

In Brief

The obesity paradox (survival advantage in overweight/obese patients with type 2 diabetes) has called into question the importance of weight loss in overweight people with diabetes. A systematic review of weight loss studies with a minimum of 1-year outcomes in people with diabetes reported inconsistent beneficial effects of weight loss on A1C, lipids, and blood pressure. To lower the risk of cardiovascular disease, a better nutrition therapy intervention may be reducing energy intake, which may or may not lead to weight loss, and selecting cardioprotective foods in appropriate portion sizes. However, any nutrition therapy intervention must be based on lifestyle changes the person with diabetes is willing and able to make.

The Obesity Paradox and Diabetes

Marion J. Franz, MS, RD, CDE

It is being called the “obesity paradox”: research findings that people with obesity-related illnesses and who are overweight or obese have better outcomes, including less mortality, than their normal-weight peers.¹ These seemingly contradictory results have also been shown to apply to people with acute coronary syndrome, stroke, and diabetes.²⁻⁹ The consequences of obesity are clear: increased risk for diabetes, high blood pressure, heart disease, stroke, and kidney disease.¹⁰ So, the question of emerging interest becomes why, then, once the disease develops, does being overweight appear to be beneficial?

Flegal et al.,¹ in a systematic review and meta-analysis, reported that, in the general public, severe obesity was associated with an increased risk for death from all causes but that lesser amounts of excess weight either did not increase this risk or were protective. They concluded, “. . . excess mortality in obesity may predominantly be due to elevated mortality at higher body mass index (BMI) levels. Overweight was associated with significantly lower all-cause mortality.” They further noted that these results are consistent with previous findings that have also shown lower mortality among overweight and moderately obese patients. Possible explanations include earlier medical care and aggressive risk factor treatment given

to heavier patients, cardioprotective metabolic effects of increased body fat, and/or beneficial effects of higher metabolic reserves.¹

A U-shaped association of weight with mortality is reported in people with diabetes. In a recent study,⁴ the records of 106,640 people with type 2 diabetes in Scotland were reviewed and BMI recorded around the time of diagnosis and mortality throughout the next ~ 5 years was assessed. Mortality risk was higher in people with a BMI of 20 to < 25 kg/m² and in those with a BMI ≥ 35 kg/m². Vascular mortality was also higher for each 5-kg/m² increase in BMI > 30 kg/m² but was lower below this level. Another study⁵ assessed the relationships between BMI and all-cause mortality in African-American and white men with type 2 diabetes and observed a significantly higher mortality risk (70%) in those with a BMI within the normal range (18.5–24.9 kg/m²) than in heavier subjects, with a higher mortality rate in African Americans (95%) than in whites (53%). In a study of patients who developed diabetes,⁶ total, cardiovascular, and noncardiovascular mortality rates were higher among normal-weight than among obese subjects. This finding was true regardless of diabetes type. Two other recent studies, Translating Research into Action for Diabetes⁷ and the

PROactive trial,⁸ also observed that participants who were of normal weight at the baseline examination or who lost weight during the trial (PROactive) experienced higher mortality than participants who were overweight or obese. This obesity paradox (survival advantage in obese patients with type 2 diabetes) was also shown to exist in patients with diabetes and cancer.⁹ Similar findings had been reported in two earlier studies.^{11,12} All of the above brings into question the role of weight management in people with chronic diseases, especially in people with diabetes.

Weight Loss/Management: Prevention Versus Treatment of Diabetes

The research results described above suggest that, perhaps, guidelines recommending weight loss should apply primarily to prevention and not necessarily to treatment of chronic diseases, including diabetes. Strong evidence exists for the benefits of moderate weight loss for the prevention of type 2 diabetes.¹³ The question becomes, what are the benefits from weight loss as glycemic impairments progress from prediabetes to overt type 2 diabetes?

The goals of medical nutrition therapy (MNT) for individuals with diabetes include achieving and maintaining blood glucose levels in the normal range or as close to normal as is safely possible, a lipid and lipoprotein profile that reduces the risk for cardiovascular disease (CVD), and blood pressure levels in the normal range or as close to normal as is safely possible.¹³ To achieve these goals, weight loss has been recommended for all overweight or obese individuals who have diabetes or are at risk for diabetes, with the level of evidence rated A (clear evidence from well-conducted, generalizable, randomized, controlled trials that are adequately powered).¹⁴ But perhaps the benefits of weight loss in the treatment of type 2 diabetes need to be reexamined. An inconclusive picture emerges from review of the benefits of weight loss on A1C, lipids, and blood pressure in people with type 2 diabetes. This also brings into question the effects of weight loss on risk factors for the prevention and treatment of CVD in people with diabetes.

Weight Loss and Diabetes-Related Outcomes

A PubMed search was conducted to determine the outcomes from baseline to study completion of nutrition therapy weight loss interventions in overweight or obese adults with type 2 diabetes. Research was reviewed from 1 January 2000 to 1 February 2013. Eleven randomized, clinical trials with a completion rate of $\geq 70\%$ and 12-month laboratory data reported were identified and are included in Table 1.¹⁵⁻²⁷ Because the duration of trials affects their outcomes, and to better compare outcomes among studies, 1-year outcomes are presented in the table for all trials. However, also included are data from two trials reporting 18-month data,^{18,19} two with 2-year data,^{23,24} and two with 4-year data.^{21,25-27} One trial²¹ did not report the statistical significance of intervention changes from baseline to study end, and one trial²⁵ reported statistical significance for the intervention arm only; however, both trials are included in the intervention summaries.

Eight of the studies compared varying weight loss interventions (10 different interventions),¹⁷⁻²⁴ and three studies compared the intervention to usual care or to a control group.^{15,16,25,27} Weight loss interventions implemented in 19 study arms included meal replacements (2 studies),^{15,17} individualized food plans (2 studies),^{16,17} one study with two group behavioral weight management arms,¹⁸ low fat (3 studies),^{20,21,24} high monounsaturated fat (MUFA),¹⁹ high carbohydrate (CHO) (3 studies),^{19,22,23} low CHO (2 studies),^{20,24} high protein (2 studies),^{22,23} Mediterranean-style diet (MED),²¹ and intensive lifestyle intervention (ILI).²⁵⁻²⁷ Although physical activity was suggested or encouraged in several studies,^{16,18,19,20,22} only two studies, those testing MED and ILI, included physical activity recommendations and measured and reported adherence.^{21,25,27} Four studies^{15,17,23,24} did not mention physical activity.

Weight changes

Weight losses from interventions ranged from 1.9 to 8.4 kg at 12 months; 16 of the interventions¹⁵⁻²⁴ reported weight losses ranging from 2.4 to 4.8 kg. The MED and ILI interventions reported the largest weight loss at 1 year, 6.2 and 8.4 kg, respectively,^{21,25} and a low-CHO intervention reported the smallest, 1.9

kg.²⁴ In the trials reporting data collected for > 1 year, none of the average weight losses were back to baseline by the end of the studies.

A1C results

All of the studies reported the effect of weight loss on 1-year A1C values. Improvements in A1C were reported from eight of the weight loss interventions.^{15,18,21,22,25} However, one of the trials extending to 18 months¹⁸ reported significant improvements in A1C at 12 months that were not maintained to 18 months. The MED reported the largest improvement in A1C at 1 year, -1.2% ,²¹ and the ILI reported the second largest, -0.64% .²⁵ Significant improvements in A1C were also reported from the use of meal replacements¹⁵ and one low-fat,²¹ one high-protein,²² and one high-CHO²² study. At 4 years, the MED and ILI reported continued improvements in A1C, -0.9 and -0.36% , respectively.^{21,27} Perhaps of equal importance is that nonsignificant changes in A1C were reported from 11 weight loss interventions at 1 year^{16,17,19,20,23,24} and from one study at 18 months.¹⁸

Also of interest, five trials compared weight loss interventions with differing macronutrient percentages (high-MUFA vs. high-CHO,¹⁹ low-CHO vs. low-fat,²⁰ high-protein vs. high-CHO,^{22,23} and low-CHO vs. low-fat²⁴). All five reported that weight changes did not differ statistically between arms, and weight losses ranged from 1.9 to 4.0 kg. Furthermore, eight of the intervention arms reported nonsignificant changes in A1C from baseline to study end,^{19,20,23,24} and only two intervention arms (in one study) reported significant but modest changes in A1C.²² These findings support a conclusion that a variety of eating patterns with differing macronutrient percentages are moderately effective for weight loss but may not improve A1C levels.

Weight loss interventions reporting improvements in A1C at 1 year had weight losses of 4.8, 4.2, 3.0, 2.7, 2.2, and 1.0 kg, with the MED (-6.2 kg, 7.2%) and ILI (8.4 kg, 8.6%) having larger weight losses. Weight loss interventions reporting no statistically significant improvement in A1C at 1 year had similar weight losses ranging from 1.9 to 4.4 kg. Although there is overlap in the weight loss effect on A1C, it does appear that larger amounts of weight lost are more likely

to improve A1C; weight losses of ~7–8% are needed.

Lipid levels

Ten of the trials (11 weight loss intervention arms) measured lipid levels. The most consistently reported positive change was in HDL cholesterol. However, all but the ILI and MED reported nonsignificant changes in various lipids as a result of weight loss. HDL was reported for 17 of the interventions; 10 reported positive changes in HDL,^{15,17,19,20–22,24,25} and 7 reported nonsignificant changes. Triglycerides were also reported for 17 interventions; 6 reported lowering of triglycerides,^{17,21,22,25} and 11 reported nonsignificant changes. LDL cholesterol was reported from 15 interventions, with only the ILI reporting a positive change.²⁵ Total cholesterol was reported for 16 interventions, and only the MED resulted in a positive change.²¹

Blood pressure

Eight studies (14 weight loss interventions) reported the effect of weight loss on blood pressure. Five studies reported positive blood pressure changes,^{15,19,21,24,25} and three studies reported no changes.^{20,22,23}

Medication-related effects

Weight loss may also have an effect on dosage of anti-diabetes, lipid, and blood pressure medications. However, changes in these medications can also confound results of the nutrition therapy intervention. Limited data are available on medications taken at baseline and whether medication adjustments were made as a result of weight loss. Four trials did not report on medication changes;^{15,18,19,23} two studies reported no change in medications in one study arm.^{17,22} General decreases in medications were reported in seven studies from weight loss interventions.^{16,17,20–22,24,25} Only one study reported an increase in medications and that was in the Look AHEAD trial control group for lipids and is noted in the next section.²⁷

Look AHEAD (Action for Health in Diabetes) Trial

Because of its size and duration, it is important to summarize the Look AHEAD trial.^{25–27} The trial, conducted in 16 centers in the United States and planned to last 11.5 years, was designed to test whether a life-

style intervention resulting in weight loss would reduce rates of heart disease, stroke, and CVD deaths in overweight and obese people with type 2 diabetes, a group at increased risk for such events. Half of the 5,145 people enrolled in the study were randomly assigned to receive an ILI, and the other half were assigned to a control group that received a general program of diabetes support and education (DSE). Both groups received routine medical care from their own health care providers. Participants randomized to the ILI received meal replacements or structured food plans, were encouraged to achieve 175 minutes of physical activity per week, and attended three to four education/counseling sessions per month. At 4 years, participants in the ILI group averaged a weight loss of 4.7 kg compared to 1.1 kg in the control group.²⁷

In September 2012, the National Institutes of Health stopped the ILI group early, acting on the recommendation of the study's data and safety monitoring board.²⁸ The independent advisory board found that the ILI did no harm but was not on a trajectory that would result in greater decreases in cardiovascular events compared to the control group. The board recommended continuing to follow all Look AHEAD participants to identify other potential longer-term effects of the intervention.

The benefits on A1C, HDL cholesterol, triglycerides, and blood pressure were significantly greater in the ILI group compared to the DSE group after 4 years (weight $P < 0.001$, A1C $P < 0.001$, HDL cholesterol $P < 0.001$, triglycerides $P < 0.001$, systolic blood pressure $P < 0.001$, and diastolic blood pressure $P = 0.01$), but reductions in LDL cholesterol were greater in the DSE group ($P = 0.009$) because of more aggressive use of medications to lower lipid levels in the this group.²⁷ It is encouraging and important to note that both groups had a lower number of cardiovascular events compared to previous studies of people with diabetes.²⁷

MNT for Diabetes

So, if weight loss is not the complete nutrition therapy answer for improvements in cardiometabolic outcomes for diabetes, what other interventions have been reported to be beneficial? A systematic review was conducted to determine the effectiveness of MNT

provided by a registered dietitian (or nutritionist) independently or as part of an overall diabetes self-management education (DSME) program. Although weight loss is sometimes reported, it is not the primary goal of the nutrition therapy interventions. Eleven studies published after 2000 reported improvements in A1C with independent MNT interventions, and seven studies showed improvement when MNT was part of DSME.²⁹ Randomized, clinical studies and observational outcome studies documented decreases in A1C of ~1–2% (range –0.5 to –2.6%), depending on the type and duration of diabetes and the baseline A1C value.

Of interest are the types of nutrition therapy interventions that are most effective. Interventions for people with type 2 diabetes include reduced energy/fat intake, individualized MNT, portion control and healthy food choices, and carbohydrate counting, and for people with type 1 diabetes, carbohydrate counting and matching insulin to carbohydrate intake. Although it is clear that there is not one nutrition therapy intervention that applies to all people with diabetes, a consistent theme for individuals with type 2 diabetes is that a reduced energy intake, which may or may not lead to substantial weight loss, consistently improves glycemic control.

An eating pattern designed to both lower glucose and improve lipids and blood pressure, along with regular physical activity, is the cornerstone of diabetes care.^{30,31} A cardioprotective eating pattern provides 25–35% of calories from fat, with <7% of calories from saturated and *trans* fatty acids. The majority of the fat intake is from unsaturated fatty acids. Cholesterol intake is ideally <200 mg/day. Evidence indicates that this type of eating pattern can reduce total cholesterol by 7–21%, LDL cholesterol by 7–22%, and triglycerides by 11–31%.³² In addition, controlling sodium intake to 2,400 mg/day has an approximate systolic blood pressure–lowering range of 2–8 mmHg.³¹

Although the ILI in the Look AHEAD trial resulted in weight loss and improved A1C, some lipids, and blood pressure, it did not improve these risk factors enough to result in better CVD protection than standard diabetes (education/medical) care. Equally important, it is not clear how

Table 1. Diabetes Weight Loss Trials: Outcomes of Interventions at 1 Year Compared to Baseline Values (Five Trials With > 1 Year Outcomes Also Reported)

Study	Subjects Enrolled (n [n of completers, percentage of completers])	Interventions	Weight Loss (kg)	A1C (%)	Lipids (mg/dl)	Blood Pressure (mmHg)
Metz et al. ¹⁵	119 (92, 77%)	1. Prepared meal plan (meal replacements) 2. Usual care (reduced energy intake)	1. ↓3.0 ± 5.4 2. ↓1.0 ± 3.8	1. ↓0.24 ± 1.52 (P < 0.0001) 2. ↓0.2 ± 1.30 (P < 0.0001)	1. TC ↑6.2 ± 29.2, LDL ↑7.0 ± 26.7, HDL ↑1.9 ± 5.7, TG ↓14.2 ± 126 (all NS) 2. TC ↑1.0 ± 41.7, LDL ↓0.3 ± 24.6, HDL ↑0.3 ± 5.2, TG ↑5.1 ± 274.0 (all NS)	1. SBP ↓8.8 ± 12.6 1. DBP ↓5.1 ± 5.6 (both P < 0.0001) 2. SBP ↓.9 ± 13.2 2. DBP ↓3.8 ± 6.2 (both P < 0.0001)
Wolf et al. ¹⁶	144 (115, 80%)	1. Case management (individualized food plan) 2. Usual care	1. ↓2.4 (↓4.1 to ↓0.6) 2. ↑0.6 (↑1.0 to ↑2.2)	1. ↓0.1 (NS) 2. ↓0.09 (NS)	TC, LDL, HDL, TG (both groups NS changes)	Not reported
Li et al. ¹⁷	104 (82, 79%)	1. Soy-based meal replacement 2. Individualized food plan	1. ↓4.4 ± 0.8 (P < 0.0001) 2. ↓2.4 ± 0.8 (P = 0.038)	1. ↓0.30 (NS) 2. ↓0.15 (NS)	1. TC ↓10.7, LDL ↓6.1, HDL ↑1.0 (all NS); TG ↓28 (P = 0.038) 2. TC ↓5.3, LDL ↑8.8, TG ↓28 (all NS); HDL ↑2.3 (P = 0.012)	Not reported
West et al. ¹⁸	217 (195, 90% at 12 months) 217 (202, 90% at 18 months)	1. Group behavioral weight management 2. Group behavioral weight management plus motivational interviewing	1. ↓2.7 ± 0.6 2. ↓4.8 ± 0.6 1. ↓1.7 ± 0.6 2. ↓3.5 ± 0.6	1. ↓0.6 ± 0.1 (P < 0.0001) 2. ↓0.4 ± 0.01 (P < 0.0001) 1. ↓0.2 ± 0.1 (NS) 2. ↓0.1 ± 0.1 (NS)	Not reported	Not reported
Brehm et al. ¹⁹	124 (95, 77% at 12 months)	1. High monounsaturated fat 2. High carbohydrate	1. ↓4.0 ± 0.8 (P < 0.01) 2. ↓3.8 ± 0.6 (P < 0.01)	1. ↑0.1 (NS) 2. No change (NS)	1. TC ↑5, LDL ↓3, TG ↓1 (all NS); HDL ↑5 (P < 0.01) 2. TC ↑2, LDL ↓3, HDL ↑5, TG ↓5 (all NS); HDL ↑5 (P < 0.01)	1. SBP ↓2 (NS) 1. DBP ↓5 (P < 0.01) 2. SBP ↓1 (NS) 2. DBP ↓4 (P < 0.01)
Davis et al. ²⁰	57 (38, 67% at 18-month extension) 105 (85, 81%)	1. Low carbohydrate 2. Low fat	No significant change from 12 months 1. ↓3.1 ± 4.8 (P = 0.005) 2. ↓3.1 ± 5.8 (P = 0.005)	No significant change from 12 months 1. ↓0.02 ± 0.89 (NS) 2. ↑0.24 ± 1.4 (NS)	No significant changes from 12 months 1. TC ↑3.9 ± 29.4, LDL ↓1.5, TG ↓13.3 (all NS); HDL ↑6.2 (P = 0.002) 2. TC ↓5.0 ± 27.1, LDL ↓7.0, TG ↓1.0, HDL ↑2.3 (all NS)	No significant changes from 12 months 1. SBP ↑2.0 ± 15.6 1. DBP ↓2.9 ± 9.4 2. SBP ↓1.8 ± 22.6 2. DBP ↓2.3 ± 11.6 (all NS)

Esposito et al. ^{21*}	215 (195, 91% at 1 year)	1. Mediterranean-style diet (MED) 2. Low fat	1. ↓6.2 ± 3.2 2. ↓4.2 ± 1.9	1. ↓1.2 ± 1.0 2. ↓0.6 ± 0.6	1. TC ↓15.1 ± 14.7, HDL ↑3.9 ± 4.6, TG ↓39.0 ± 50.5 2. TC ↓5.8 ± 6.6, HDL ↑1.0 ± 0.8, TG ↓19.4 ± 40.0	1. SBP ↓5.1 ± 4.2 1. DBP ↓4.0 ± 3.0 2. SBP ↓2.0 ± 1.9 2. BP ↓1.0 ± 1.0
	215 (195, 91% at 4 years)		1. ↓3.8 ± 2.0 2. ↓3.2 ± 1.9	1. ↓0.9 ± 0.6 2. ↓0.5 ± 0.4	1. TC ↓9.7 ± 7.7, HDL ↑3.5 ± 3.1, TG ↓24.8 ± 24.6 2. TC ↓3.9 ± 7.9, HDL ↑1.0 ± 0.1, TG ↓6.2 ± 8.9	1. SBP ↓2.5 ± 2.6 1. DBP ↓2.9 ± 1.9 2. SBP ↓1.0 ± 1.0 2. DBP ↓1.5 ± 1.4
Larsen et al. ²²	108 (99, 92% at 1 year)	1. High protein 2. High carbohydrate	1. ↓2.2 (P < 0.001) 2. ↓2.2 (P < 0.001)	1. ↓0.23 (P < 0.001) 2. ↓0.28 (P < 0.001)	1. TC ↓5.8 (NS), LDL ↓1.9 (NS), HDL ↑3.1 (P = 0.008), TG ↓41.6 (P < 0.001) 2. TC ↓0.4 (NS), LDL ↓1.5 (NS), HDL ↑3.1 (P = 0.008), TG ↓26.6 (P < 0.001)	1. SBP ↓5.0 1. DBP ↓0.2 2. SBP ↓0.8 2. SBP ↓0.7 (all NS)
	419 (310, 74% at 12 months)	1. High protein 2. High carbohydrate	1. ↓3.2 (P < 0.001) 2. ↓2.4 (P < 0.001)	1. ↓0.18 (NS) 2. ↓0.20 (NS)	1. TC ↓3.9, LDL ↓2.3, HDL ↑0.8, TG ↓9.7 (all NS) 2. TC ↓2.7, LDL ↓3.1, HDL ↑0.8, TG ↑1.8 (all NS)	1. SBP ↓0.2 1. DBP ↓0.1 2. SBP ↓0.5 2. DBP ↓0.5 (all NS)
Krebs et al. ²³	419 (294, 70% at 24 months)		1. ↓3.9 (P < 0.001) 2. ↓3.0 (P < 0.001)	1. ↑0.1 (NS) 2. ↑0.1 (NS)	1. TC ↓9.2 (P = 0.02), LDL ↓6.6 (NS), HDL ↓0.4 (NS), TG ↓3.5 (NS) 2. TC ↓12.8 (P = 0.02), LDL ↓7.7 (NS), HDL ↑0.7 (NS), TG ↓0.9 (NS)	1. SBP ↑2.0 1. DBP ↓0.3 2. SBP ↑1.0 2. DBP ↓0.4 (all NS)
	61 (54, 89% at 12 months)	1. Low carbohydrate 2. Low fat	1. ↓1.9 (P < 0.001) 2. ↓3.9 (P < 0.001)	1. ↓0.2 (NS) 2. ↑0.1 (NS)	1. TC ↓7.7 (NS), LDL ↓7.7 (NS), HDL ↑4.3 (P = 0.024), TG ↓26.6 (NS) 2. TC 0.0 (NS), LDL ↓3.9 (NS), HDL ↑3.1 (P = 0.029), TG ↓8.9 (NS)	1. SBP ↓8 (P = 0.003) 1. DBP ↓6 (P = 0.002) 2. SBP ↓10 (P < 0.001) 2. DBP ↓8 (P < 0.001)
Guldbrand et al. ²⁴	61 (47, 77% at 24 months)		1. ↓2.0 (P = 0.020) 2. ↓2.9 (P = 0.002)	1. 0.0 (NS) 2. ↑0.2 (NS)	1. TC ↓3.9 (NS), LDL ↓11.6 (P = 0.020), HDL ↑8.9 (P < 0.001), TG ↓17.7 (NS) 2. TC ↓11.6 (NS), LDL ↓11.6 (P = 0.017), HDL ↑4.3 (P = 0.050), TG ↓8.9 (NS)	1. SBP ↓9 (P = 0.012) 1. DBP ↓5 (P = 0.004) 2. SBP ↓11 (P < 0.001) 2. DBP ↓6 (P = 0.001)
	5,145 (4,959, 96% at 1 year)	1. Intensive lifestyle intervention (ILI) 2. Diabetes support/education (control)	1. ↓8.7 ± 6.9 (P < 0.0001) 2. ↓0.7 ± 4.8	1. ↓0.64 ± 1.02 (P < 0.0001) 2. ↓0.14	1. LDL ↓5.2 ± 28.63, HDL ↑3.4 ± 0.2, TG ↓30.3 ± 2.0 (all P < 0.0001) 2. LDL ↓5.7 ± 0.6, HDL ↑1.4 ± 0.1, TG ↓14.6 ± 1.8	1. SBP ↓6.8 ± 0.4 1. DBP ↓3.0 ± 0.2 (both P < 0.0001) 2. SBP ↓2.8 ± 0.3 2. DBP ↓1.8 ± 0.2
Look AHEAD Research Group ^{25-27**}	5,145 (4,815, 94% at 4 years)		1. ↓4.7 ± 0.2 2. ↓1.1 ± 0.2	1. ↓0.36 (0.4-0.33) 2. ↓0.09 (0.13-0.06)	1. LDL ↓8.8 (12.1-10.4), HDL ↑3.7 (3.43-3.91), TG ↓25.6 (27.9-23.2) 2. LDL ↓9.2 (10.0-8.4), HDL ↑2.0 (1.73-2.22), TG ↓19.8 (22.11-23.21)	1. SBP ↓5.3 (5.8-4.96) 1. DBP ↓2.9 (3.2-2.7) 2. SPB ↓3.0 (3.4-2.5) 2. DBP ↓2.5 (2.8-2.2)

*Statistical significance from baseline not reported.

**Statistical significance from baseline only reported for 1-year ILI.

DBP, diastolic blood pressure; HDL, HDL cholesterol; LDL, LDL cholesterol; NS, not significant; PA, physical activity; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides.

the intervention package might be delivered in a real-world medical setting. It involved weekly sessions for the first 6 months, sessions three times per month for the next 6 months, and, for years 2–4, at least monthly contact by telephone or e-mail, as well as a variety of ancillary group classes in between contacts.²⁷

In contrast, the MED (plus physical activity) intervention, which also improved A1C, lipids, and blood pressure, involved monthly sessions for the first year and bimonthly sessions thereafter. An interesting side note is that, although the authors described the Mediterranean-style eating plan as low carbohydrate, the actual intent was to have the carbohydrate content be < 50% of the reduced daily energy, with added fat being 30–50 g of olive oil.²¹ Reported intake at 1 year included an average carbohydrate intake of 43% of calories, protein 28%, polyunsaturated fats 29%, and saturated fats 10%. This could better be described as a moderate-carbohydrate eating pattern, which is typical of people with type 2 diabetes.³³

In summary, the most common nutrition advice given to people with type 2 diabetes involves weight loss. But weight loss is atypically substantial (< 5%) over the long term and, even if successful, may not result in cardiometabolic outcomes as strong as clinicians and individuals with diabetes would like to see. More realistic and helpful nutrition advice for overweight individuals with diabetes may be to pay less attention to the scale and concentrate more on eating smaller portions while choosing healthy foods such as fruits, vegetables, whole grains, legumes, low-fat dairy products, lean meats, and unsaturated fats in appropriate portion sizes.

To prevent and treat CVD, nutrition therapy for diabetes, instead of focusing on weight loss, should focus on 1) nutrition interventions shown to improve metabolic outcomes (glycemia, lipids, and blood pressure), 2) prioritizing goals for individuals, 3) negotiating lifestyle changes individuals are willing and able to make, and 4) assisting patients in choosing appropriate portion sizes of foods shown to have health benefits.

References

¹Flegal KM, Kit B, Graubard B: Association for all-cause mortality with overweight and

obesity using standard body mass index categories: systematic review and meta-analysis. *JAMA* 309:71–82, 2013

²Angerås O, Albertsson P, Karason K, Råmandal T, Matejka G, James S, Lagervist B, Rosengren A, Omerovis E: Evidence for obesity paradox in patients with acute coronary syndrome: a report from the Swedish Coronary Angiography and Angioplasty Registry. *Eur Heart J* 34:345–353, 2013

³Doehner W, Schenkel J, Anke S, Springer J, Audebert H: Overweight and obesity are associated with improved survival, functional outcomes, and stroke recurrence after acute stroke or transient ischaemic attack: observations from the TEMPIs trial. *Eur Heart J* 34:268–277, 2013

⁴Logue J, Walker JJ, Leese G, Lindsay R, McKnight J, Morris A, Philip S, Wild S, Sattar N, on behalf of the Scottish Diabetes Research Network Epidemiology Group: Association between BMI measured within a year after diagnosis of type 2 diabetes and mortality. *Diabetes Care* 36:887–893, 2013

⁵Kokkinos P, Myers J, Faselies C, Doumas M, Kheifbek R, Nylen E: BMI-mortality paradox and fitness in African American and Caucasian men with type 2 diabetes. *Diabetes Care* 35:1021–1027, 2012

⁶Carnethon MR, De Chavez PJ, Biggs ML, Lewis CE, Pankow JS, Bertoni AG, Golden SH, Liu K, Mukamal KJ, Campbell-Jenkins B, Dyer AR: Association of weight status with mortality in adults with incident diabetes. *JAMA* 308:581–590, 2012

⁷McEwen LN, Kim C, Karter AJ, Haan MN, Ghosh D, Llantz PM, Mangione CM, Thompson TJ, Herman WH: Risk factors for mortality among patients with diabetes. *Diabetes Care* 30:1736–1741, 2007

⁸Doehner W, Erdman E, Cairns R, Clark AL, Dormandy JA, Ferrannini E, Anker SD: Inverse relation of body weight and weight change with mortality and morbidity in patients with type 2 diabetes and cardiovascular co-morbidity: an analysis of the PROactive study population. *Int J Cardiol* 162:20–26, 2012

⁹Tseng CH: Obesity paradox: differential effects on cancer and noncancer mortality in patients with type 2 diabetes mellitus. *Atherosclerosis* 226:186–192, 2013

¹⁰Centers for Disease Control and Prevention: Overweight and obesity: causes and consequences. Available from <http://www.cdc.gov/obesity/adult/causes/index.html>. Accessed 10 February 2013

¹¹Klein R, Klein BE, Moss SE: Is obesity related to microvascular and macrovascular complications in diabetes? The Wisconsin Epidemiologic Study of Diabetic Retinopathy. *Arch Intern Med* 157:650–656, 1997

¹²Ross C, Langer RD, Barrett-Connor E: Given diabetes, is fat better than thin? *Diabetes Care* 20:650–652, 1997

¹³American Diabetes Association: Nutrition recommendations and interventions for diabetes: a position statement of the American Diabetes Association. *Diabetes Care* 31 (Suppl. 1):S61–S78, 2008

¹⁴American Diabetes Association: Standards of medical care in diabetes—2013 (Position Statement). *Diabetes Care* 36 (Suppl. 1):S11–S66, 2013

¹⁵Metz JA, Stern JS, Kris-Etherton P, Reusser ME, Morris CD, Hatton DC, Haynes B, Resnick LM, Pi-Sunyer X, Clark S, Chester L, McMahon M, Snyder GW, McCarron DA: A randomized trial of improved weight loss with a prepared meal plan in overweight and obese patients. *Arch Intern Med* 160:2150–2158, 2000

¹⁶Wolf AM, Conaway MR, Crowther JQ, Hazen KY, Nadler JL, Oneida B, Bovbjerg VE: Translating lifestyle intervention to practice in obese patients with type 2 diabetes. *Diabetes Care* 27:1570–1576, 2004

¹⁷Li Z, Hong K, Saltsman P, DeShields S, Bellman M, Thames G, Liu Y, Wang H-J, Elashoff R, Heber D: Long-term efficacy of soy-based meal replacement vs an individualized diet plan in obese type II DM patients: relative effects on weight loss, metabolic parameters, and C-reactive protein. *Eur J Clin Nutr* 59:411–418, 2005

¹⁸West DS, DiLillo V, Bursac Z, Gore SA, Greene PG: Motivational interviewing improves weight loss in women with type 2 diabetes. *Diabetes Care* 30:1081–1087, 2007

¹⁹Brehm BJ, Lattin BL, Summer SS, Boback JA, Gilchrist GM, Jandacek RJ, D'Alessio DA: One-year comparison of a high-mono-unsaturated fat diet with a high-carbohydrate diet in type 2 diabetes. *Diabetes Care* 32:215–220, 2009

²⁰Davis NJ, Tomuta N, Schechter C, Isasi CR, Segal-Isaacson CJ, Stein D, Zonszein JZ, Wylie-Rosett J: Comparative study of the effects of a 1-year dietary intervention of a low-carbohydrate diet versus a low-fat diet on weight and glycemic control in type 2 diabetes. *Diabetes Care* 32:1147–1152, 2009

²¹Esposito K, Maiorino MI, Ciotola M, Di Paol C, Scognamiglio P, Gicchino M, Petrizzo M, Saccomanno F, Beneduce F, Ceriello A, Giuffliano D: Effects of a Mediterranean-style diet on the need for antihyperglycemic drug therapy in patients with newly diagnosed type 2 diabetes: a randomized trial. *Ann Intern Med* 151:306–314, 2009

²²Larsen RN, Mann NJ, Maclean E, Shaw JE: The effect of a high-protein, low-carbohydrate diets in the treatment of type 2 diabetes: a 12 month randomized controlled trial. *Diabetologia* 54:731–740, 2011

²³Krebs JD, Elley CR, Parry-Strong A, Lunt H, Drury PL, Bell DA, Robinson E, Moyes SA, Mann JI: The Diabetes Excess Weight Loss (DEWL) Trial: a randomized controlled trial of high-protein versus high-carbohydrate diets over 2 years in type 2 diabetes. *Diabetologia* 55:905–914, 2012

²⁴Guldbrand H, Dizdar B, Bunjaku B, Lindström T, Bachrach-Lindström M, Fredrikson M, Fredrikson M, Östgren CJ, Nystrom FH: In type 2 diabetes, randomization to advice to follow a low-carbohydrate diet transiently improves glycaemic control compared with advice to follow a low-fat diet producing similar weight loss. *Diabetologia* 55:2118–2127, 2012

²⁵Look AHEAD Research Group: Reduction in weight and cardiovascular disease risk factors in individuals with type 2 diabetes: one-year results of the Look AHEAD trial. *Diabetes Care* 30:1374–1383, 2007

²⁶Wing RR, Lang W, Wadden TA, Safford M, Knowler WC, Bertoni AG, Hill JO, Brancati FL, Peters A, Wagenknecht L, The Look AHEAD Research Group: Benefits of modest weight loss in improving cardiovascular risk factors in overweight and obese individuals with type 2 diabetes. *Diabetes Care* 34:1481–1486, 2011

²⁷Look AHEAD Research Group: Long-term effects of a lifestyle intervention on weight and cardiovascular risk factors in individuals with type 2 diabetes mellitus: four-year results of the Look AHEAD trial. *Arch Intern Med* 170:1566–1575, 2010

²⁸National Institute of Diabetes and Digestive and Kidney Diseases: Weight loss does not lower heart disease risk from type 2 diabetes.

Available from <http://www.nih.gov/news/health/oct2012/niddk-19.htm>. Accessed 13 February 2013

²⁹Pastors JG, Franz MJ: Effectiveness of medical nutrition therapy in diabetes. In *American Diabetes Association Guide to Nutrition Therapy for Diabetes*. 2nd ed. Franz MJ, Evert AB, Eds. Alexandria, Va., American Diabetes Association, 2012, p. 1–18

³⁰Karmally E, Zimmerman JS: Nutrition therapy for diabetes and lipid disorders. In *American Diabetes Association Guide to Nutrition Therapy for Diabetes*. 2nd ed. Franz MJ, Evert AB, Eds. Alexandria Va., American Diabetes Association, 2012, p. 265–294

³¹Aebersold K, Ostrovsky N, Wylie-Rosett J: Nutrition therapy for diabetes and hypertension. In *American Diabetes Association Guide to Nutrition Therapy for Diabetes*. 2nd ed. Franz MJ, Evert AB, Eds. Alexandria

Va., American Diabetes Association, 2012, p. 295–306

³²Academy of Nutrition and Dietetics: Disorders of lipid metabolism evidence-based nutrition practice guidelines update, 2010. Available from <http://www.adaevidence.library.com/topic.ctm?cat=4528>. Accessed 20 February 2013

³³Vitolins MZ, Anderson AM, Delahanty L, Raynor H, Miller GD, Mobley C, Reeves R, Yamamoto M, Champagne C, Wing RR, Mayer-Davis E, and the Look AHEAD Research Group: Action for Health in Diabetes (Look AHEAD) Trial: baseline evaluation of selected nutrients and food groups. *J Am Diet Assoc* 109:1367–1375, 2009

Marion J. Franz, MS, RD, CDE, is a nutrition/health consultant at Nutrition Concepts by Franz, Inc., in Minneapolis, Minn.