

# Dietary Treatment and Long-Term Weight Loss and Maintenance in Type 2 Diabetes

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## Abstract

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Increasing body weight, particularly abdominal weight, is associated with increasing risk for type 2 diabetes, and 80% of people with type 2 diabetes are overweight or obese. Weight loss and maintenance are challenging in the obese population without diabetes, and data suggest that this may be more difficult in obese people with diabetes. Various weight-loss strategies with follow-up for at least 1 year have been evaluated in people with diabetes with mixed results. Diet is most effective in promoting initial weight loss. Energy restriction will improve glycemic control within days of initiation, independent of weight loss. There is deterioration of the effects of dietary therapy on glycemic control over time, even with partial weight maintenance, because of the relaxation of energy restriction. Diet composition has little effect on glycemic control independent of total calories. Very-low-calorie diets lead to better initial weight loss and glycemic control but yield no better long-term results than more moderate treatment. The initial results from studies using prepared meals and liquid meal replacements show that weight loss and glycemic control are comparable with conventional dietary treatment. Comprehensive lifestyle therapies, involving diet, exercise, and behavioral modification, can lead to weight losses of ~2 to 10 kg over 10 to 20 weeks, with regain over 1 year of one-third to one-half of weight initially lost. The net improvement on glycemic control is usually small 1 year after weight loss. Creative strategies using these and other modalities are needed to improve long-term weight loss, weight maintenance, and glycemic control in patients with type 2 diabetes. Greater efforts in primary prevention are

also needed because of the increasing prevalence of obesity and type 2 diabetes.

**Key words:** type 2 diabetes, weight loss, glycemic control

## Introduction

Increasing body weight is associated with increasing risk for glucose intolerance and type 2 diabetes (1–6). Conversely, ~80% of patients with type 2 diabetes are overweight or obese. The prevalence of type 2 diabetes in the United States increased by one-third in the 1990s and in concert with the increasing prevalence of obesity over the past few decades (7,8). Greater abdominal distribution of weight and visceral obesity also increase the risk for type 2 diabetes (4,9,10). Other modifiable factors that may increase the risk for type 2 diabetes include low levels of physical activity (11–14) and a diet low in fiber and high in glycemic index (15,16).

Both obesity and type 2 diabetes are associated with increased morbidity and mortality (17,18). Greater amounts of abdominal obesity is associated with insulin resistance, which plays a central role in the metabolic constellation that includes dyslipidemia, hypertension, and increased risk of cardiovascular disease, in addition to type 2 diabetes (19–22). Therefore, weight loss is extremely important to improve glycemic control and to decrease other risks associated with diabetes and obesity.

Short-term weight-loss results in improved glycemic control in patients with diabetes by decreasing insulin resistance, decreasing hepatic glucose production, and possibly increasing insulin secretion (23–25). The effect of energy restriction on improving glycemic control is apparent within days after initiation of dietary change and is independent of weight loss (25,26). Physical activity and physical training increase glucose uptake and insulin sensitivity (27,28). Sustaining the short-term benefits of glycemic control requires long-term maintenance of weight loss, although there is some deterioration of the effects of dietary treatment over time primarily due to the inevitable relaxation of energy restriction as weight plateaus (29–31). Weight loss in pa-

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tients with diabetes improves control of associated comorbidities, including hypertension and dyslipidemia (32). Intentional weight loss by patients with type 2 diabetes also seems to decrease mortality (33).

Not all patients with type 2 diabetes respond to weight loss with adequate improvement in glycemic control (34). Moreover, in patients that do respond, the magnitude of improvement in glycemic control is related to the degree of weight loss, and relatively large amounts of weight loss and considerable maintenance are needed to maintain near-normal levels of fasting glucose (29,31). However, these considerations need to be balanced against the practical likelihood of achieving and maintaining significant amounts of weight loss. In addition, modest long-term weight loss of 5% to 10% should result in improvement of glycemic control in most patients (29,35).

Many studies have shown that weight loss maintenance is challenging in the general population (36–38). Weight loss and maintenance are more imperative in type 2 diabetes because of the increased health risks associated both with diabetes and obesity. Despite the increased need for weight management in overweight people with diabetes, weight loss may be more difficult to initiate and maintain than in people without diabetes (39–42). It has been suggested that having diabetes for a long duration or treatment with insulin may contribute to this difficulty in losing weight. However, data are not consistent with this contention (43).

The treatment of obesity in type 2 diabetes should involve diet, physical activity, and behavioral modification and may involve pharmacotherapy or surgery. Previous reviews have discussed the prevention and treatment of obesity in type 2 diabetes in detail (32,44). This article will briefly review the effect of dietary treatment on weight loss and long-term weight maintenance in overweight and obese patients with type 2 diabetes.

### ***The UK Prospective Diabetes Study***

The UK Prospective Diabetes Study provided useful information on initial dietary treatment of type 2 diabetes (31). In this study, of the initial 3044 newly diagnosed patients with diabetes, 447 were excluded because of an inability to achieve a fasting blood glucose level of <15 mM after 3 months of dietary therapy. Of the remaining 2597 patients (mean body weight,  $132 \pm 26\%$  ideal body weight), mean weight loss was 5 kg over these initial 3 months.

Four hundred eighty-two patients (16%) achieved a fasting glucose level of <6 mM after 3 months of dietary treatment. Patients who had higher initial fasting blood glucose levels and patients who lost more weight experienced the greatest decrease in levels of fasting blood glucose. Initial body weight showed no relationship to glycemic control. From a different perspective, to achieve a normal (<6 mM) fasting blood glucose level required relatively large amounts of weight loss (e.g., 19 kg with a

baseline fasting blood glucose of 12 mM), and the greater the initial fasting blood glucose level, the larger the amount of weight loss that was required. In 823 subjects who continued on dietary treatment, weight 1 year later was virtually the same as after the first 3 months, although there was slight worsening of fasting blood glucose levels.

### ***Long-Term Diet Studies***

For methodological reasons, it is difficult to accurately determine the independent effect of long-term dietary treatment on weight and glycemic control in patients with diabetes. Studies have used different designs, settings, diets, adjuvant treatments, or have not used controls. Often, diet was appropriately combined with exercise and behavioral modification techniques. Finally, subjects lost to follow-up make it more difficult to determine true effect.

In summarizing the results of previous studies in which the effect of diet could be estimated, weight loss usually is maximized by 3 to 6 months and ranges from none up to ~8 kg, depending on the treatment used (32,45). More intensive treatment increases weight loss. Regardless of the approach, one-third to one-half of the weight initially lost is regained within the first year, so that weight loss from baseline by 1 year rarely exceeds 5 kg.

### ***Diet Composition***

The long-term effects of diet composition on weight loss and glycemic control in patients with type 2 diabetes have been addressed in few studies. There is consistent agreement that dietary saturated fat should be low, particularly because of the increased risk of cardiovascular disease in patients with diabetes (46). To promote weight loss, total calories should be limited, regardless of the source. This can be accomplished by decreasing the intake of saturated fat and not replacing these calories.

Beyond decreasing dietary saturated fat and total calories, controversy exists as to whether the diet should be low in total fat and high in carbohydrate, or moderate to high in total fat with a relatively high proportion of monounsaturated fat (47). Consuming a low-fat diet may or may not promote weight loss, depending on the amount of nonfat calories that are consumed (48). A randomized trial of 44 obese women with type 2 diabetes assessed weight loss and glycemic control on a calorie-restricted diet vs. a calorie-plus fat-restricted diet (49). After 1 year, those following the calorie plus fat restriction lost more weight (5.2 kg vs. 1.0 kg,  $p < 0.05$ ) but there was no significant difference in glycemic control. Subjects in the calorie plus fat restriction group consumed ~200 fewer calories/d at the end of the 16-week program ( $p < 0.05$ ) and at 1-year follow-up, although the difference was not significantly different at 1 year. Thus, decreasing dietary fat intake may help reduce body weight if it helps to decrease total calorie intake.

Short-term studies suggest similar glycemic control but greater improvement of serum lipids when saturated fat is

replaced by monounsaturated fat vs. carbohydrate (50). However, it may be more difficult for patients to self-select a higher proportion of monounsaturated fat, yet reduce total calories. Concern has been expressed that high-carbohydrate diets will raise serum triglycerides. However, if weight loss is realized, the effect of weight loss seems to predominate over the hypertriglyceridemic effect of carbohydrate (51). In addition, simple sugars can be incorporated into an overall dietary plan without adversely affecting blood glucose levels (52,53).

Energy density refers to the number of calories in a given weight or volume of food. Fat, sugar, and processed food contain a relatively large amount of calories in a small volume and, therefore, a high energy density. Fruits and vegetables contain a relatively small amount of calories in a large volume because of the high content of water and fiber and, therefore, have a low energy density. Consuming a low-energy dense diet seems to promote increased satiety, decreased energy intake, and weight loss in nondiabetic subjects, but it has not been adequately studied in people with diabetes (54). A low-energy dense diet that is high in vegetables and fruits contains an increased amount of fiber, which has an independent effect on improving glycemic control and serum lipids (55,56). It is controversial whether consuming foods with a low glycemic index will improve glycemic control. This controversy notwithstanding, it is unlikely that changes in dietary glycemic index will affect body weight without decreasing total calories.

**Prepared Meals**

A randomized trial of 119 people with type 2 diabetes that were not on insulin and 183 people with hypertension and dyslipidemia evaluated the impact of a prepared meal plan compared with a usual care diet plan (51). After 1 year, weight loss in the subjects with diabetes was 3.0 kg with prepared meals compared with 1.0 kg with the usual care diet plan (Table 1). Although subjects with diabetes who were consuming prepared meals experienced significantly greater reductions in weight, fasting blood glucose levels, and hemoglobin A1c during the study, absolute reductions at 1 year were similar between groups. Subjects with diabetes lost approximately one-half the amount of weight that subjects without diabetes lost, consistent with previous studies.

**Liquid Meal Replacements**

A study of 29 obese subjects with type 2 diabetes controlled by diet or oral medications evaluated liquid meal replacements compared with an isocaloric energy-restricted diet (57). Maximum weight loss was achieved after 3 months on both programs and glycemic control improved in both groups (Table 2). Overall, there were no significant differences in weight or glycemic control between the groups at 1 year.

Prepared meals and liquid meal replacements offer alternatives to traditional dietary plans for patients with diabetes.

**Table 1.** Changes in weight and glycemic control in a randomized trial of a prepared meal plan in type 2 diabetes (51)

	LCD	Prepared meal plan
Weight, kg		
3 months	-1.3	-4.7†
1 year	-1.0	-3.0
Glucose, mg/dL		
3 months	-11	-39*
1 year	-11	-11
Glycosylated hemoglobin, %		
3 months	-0.3	-1.0*
1 year	-0.2	-0.2

\*  $p < 0.05$  during the study.

†  $p < 0.001$  during the study.

The results on weight loss and glycemic control are as good or, in some cases, slightly better than traditional dietary treatment. Both plans offer increased convenience. Prepared meals may cost approximately the same, whereas liquid meal replacements cost slightly less than traditional dietary plans.

**Very-Low-Calorie Diets (VLCD)**

VLCDs contain 400 to 800 kcal/d. A study with particularly favorable results involved 93 subjects with type 2 diabetes and evaluated a year-long treatment of a low-calorie diet (LCD) of 1000 to 1200 kcal/d, or an LCD that included two 12-week periods of a VLCD (400 to 500 kcal/d) (30). At 1 year, weight loss was 14.2 kg in the VLCD group compared with 7.2 kg in those following the LCD; however, there were no significant differences between groups in fasting blood glucose or glycosylated hemoglobin levels. After 2 years, weight loss, fasting glucose, and glycosylated hemoglobin levels were not significantly different between the two groups.

Overall, the results of these and other studies in patients with type 2 diabetes suggest that a program incorporating a VLCD results in greater initial improvement in weight loss and glycemic control compared with a LCD (32). Over time, there is greater recidivism after a VLCD, so that long-term weight loss is no different compared with a LCD. Repeated VLCDs in patients with diabetes seem to have a reduced effect on weight loss after the first VLCD, primarily due to decreased adherence (30,58). Whether the temporary benefits on weight loss and glycemic control justify the use of a VLCD with the associated greater cost is not clear.

**Comprehensive Approach**

Most weight management programs in patients with diabetes have appropriately used a comprehensive approach

**Table 2.** Changes in weight and glycemic control in a randomized trial of liquid meal replacements in type 2 diabetes (57)

	LCD	Liquid meal replacements
Weight, kg		
3 months	-4.3	-6.9
1 year	-1.7	-2.1
Glucose, mg/dL		
3 months	-29	-39
1 year	-22	-4
Glycosylated hemoglobin, %		
3 months	-1.2	-1.7
1 year	-0.8	0.0

No significant differences between groups.

involving diet, exercise, and behavioral modification to try to maximize results. Exercise adds only a small amount of weight loss beyond dietary treatment in people without diabetes and probably diabetes as well, although not all data are consistent (48,59,60). However, exercise can improve glycemic control and can help with weight maintenance (60). Behavioral modification may increase weight loss slightly in proportion to the length of the program (61). Weight loss of from 2 to 10 kg along with improvements in glycemic control can be expected in comprehensive programs from 10 to 20 weeks in duration (32,43,44). By 1 year, most programs show that patients regain approximately one-third to one-half of the weight that was originally lost.

### Discussion

It is clear that energy restriction is the major dietary change needed to reduce body weight and to improve glycemic control among patients with type 2 diabetes who are overweight or obese. Individualization of recommendations is important for practical as well as metabolic reasons. Additional studies are needed to adequately address the long-term effects of changes in diet composition. Additional studies should also evaluate the role that prepared meals and liquid meal replacements can play as alternatives to traditional dietary therapy.

Glycemic control shows more recidivism than does weight loss and may revert to normal by 1 year. This is most likely because initial short-term results reflect the effect of energy restriction as well as weight loss, whereas long-term follow-up results are usually not affected by energy restriction. Glycemic control may even be slightly greater at follow-up than at baseline, whereas body weight remains

less than baseline if subjects are in positive energy balance and gaining weight at the time of measurement.

In addition to weight loss and glycemic control, diet and other lifestyle treatments in people with diabetes may improve dyslipidemia, hypertension, and the risk for other health conditions. Individual results vary, and, despite modest long-term results, the benefits over time may still be greater than making no attempt at weight loss.

Many of the issues related to the long-term management of type 2 diabetes are similar to those for obesity and revolve around improved maintenance of weight loss. However, other issues, such as glycemic control and the relationship of diet composition to glycemic control, are specific to diabetes. In addition, associated comorbidities along with increased morbidity and mortality from diabetes and obesity make long-term management more important, although it may be more difficult to achieve.

Achieving satisfactory long-term management of type 2 diabetes will become more critical in the future in view of the increasing prevalence of obesity and type 2 diabetes. Creative approaches are needed to improve long-term management. Also, with the prevalence of diabetes increasing, prevention becomes more important. Future studies should consider using different modalities, including more intensive lifestyle approaches, such as residence programs; public health and community programs that change environmental factors contributing to decreased physical activity and increased energy intake; increased efforts in the area of weight maintenance; long-term dietary studies evaluating diet composition, liquid meal replacements, prepared meals, and energy density and how they affect weight management and glycemic control; and finally, increased efforts in primary prevention, including children, which will hopefully help to counteract the ominous rise of obesity and type 2 diabetes and their associated increased health risks.

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### References

1. **Van Itallie TB.** Health implications of overweight and obesity in the United States. *Ann Intern Med.* 1985;103:983-8.
2. **Colditz GA, Willett WC, Rotnitzky A, Manson JE.** Weight gain as a risk factor for clinical diabetes in women. *Ann Intern Med.* 1995;122:481-6.
3. **Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC.** Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. *Diabetes Care.* 1994;17:961-9.
4. **Pi-Sunyer FX.** Weight and non-insulin-dependent diabetes mellitus. *Am J Clin Nutr.* 1996;63(suppl):426S-9.
5. **Ford ES, Williamson DF, Liu S.** Weight change and diabetes incidence: findings from a national cohort of US adults. *Am J Epidemiol.* 1997;146:214-22.

6. **Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH.** The disease burden associated with overweight and obesity. *J Am Med Assoc.* 1999;282:1523–9.
7. **Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL.** Overweight and obesity in the United States: prevalence and trends, 1960–1994. *Int J Obes Relat Metab Disord.* 1998;22:39–47.
8. **Mokdad AH, Ford ES, Bowman BA, et al.** Diabetes trends in the U.S.: 1990–1998. *Diabetes Care.* 2000;23:1278–83.
9. **Kissebah AH, Vydelingum N, Murray R, et al.** Relation of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab.* 1982;54:254–60.
10. **Ohlson LO, Larsson B, Svardsudd K, et al.** The influence of body fat distribution on the incidence of diabetes mellitus—13.5 years of follow-up of the participants in the study of men born in 1913. *Diabetes.* 1985;34:1055–8.
11. **Helmrich SP, Ragland DR, Leung RW, Paffenbarger RS Jr.** Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. *N Engl J Med.* 1991;325:147–52.
12. **Manson JE, Rimm EB, Stampfer MJ, et al.** Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet.* 1991;338:774–8.
13. **Manson JE, Nathan DM, Krolewski AS, Stampfer MJ, Willett WC, Hennekens CH.** A prospective study of exercise and incidence of diabetes among US male physicians. *J Am Med Assoc.* 1992;268:63–7.
14. **Hu FB, Sigal RJ, Rich-Edwards JW, et al.** Walking compared with vigorous physical activity and risk of type 2 diabetes in women. *J Am Med Assoc.* 1999;282:1433–9.
15. **Salmeron J, Ascherio A, Rimm EB, et al.** Dietary fiber, glycemic load, and risk of NIDDM in men. *Diabetes Care.* 1997;20:545–50.
16. **Salmeron J, Manson JE, Stampfer MJ, et al.** Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *J Am Med Assoc.* 1997;277:472–7.
17. **National Task Force on the Prevention and Treatment of Obesity.** Overweight, obesity, and health risk. *Arch Intern Med.* 2000;160:898–904.
18. **Harris MI, Cowie CC, Stern M, et al, eds.** *Diabetes in America.* 2nd ed. Washington, DC: U.S. Government Printing Office; 1995.
19. **Després J-P.** The insulin resistance-dyslipidemic syndrome of visceral obesity: effect on patients' risk. *Obes Res.* 1998;6(suppl 1):8S–17.
20. **Reaven GM.** Syndrome X: 6 years later. *J Intern Med.* 1994;236(suppl):13–22.
21. **Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Björntorp P, Tibblin G.** Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13-year follow-up of participants in the study of men born in 1913. *Br Med J (Clin Res Ed).* 1984;288:1401–4.
22. **Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjöström L.** Distribution of adipose tissue and risk of cardiovascular disease and death: a 12-year follow-up of participants in the population study of women in Gothenburg, Sweden. *Br Med J (Clin Res Ed).* 1984;289:1257–61.
23. **Henry RR, Wallace P, Olefsky JM.** Effects of weight loss on mechanisms of hyperglycemia in obese non-insulin dependent diabetes mellitus. *Diabetes.* 1986;35:990–8.
24. **Gumbiner B, Polonsky KS, Beltz WF, et al.** Effects of weight loss and reduced hyperglycemia on the kinetics of insulin secretion in obese non-insulin dependent diabetes mellitus. *J Clin Endocrinol Metab.* 1990;70:1594–602.
25. **Kelley DE, Wing R, Bronocore C, Sturis J, Polonsky K, Fitzsimmons M.** Relative effects of calorie restriction and weight loss in noninsulin-dependent diabetes mellitus. *J Clin Endocrinol Metab.* 1993;77:1287–93.
26. **Wing RR, Blair EH, Bononi P, Marcus MD, Watanabe R, Bergman RN.** Caloric restriction per se is a significant factor in improvements in glycemic control and insulin sensitivity during weight loss in obese NIDDM patients. *Diabetes Care.* 1994;17:30–6.
27. **Borghouts LB, Keizer HA.** Exercise and insulin sensitivity: a review. *Int J Sports Med.* 2000;21:1–12.
28. **Albright A, Franz M, Hornsby G, et al.** American College of Sports Medicine position stand: exercise and type 2 diabetes. *Med Sci Sports Exerc.* 2000;32:1345–60.
29. **Wing RR, Koeske R, Epstein LG, Nowalk MP, Gooding W, Becker D.** Long-term effects of modest weight loss in type II diabetic patients. *Arch Intern Med.* 1987;147:1749–53.
30. **Wing RR, Blair E, Marcus M, Epstein LH, Harvey J.** Year-long weight loss treatment for obese patients with type II diabetes: does including an intermittent very-low-calorie diet improve outcome? *Am J Med.* 1994;97:354–62.
31. **United Kingdom Prospective Diabetes Study Group.** UK prospective diabetes study 7: response of fasting plasma glucose to diet therapy in newly presenting type II diabetic patients. *Metabolism.* 1990;39:905–12.
32. **Maggio CA, Pi-Sunyer FX.** The prevention and treatment of obesity: application to type 2 diabetes. *Diabetes Care.* 1997;20:1744–65.
33. **Williamson DF, Thompson TJ, Thun M, Flanders D, Pamuk E, Byers T.** Intentional weight loss and mortality among overweight individuals with diabetes. *Diabetes Care.* 2000;23:1499–504.
34. **Watts NB, Spanheimer RG, DiGirolamo M, et al.** Prediction of glucose response to weight loss in patients with non-insulin-dependent diabetes mellitus. *Arch Intern Med.* 1990;150:803–6.
35. **Goldstein DJ.** Beneficial health effects of modest weight loss. *Int J Obes Relat Metab Disord.* 1992;16:397–415.
36. **Hensrud DD, Weinsier RL, Darnell BE, Hunter GR.** A prospective study of weight maintenance in obese subjects reduced to normal body weight without weight loss training. *Am J Clin Nutr.* 1994;6:688–94.
37. **National Institutes of Health Technology Assessment Conference Panel.** Methods for voluntary weight loss and control. *Ann Intern Med.* 1993;119:764–70.
38. **Safer DJ.** Diet, behavior modification, and exercise: a review of obesity treatments from a long-term perspective. *South Med J.* 1991;84:1470–4.
39. **Wing RR, Marcus MD, Epstein LH, Salata R.** Type II diabetic subjects lose less weight than their overweight non-diabetic spouses. *Diabetes Care.* 1987;10:563–6.
40. **Guare JC, Wing RR, Grant A.** Comparison of obese NIDDM and nondiabetic women: short-and long-term weight loss. *Obes Res.* 1995;3:329–35.

41. **Khan MA, St Peter JV, Breen GA, Hartley GG, Vessey JT.** Diabetes disease stage predicts weight loss outcomes with long-term appetite suppressants. *Obes Res.* 2000;8:43–8.
42. **Lindgarde F.** The effect of orlistat on body weight and coronary heart disease risk profile in obese patients: the Swedish Multimorbidity Study. *J Intern Med.* 2000;248:245–54.
43. **Wing RR, Shoemaker M, Marcus MD, McDermott M, Gooding W.** Variables associated with weight loss and improvements in glycemic control in type II diabetic patients in behavioral weight control programs. *Int J Obes Relat Metab Disord.* 1990;14:495–503.
44. **Brown SA, Upchurch S, Anding R, Winter M, Ramirez G.** Promoting weight loss in type II diabetes. *Diabetes Care.* 1996;19:613–24.
45. **Milne RM, Mann JI, Chisholm AW, Williams SM.** Long-term comparison of three dietary prescriptions in the treatment of NIDDM. *Diabetes Care.* 1994;17:74–80.
46. **American Diabetes Association.** Nutrition recommendations and principles for people with diabetes mellitus (position statement). *Diabetes Care.* 2001;24:S44–7.
47. **Muls E.** Nutrition recommendations for the person with diabetes. *Clin Nutr.* 1998;17(suppl 2):18–25.
48. **National Institutes of Health, National Heart, Lung, and Blood Institute.** *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report.* Bethesda, MD: National Institutes of Health; 1998.
49. **Pascale RW, Wing RR, Butler BA, Mullen M, Bononi P.** Effects of a behavioral weight loss program stressing calorie restriction versus calorie plus fat restriction in obese individuals with NIDDM or a family history of diabetes. *Diabetes Care.* 1995;18:1241–8.
50. **Heilbronn LK, Noakes M, Clifton PM.** Effect of energy restriction, weight loss, and diet composition on plasma lipids and glucose in patients with type 2 diabetes. *Diabetes Care.* 1999;22:889–95.
51. **Metz JA, Stern JS, Kris-Etherton P, et al.** A randomized trial of improved weight loss with a prepared meal plan in overweight and obese patients: impact on cardiovascular risk reduction. *Arch Intern Med.* 2000;160:2150–8.
52. **Franz MJ, Horton ES, Bantle JP, et al.** Nutrition principles for the management of diabetes and related complications (technical review). *Diabetes Care.* 1994;17:490–518.
53. **Malerbi DA, Paiva ESA, Duarte AL, et al.** Metabolic effects of dietary sucrose and fructose in type II diabetic subjects. *Diabetes Care.* 1996;19:1249–56.
54. **Rolls BJ, Bell EA.** Dietary approaches to the treatment of obesity. *Med Clin North Am.* 2000;84:401–18.
55. **Wursch P, Pi-Sunyer FX.** The role of viscous soluble fiber in the metabolic control of diabetes. *Diabetes Care.* 1997;20:1774–80.
56. **Chandalia M, Garg A, Lutjohann D, Von Bergmann K, Grundy SM, Brinkley LJ.** Beneficial effects of high dietary fiber intake in patients with type 2 diabetes mellitus. *N Engl J Med.* 2000;342:1392–8.
57. **Hensrud DD.** Weight loss, weight maintenance, and blood glucose control using liquid meal replacements in patients with type 2 diabetes mellitus. In: *Programs and Abstracts of the 82nd Annual Meeting of the Endocrine Society.* Toronto, Canada: 2000, p. 499.
58. **Smith DE, Wing RR.** Diminished weight loss and behavioral compliance during repeated diets in obese patients with type II diabetes. *Health Psychol.* 1991;10:378–83.
59. **Blonk MC, Jacobs MAJM, Biesheuvel EHE, Weeda-Mannak WL, Heine RJ.** Influences on weight loss in type 2 diabetic patients: little long-term benefit from group behavior therapy and exercise training. *Diabet Med.* 1994;11:449–57.
60. **Wing RR, Epstein LH, Paternostro-Bayles M, Kriska A, Nowalk MP, Gooding W.** Exercise in a behavioural weight control programme for obese patients with type 2 (non-insulin-dependent) diabetes. *Diabetologia.* 1988;31:902–9.
61. **Wing RR.** Behavioral treatment of obesity: its application to type 2 diabetes. *Diabetes Care.* 1993;16:193–9.