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Coronary Health Improvement Project (CHIP) is associated with improved nutrient intake and decreased depression

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Abstract

Objective: We evaluated the efficacy of the Coronary Health Improvement Project (CHIP) at lowering depression by modifying selected daily nutrients from food.

Methods: A randomized controlled trial was used, with 348 participants 24 to 81 y of age from metropolitan Rockford, Illinois. Participants were assessed at baseline and at 6 wk and 6 mo of follow-up. The Beck Depression Inventory (BDI) was used to measure depression.

Results: Those in the intervention group compared with the control group were 63% more likely to show a decrease in BDI through 6 wk ($P < 0.0001$) and 34% more likely to experience a decrease through 6 mo ($P < 0.0001$). Baseline levels of saturated fat were negatively associated with BDI ($P < 0.0001$) after adjusting for age, sex, exercise, body mass index, and exercise. Decrease in saturated fat over the study period was directly associated with a decrease in BDI. Increase in pyridoxine (B6) was also associated with a decrease in BDI. The intervention indirectly lowered BDI by lowering saturated fat and increasing dietary pyridoxine (B6). It also directly lowered BDI, possibly because of social interaction, positive reinforcement, and distraction.

Conclusion: The CHIP, which improves daily nutrients from food and cardiovascular risk factors, also lowers depression.

Keywords: Cardiovascular risk factors; Coronary Health Improvement Project; Depression; Nutrition

Introduction

Coronary heart disease, sometimes referred to as ischemic heart disease, is the leading cause of death in the United States. It refers to the accumulation of atheromatous plaques within the walls of the arteries that supply the heart muscle with oxygen and nutrients. Modifiable risk factors for this disease include high blood pressure, high blood cholesterol, cigarette smoking, physical inactivity, diabetes, obesity, dietary factors, and environmental tobacco smoke. Alcohol use and stress may also contribute to the disease. These risk factors have also been associated with a host of other chronic conditions, including hypertension, sickle cell disease, stroke, diabetes mellitus, and cancer.

Studies have demonstrated that health promotion efforts can positively affect many different disease states [1–7]. Most of these studies broadly address chronic conditions because behavior change has the potential to affect many different types of chronic illness. Behavior change has been shown to prevent and reverse chronic conditions. Slowing the progression of disease has also been shown to improve the success of revascularization procedures (e.g., angioplasty or coronary bypass) among patients with heart disease. However, despite the efficacy of chronic-disease prevention programs, few have been successfully translated and disseminated to benefit large numbers of people [8–10].

The Coronary Health Improvement Project (CHIP) is a health education intervention that promotes better choices in nutrition, physical activity, and tobacco use. Its goal is to improve patient understanding of the importance of living a healthy lifestyle, which includes appropriate nutrition and physical activity and understanding risk factors associated with coronary heart disease [11,12]. To date, more than 40000 individuals have participated in the CHIP in a number

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of settings, including faith-based communities, worksites, hospitals, and municipalities. The efficacy of the CHIP at improving health knowledge, physical activity, and diet has been demonstrated through 6 mo of follow-up in a randomized controlled trial [13–15].

Many of the risk factors the CHIP is designed to affect are also related to depression. Some studies have associated high cholesterol, body mass index (BMI), smoking, physical inactivity, and inadequate nutrient intake with depression [16–22]. This study evaluated the efficacy of the CHIP at lowering depression by modifying selected daily nutrients from food.

Materials and methods

Subject recruitment and design

Study participants were recruited through the Swedish-American Center for Complementary Medicine in Rockford, Illinois and surrounding metropolitan areas. Methods for recruitment included targeted advertising and marketing through the Swedish-American Medical Group, corporate client sites, the Centers for Excellence, and CHIP alumni groups. The cost for participation was $290, which was returned if the person attended at least three-fourths of the classes. Inclusion into the study required that the individual be at least 18 y of age. In addition, participants were encouraged to participate in the program with a spouse or significant other. Those who participated as a couple were randomly assigned as a couple to begin the CHIP. In previous studies, those in the latter group served as controls through 6 mo of follow-up [13–15]. No significant difference was seen in basic demographics between the couples and individuals. All participants provided informed consent and the study was approved by the institutional review board of the Swedish-American Health System on August 29, 2002.

The study coordinator supervised participant sign-up and randomization. Three hundred seventy-seven individuals completed baseline data collection. Randomization was completed using a random number generator. The intervention group started the class in March 2003 and the control group started the class 6 mo later. The controls were instructed to continue with their current lifestyle habits during the 6-mo wait. Twenty-nine individuals were lost to follow-up, leaving 348 participants for evaluation. This study used the intent-to-treat method, and baseline scores were carried forward through 6 wk and 6 mo of follow-up.

Intervention

The CHIP was advertised as a health education, cardiovascular, community-based primary prevention program. Potential participants were informed of the topics covered in the program, which included atherosclerosis, coronary risk factors, obesity, dietary fiber, smoking, diabetes, hypertension, hypercholesterolemia, dietary fat and cholesterol, exercise, osteoporosis, cancer, lifestyle and health, the optimal diet, behavioral change, and self-worth. Individuals who enrolled in the program participated in an intensive 4-wk class, which met 2 h/d, four times per week, totaling approximately 32 h in class. Development of the curriculum, class design, alumni association, and take-home assignments stemmed from theory-based intervention planning [23–25]. The CHIP incorporated the learning theory (behaviorism) where changes were encouraged in physical and dietary behaviors using health education and positive reinforcement. The CHIP alumni program was designed to help participants further maintain positive behavior changes through peer influence and positive support.

Participants began by making dietary and exercise goals. The dietary goals involved adopting a diet that emphasizes unrefined food, with more consumption of whole grains, legumes, vegetables, and fresh fruits. The recommended diet was largely unrefined complex carbohydrates (65% to 70% of total calories), low in fat (i.e., <20% of total daily caloric intake), animal protein, sugar, and salt, and virtually free of cholesterol. The exercise goal consisted of exercising at least 30 min/d. A pedometer was provided and an exercise log kept to record the distance walked each day. Participants received considerable encouragement from staff to maintain these goals. After completing the 4-wk course, participants were encouraged to join the Rockford CHIP alumni organization per annual cost of $25 for an individual or $35 per couple. Alumni were invited to several lectures covering selected health living topics and were provided with ways to maintain their dietary and exercise goals. A monthly newsletter was also sent to alumni, with information about healthy dinners, walking groups, support-group meetings, and other health-promoting community events.

Measurements

Data were collected by a registered nurse at baseline, 6 wk, and 6 mo. Selected demographic data were recorded at baseline (age, sex, race, marital status, income, education, and employment status). An indicator variable was created to identify if a person had a history of cardiovascular disease based on responses to whether or not that person had a history of angina, myocardial infarction, heart failure, or stroke.

Physical activity was based on a 7-d self-recorded pedometer log, which was maintained by each participant. Participants wore the Walk4Life Model 2000 Life Stepper pedometer (Plainfield, IL, USA) on a belt at the right hip directly above the right knee cap each day. Immediately before going to bed, the pedometer counts for the day were recorded and the number reset. Strike counts from pedometers are a valid and reliable method of monitoring and measuring free-living physical activity [26–28]. Ex-

ercise was also self-reported using the categories none, mild (2–3 d/wk), moderate (3–5 d/wk), and vigorous (4–6 d/wk).

Weight and height were measured using standard medical scales recently calibrated by the Biometrics Department of the Swedish-American Health System. BMI was determined by dividing body weight (kilograms) by height (meters) squared. This study also considered current tobacco smoking and alcohol drinking. Smoking status and the level of alcohol drinking were self-reported, with alcohol consumption reflecting the number of drinks weekly.

To assess dietary intake, the Block 98 full-length dietary questionnaire (Block 98.2, Block Dietary Data Systems, Berkeley, CA, USA) was used. The Block 98 questionnaire has been extensively studied and validated [29–31]. It is self-reported and optically scanned and scored. Daily nutrients from food, obtained from the Block 98, considered in this study were calories, protein, calcium, total fat, cholesterol, saturated fat, polyunsaturated fat, monounsaturated fat, fiber, thiamin (B1), riboflavin (B2), and pyridoxine (B6).

The Beck Depression Inventory (BDI) is a widely accepted, validated instrument appropriate for assessing depressive symptoms in a "normal" population [32,33]. The present study used the shorter BDI (BDI-SF), which has been shown to have a high degree of internal consistency and correlation with the original BDI [34]. The reliability and validity of the BDI-SF in older adults has been established previously [35]. Categories of depression for the BDI-SF have been previously established as 0–4 (not depressed), 5–7 (mildly depressed), 8–15 (moderately depressed), and 16–39 (severely depressed).

Lipid analysis followed the lipid standards provided by the Centers for Disease Control and Prevention. Glucose, total cholesterol, high-density lipoprotein (HDL), and triacylglycerol concentrations were determined using Beckman-Coulter LX-20 instrumentation (Beckman Coulter, Inc., Fullerton, CA, USA). Glucose measurements were obtained with the oxygen-rate method using a Beckman oxygen electrode; cholesterol measurements were obtained with the timed-endpoint enzymatic method using cholesterol oxidase; triacylglycerol measurements were obtained with the timed-endpoint enzymatic method using glycerol kinase; and HDL measurements were obtained with the homogeneous timed-endpoint method using polyanion detergent to separate HDL and non-HDL lipids. For participants with triacylglycerol values below 400 mg/dL, low-density lipoprotein values were calculated as follows: low-density lipoprotein = total cholesterol − HDL − (triacylglycerols/5) [36]. Glucose measurements were determined using a Kodak Ektachem (Kodak, Rochester, NY, USA). Trained program staff took blood pressure measurements. Blood pressure was measured in participants after a 5-min rest using the guidelines set forth by the American Heart Association.

Statistical methods

Frequency distributions and means were generated to describe the variables. Mean differences between independent groups were assessed using the t and F statistics. Cross-tabulations were used to perform bivariate analyses between selected variables, with statistical significance based on the chi-square test for independence. Modeled repeated measurements data at baseline, 6 wk, and 6 mo were considered, with Wilks' λ used for assessment. The polynomial option was used to assess trend. Regression techniques were used to evaluate the association between 6-wk and 6-mo change scores in BDI and changes in selected nutrients from foods. Analyses were performed using SAS 9.1 (SAS Institute, Cary, NC, USA). Statistical significance was based on the 0.05 level.

Results

A summary of demographic, behavior, and health variables at baseline is presented in Table 1. Intervention and control participants were combined because there was no significant difference in the baseline levels of these variables, with the exception of BMI and glucose. Participants in the intervention group had significantly higher levels of BMI ($P = 0.0030$) and glucose ($P = 0.0247$) at baseline. Average daily vitamins from supplements (vitamin C, vitamin D, zinc, vitamin B1, pyridoxine [B6], and vitamin B12) were also collected at baseline. There were no significant differences in supplement use between intervention and control participants.

The BDI scores at baseline ranged from 0 to 27 (mean ± SD 4.4 ± 4.6, median 3.0). Repeated measures analysis indicated a significant time effect ($P < 0.0001$) and group by time effect ($P < 0.0001$) for BDI. Decrease in mean BDI through 6 wk was significantly greater for those in the intervention group than in the control group (−2.6 versus −0.4, $P < 0.0001$), with those in the intervention group 63% more likely to show a decrease ($P < 0.0001$). After 6 mo, those in the intervention group continued to show a significantly greater decrease in BDI (−2.4 and −0.7, $P < 0.0001$), with those in the intervention group 34% more likely to show a decrease ($P < 0.0001$).

Of the variables listed in Table 1 and the vitamin supplements considered, BDI was significantly associated only with age, sex, education, exercise, and BMI (Table 2). The BDI was highest in younger groups, females, those with a high school degree or some college education, those with lower levels or no exercise, and those with higher BMI. Using stepwise regression with these variables retained in the model and selected nutrients from foods also considered (calories, protein, cholesterol, carbohydrates, calcium, total dietary fat, saturated fat, polyunsaturated fat, monounsaturated fat, thiamin [B1], riboflavin [B2], niacin [B3], pyri-

Table 1
Summary of demographic, behavior, and health variables at baseline among intervention and control participants in the Coronary Health Improvement Project, Rockville, Illinois

Demographics	No. (%)	Behaviors	No. (%)	Health	No. (%)
Age group		Exercise		Beck Depression Inventory	
24–<40	56 (16)	None	135 (39)	Not depressed (0–4)	222 (64)
40–<50	104 (30)	Mild 2–3 d/wk	115 (33)	Mildly depressed (5–7)	56 (16)
50–<60	114 (33)	Moderate 3–5 d/wk	76 (22)	Moderately depressed (8–15)	58 (17)
≥60	74 (21)	Vigorous 4–6 d/wk	20 (6)	Severely depressed (16–39)	12 (3)
Sex		Tobacco smoker		History of cardiovascular disease	
Male	98 (28)	Yes	9 (3)	Yes	36 (10)
Female	250 (72)	No	339 (97)	No	312 (90)
Race		Alcohol drinker		Body mass index	
White	327 (95)	Yes	153 (45)	Normal	67 (19)
Non-white	19 (5)	No	190 (55)	Overweight	101 (29)
Married		Coffee/tea		Obese	180 (52)
Yes	261 (76)	Yes	261 (76)	Systolic blood pressure (mmHg)	
No	81 (24)	No	81 (24)	Normal (<120)	112 (32)
Employed				Prehypertensive (120–139)	148 (43)
Yes	278 (81)			High (≥140)	88 (250)
No	64 (19)			Diastolic blood pressure (mmHg)	
Annual income				Normal (<80)	199 (57)
$0–$40 000	88 (26)			Prehypertensive (80–89)	107 (31)
$40 001–$60 000	78 (23)			High (≥90)	42 (12)
≥$60 000	174 (51)			Serum total cholesterol (mg/dL)	
Education				Normal (<200)	207 (60)
Less than high school	11 (3)			Borderline (200–239)	106 (31)
High school	83 (24)			High (≥240)	30 (9)
Some college	97 (28)			Triacylglycerols (mg/dL)	
College	77 (22)			Normal (<150)	257 (75)
Postgraduate	77 (22)			Borderline (150–199)	43 (12.5)
				High (≥200)	43 (12.5)
				Glucose (mg/dL)	
				Normal (<110)	281 (82)
				Impaired fasting glucose (110–125)	34 (10)
				Diabetes (≥126)	28 (8)

doxine [B6], and zinc), only saturated fat significantly added to fit of the model. Exercise became insignificant.

Repeated measures analysis showed that the time effect of BDI was not significantly associated with age, sex, education, baseline levels of exercise, or BMI. However, it was significantly associated with the baseline level of saturated fat ($P = 0.0127$). After saturated fat, none of the other selected nutrients from food were significant. Mean BDI according to quartile groupings of saturated fat is presented Table 3. Change in BDI is also presented across the quartile groupings of saturated fat. There was no difference between the intervention and control groups in BDI across the quartile groupings at baseline, but there was at 6 wk and 6 mo. In addition, through 6 wk and 6 mo the significant intervention effect was more pronounced at higher levels of saturated fat.

Repeated measures analysis showed that the time effect for selected nutrients from food was significantly influenced by intervention status (Table 4). The intervention significantly lowered calories, protein, dietary cholesterol, calcium, total fat, saturated fat, monounsaturated fat, polyunsaturated fat, total dietary fiber, riboflavin (B2), niacin (B3), and zinc through 6 wk and 6 mo of follow-up. In addition, the intervention significantly increased vitamin C and folate through 6 wk and 6 mo of follow-up.

Through 6 wk of follow-up, 10.4% of the variation in change of BDI was explained by the group variable, with a slope estimate from the regression model of -2.2 ($P < 0.0001$). Through 6 mo of follow-up, 7.1% of the variation in change in BDI was explained by the intervention, with the slope estimate of -1.7 ($P < 0.0001$). Stepwise regression was used with change in BDI through 6 wk as the dependent variable and intervention status and change scores through 6 wk in BMI, total steps, calories, protein calcium, carbohydrates, total fat, saturated fat, monounsaturated fat, polyunsaturated fat, thiamin (B1), riboflavin (B2), niacin (B3), zinc, vitamin C, and folate as the independent variables. The best fitting model indicated that 1.5% of the variation in change in BDI was explained by change in saturated fat (slope = 0.06, $P = 0.0006$), 1.9% was explained by the change in pyridoxine (B6; slope = -0.8, $P = 0.0064$), and 10.4% was explained by group (slope = -1.6, $P < 0.0001$). Using stepwise regression with the same variables, but reflecting change scores through 6 mo, 8.2% of the variation in change in BDI was explained by the change in saturated fat (slope = 0.08, $P <$

Table 2
Beck Depression Inventory at baseline according to selected variables among intervention and control participants in the Coronary Health Improvement Project, Rockville, Illinois

Variable	Mean	F statistic	
		P^*	$P^\dagger$
Age group			
24–<40	6.2	0.0018	0.0004
40–<50	4.6		
50–<60	3.7		
≥60	3.6		
Sex			
Male	3.4	0.0098	0.0227
Female	4.7		
Education			
Less than high school	2.7	0.0052	0.0036
High school	4.9		
Some college	5.6		
College	3.3		
Postgraduate	3.6		
Exercise			
None	5.6	<0.0001	0.0001
Mild 2–3 d/wk	4.6		
Moderate 3–5 d/wk	2.3		
Vigorous 4–6 d/wk	3.0		
Body mass index			
Normal	2.9	0.0032	0.0061
Overweight	3.6		
Obese	5.3		

* Based on type I sums of squares.
† Based on type III sums of squares after adjusting for the other variables listed.

0.0001), 2.6% was explained by the change in pyridoxine (B6; slope = −0.6; $P = 0.0217$), but only 1.0% was explained by group (slope = −0.7, $P = 0.0599$).

Discussion

The primary aim of this study was to evaluate the efficacy of the CHIP at lowering depression by modifying daily nutrients from selected foods. Baseline levels of saturated fat and change in saturated fat and pyridoxine (B6) were the primary contributors for lower BDI. There was also a significant group effect on the change in BDI through 6 wk, but the group effect became insignificant after 6 mo after adjusting for change in saturated fat and pyridoxine (B6).

The observed association between BDI and exercise at baseline is consistent with other studies. Research has shown that exercise improves mood and decreases anxiety and depression [37–40]. This may be partly explained by the fact that physical activity releases endorphins and monoamines, which help to decrease clinical depression and depressive symptoms [41,42]. However, exercise at baseline was not associated with change in BDI over time, nor was change in total steps associated with change in BDI, after adjusting for change in saturated fat and change in pyridoxine (B6).

Although BDI and BMI were significantly correlated at baseline, the stepwise regression analysis did not identify change in BMI as being associated with change in BDI after inclusion of group, saturated fat, and pyridoxine (B6) in the model. Nevertheless, research has shown that when obese or overweight individuals lose weight, health-related quality of life, including mental well-being, is improved and anxiety and depressive symptoms decrease [43–45]. Further, weight loss among overweight and obese individuals can decrease blood pressure, lower persistent high levels of blood glucose associated with diabetes, bring blood concentrations of cholesterol and triacylglycerols down, improve sleep, decrease osteoarthritis of the weight-bearing joints, and increase self-esteem while lowering depression [46,47].

Those in the intervention group compared with the control group showed a significantly greater decrease in BDI through 6 wk and 6 mo of follow-up. The significantly greater decrease in BDI for those in the intervention group compared with the control group was more pronounced for those with higher levels of saturated fat at baseline. This may be because individuals with high levels of saturated fat at baseline had the greatest potential for decreasing their saturated fat level and, consequently, decreasing their BDI.

Through 6 wk of follow-up, decrease in BDI was significantly greater for those in the intervention group, for those who had a decrease in saturated fat, and for those who had

Table 3
Mean Beck Depression Inventory at baseline and their change scores through 6 wk and 6 mo of follow-up according to saturated fat and intervention status among participants in the Coronary Health Improvement Project, Rockville, Illinois

Food nutrient	Baseline		Change through 6 wk		Change through 6 mo	
	Intervention	Control	Intervention	Control	Intervention	Control
Saturated fat (g)						
Quartile 1	2.9†	3.5	−1.4*‡	−0.7	−1.2*‡	−0.9
Quartile 2	3.7	3.5	−1.9	−0.2	−1.9	−0.9
Quartile 3	5.0	4.2	−3.2	−0.7	−2.5	−0.2
Quartile 4	7.0	4.4	−3.5	−0.2	−3.5	−0.7

* $P < 0.05$ for group effect.
† $P < 0.05$ for food nutrient effect.
‡ $P < 0.05$ for group by food nutrient interaction effect.

Table 4
Repeated measures for baseline, 6-wk, and 6-mo measurements of daily nutrients from foods and change in these nutrients over time according to intervention status among participants in the Coronary Health Improvement Project, Rockville, Illinois*

Nutrients from foods	Repeated measures		6 wk		6 mo	
	Time effect	Group × time effect	Mean change	P	Mean change	P
Kilocalories						
Intervention	<0.0001	<0.0001	−406	0.0006	−556	<0.0001
Control			−150		−142	
Protein (g)						
Intervention	<0.0001	<0.0001	−18	<0.0001	−24	<0.0001
Control			−5		−3	
Calcium (mg)						
Intervention	<0.0001	<0.0001	−177	0.0004	−270	<0.0001
Control			−53		−62	
Dietary cholesterol (mg)						
Intervention	<0.0001	<0.0001	−98	<0.0001	−121	<0.0001
Control			−12		10	
Total fat (g)						
Intervention	<0.0001	<0.0001	−33	<0.0001	−38	<0.0001
Control			−7		−5	
Saturated fat (g)						
Intervention	<0.0001	<0.0001	−11	<0.0001	−13	<0.0001
Control			−3		−1	
Monounsaturated fat (g)						
Intervention	<0.0001	<0.0001	−13	<0.0001	−15	<0.0001
Control			−3		−2	
Polyunsaturated fat (g)						
Intervention	<0.0001	<0.0001	−7	<0.0001	−8	<0.0001
Control			−2		−2	
Total dietary fiber (g)						
Intervention	<0.0001	<0.0001	8	<0.0001	−7	<0.0001
Control			0		−1	
Thiamin (B1) (mg)						
Intervention	0.3347	0.4057				
Control						
Riboflavin (B2) (mg)						
Intervention	<0.0001	<0.0001	−0.24	0.0204	−0.40	<0.0001
Control			−0.09		−0.08	
Niacin (B3) (mg)						
Intervention	<0.0001	<0.0001	−4	0.0018	−5	<0.0001
Control			−1		−1	
Pyridoxine (B6) (mg)						
Intervention	0.0649	0.0555				
Control						
Vitamin C (mg)						
Intervention	<0.0001	<0.0001	27	<0.0001	9	<0.0001
Control			−2		−15	
Vitamin D (IU)						
Intervention	<0.0001	0.2377	−2	0.8737	−8	0.3433
Control			−2		−9	
Folate (μg)						
Intervention	0.0046	<0.0001	66	0.0003	36	<0.0001
Control			−9		−24	
Magnesium (mg)						
Intervention	0.3181	0.1476				
Control						
Zinc (mg)						
Intervention	<0.0001	<0.0001	−2.0	0.0060	−3.0	<0.0001
Control			−0.5		−0.4	

* Repeated measures *P* values are based on Wilks' λ. The *P* values assessing mean change scores are based on the F statistic.

an increase in pyridoxine (B6). Three hypotheses may help explain the significant group effect. First, the social interaction hypothesis claims that increased interaction and mutual support can contribute to positive mental health [48]. Second, the self-efficacy hypothesis claims that meeting challenging experiences may improve mood and self-

confidence as the effort is maintained [49]. Third, the distraction hypothesis claims that mental diversion from challenging experiences, such as participation in the intervention, may result in improved mood [50]. Because the class lasted 4 wk, the benefits of social interaction, positive reinforcement, and distraction from the intervention were likely experienced during this time. Participation in the alumni program thereafter would also contribute to social interaction, positive reinforcement, and distraction, but to a much lesser degree. Through 6 mo, change in BDI was associated only with change in saturated fat and change in fiber. The finding that the group variable was significant only through 6 wk supports the idea that social interaction, positive reinforcement, and distraction were insufficient after the classroom intervention to maintain significantly lower BDI for those in the intervention group.

Few studies have addressed the association between dietary fat and depression. Yet of those studies available, they conclude that saturated fat intake is associated with depression, but varying results exist regarding mono- and polyunsaturated fats [51,52]. In a consistent manner, this study found that those who decreased their dietary saturated fat through 6 mo of follow-up were 34% more likely to lower their depression score. Also, after including change in saturated fat in the model, change in mono- and polyunsaturated fats were not significantly associated with change in BDI.

The results are consistent with other studies showing that deficiency in these B vitamins can cause depression [53–55]. At baseline, BDI was significantly associated with thiamine (B1), riboflavin (B2), niacin (B3), and pyridoxine (B6; data not shown). These B vitamins were highly positively correlated among themselves at baseline, as were their change scores through 6 wk and 6 mo (data not shown). Recall that the best fitting models showed that decrease in BDI was associated with being in the intervention group, decrease in saturated fat, and increase in pyridoxine (B6). If change in pyridoxine (B6) were not included in the stepwise models, change in thiamin (B1) would have been significant. If changes in pyridoxine (B6) and change in thiamin (B1) were not included in the stepwise model, then change in niacin (B3) would have been significant. However, change in riboflavin would not have entered into the 6-wk or 6-mo models in the absence of the other B vitamins.

Limitations of this study include self-selection and self-reported responses. Participants in the intervention and control groups were interested in making health behavior changes. They were also primarily white, married, and had an annual income of at least $60 000. Hence, generalization of the results should consider this. In addition, self-reported responses may be biased. However, weight and height, which were used to compute BMI, were acquired objectively from a trained nurse, physical activity was monitored using pedometer readings, cardiovascular risk information was collected from a blood draw and standardized tests, and individuals were encouraged to complete the questionnaires as accurately and honestly as possible.

Conclusion

This study shows that daily intake of saturated fat is directly associated with depression. Decrease in saturated fat over the study period was directly associated with a decrease in BDI. Increase in pyridoxine (B6) was also associated with a decrease in BDI. In addition to the intervention indirectly lowering BDI by lowering saturated fat and increasing dietary pyridoxine (B6), it appears to have directly lowered BDI through 6 wk because of social interaction, positive reinforcement, and distraction. This direct effect on lowering BDI was marginally insignificant through 6 mo.

References

[1] Roberts CK, Barnard RJ. Effects of exercise and diet on chronic disease. J Appl Physiol 2005;98:3–30.
[2] Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med 1997; 336:1117–24.
[3] Appel LJ, Champagne CM, Harsha DW, Cooper LS, Obarzanek E, Elmer PJ, et al. Effects of comprehensive lifestyle modification on blood pressure control: main results of the PREMIER clinical trial. JAMA 2003;289:2083–93.
[4] Appel LJ, Sacks FM, Carey VJ, Obarzanek E, Swain JF, Miller ER III, et al. Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. JAMA 2005;294:2455–64.
[5] Howard BV, Van Horn L, Hsia J, Manson JE, Stefanick ML, Wassertheil-Smoller S, et al. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. JAMA 2006;295:655–66.
[6] Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med 2002;346: 393–403.
[7] de Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. Circulation 1999;99:779–85.
[8] Clark GN. Improving the transition from basic efficacy research to effectiveness studies: methodological issues and procedures. J Consult Clin Psychol 1995;63:718–25.
[9] Glasgow RE, Lichtenstein E, Marcus AC. Why don't we see more translation of health promotion research to practice? Rethinking the efficacy-to-effectiveness transition Am J Public Health 2003;93:1261–7.
[10] Committee on Quality Health Care in America. Crossing the quality chasm: a new health system for the 21st century. Washington, DC: National Academy Press; 2001.
[11] Englert HS, Diehl HA, Greenlaw RL. Rationale and design of the Rockford CHIP, a community-based coronary risk reduction program: results of a pilot phase. Prev Med 2004;38:432–41.
[12] Diehl HA. Coronary risk reduction through intensive community-based lifestyle intervention: the Coronary Health Improvement Project (CHIP) experience. Am J Cardiol 1998;82(suppl 10B):83T–7.

[13] Aldana SG, Greenlaw RL, Diehl HA, Salberg A, Merrill RM, Ohmine S, et al. Effects of an intensive diet and physical activity modification program on the health risks of adults. J Am Diet Assoc 2005;105: 371–81.
[14] Aldana SG, Greenlaw RL, Diehl HA, Salberg A, Merrill RM, Ohmine S, et al. The behavioral and clinical effects of therapeutic lifestyle change on middle-aged adults. Prev Chronic Dis 2006;3:A05.
[15] Aldana SG, Greenlaw RL, Diehl HA, Merrill RM, Salberg A, Englert H. A video-based lifestyle intervention and changes in coronary risk. Health Educ Res 2008;23:115–24.
[16] Bodnar LM, Wisner KL. Nutrition and depression: implications for improving mental health among childbearing-aged women. Biol Psychiatry 2005;58:679–85.
[17] Cassidy K, Kotynia-English R, Acres J, Flicker L, Lautenschlager NT, Almeida OP. Association between lifestyle factors and mental health measures among community-dwelling older women. Aust N Z J Psychiatry 2004;38:940–7.
[18] Ahlberg A, Ljung T, Rosmond R, McEwen B, Holm G, Akesson HO, et al. Depression and anxiety symptoms in relation to anthropometry and metabolism in men. Psychiatry Res 2002;112:101–10.
[19] Strawbridge WJ, Deleger S, Roberts RE, Kaplan GA. Physical activity reduces the risk of subsequent depression for older adults. Am J Epidemiol 2002;156:328–34.
[20] Glassman AH. Cigarette smoking: implications for psychiatric illness. Am J Psychiatry 1993;150:546–53.
[21] Nakao M, Yano E. Relationship between major depression and high serum cholesterol in Japanese men. Tohoku J Exp Med 2004;204: 273–87.
[22] Weidner G, Connor SL, Hollis JF, Connor WE. Improvements in hostility and depression in relation to dietary change and cholesterol lowering. The Family Heart Study. Ann Intern Med 1992;117:820–3.
[23] Armitage CJ, Conner M. Social cognition models and health behaviour: a structured review. Psychol Health 2000;15:173–89.
[24] Green LW, Kreuter NW. Health promotion planning: an educational and ecological approach. Mountain View, CA: Mayfield; 1999.
[25] McKenzie JF, Smeltzer JL. Planning, implementing, and evaluating health promotion programs: a primer. Boston, MA: Allyn & Bacon; 2001.
[26] Sieminski DJ, Cowell LL, Montgomery PS, Pillai SB, Gardner AW. Physical activity monitoring in patients with peripheral arterial occlusive disease. J Cardiopulm Rehabil 1997;17:43–7.
[27] Hendelman D, Miller K, Baggett C, Debold E, Freedson P. Validity of accelerometry for the assessment of moderate intensity physical activity in the field. Med Sci Sports Exerc 2000;32(suppl 9):S442–9.
[28] Mizuno C, Yoshida T, Udo M. Estimation of energy expenditure during walking and jogging by using an electro-pedometer. Ann Physiol Anthropol 1990;9:283–9.
[29] Block G, Woods M, Potosky A, Clifford C. Validation of a self-administered diet history questionnaire using multiple diet records. J Clin Epidemiol 1990;43:1327–35.
[30] Block G, Thompson FE, Hartman AM, Larkin FA, Guire KE. Comparison of two dietary questionnaires validated against multiple dietary records collected during a 1-year period. J Am Diet Assoc 1992;92:686–93.
[31] Mares-Perlman JA, Klein BE, Klein R, Ritter LL, Freudenheim JL, Luby MH. A diet history questionnaire ranks nutrient intakes in middle-aged and older men and women similarly to multiple food records. J Nutr 1993;123:489–501.
[32] Steer RA, Beck AT, Garrison B. Applications of the Beck Depression Inventory. In: Sartorius N, Ban TA, editors. Assessment of depression. New York: Springer-Verlag; 1985, p. 121–42.
[33] Steer RA, Beck AT, Riskind J, Brown G. Differentiation of depressive disorders from generalized anxiety by the Beck Depression Inventory. J Clin Psychol 1986;40:475–8.
[34] Beck AT, Rial WY, Rickels K. Short form of depression inventory: cross-validation. Psychol Rep 1974;34:1184–6.
[35] Scogin F, Beutler L, Corbishley A, Hamblin D. Reliability and validity of the short form Beck Depression Inventory with older adults. J Clin Psychol 1988;44:853–6.
[36] Sinha R, Block G, Taylor PR. Determinants of plasma ascorbic acid in a healthy male population. Cancer Epidemiol Biomarkers Prev 1992;1:297–302.
[37] Blumenthal JA, Babyak MA, Moore KA, Craighead WE, Herman S, Khatri P, et al. Effects of exercise training on older patients with major depression. Arch Intern Med 1999;159:2349–56.
[38] Galper DI, Trivedi MH, Barlow CE, Dunn AL, Kampert JB. 2. Inverse association between physical inactivity and mental health in men and women. Med Sci Sports Exerc 2006;38:173–8.
[39] Paluska SA, Schwenk TL. Physical activity and mental health: current concepts. Sports Med 2000;29:167–80.
[40] Strawbridge WJ, Deleger S, Roberts RE, Kaplan GA. Physical activity reduces the risk of subsequent depression for older adults. Am J Epidemiol 2002;156:328–34.
[41] Sjosten N, Kivela SL. The effects of physical exercise on depressive symptoms among the aged: a systematic review. Int J Geriatr Psychiatry 2006;21:410–8.
[42] Penedo FJ, Dahn JR. Exercise and well-being: a review of mental and physical health benefits associated with physical activity. Curr Opin Psychiatry 2005;18:189–93.
[43] Dixon JB, Dixon ME, O'Brien PE. Depression in association with severe obesity: changes with weight loss. Arch Intern Med 2003;163: 2058–65.
[44] Koeppl PM, Heller J, Bleecker ER, Meyers DA, Goldberg AP, Bleecker ML. The influence of weight reduction and exercise regimes upon the personality profiles of overweight males. J Clin Psychol 1992;48:463–71.
[45] Weighill VE, Buglass D. Weight change and psychological state in obese women. Appetite 1984;5:95–102.
[46] Oster G, Thompson D, Edelsberg J, Bird AP, Colditz GA. Lifetime health and economic benefits of weight loss among obese persons. Am J Public Health 1999;89:1536–42.
[47] Pi-Sunyer FX. Short-term medical benefits and adverse effects of weight loss. Ann Intern Med 1993;119:722–6.
[48] Ransford CP. A role for amines in the antidepressant effect of exercise: a review. Med Sci Sports Exerc 1982;4:1–10.
[49] North TC, McCullagh P, Tran ZV. Effect of exercise on depression. Exerc Sport Sci Rev 1990;18:379–415.
[50] Morgan WP. Affective beneficence of vigorous physical activity. Med Sci Sports Exerc 1985;17:94–100.
[51] Anton SD, Miller PM. Do negative emotions predict alcohol consumption, saturated fat intake, and physical activity in older adults? Behav Modif 2005;29:677–88.
[52] Payne ME, Hybels CF, Bales CW, Steffens DC. Vascular nutritional correlates of late-life depression. Am J Geriatr Psychiatry 2006;14: 787–95.
[53] Bell IR, Edman JS, Morrow FD, et al. B complex vitamin patterns in geriatric and young adult inpatients with major depression. J Am Geriatr Soc 1991;39:252–7.
[54] Coppen A, Bolander-Gouaille C. Treatment of depression: time to consider folic acid and vitamin B12. J Psychopharmacol 2005;19: 59–65.
[55] Murray M, Pizzorno J. Encyclopedia of natural medicine. Rocklin, CA: Prima Publishing; 1991.