

Long-term Effects of Dieting: Is Weight Loss Related to Health?

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Abstract

“Success” in dieting interventions has traditionally been defined as weight loss. It is implicit in this definition that losing weight will lead to improved health, and yet, health outcomes are not routinely included in studies of diets. In this article, we evaluate whether weight loss improves health by reviewing health outcomes of long-term randomized controlled diet studies. We examine whether weight-loss diets lead to improved cholesterol, triglycerides, systolic and diastolic blood pressure, and fasting blood glucose and test whether the amount of weight lost is predictive of these health outcomes. Across all studies, there were minimal improvements in these health outcomes, and none of these correlated with weight change. A few positive effects emerged, however, for hypertension and diabetes medication use and diabetes and stroke incidence. We conclude by discussing factors that potentially confound the relationship between weight loss and health outcomes, such as increased exercise, healthier eating, and engagement with the health care system, and we provide suggestions for future research.

When physicians recommend that their patients go on diets, their implicit goal is unlikely to be to help these patients improve their appearance or body image. The assumption in recommending diets is that losing weight will lead to improved health, and yet, it is far less common for studies of the effectiveness of diets to directly measure health outcomes than to measure weight. There is ample evidence that diets do not lead to long-term weight loss in the majority of people (Mann et al., 2007), but what does this mean for health? Is losing weight closely tied to health benefits? In this paper, we attempt to answer this question by reviewing evidence on the long-term effects of weight-loss diets on health outcomes.

Traditional Definitions of Dieting Success

Historically, the criterion that diets – defined as a change in eating, most often a reduction in calories with a goal of weight loss – have been judged on has been weight loss. The necessary amount of weight loss, however, has been somewhat arbitrary and has changed dramatically since dieting first started being routinely studied. The original standard weight recommended by physicians was based on the Metropolitan Life Insurance Tables requiring particular weights for any given height and body frame size. For example, the tables designated 134 lb as the expected weight for an average-height woman (5’5”) of medium body frame. Whatever her starting weight, 134 lb would be her goal (Metropolitan Life Insurance Company, 1942).

Obese dieters, however, rarely achieved these standards (Stunkard & McLaren-Hume, 1959). Researchers turned to what they considered to be the more realistic goal of 20% weight loss, but only 5% of obese dieters succeeded by that definition (Stunkard & McLaren-Hume, 1959). Over the next 30 years, reviews of diet studies showed that individuals tended to lose an average of about 8% of their starting weight on most diets (Bennett, 1987; Wadden, 1993;

Wing & Jeffery, 1979). In an effort to create a more achievable goal, but without any particular medical reason, researchers lowered the standard to just 5% of one's starting weight (Institute of Medicine, 1995). By that standard, an average-height woman weighing 200 lb would need to lose just 10 lb to be considered a successful dieter, even though her BMI of 32 at that weight is still in the obese category (according to the World Health Organization, 2000, definition).

Given the arbitrary and non-medical nature of the currently accepted definition of dieting success as 5% weight loss, along with the fact that dieting does not appear to promote weight loss in the long term, we propose that the focus of dieting research – even weight-loss dieting – should be to promote *health*, rather than merely to reduce weight. The purpose of this review is therefore to assess the long-term health outcomes of weight-loss diets.

Method

To identify the studies to analyze, we first searched online databases (Google Scholar and PubMed) to locate reviews of diet studies such as comprehensive Cochrane Reports. We then searched their respective reference lists for candidate trials (Astrup & Rossner, 2000; Black, Gleser, & Kooyers, 1990; Foreyt, Goodrick, & Gotto, 1981; Jeffery et al., 2000; Leon, 1976; Mann et al., 2007; Norris et al., 2004; Norris, Zhang, Avenell, Gregg, Brown, et al., 2005; Norris, Zhang, Avenell, Gregg, Schmid, et al., 2005; Perri, 1998; Perri & Fuller, 1995; Saris, 2001; Siebenhofer et al., 2011). We also examined the reference lists of the studies themselves to locate additional potential studies. We then conducted forward reference searching to identify articles that had cited the studies included in our analyses. We searched only full articles and not abstracts. We also did not search for unpublished studies, and given publication bias, the results below might be considered an upper limit to the effects of dieting interventions on health outcomes. Diets included in our review had to meet the following three primary criteria.

Randomized controlled trial with a non-diet control group

The most rigorous form of study to determine the effectiveness of a treatment is a randomized controlled trial (RCT), and therefore, only RCTs were eligible for this review. In addition, to demonstrate that a weight-loss diet is truly beneficial, it must be shown to be more effective than no diet at all. For this reason, we only included studies that included a non-diet control group. This excluded randomized trials that only compared diet interventions to another type of diet (such as McManus, Antinoro, & Sacks, 2001).

Goal of weight loss

The word “diet” has numerous meanings, which include both “weight-loss diets” and “healthy diets” with no intent of weight loss. The objective of this review is to assess the long-term health outcomes of weight-loss diets, and we have therefore only included interventions where the goal was to lose weight or where participants were put on a typical low-calorie or low-fat diet. We did not include interventions that merely encouraged consumption of foods like olive oil or fish. Nor did we include interventions that relied exclusively upon exercise without alterations to diet.

Follow-up of at least 2 years

The most widely accepted standard for a “successful diet” is that set by the Institute of Medicine (1995), which states that an individual must maintain weight loss for a year. Although our focus is on long-term health outcomes rather than on weight loss, we use the same timeframe of assessing outcomes at least one year post-diet. The Institute of Medicine, however, counts the one year as beginning when the diet *begins*, rather than when the target weight is reached. This odd convention is likely used to make it easier to evaluate diets, as reaching a target weight happens at different time points for different individuals. Given that dieting interventions usually last anywhere from a few months to 1 year, we retain this convention of describing follow-up periods as starting from the *beginning* of the diet. Therefore, what is referred to in the literature as a two-year follow-up would conservatively capture health change sustained over at least 1 year. We therefore only included studies that are described as having at least a two-year follow-up period.

Eligible studies

Twenty-one diet papers met the inclusion criteria listed above (Diabetes Prevention Program Research Group, 2002; Hanefeld et al., 1991; Heshka et al., 2003; Howard et al., 2006, 2013; Hypertension Prevention Trial Research Group, 1990; Jarrett, Keen, & Murrells, 1987; Jeffery & French, 1999; Jeffery & Wing, 1995; Jones et al., 1999; Kuller, Simkin-Silverman, Wing, Meilahn, & Ives, 2001; Lindstrom et al., 2003; Mensink et al., 2003; Miettinen et al., 1985; Page, Harnden, Cook, & Turner, 1992; Pissarek, Panzram, Lundershausen, Adolph, & Senf, 1980; Sone et al., 2010; Stamler et al., 1987; Stevens et al., 2001; Trento et al., 2002; Uusitupa, Laitinen, Siitonen, Vanninen, & Pyörälä, 1993; Whelton et al., 1998). Details of these trials appear in Table 1.

Selection of health outcomes

The health outcomes reported in each article varied widely, from those that were often reported (e.g., blood pressure) to those reported by just one or two studies (e.g., retinopathy). We focus on outcomes that were reported in at least five studies. When outcomes overlapped, we included the outcome that was reported by the greater number of studies. For instance, HbA1c, an indicator of average blood glucose over 8–12 weeks, was reported in six studies. However, every study that measured HbA1c also reported fasting blood glucose (which was included in 12 studies), so we analyzed fasting blood glucose only. Finally, we excluded outcomes that were reported but not considered an endpoint from the standpoint of the study design; an example is mortality, which some papers reported but did not target as a study outcome.

We therefore examined five specific health outcomes: total cholesterol, triglycerides, systolic and diastolic blood pressure, and fasting blood glucose. We also discuss disease incidence and medication use as they relate to the five main health outcomes.

Analyses

Each of the five main health outcomes was treated as a continuous variable. As the outcome units in our analysis are directly interpretable (e.g., kg and mmHg), we calculated means rather than effect sizes such as Cohen's *d*. Means were calculated for both (i) the change over time among participants in the diet groups (mean difference from baseline to follow-up) and (ii) the difference between the diet and control groups' changes. Because of the wide

Table 1. Included studies and their characteristics.

Author	Diet	Control	Baseline N	Follow-up N ^a	Years follow-up	Group sessions	Exercise
Statistically significant weight-loss difference between intervention and control groups							
Diabetes Prevention Group (2002)	Low calorie, low fat	Standard lifestyle recommendations + placebo	2161	1969	2.8	Y	3
Howard et al. (2006)	Low fat, F&V	Information	48,835	44,351	3	Y	1
Kuller et al. (2001)	Low calorie, low fat, low cholesterol	No treatment	535	508	4.5	Y	2
Lindstrom et al. (2003)	Low fat, fiber	Information	522	434	3	Y	3
Mensink et al. (2003)	Low fat and cholesterol, fiber, protein, low alcohol	Information	114	88	2	Y	3
Miettinen et al. (1985)	Low calorie, low fat, V, low alcohol, good fats, lean meat	Screening and usual care	1222	1155	5	N	2
Pissarek et al. (1980)	Low calorie	Usual care	150	118	2	N	1
Stamler et al. (1987)	Low calorie, low sodium, low alcohol	No treatment	189	158	4	N	1
Stevens et al. (2001)	Low calorie, low fat, low sodium, low alcohol	Usual care	1191	1101	3	Y	2
Whelton et al. (1998)	Weight-loss diet but not described, low sodium	Usual care	585	540	2.5	Y	2
No statistically significant weight-loss difference between intervention and control groups							
Hanefeld et al. (1991)	Low calorie, low fat	Usual care	760	645	5	Y	3
Heshka et al. (2003)	Low calorie	Information, two 20-min dietitian consultations	423	307	2	Y	2
HPTRG (1990)	Low calorie	No treatment	506	463	3	Y	1
Jeffery and French (1999)	Low fat, F&V	No treatment	1226	809	3	Y	3
Jeffery and Wing (1995)	Low calorie	No treatment	202	153	2.5	Y	3
Jones et al. (1999)	Low calorie, low fat	Told patients to "lose weight", otherwise no treatment	112	102	2.5	Y	1

Page et al. (1992)	Low calorie, low fat, fiber, whole grains	No treatment	31	23	2	Y	3
Sone et al. (2010)	Comprehensive diet counseling, low alcohol	Usual care	2033	1319	8	Y	3
Trento et al. (2002)	Intensive education and nutritional goal setting	Individualized information and usual care	112	90	4.5	Y	2
Uusitupa et al. (1993)	Low calorie, low fat, fiber	Information	86	82	2	Y	2
Unknown statistical significance of weight-loss difference between intervention and control groups							
Jarrett et al. (1987)	Low carbohydrate	"Reduce table sugar" + placebo or 50 mg phenformin SA q.d.	204	117	10	Y	1

Notes: HPTRG = Hypertension Prevention Trial Research Group; F&V = Increased fruit and vegetable consumption; V = Increased vegetable consumption; 1 = Intervention did not include recommendations to exercise; 2 = Intervention included recommendations to exercise, but there were no formal exercise components; 3 = Intervention included formal exercise components (e.g., group exercise classes, exercise diaries, and pedometers).
^aReflects sample size for weight data.

variation in sample size ($n = 31$ to $n = 48,835$ participants), we weighted each mean by sample size. When possible, we used the sample sizes available for each specific health outcome to calculate weighted means, but in some studies, that information was not provided. In those cases, we used the sample sizes available for weight loss. To assess the relationship between weight loss and health outcomes, we calculated correlation coefficients between weight change in the diet groups and each health outcome.

Results

Weight

Across the 21 trials, the average amount of weight loss maintained among participants in diet conditions from baseline to follow-up was 0.94 kg. Participants in the diet conditions averaged a weight loss of 1.49 kg more than that of participants in the control groups. Results of the 21 included trials appear in Table 2.

Blood pressure

Hypertension, defined as systolic blood pressure of ≥ 140 mmHg and diastolic blood pressure of ≥ 90 mmHg or currently taking blood pressure medication (Chobanian et al., 2003), precedes first heart attacks 69% of the time, first stroke 77% of the time, and congestive heart failure 77% of the time (Roger et al., 2012). Thirteen of the 21 studies had information on diastolic and systolic blood pressure outcomes (Hanefeld et al., 1991; Heshka et al., 2003; Howard et al., 2006; Hypertension Prevention Trial Research Group, 1990; Jarrett et al., 1987; Miettinen et al., 1985; Page et al., 1992; Sone et al., 2010; Stamler et al., 1987; Stevens et al., 2001; Trento et al., 2002; Uusitupa et al., 1993; Whelton et al., 1998). Of these, 12 had information on blood pressure changes unconfounded by hypertension medication use (all but Stamler et al., 1987). One only included usable data to calculate the blood pressure change in the intervention group (Whelton et al., 1998) while another only had usable data to calculate the mean changes between groups (Stevens et al., 2001). Among intervention participants, the average difference between pre- and post-diet blood pressure, weighted by sample size, was a reduction of 2.37 mmHg for systolic and 2.71 mmHg for diastolic blood pressure (see Figure 1). Weight change (in the diet groups) and change in systolic blood pressure were not significantly correlated ($r = -0.08$, $k = 12$, $p = 0.79$) nor were weight change and change in diastolic blood pressure ($r = -0.07$, $k = 12$, $p = 0.83$).

At follow-up, the systolic blood pressure change for diet groups was 2.21 mmHg lower than that of control groups, and diastolic blood pressure was 0.50 mmHg lower than that of control groups, weighted by sample size. Given that the average (weighted) antihypertensive medication efficacy was 14.5 mmHg for systolic and 10.7 mmHg for diastolic blood pressure in a meta-analysis (Baguet, Legallier, Auquier, and Robitail, 2007), the blood pressure results attributed to these diets are small in comparison.

Six studies reported blood pressure medication use in relation to the diets, although the method of reporting this information varied. These measures included the percentage of participants using medication (Hanefeld et al., 1991; Hypertension Prevention Trial Research Group, 1990; Sone et al., 2010; Trento et al., 2002), the percentage remaining off medication (Stamler et al., 1987), and the percentage able to drop medication (Whelton et al., 1998). Of the four studies reporting simple percentages of medication use for all groups at baseline and follow-up, the most successful diet had 19.2% fewer participants on medication

Table 2. Weight loss and health outcomes.

Author	Weight loss (kg)		Total cholesterol (mmol/L)		Triglycerides (mmol/L)		Systolic blood pressure (mmHg)		Diastolic blood pressure (mmHg)		Fasting blood glucose (mmol/L)	
	Pre/Post	Int versus Cont	Pre/Post	Int versus Cont	Pre/post	Int versus Cont	Pre/Post	Int versus Cont	Pre/Post	Int versus Cont	Pre/Post	Int versus Cont
Statistically significant weight-loss difference between intervention and control groups												
Diabetes Prevention Group (2002) ^a	-4	-4.5									0.08	-0.35
Howard et al. (2006)	-0.7	-1.29	-0.26	-0.08	0	0	-2.2	-0.17	-2.6	-0.31	-0.09	-0.06
Kuller et al. (2001)	-0.1	-2.45			0.21	-0.13	-0.12	-0.32	1.5	-0.7	0.09	-0.09
Lindstrom et al. (2003)	-3.5	-2.6	-0.1	-0.2	-0.1	-0.1					0	-0.1
Mensink et al. (2003)	-2.7	-2.5	0.3	-0.1	-0.30	-0.55					0.2	-0.3
Miettinen et al. (1985) ^b	-1	-2	-0.4	-0.45	-0.3	-0.27	-10	-6	-8	-5		
Pissarek et al. (1980)	-9	-7										
Stamler et al. (1987) ^c	-1.8	-3.6	.02	-0.23	-0.03	-0.26		3.85		1.35		
Stevens et al. (2001)	-0.2	-2						-1.3	-2.25	-0.9		
Whelton et al. (1998) ^d	-4.7	-3.8					-4.65					
No statistically significant weight-loss difference between intervention and control groups												
Hanefeld et al. (1991) ^e	-1.1	-0.18	0.35	-0.12	0.23	-0.34	-5.6	-9.9	-3.0	-4.4	1.6	-0.12
Heshka et al. (2003)	-3	-2.9	-0.27	0.02	0	-0.2	-2.2	0.2	-0.6	-0.6	0.29	0.03
HPTRG (1990) ^f	-0.89	-3.6					-5.0	-2.4	-4.2	-1.8		
Jeffery and French (1999)	1.6	-0.25										
Jeffery and Wing (1995)	-1.7	-2.3										
Jones et al. (1999)	-1.2	1										
Page et al. (1992)	0	0	-0.4	0	-0.2	-0.3	-6	-1	-3	0	0.5	1
Sone et al. (2010)	-0.45	-0.45	-0.1	0	-0.06	-0.05	1	1	-3	0	-0.2	0.1
Trento et al. (2002)	-1.5	-0.5	-0.07	-0.2	-0.43	-0.26	-5.9	-4	-7.1	-0.8	-0.5	-1.3
Uusitupa et al. (1993)	-0.62	-2.71	0.3	0.1	-0.16	-0.15	6	1	1	0	0.5	0.2

(Continues)

Table 2. Continued

Author	Weight loss (kg)		Total cholesterol (mmol/L)		Triglycerides (mmol/L)		Systolic blood pressure (mmHg)		Diastolic blood pressure (mmHg)		Fasting blood glucose (mmol/L)	
	Pre/Post	Int versus Cont	Pre/Post	Int versus Cont	Pre/post	Int versus Cont	Pre/Post	Int versus Cont	Pre/Post	Int versus Cont	Pre/Post	Int versus Cont
Jarrett et al. (1987)	-8.74	-4.3					-2.96	-10.5	-3.56	-6.07		

Unknown statistical significance of weight-loss difference between intervention and control groups

Notes: HPTRG = Hypertension Prevention Trial Research Group; Pre/Post = Intervention group; Int versus Cont = Difference in outcome between Intervention and Control groups.

^aMetformin group excluded in the analysis.

^bLow-risk control group excluded in the analysis.

^cContinued hypertensive medication group (blood pressure analysis only) excluded in the analysis.

^dNon-obese subjects excluded in the analysis.

^eClofibric acid group excluded in the analysis.

^fNon-obese subjects excluded from all analyses. Averaged across Cal/NaCal versus control/Na with the exception of blood pressure, which reflects Cal versus control groups.

^gIntervention: averaged across Placebo+Diet and Phenformin+Diet groups. Control: averaged across Placebo+No diet and Phenformin+No diet groups.

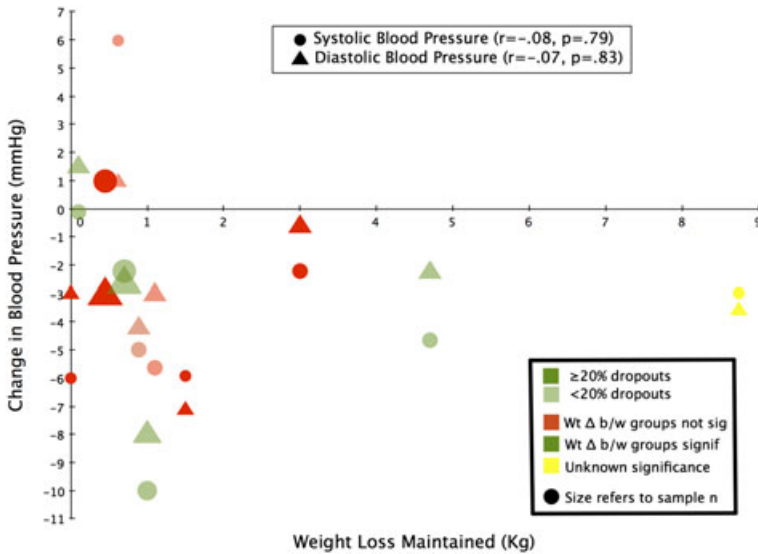


Figure 1 Change in systolic and diastolic blood pressure (mmHg) by amount of weight loss (kg) maintained. Symbol size refers to sample size, with, from smallest to largest symbol, $n \leq 100$, $100 < n \leq 500$, $500 < n \leq 1000$, and $n > 1000$. Percent dropouts from each study are depicted by the opacity of the symbol ($>20\%$ or $<20\%$). The statistical significance of the difference between diet and control groups in amount of weight lost is depicted by color, with green = statistically significant difference, red = no statistically significant difference, and yellow = unknown statistical significance.

than control participants (Hanefeld et al., 1991), and the least successful had 0.96% more diet participants on medication than control participants (Sone et al., 2010). Across all six studies, two showed statistically significantly better medication outcomes in the diet group than in the control group at follow-up (Hanefeld et al., 1991; Stamler et al., 1987), and four showed no difference (Hypertension Prevention Trial Research Group, 1990; Sone et al., 2010; Trento et al., 2002; Whelton et al., 1998).

Fasting blood glucose and diabetes incidence

The disease most closely linked to obesity is type 2 diabetes, which is the seventh leading cause of death in the United States (Centers for Disease Control and Prevention, 2000). Individuals with diabetes have elevated levels of fasting blood glucose and impaired glucose tolerance. Individuals with higher than average levels of fasting blood glucose or glucose intolerance (but not as high as those with diabetes) are considered to have pre-diabetes. Weight-loss diets have been tested as a way to treat diabetes as well as a way to prevent the development of diabetes among those with pre-diabetes.

Eleven of the 21 studies (Diabetes Prevention Program Research Group, 2002; Hanefeld et al., 1991; Heshka et al., 2003; Howard et al., 2006; Kuller et al., 2001; Lindstrom et al., 2003; Mensink et al., 2003; Page et al., 1992; Sone et al., 2010; Trento et al., 2002; Uusitupa et al., 1993) included measures of fasting blood glucose (see Figure 2). Participants in the diet conditions of these studies averaged a loss of 0.05 mmol/L (weighted by sample size), a change that did not significantly correlate with weight loss maintained ($r = -0.14$, $k = 11$, $p = 0.69$).

Although fasting blood glucose was not significantly related to weight loss maintained, the diets appeared to be beneficial for reducing diabetes diagnosis. Although only two studies

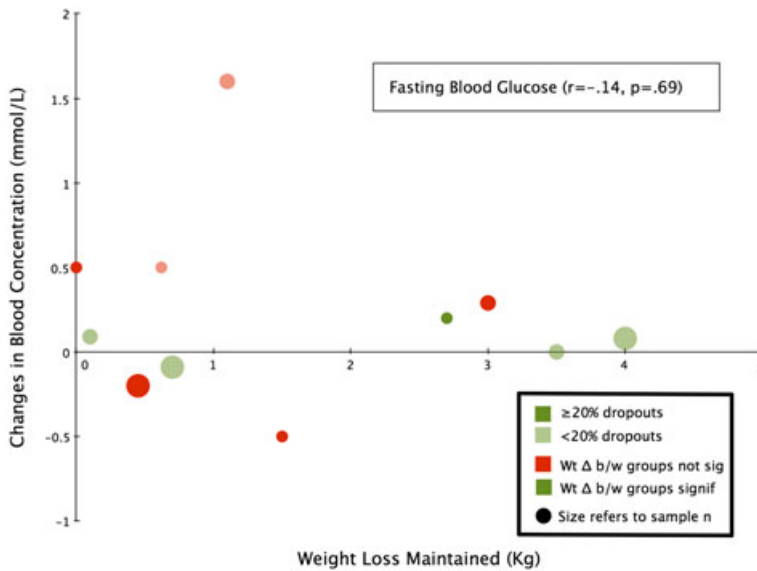


Figure 2 Change in blood glucose (mmol/L) by amount of weight loss (kg) maintained. Symbol size refers to sample size, with, from smallest to largest symbol, $n \leq 100$, $100 < n \leq 500$, $500 < n \leq 1000$, and $n > 1000$. Percent dropouts from each study are depicted by the opacity of the symbol ($>20\%$ or $<20\%$). The statistical significance of the difference between diet and control groups in amount of weight lost is depicted by color, with green = statistically significant difference and red = no statistically significant difference.

reported on incidence outcomes, the incidence of diabetes was significantly reduced among participants in both of them. In the Diabetes Prevention Program (DPP; Diabetes Prevention Program Research Group, 2002), incidence was reduced by 58% compared to a placebo control group and by 39% compared to participants given metformin, an antihyperglycemic medication. In the Finnish Diabetes Prevention Study (FDPS; Lindstrom et al., 2003), diabetes incidence was reduced by 58% compared to the control group.

The use of antidiabetic medication is another important outcome, and three studies reported on the percentage of participants who needed to start such medications. Two of those studies found that significantly fewer participants in the diet than in the control groups needed to start these medications (Uusitupa et al., 1993) or increase their dosage (Trento et al., 2002), whereas the third found no differences (Sone et al., 2010).

Lipid levels and cardiovascular events

Obesity has also been linked to elevated cholesterol and triglycerides (Malnick & Knobler, 2006), two risk factors for cardiovascular disease (Austin, Hokanson, & Edwards, 1998). In the studies that reported outcomes for cholesterol ($k = 11$) and triglycerides ($k = 12$; Hanefeld et al., 1991; Heshka et al., 2003; Howard et al., 2006; Kuller et al., 2001; Lindstrom et al., 2003; Mensink et al., 2003; Miettinen et al., 1985; Page et al., 1992; Sone et al., 2010; Stamler et al., 1987; Trento et al., 2002; Uusitupa et al., 1993), the diets led to minimal or no improvement in these lipid levels (see Figure 3). The diets led to small decreases in both cholesterol (mean change = -0.24 mmol/L, weighted by sample size) and triglyceride (mean change = -0.01 mmol/L, weighted by sample size) levels. These changes were not due to increased lipid levels among the controls, as is evident from comparing the mean changes between the two groups (cholesterol mean difference = -0.09 mmol/L; triglycerides mean

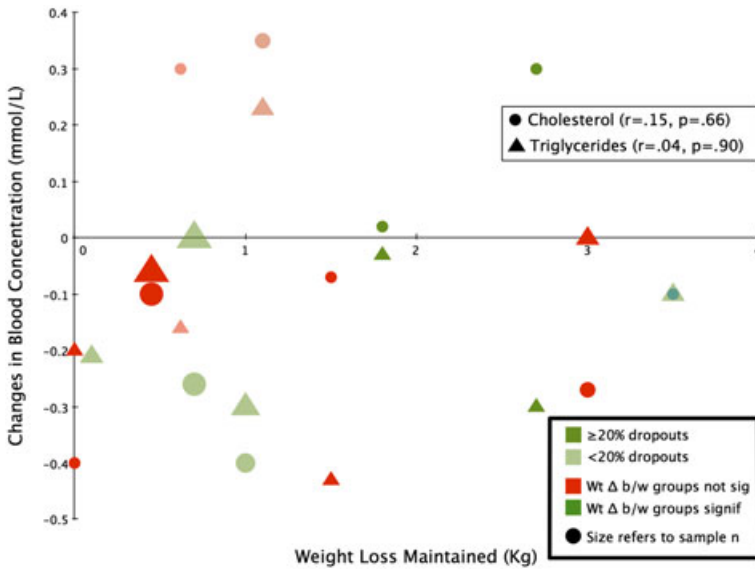


Figure 3 Change in cholesterol and triglycerides (mmol/L) by amount of weight loss (kg) maintained. Symbol size refers to sample size, with, from smallest to largest symbol, $n \leq 100$, $100 < n \leq 500$, $500 < n \leq 1000$, and $n > 1000$. Percent dropouts from each study are depicted by the opacity of the symbol ($>20\%$ or $<20\%$). The statistical significance of the difference between diet and control groups in amount of weight lost is depicted by color, with green = statistically significant difference and red = no statistically significant difference.

difference = -0.01). In addition, weight change in the diet conditions did not significantly correlate with changes in cholesterol ($r = 0.15$, $k = 11$, $p = 0.66$) or triglycerides ($r = 0.04$, $k = 12$, $p = 0.90$) in those conditions.

Of the five studies reporting the use of lipid-lowering medication at follow-up, three of them found no significant group differences in the use of lipid-lowering medication (Howard et al., 2006; Sone et al., 2010; Trento et al., 2002), and in two studies (Miettinen et al., 1985; Uusitupa et al., 1993), the diet participants were *more* likely to take lipid-lowering medication at follow-up.

The diets did not appear to meaningfully lower lipid levels, and accordingly, improvements in coronary morbidity/mortality and stroke were minimal. In all five studies that reported on these outcomes (Hanefeld et al., 1991; Howard et al., 2006; Miettinen et al., 1985; Sone et al., 2010; Whelton et al., 1998), the diets did not lead to significant reductions in coronary morbidity or mortality. Furthermore, in only two (Miettinen et al., 1985; Sone et al., 2010) of the five studies did the diet lead to significant reductions in stroke, and the researchers for one of these studies (Sone et al., 2010) noted that the significant finding should be treated with caution, as there were no group differences on most of the risk factors for stroke. A sixth study (Trento et al., 2002) reported on cardiovascular risk scores (rather than morbidity or mortality) and found no significant group differences.

Confounding Factors

Higher levels of exercise tend to lead to more weight loss (Anderson, Konz, Frederich, & Wood, 2001), but exercise also leads to health benefits in the absence of weight change (King, Hopkins, Caudwell, Stubbs, & Blundell, 2007). As a result, participants who lose weight *and* increase exercise may experience health benefits, and both researchers and diet participants may attribute the improved health to the weight loss rather than to the exercise.

This is evident in the two studies that reported and demonstrated favorable diabetes incidence outcomes, in which it is not possible to separate the effects of diet from those of exercise. In the DPP (Diabetes Prevention Program Research Group, 2002), the intervention included a low-fat, low-calorie diet, plus a goal of 150 min of moderate exercise per week. In the FDPS (Lindstrom et al., 2003), participants in the intervention were offered supervised, individually tailored, circuit training sessions. Both interventions are notable for their extensive exercise components, and exercise adherence was exceptionally high in both studies. Seventy-four percent of the participants in the DPP intervention reported engaging in at least 150 min of exercise per week during the first 6 months of the study, and 58% reported doing so at the 2.8-year follow-up. In the FDPS, 86% of participants achieved the goal of exercising at least 4 h per week during the first year of the study.

Diet interventions usually encourage the consumption of fruit, vegetables, and fiber, and these foods help regulate blood glucose and may play a role in preventing cancer, stroke, and heart disease (Mozaffarian et al., 2003; Steinmetz & Potter, 1996). Some interventions also discourage sodium and cholesterol intake as these nutrients have been linked with cardiovascular outcomes (August, 2003; Downs, 1998; Grollman, 1945). Thus, diet participants who adhere to the recommended meal plan may benefit, regardless of whether they lose weight.

Diet interventions often include health checkups as part of measuring study outcomes. These meetings may be one-on-one sessions with a nutritionist or physician (Lindstrom et al., 2003), group sessions with other participants (Diabetes Prevention Program Research Group, 2002; Hanefeld et al., 1991; Heshka et al., 2003; Howard et al., 2006; Hypertension Prevention Trial Research Group, 1990; Jeffery & French, 1999; Jeffery & Wing, 1995; Jones et al., 1999; Lindstrom et al., 2003; Stevens et al., 2001; Trento et al., 2002), and/or phone meetings with a researcher (Howard et al., 2006; Hypertension Prevention Trial Research Group, 1990; Lindstrom et al., 2003). The majority of the RCTs we examined used a wait-list control group, and diet participants received many more checkups than control participants. In these sessions, participants received information about dieting, but they were also often checked for hypertension (Lindstrom et al., 2003), high cholesterol (Lindstrom et al., 2003), and abnormal blood glucose levels (Hanefeld et al., 1991; Uusitupa et al., 1993). This likely resulted in diet participants receiving more prompt care for illness than control participants. Since many participants were obese and since obese individuals are less likely to receive preventative care (Jones, 2010) and more likely to cancel or delay medical appointments (Alegria Drury & Louis, 2002), this prompt care could lead to better health through appropriate medication and treatment irrespective of participants' weight or diet.

Finally, social support has been associated with beneficial cardiovascular and immune system health, independent of health behaviors (Uchino, Cacioppo, & Kiecolt-Glaser, 1996), and all but three studies administered their interventions in group settings. Increased social support from engagement in group sessions and meetings with study staff could also have participants' health outcomes.

Summary

Overall, there were only slight improvements in most health outcomes studied. Changes in diastolic and systolic blood pressure, fasting blood glucose, cholesterol, and triglyceride levels were small, and none of these correlated with weight change. There were also very small effects of these diets on lipid-lowering medication use and coronary morbidity and mortality. There were a few larger positive effects for hypertension and diabetes medication use, as well

as diabetes and stroke incidence. In correlational analyses, however, we uncovered no clear relationship between weight loss and health outcomes related to hypertension, diabetes, or cholesterol, calling into question whether weight change *per se* had any causal role in the few effects of the diets. Increased exercise, healthier eating, engagement with the health care system, and social support may have played a role instead.

Our findings are in line with a recent meta-analysis (Flegal, Kit, Orpana, & Graubard, 2013) that found that overweight and class I obesity were not associated with higher all-cause mortality. Moreover, Ortega and colleagues (2013) have documented metabolically healthy but obese individuals, and an emerging literature on the “obesity paradox”, whereby obesity appears to confer health benefits in certain diseases (Amundson, Djurkovic, & Matwiyoff, 2010), suggests that a disconnect between weight loss and health outcomes should not be surprising.

Suggestions for future work

In this paper, our goal was to answer the question of whether losing weight leads to health benefits in the long term. We note, however, that the number of studies that had long-term follow-up measurements was surprisingly low given the large number of published dieting interventions. Accordingly, our first suggestion for future work is to ensure that diet studies have follow-up measurement points at least one year after the diet has *concluded*. Small, short-term improvements in health that revert back to baseline levels immediately after the study has concluded do not represent treatments worthy of the time, money, and effort involved in undergoing them. The dearth of long-term follow-up measurement may represent a structural problem with the five-year NIH funding cycle, in which long-term follow-ups are not feasible for most in the first funding period given how long it takes to initiate intervention studies. Just as the R03 and R21 mechanisms exist for pilot work, perhaps so too should a dedicated mechanism be developed for follow-up measurements for concluded intervention studies.

A second clear suggestion for future work is to measure health outcomes along with weight. Weight, as we reviewed here, turns out to be an inadequate proxy for health outcomes. Given that weight loss appears to be elusive for the majority of dieters (Mann et al., 2007), measuring health outcomes is the only way to detect improvements in individuals who would otherwise be deemed “failures” for not losing weight. Indeed, it may be the case that weight loss is simply unnecessary for health improvements. Proponents of the Health at Every Size paradigm (HAES; Bacon, 2010) argue exactly that. Studies testing HAES interventions that emphasize healthy behaviors, size acceptance, and *non*-dieting have shown health benefits independent of weight loss (e.g., improved blood lipid levels; Bacon et al., 2002; Rapoport, Clark, & Wardle, 2000). As this literature is relatively new, long-term studies of HAES interventions are not yet available.

We believe the ultimate goal of diets is to improve people’s long-term health, rather than to reduce their weight. Our review of randomized controlled trials of the effects of dieting on health finds very little evidence of success in achieving this goal. If diets do not lead to long-term weight loss or long-term health benefits, it is difficult to justify encouraging individuals to endure them.

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Short Biographies

A. Janet Tomiyama's research lies at the intersection of eating behavior, psychological stress, and cellular aging, and her work spans macro factors like socioeconomic status to micro factors like telomere shortening. Obtaining her PhD in Social and Health Psychology from UCLA in 2009, Dr. Tomiyama was first a Robert Wood Johnson Foundation Health and Society Scholar at UCSF and UC Berkeley and then an Assistant Professor of Psychology and Nutritional Sciences at Rutgers University in New Brunswick. She is now an Assistant Professor of Psychology at the University of California, Los Angeles. Her research is designed to test questions such as the following. Is "comfort food" really comforting? Is dieting stressful? Does stress age our immune system and, if so, how? Can calorie restriction reverse this type of aging? Who are our society's most successful dieters, and what can we learn from them? Is stress to blame for racial disparities in obesity? What are the health consequences of weight stigma?

Britt Ahlstrom's research centers on evolutionary theories of food preferences. She previously worked as a laboratory manager of the Health and Eating Laboratory at the University of Minnesota, where she was involved in studies funded by the NIH and NASA. She is currently pursuing a PhD in Health Psychology at the University of California, Los Angeles. Her most recent work focuses on how evolutionary-developmental theories can predict individual variation in eating, health behaviors, and aging.

Traci Mann's research explores health behavior change, broadly, with a focus on the self-control of eating. She obtained her PhD in Psychology from Stanford University in 1995 and was a postdoctoral fellow in the National Institute of Mental Health HIV Prevention Training Program at UCLA. In 1998, she became an Assistant Professor in the Department of Psychology at UCLA and received tenure in 2004. She joined the Department of Psychology at the University of Minnesota in 2007 and was promoted to Full Professor in 2012. Her work has been published in a variety of psychology and medical journals, including the *Journal of the American Medical Association*, *American Psychologist*, and *Psychological Science*, and she has been funded by the NIH, the USDA, and NASA. Her current research examines basic issues in self-control processes, as well as applied issues involving promoting vegetable consumption in children and sufficient calorie consumption in astronauts.

Note

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