano LA, He J, Ogden LG *et al.* (2002) Dietary intake of folate and risk of stroke in US men and women: NHANES I Epidemiologic Follow-up Study. National Health and Nutrition Examination Survey. *Stroke* **33**, 1183–1188.
16. Voutilainen S, Rissanen TH, Virtanen J *et al.* (2001) Low dietary folate intake is associated with an excess incidence of acute coronary events: The Kuopio Ischemic Heart Disease Risk Factor Study. *Circulation* **103**, 2674–2680.
17. Witte KK, Clark AL & Cleland JG (2001) Chronic heart failure and micronutrients. *J Am Coll Cardiol* **37**, 1765–1774.
18. Joshipura KJ, Hu FB, Manson JE *et al.* (2001) The effect of fruit and vegetable intake on risk for coronary heart disease. *Ann Intern Med* **134**, 1106–1114.
19. Fang J, Madhavan S & Alderman MH (2000) Dietary potassium intake and stroke mortality. *Stroke* **31**, 1532–1537.
20. Khaw KT & Barrett-Connor E (1987) Dietary potassium and stroke-associated mortality. A 12-year prospective population study. *N Engl J Med* **316**, 235–240.
21. Whelton PK, He J, Appel LJ *et al.* (2002) Primary prevention of hypertension: clinical and public health advisory from the National High Blood Pressure Education Program. *JAMA* **288**, 1882–1888.
22. He J & Whelton PK (1999) What is the role of dietary sodium and potassium in hypertension and target organ injury? *Am J Med Sci* **317**, 152–159.
23. Adrogue HJ & Wesson DE (1996) Role of dietary factors in the hypertension of African Americans. *Semin Nephrol* **16**, 94–101.
24. Geleijnse JM, Kok FJ & Grobbee DE (2004) Impact of dietary and lifestyle factors on the prevalence of hypertension in Western populations. *Eur J Public Health* **14**, 235–239.
25. Forman JP, Rimm EB, Stampfer MJ *et al.* (2005) Folate intake and the risk of incident hypertension among US women. *JAMA* **293**, 320–329.

26. Block G (2002) Ascorbic acid, blood pressure, and the American diet. *Ann N Y Acad Sci* **959**, 180–187.
27. Chen J, He J, Hamm L *et al.* (2002) Serum antioxidant vitamins and blood pressure in the United States population. *Hypertension* **40**, 810–816.
28. Davey GK, Spencer EA, Appleby PN *et al.* (2003) EPIC-Oxford: lifestyle characteristics and nutrient intakes in a cohort of 33 883 meat-eaters and 31 546 non meat-eaters in the UK. *Public Health Nutr* **6**, 259–269.
29. Turner-McGrievy GM, Barnard ND, Cohen J *et al.* (2008) Changes in nutrient intake and dietary quality among participants with type 2 diabetes following a low-fat vegan diet or a conventional diabetes diet for 22 weeks. *J Am Diet Assoc* **108**, 1636–1645.