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Longitudinal changes in dietary fat intake and associated changes in cardiovascular risk factors in adults with type 2 diabetes: The ACCORD trial

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Abstract

Aims—To measure dietary fat intake using the Puget Sound Eating Patterns (PEP) questionnaire, a validated 19-item food questionnaire, and to quantify how reduced dietary fat intake affects cardiovascular risk factors in adults with type 2 diabetes.

Methods—Randomized controlled trial including a subsample of 1781 Action to Control Cardiovascular Risk in Diabetes (ACCORD) participants. Participants received dietary counseling to consume a reduced-fat diet. Outcome measures included HbA1c, fasting lipid profile, blood

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Conflicts of interest

There are no conflicts of interest.

pressure, and weight. Longitudinal linear regression analyses were used to evaluate relationships between baseline and follow-up PEP scores and cardiovascular risk factors.

Results—PEP scores decreased significantly from baseline to 12-month follow up with a mean difference of -0.09 ± 0.39 , $P < 0.001$. All of the fat intake subscales showed significant improvement at 12 months from baseline. White race, female gender, and more hours per week of physical activity were correlated with a decline in PEP scores at 1-year. A longitudinal decrease in dietary fat intake was associated with significantly less weight gain at 12- and 36-months and lower serum triglycerides at 1 year.

Conclusions—Reduced fat intake as measured by a brief questionnaire was associated with significant improvement in some cardiovascular risk factors (triglycerides and weight), but not in others.

Keywords

Dietary fats; Diabetes mellitus type 2; Cardiovascular disease; Risk factors

Accurate assessment of dietary intake in large multicenter studies of subjects consuming a wide variety of foods is very challenging. Comprehensive methods of dietary assessment such as 4-day food records and 24-h diet recalls are often quite time-consuming for participants and expensive for the study, but less arduous and expensive methods may be considered to have unacceptably high measurement error. Food frequency questionnaires are often used despite substantial measurement error in estimating individual dietary intake [1–4], but even these are too costly or burdensome for many large studies. Brief questionnaires are another method of obtaining key dietary assessments while imposing less burden on the participants, but valid instruments are limited to a relatively small number of nutrients and food types [5–7]. Consequently, some large cardiovascular disease (CVD) studies have not assessed diet at all, even when dietary recommendations are an integral part of the protocol [8,9].

The Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial was initiated in 1999 to determine whether intensive glycemic control with a goal of glycated hemoglobin A1c (HbA1c) $< 6\%$ vs. standard control (HbA1c 7.0–7.9%) would reduce CVD morbidity and mortality in patients with type 2 diabetes [10,11]. Participants were also randomly assigned to either an intensive or standard intervention strategy for blood pressure or lipid management. In view of the importance of diet for all three aspects of risk factor management, dietary counseling was a key component of the background treatment in all of the interventions. However, because of the study's time demands, only a brief dietary assessment was included for a subgroup of the participants to minimize additional participant and staff burden. The self-administered Puget Sound Eating Patterns (PEP) 19-item questionnaire focusing on total fat consumption was selected as a measure of behavior change, because fat consumption was an aspect of the dietary recommendations and there was no comparable brief validated instrument for any other major aspect of the recommendations [7].

The purposes of the current study were (1) to examine longitudinal self-reported fat intake using a brief assessment tool (PEP) as a proxy for dietary behavior change in ACCORD, (2) to determine which participant characteristics were related to greater change in self-reported fat intake, and (3) to determine whether changes in self-reported fat intake were related to changes in CVD risk factors and metabolic outcomes during follow up in ACCORD.

1. Methods

1.1. Study design

The ACCORD study design has previously been described in detail [10,11]. Briefly, 10,251 participants with type 2 diabetes at high risk for CVD (previous history of CVD or multiple risk factors such as hyperlipidemia, hypertension, etc.) were randomly assigned to one of two therapeutic strategies for glycemic control: an intensive strategy to achieve nearly normal glycemic control with HbA1c < 6.0% or a standard strategy to maintain HbA1c at 7.0–7.9%. Patients received glucose-lowering medications from a wide variety of therapeutic classes at the discretion of the investigators on the basis of study-group assignment and their response to therapy. Both glycemia treatment groups in ACCORD received adjunctive lifestyle therapy, including medical nutrition therapy (MNT) and physical activity counseling. Lifestyle and MNT recommendations, including a modestly low-fat, heart-healthy diet equivalent to an American Heart Association (AHA) Step 1 diet (30 total fat) were similar in the intensive and standard treatment arms but differed in the intensity of reinforcement, as participants in the intensive treatment arm had more frequent follow-up visits [12].

With the use of a double two-by-two factorial design, each ACCORD participant was also randomly assigned to one of two subtrials: (1) 4733 were assigned to lower their blood pressure (BP) by receiving either intensive therapy (systolic BP target, <120 mm Hg) or standard therapy (systolic BP target, <140 mm Hg) and (2) 5518 were assigned to receive either fenofibrate or placebo while maintaining good control of low-density lipoprotein (LDL) cholesterol with simvastatin. As in the glycemia trial, patients in the BP trial received BP-lowering medications from a wide variety of therapeutic classes at the discretion of the investigators on the basis of study-group assignment and their response to therapy.

Of 10,251 participants randomized in the ACCORD trial, a subsample of 2053 was randomly selected to complete additional questionnaires on diet, physical activity and quality of life. Assessments were administered at baseline and at 12 and 36 months of follow up. Of these 2053 participants, 1781 (86%) completed the PEP questionnaire at baseline and are included in these analyses. The PEP questionnaire has been shown to have high test-retest and internal consistency reliabilities and to correlate reasonably well ($r = 0.68$) with the percentage of total energy intake consumed as fat measured by a 4-day food record [7]. When scored, the PEP questionnaire provides an overall summary measure of fat intake, as well as five subscales designed to describe: (1) substitution of lower-fat alternatives; (2) modification or reduction of meat intake; (3) avoidance of fried foods; (4) replacement of fat with non-fat alternatives; and (5) avoidance of dietary fat. Each subscale score and the summary score ranges from 1 to 4, with lower scores indicating a behavioral pattern leading

to lower fat intake (each 1 point lower on the summary score corresponds to 13% lower percentage of total energy intake consumed as fat) [13,14].

The Protocol Review Committee, appointed by the National Heart, Lung, and Blood Institute, approved the ACCORD study protocol. Each participant gave written informed consent using procedures reviewed and approved by local institutional review boards and based on a template from the study group that was approved and centrally monitored by the Coordinating Center and the National Heart, Lung, and Blood Institute.

1.2. Data collection

Demographic data, diabetes duration, smoking status, and alcohol consumption were assessed at baseline by questionnaires. A medication inventory was conducted by trained study staff to assess diabetes and antihypertensive and lipid-lowering treatments and other concomitant medications used at study enrollment. Previous CVD at enrollment was assessed on the basis of self-reported history and review of medical records. Body size measurements were performed at baseline and annual visits. Weight and height were obtained by trained staff using a stadiometer for height and a balance beam scale for weight. Waist circumference was measured to the nearest 0.1 cm at the smallest point between the 10th rib and iliac crest while the subject was standing. BP was measured as the average of three readings using the Omron HEM-907 automated device according to standard protocol. Physical activity level was measured using a modified version of the Community Healthy Activities Model Program for Seniors (CHAMPS) physical activity questionnaire for older adults [15]. Data regarding depression and quality of life was collected using the Personal Health Questionnaire (PHQ9) and the Short-Form 36 Health Survey Questionnaire (SF36) [16]. HbA1c and fasting lipid profile measurements were performed at the ACCORD Central Laboratory at the University of Washington. Duration of nutrition education time for each visit was recorded by study staff.

1.3. Data analysis

Descriptive analyses included means, standard deviations (SDs), percentiles, ranges of continuous factors, frequencies, and percentages of categorical factors. PEP scores and subscores were analyzed as ordinal/continuous variables. Characteristics of participants selected for inclusion in the diet substudy were compared with those not selected using *t*-tests (with Satterthwaite's adjustment for unequal variances where warranted) for continuous factors and chi-square tests for categorical ones. Longitudinal, within-subject change in PEP scores and change in CVD risk factors were computed by subtracting the baseline value from months 12 and 36 follow-up values; thus, negative changes indicated lower values at follow up (eg., changes in PEP values less than zero indicated lower fat intake at follow-up). To explore potential predictors of change in PEP summary scores, a maximum-likelihood (ML) repeated measures analysis-of-covariance (ANCOVA) model was fitted that included baseline PEP score as a covariate [17]. Relationships between PEP scores and change in CVD risk factors from baseline to 12 and 36 months were evaluated for the following outcomes: HbA1c, LDL cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, systolic BP, diastolic BP, weight, and waist circumference using ML repeated measures ANCOVA models as well. Models were fitted using an unstructured

variance–covariance matrix, all continuous covariates were standardized by dividing by their respective standard deviations. Models for CVD outcomes included the baseline outcome, baseline and longitudinal change in PEP scores, randomized treatment groups, age, sex, race, and baseline hours of any physical activity and moderate-intensity activity. Regression analyses only included participants with observed values for all covariates included in models. All analyses were performed using SAS Version 9.2 software (SAS Institute Inc., Cary, NC).

2. Results

Participant characteristics and diabetes risk factors are shown in Table 1. The treatment groups were well balanced with respect to covariates, with most individuals having at least a high school education; more than half were male; and participants in both groups had long-standing diabetes. The baseline characteristics reflected the ACCORD population overall, except that a slightly higher proportion of participants randomly selected for the diet substudy were in the BP rather than the lipid trial (50.1% vs. 45%). Compared with those selected for the diet substudy who did not complete the baseline PEP questionnaire, the group who completed it had lower HbA1c ($8.2 \pm 1.0\%$ vs. $8.6 \pm 1.2\%$), lower LDL cholesterol (2.7 ± 0.9 vs. 2.8 ± 0.9 mmol/l), higher triglycerides (4.9 ± 3.7 vs. 4.5 ± 2.4 mmol/l), lower systolic BP (136 ± 17 vs. 140 ± 18 mm Hg), a lower proportion of Hispanics (6.2% vs. 10.7%), and a higher proportion assigned to the BP rather than the lipid trial (51.2% vs. 42.6%).

PEP questionnaire summary score and subscores are shown in Table 2. Results were nearly identical at each time point in both the intensive and standard glycemia groups and have therefore been combined. Fat intake was significantly decreased overall and in all subscores between baseline and 12-month follow up, but there was no further change between 12-month and 36-month follow up.

Results of a repeated measures regression model to predict longitudinal change in PEP summary score at 12 and 36 months are shown in Table 3. As noted previously, glycemia treatment group assignment had no effect on change in PEP score, nor was there an effect of BP or lipid trial assignment. After controlling for baseline PEP scores, female sex and more time spent performing moderate-intensity physical activity at baseline were associated with a greater decrease in fat intake, whereas both Asian and African-American participants had less overall declines in fat intake than white participants.

Relationships between longitudinal changes in PEP scores (i.e., reductions in fat intake) and longitudinal changes in CVD risk factors were examined (Table 4), and no association was observed for A1c, LDL cholesterol, systolic BP, diastolic BP, or waist circumference. Although higher baseline PEP score was associated with a reduction in HDL cholesterol over time (0.011 ± 0.005 mmol/L per SD higher baseline PEP score, $P = 0.024$, Table 4), there was no association between change in PEP score and changes in HDL cholesterol during follow up. Significant weight gain was observed between baseline and 12 months (mean $\pm$ SE: 1.21 ± 0.17 kg, $P < 0.001$), and there was additional gain between 12 and 36 months (0.81 ± 0.25 kg, $P = 0.001$). Participants with less reduction (or increases) in PEP

scores exhibited greater weight gain (0.37 ± 0.16 kg per SD increase in PEP score change, $P = 0.017$) (Table 4).

Overall, significant changes in triglycerides were observed at both 12 months (mean $\pm$ SE change from baseline -0.3 ± 0.08 mmol/l, $P < 0.001$) and 36 months of follow up (-0.7 ± 0.06 mmol/l, $P < 0.001$). The association between change in PEP score and change in triglycerides differed between the first year of follow up and the subsequent 2 years ($P = 0.029$ for interaction). A strong relationship was observed between increases (or smaller decreases) in fat intake and increases in triglycerides in the first year of follow-up (0.123 ± 0.040 mmol/l per SD increase in PEP score change, $P = 0.002$); however, no relationship was observed at year 3 (Table 4).

3. Discussion

The purpose of the current study was to examine the pattern of dietary fat intake in a population of patients with diabetes at high risk for CVD who were counseled to reduce fat intake, and to examine the effect of changes in fat intake on CVD risk factors and metabolic outcomes. Despite the limitations in the brief dietary assessment PEP questionnaire that was used in the ACCORD trial, we were able to observe several key findings in this analysis: (1) Participants in both intensive and standard glycemia treatment groups reported a similar eating pattern consistent with significantly reduced fat intake in the first year of follow up, and the pattern was sustained through 3 years; (2) Subjects who were female, white, or had higher baseline hours per week of moderate-intensity physical activity were more likely to reduce their fat intake; (3) In this population, which was simultaneously treated in a lipid or BP trial, longitudinal reductions in dietary fat intake were associated with less weight gain at both 12 and 36 months and with lower triglycerides at 12 months but not with significant differences in BP, LDL cholesterol, HDL cholesterol, or HbA1c.

We interpret these findings to suggest that the dietary counseling participants received in ACCORD was associated with real and sustained changes in dietary fat intake. The findings also suggest that the goal to impart similar dietary advice to all participants, regardless of their treatment assignment, was achieved. In this study, women reported more success in lowering fat intake, which could reflect biased reporting by women, greater motivation to change diet among women, or better interactions between dietitians and female patients. The association between physical activity and lower fat intake likely reflects a similar orientation to better lifestyle habits in certain individuals. Less change in fat intake among African-Americans and Asians suggests the need for improved, culturally specific dietary counseling interventions for these patients.

In this study, participants gained an average of 1.9 kg (4.2 lbs.) over 36 months, and reductions in dietary fat were associated with less weight gain at both 12 and 36 months. This may relate to broader unmeasured changes in diet, given that we measured only dietary behavior change related to fat intake. However, weight loss has been observed by others with ad libitum low-fat diets [18]. The association between fat intake and triglyceride levels was significant at 12 months but dissipated by 36 months. A reduction in the serum

triglyceride level similar to that observed in the current study has been shown to result from an AHA Step 1 low-fat diet regardless of, but amplified by, weight loss [19].

Various dietary intervention studies in subjects at risk for diabetes have used fat-restricted and carbohydrate-restricted diets to promote weight loss. The Diabetes Prevention Program utilized a questionnaire of 117 items plus open-ended food queries among 3175 participants at risk for diabetes [20]. Adherence to dietary intervention was associated with weight loss and improvement in CVD risk factors in the intensive lifestyle treatment group [20,21]. The Finnish Diabetes Prevention Study randomly assigned 172 men and 350 women with impaired glucose tolerance to intensive dietary and exercise counseling compared with standard care [22]. Three-day food records showed that high fiber and low fat intake were significant predictors of sustained weight reduction, lessened progression to type 2 diabetes, and lower triglyceride levels. The latter was most marked in the first year of the study and attenuated at year 3 [22].

The Look AHEAD Trial investigated the long-term effects of a lifestyle intervention on weight and CVD risk factors in individuals with type 2 diabetes. It also found a reduction in serum triglyceride levels (with a modestly fat-restricted diet) that was readily apparent at 1 year of follow up, but this attenuated over time and was not significant at 4 years of follow up [23]. A significant reduction in HbA1c in both the Finnish Diabetes Prevention Study and Look AHEAD persisted for 3 or 4 years of follow up, but unlike the ACCORD Trial, both employed an intensive combined exercise and diet lifestyle modification intervention [22,23]. The current observational findings of no significant effect of diet modification on HbA1c may be due to the fact that subjects on average gained weight, rather than losing weight. Moreover, major changes in HbA1c-lowering drug therapy in ACCORD may have made it difficult to detect an independent effect of diet on HbA1c. Similarly, use of an average of 2 or 3 antihypertensive drugs and moderate doses of simvastatin in the ACCORD BP and lipid subtrials may have contributed to difficulty detecting an effect of dietary changes on BP and LDL cholesterol [24,25].

The PEP score is a measure of behavior change rather than a direct measure of fat intake. Each 1-point unit lower on the summary score corresponds to 13% lower percentage of total energy intake consumed as fat. Other studies using PEP have reported generally similar baseline scores to those observed in ACCORD. These include an interventional trial of educational materials in 1459 generally healthy adults ages 18–69 years who had a mean summary fat score of 2.29 ± 0.49 at baseline and a decrease in fat score of -0.09 ± 0.38 at 12 months [26]. Although the mean score at baseline was somewhat higher in the current study (2.41 ± 0.45), the reduction in summary fat score at 12 months was similar (-0.09 ± 0.39). The Women's Health Trial Feasibility Study in Minority Populations studied women whose fat intake, based on a food frequency questionnaire, was about 40% of total energy intake [13]. Mean PEP scores from a modified version of the validated instrument in white, black, and Hispanic women were 2.55, 2.64, and 2.50, respectively. The study intervention was designed to reduce fat intake to 20% or less of total energy and resulted in mean reductions in PEP scores of 0.39–0.48, much larger than those observed in ACCORD.

Several factors limit the interpretation of our results. The dietary assessment in ACCORD was limited to a brief questionnaire focused on fat intake. Although PEP has been validated and used in numerous studies [7,13,14,26] fat intake is a difficult construct to assess. In addition, a brief, self-report questionnaire may not capture information about actual dietary behavior and may be subject to significant recall bias. ACCORD did not quantify participants' caloric intake or consumption in grams of either total or saturated fat or assess intake of other macronutrients or foods. Furthermore, nutritional advice given to participants in ACCORD was not standardized, and the impact of post-baseline medication regimens is not considered in the analysis due to the complexity of the medication interventions in ACCORD. We did not examine the effect of dietary change on CVD outcomes or mortality due to limited power. Finally, *P*-values in Table 4 are from examination of epidemiologic relationships between dietary fat intake and seven separate outcomes without adjustment for potential inflation of type I error due to multiple hypothesis tests and should be interpreted accordingly.

This study has several strengths, including a large sample of patients with diabetes and CVD risk factors. The data provide a valuable opportunity to characterize longitudinal changes in behavior and diet in a large population of at-risk subjects with type 2 diabetes enrolled in a multicenter interventional trial. In addition, this substudy of ACCORD participants includes a relatively diverse population of minority individuals, who are often understudied in large diabetes trials. The magnitude of the problem of under representation of minority populations in studies of type 2 diabetes has been reviewed recently by Lirussi [27]. Ethnicity-specific dietary interventions that emphasize healthy food choices rather than exchange-based meal plans likely have a positive impact on type 2 diabetes self-management [28].

4. Conclusions

ACCORD participants successfully modified dietary patterns during the study, with a modest but significant reduction in dietary fat intake at 1 year of follow up that was sustained throughout the 3-year follow-up period. This modest reduction in fat was associated with significant improvement in some CVD risk factors (triglycerides and weight) at 1 year, but not in others (HbA1c, blood pressure, or high-density or low-density lipoprotein cholesterol). Reduced fat intake in a large clinical trial can be achieved at 1 year and sustained for 3 years. The observed impact of ethnicity and sex on behavior modification of CVD risk reduction suggest the need for additional study and perhaps of development of more tailored approaches to modification of fat intake.

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References

1. Dolecek TA, Stamler J, Gaggiula AW, Tillotson JL, Buzzard IM. Methods of dietary and nutritional assessment and intervention and other methods in the Multiple Risk Factor Intervention Trial. *Am J Clin Nutr.* 1997; 65(January Suppl 1):196s–210s. [PubMed: 8988938]
2. Kristal AR, Peters U, Potter JD. Is it time to abandon the food frequency questionnaire? *Cancer Epidemiol Biomarkers Prev.* 2005; 14(December):2826–8. [PubMed: 16364996]
3. Prentice RL, Thomson CA, Caan B, Hubbell FA, Anderson GL, Beresford SA, et al. Low-fat dietary pattern and cancer incidence in the Women's Health Initiative Dietary Modification Randomized Controlled Trial. *J Natl Cancer Inst.* 2007; 99(October):1534–43. [PubMed: 17925539]
4. Tinker LF, Rosal MC, Young AF, Perri MG, Patterson RE, Van Horn L, et al. Predictors of dietary change and maintenance in the Women's Health Initiative Dietary Modification Trial. *J Am Diet Assoc.* 2007; 107(July):1155–65. [PubMed: 17604744]
5. Shannon J, Kirkley B, Ammerman A, Keyserling T, Kelsey K, DeVellis R, et al. Self-efficacy as a predictor of dietary change in a low-socioeconomic-status southern adult population. *Health Educ Behav.* 1997; 24(June):357–68. [PubMed: 9158979]
6. Glasgow RE, Perry JD, Toobert DJ, Hollis JF. Brief assessments of dietary behavior in field setting. *Addict Behav.* 1996; 21(March/April):239–47. [PubMed: 8730527]
7. Kristal AR, Shattuck AL, Henry HJ. Patterns of dietary behavior associated with selecting diet low in fat: reliability and validity of a behavioral approach to dietary assessment. *J Am Diet Assoc.* 1990; 90(February):214–20. [PubMed: 2303658]
8. Davis BR, Cutler JA, Gordon DJ, Furberg CD, Wright JT Jr, Cushman WC, et al. ALLHAT research group. Rationale and design for the Antihypertensive and Lipid Lowering Treatment to Prevent Heart Attack Trial (ALLHAT). *Am J Hypertens.* 1996; 9(April 4 Pt 1):342–60. [PubMed: 8722437]
9. Gerstein HC, Yusuf S, Bosch J, Pogue J, Sheridan P, Diccag N, et al. DREAM (Diabetes REduction Assessment with ramipril and rosiglitazone Medication) Trial Investigators. Effect of rosiglitazone on the frequency of diabetes in patients with impaired glucose tolerance or impaired fasting glucose: a randomized controlled trial. *Lancet.* 2006; 368(September 9541):1096–105. [PubMed: 16997664]
10. Buse JB, Bigger JT, Byington RP, Cooper LS, Cushman WC, Friedwald WT, et al. ACCORD study group. Action to Control Cardiovascular Risk in Diabetes (ACCORD) Trial: design and methods. *Am J Cardiol.* 2007; 99(June 12A):21i–3i.
11. Gerstein HC, Miller ME, Byington RP, Goff DC Jr, Bigger JT, et al. Action to Control Cardiovascular Risk in Diabetes study group. Effects of intensive glucose lowering in type 2 diabetes. *N Engl J Med.* 2008; 358(June):2545–59. [PubMed: 18539917]
12. Krauss RM, Eckel RH, Howard B, Appel LJ, Daniesl SR, Deckelbaum RJ, et al. AHA dietary guidelines: revision 2000: a statement for healthcare professionals from the Nutrition Committee of the American Heart Association. *Circulation.* 2000; 102(October):2284–99. [PubMed: 11056107]
13. Kristal AR, Shattuck AL, Patterson RE. Differences in fat-related dietary patterns between black, Hispanic and White women: results from the Women's Health Trial Feasibility Study in minority populations. *Public Health Nutr.* 1999; 2(September):253–62. [PubMed: 10512559]
14. Kristal AR, Beresford SA, Lazovich D. Assessing change in diet-intervention research. *Am J Clin Nutr.* 1994; 59(January Suppl 1):S185–9.
15. Stewart AL, Mills KM, King AC, Haskell WL, Gillis D, Ritter PL. CHAMPS physical activity questionnaire for older adults: outcomes for interventions. *Med Sci Sports Exerc.* 2001; 33(July):1126–41. [PubMed: 11445760]
16. Ware JE, Snyder MK, Wright WR, Davies AR. Defining and measuring patient satisfaction with medical care. *Eval Program Plann.* 1983; 6(3/4):247–63. [PubMed: 10267253]
17. Fitzmaurice, GM.; Laird, NM.; Ware, JH. *Applied Longitudinal Analysis.* New York: Wiley; 2004. p. 49-134.

18. Astrup A, Grunwald GK, Melanson EL, Saris WHM, Hill JO. The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *Int J Obesity*. 2000; 24(March):1545c–52c.
19. Dengel JL, Katzell LI, Goldberg AP. Effect of an American Heart Association diet, with or without weight loss on lipids in obese middle-aged and older men. *Am J Clin Nutr*. 1995; 62(October): 715–21. [PubMed: 7572698]
20. Mayer-Davis EJ, Sparks KC, Hirst K, Costacou T, Lovejoy JC, Regensteiner JG, et al. Dietary intake in the diabetes prevention program cohort: baseline and 1-year post randomization. *Ann Epidemiol*. 2004; 14(vember):763–72. [PubMed: 15573453]
21. Goldberg RB, Temprosa M, Haffner S, Orchard TJ, Ratner RE, Fowler SE, et al. Effect of progression from impaired glucose tolerance to diabetes on cardiovascular risk factors and its amelioration by lifestyle and metformin intervention. *Diabetes Care*. 2009; 32(April):726–32. [PubMed: 19171717]
22. Lindstrom J, Louheranta A, Mannelin M, Rastas M, Salminen V, Eriksson J, et al. The Finnish diabetes prevention group. Lifestyle intervention and 3-year results on diet and physical activity. *Diabetes Care*. 2003; 26(December):3230–6. [PubMed: 14633807]
23. Wing RR. The look AHEAD research group. Long-term effects of a lifestyle intervention on weight and cardiovascular risk factors in individuals with type 2 diabetes mellitus. *Arch Intern Med*. 2010; 170(September):1566–75. [PubMed: 20876408]
24. Ginsberg HN, Elam MB, Lovato LC, Crouse JR 3rd, Leiter LA, et al. The ACCORD study group. Effects of combination lipid therapy in type 2 diabetes mellitus. *N Engl J Med*. 2010; 362(April): 1563–74. [PubMed: 20228404]
25. Cushman WC, Evans GW, Byington RP, Goff DC Jr, Cutler JA, et al. The ACCORD study group. Effects of intensive blood-pressure control in type 2 diabetes mellitus. *N Engl J Med*. 2010; 362(April):1575–85. [PubMed: 20228401]
26. Kristal AR, White E, Shattuck AL, Curry S, Anderson GL, Fowler A, et al. Long-term maintenance of a low-fat diet: durability of fat-related dietary habits in the Women's Health Trial. *J Am Diet Assoc*. 1992; 92(May):553–9. [PubMed: 1573135]
27. Lirussi F. The global challenge of type 2 diabetes and the strategies for response in ethnic minority groups. *Diabetes Metab Res Rev*. 2010; 26(September):421–32. [PubMed: 20641140]
28. Ziemer DC, Berkowitz KJ, Panayiotou RM, El-Kebbi IM, Musey VC, Anderson LA, et al. A simple meal plan emphasizing healthy food choices is as effective as an exchange-based meal plan for urban African Americans with type 2 diabetes. *Diabetes Care*. 2003; 26(June):1719–24. [PubMed: 12766100]

Table 1

Diet participant baseline characteristics.

Variable ^a	Overall		Intensive glycemia		Standard glycemia	
	N	Mean (SD) or Frequency (%)	N	Mean (SD) or Frequency (%)	N	Mean (SD) or Frequency (%)
Age (years)	1781	62.1 (6.7)	883	62.2 (6.7)	898	62.0 (6.6)
Race/Ethnicity	1781		883		898	
White		1189 (66.8)		586 (66.4)		603 (67.1)
Black		336 (18.9)		170 (19.3)		166 (18.5)
Hispanic		110 (6.2)		52 (5.9)		58 (6.5)
Asian		95 (5.3)		46 (5.2)		49 (5.5)
Other		174 (9.8)		87 (9.9)		87 (9.7)
Female sex (%)	1781	705 (39.6)	883	356 (40.3)	898	349 (38.9)
History of cardiovascular disease	1781	638 (35.8)	883	325 (36.8)	898	313 (34.9)
Alcohol Use (number of drinks/week)	1781	0.96 (2.74)	883	0.94 (2.73)	898	0.98 (2.76)
Education	1779		881		889	
<High school graduate		241 (13.5)		116 (13.2)		125 (13.9)
High school graduate		466 (26.2)		236 (26.8)		230 (25.6)
Some college		591 (33.2)		304 (34.5)		287 (32.0)
College graduate		481 (27.0)		225 (25.5)		256 (28.5)
History of smoking	1780	1042 (58.5)	882	512 (58.0)	898	530 (59.0)
On statin	1781	1158 (65.0)	883	578 (65.5)	898	580 (64.6)
On antidepressant	1781	258 (14.5)	883	127 (14.4)	898	131 (14.6)
On insulin	1781	643 (36.1)	883	326 (36.9)	898	317 (35.3)
Diabetes duration (years)	1756	11.0 (7.8)	871	11.3 (7.9)	885	10.8 (7.7)
Waist circumference (cm)	1767	107 (14)	876	107 (14)	891	107 (10)
Body mass index (kg/m ²)	1781	32.5 (5.6)	883	32.4 (5.6)	898	32.6 (5.5)
Systolic blood pressure (mm Hg)	1777	136 (17)	880	136 (17)	897	136 (17)
Diastolic blood pressure (mm Hg)	1777	74 (11)	880	74 (11)	897	74 (11)
HbA1c (%)	1778	8.2 (1.0)	881	8.2 (1.0)	897	8.3 (1.1)
LDL cholesterol (mmol/l)	1772	2.7 (0.9)	880	2.7 (0.9)	892	2.7 (0.9)
HDL cholesterol (mmol/l)	1772	1.1 (0.3)	880	1.1 (0.3)	892	1.1 (0.3)

Variable ^a	Overall		Intensive glycemia		Standard glycemia	
	N	Mean (SD) or Frequency (%)	N	Mean (SD) or Frequency (%)	N	Mean (SD) or Frequency (%)
Triglycerides (mmol/l)	1772	4.9 (3.7)	880	4.8 (3.6)	892	5.1 (3.6)
PHQ-9 depression scale	1765	5.45 (5.12)	875	5.69 (5.35)	890	5.21 (4.87)
SF36 Mental function subscale	1763	20.1 (3.7)	875	20.0 (3.7)	888	20.2 (3.6)
SF36 Physical function subscale	1765	23.3 (5.2)	875	23.1 (5.4)	890	23.4 (5.0)
Hours/week of any physical activity	1762	8.0 (8.2)	874	7.8 (8.1)	888	8.3 (8.4)
Hours/week of moderate intensity activity	1762	3.2 (4.8)	874	3.0 (4.7)	888	3.4 (4.8)

HDL, high-density lipoprotein; LDL, low-density lipoprotein; PHQ9, Personal Health Questionnaire; SD, standard deviation; SF36, Short-Form 36 Health Survey Questionnaire.

^aNo statistical difference in baseline values between intensive and standard arm.

Modified PEP Questionnaire Scores at Baseline, 12, and 36 Months.

PEP score component	Baseline			Month 12			Month 36					
	N ^a	Mean	SD	Range	N ^a	Mean	SD	Range	N ^a	Mean	SD	Range
Summary score	1781	2.41	0.45	1.17–3.64	1633	2.33	0.43	1.05–3.62	1525	2.33	0.43	1.20–3.60
Substitution subscore	1774	2.61	0.75	1.00–4.00	1628	2.51	0.77	1.00–4.00	1519	2.53	0.75	1.00–4.00
Modify meat subscore	1760	2.00	0.87	1.00–4.00	1611	1.91	0.83	1.00–4.00	1506	1.90	0.81	1.00–4.00
Avoid frying subscore	1780	1.86	0.65	1.00–4.00	1632	1.79	0.61	1.00–4.00	1522	1.77	0.60	1.00–4.00
Replacement subscore	1750	2.70	0.73	1.00–4.00	1602	2.59	0.71	1.00–4.00	1488	2.58	0.70	1.00–4.00
Avoid fat subscore	1780	2.89	0.63	1.00–4.00	1630	2.85	0.61	1.00–4.00	1521	2.86	0.60	1.00–4.00

PEP score component	Month 12 Minus Baseline				Month 36 Minus Baseline			
	N ^a	Mean	SD	Range	N ^a	Mean	SD	Range
Summary score	1633	-0.09 ^b	0.39	-1.26-1.52	1464	-0.00	0.38	-1.35-1.49
Substitution subscore	1622	-0.10 ^b	0.75	-3.00-3.00	1454	0.02	0.72	-3.00-3.00
Modify meat subscore	1599	-0.10 ^b	0.83	-3.00-3.00	1434	-0.01	0.81	-3.00-3.00
Avoid frying subscore	1631	-0.07 ^b	0.63	-2.67-2.50	1461	-0.02	0.63	-3.00-2.50
Replacement subscore	1578	-0.12 ^b	0.79	-2.67-3.00	1408	-0.01	0.78	-3.00-3.00
Avoid fat subscore	1629	-0.06 ^b	0.65	-2.25-2.67	1458	0.00	0.64	-3.00-2.67

A summary score was observed if at least one of its components was non-missing. A change score was observed if both elements of change were non-missing.

P value < 0.001 for test of mean change equal to zero.

Table 3

Multivariable Linear Regression of Effect of Baseline Factors on Post-Randomization Change (12 and 36 months minus baseline) in PEP summary score ($n = 1524$).

Baseline factor ^b	Regression coefficient ^a	SE (Beta)	P value
Baseline PEP summary score	−0.4592	0.0175	<0.001
Month 12 vs. Month 36	0.0030	0.0099	0.766
Intensive vs. standard glycemia arm	−0.0150	0.0163	0.357
Blood pressure vs. lipid trial participant	−0.0011	0.0165	0.949
Race/ethnicity			
Hispanic vs. white ^b	−0.0368	0.0336	0.273
African-American vs. white ^b	0.0542	0.0218	0.012
Asian vs. white ^b	0.0967	0.0353	0.006
Other vs. white ^b	0.0009	0.0345	0.980
Female sex	−0.0584	0.0197	0.003
History of clinical cardiovascular disease vs. no history	−0.0224	0.0168	0.179
Ever vs. never smoker	0.0260	0.0160	0.104
Education			
<High school vs. college grad ^c	0.0318	0.0264	0.104
High school grad vs. college grad ^c	0.0134	0.0211	0.527
Some college vs. college grad ^c	0.0074	0.0195	0.706
On antihypertensive vs. not on antihypertensive	−0.0080	0.0222	0.720
On statin vs. not on statin	−0.0077	0.0173	0.657
On antidepressant vs. not on statin	−0.0237	0.0228	0.299
On insulin vs. not on insulin	−0.0067	0.0173	0.699
Age: 7 year increase	0.0051	0.0089	0.569
Body mass index: 6 kg/m ² increase	−0.0257	0.0157	0.103
Waist circumference: 15 cm increase	0.0161	0.0154	0.296
Diabetes duration: 8 year increase	−0.0116	0.0085	0.173
Systolic blood pressure: 17 mm Hg increase	−0.0074	0.0097	0.447
Diastolic blood pressure: 11 mm Hg increase	0.0025	0.0103	0.806
HbA1c: 1% increase	−0.0013	0.0074	0.859
LDL cholesterol: 0.9 mmol/l increase	−0.0115	0.0084	0.174
HDL cholesterol: 9.3 mmol/l increase	0.0045	0.0092	0.623
Triglycerides: 3.7 mmol/l increase	−0.0069	0.0087	0.428
Alcohol consumption: three drinks/week increase	0.0026	0.0086	0.761
PHQ-9 depression scale: five-unit increase	0.0205	0.0106	0.055
SF-36 mental health subscale: four-unit increase	−0.0015	0.0113	0.893
SF-36 physical function subscale: five-unit increase	0.0031	0.0087	0.720
Hours/week of any physical activity	−0.0003	0.0017	0.874
Hours/week of moderate intensity physical activity	−0.0071	0.0030	0.017
Total nutrition education time first year: 78 min increase	−0.0079	0.0083	0.338

HDL, high-density lipoprotein; LDL, low-density lipoprotein; PHQ9, Personal Health Questionnaire; SD, standard deviation; SF36, Short-Form 36 Health Survey Questionnaire.

^aExpected change in PEP score for specified change in baseline factor.

^b4 degrees of freedom, test for race/ethnicity: *P* value = 0.003.

^c3 degrees of freedom test for education: *P* value = 0.677.

Table 4

Effect of PEP summary score on baseline value and post-randomization change in selected cardiovascular and metabolic risk factors at 12- and 36-months.

Cardiovascular and metabolic risk factors	PEP summary score ^a	Regression coefficient ^b	SE coefficient	P value
HbA1c (%) (<i>n</i> = 1672)	Baseline ^c	−0.0330	0.0225	0.143
	Change ^d	0.0182	0.0194	0.350
LDL cholesterol (mmol/L) (<i>n</i> = 1666)	Baseline ^c	−0.0279	0.0183	0.127
	Change ^d	−0.0052	0.0157	0.739
HDL cholesterol (mmol/L) (<i>n</i> = 1666)	Baseline ^c	−0.0106	0.0047	0.024
	Change ^d	−0.0042	0.0039	0.282
Triglycerides ^e at month 12 (mmol/L) (<i>n</i> = 1608)	Baseline ^c	0.0416	0.0418	0.319
	Change ^d	0.1227	0.0400	0.002
Triglycerides ^e at month 36 (mmol/L) (<i>n</i> = 1499)	Baseline ^c	0.0022	0.0301	0.943
	Change ^d	0.0005	0.0292	0.987
Systolic blood pressure (mmHg) (<i>n</i> = 1671)	Baseline ^c	0.3167	0.3445	0.358
	Change ^d	−0.2759	0.3134	0.379
Diastolic blood pressure (mmHg) (<i>n</i> = 1671)	Baseline ^c	0.2674	0.2060	0.194
	Change ^d	−0.0727	0.1832	0.692
Weight (kg) (<i>n</i> = 1675)	Baseline ^c	0.1861	0.1815	0.306
	Change ^d	0.3705	0.1551	0.017
Waist circumference (cm) (<i>n</i> = 1661)	Baseline ^c	0.2527	0.1787	0.158
	Change ^d	0.0857	0.1476	0.562

HDL, high-density lipoprotein; LDL, low-density lipoprotein; PEP, Puget Sound Eating Patterns questionnaire; PHQ9, Personal Health Questionnaire; SD, standard deviation; SF36, Short-Form 36 Health Survey Questionnaire.

^a In addition to PEP scores listed, each model includes baseline outcome, glycemia arm assignment, lipid trial arm assignment, age, sex, race, and baseline hours per week of any physical activity.

^b Expected change in risk factor for a standard deviation increase in baseline PEP score (0.45 unit) or change in PEP score (0.35 unit).

^c Baseline summary score standardized to 0.45 unit increment.

^d Change standardized to 0.39 unit increment.

^e The (follow-up time) × (PEP score change) interaction was significant (*P* = 0.029); therefore, models for triglycerides re-stratified by follow-up measurement. No other 2-way interactions between PEP score or change in PEP score and follow-up time were statistically significant.