Is long-term weight loss possible?

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Any intervention which causes negative energy balance is guaranteed to be efficacious in producing weight loss, which will continue while there is negative energy balance or be maintained as long as the new energy balance is maintained. In clinical practice compliance is rarely 100% so the efficiency of even the most efficacious treatment is usually low. However, recent evidence-based guidelines have recognized the clinical benefits of moderate (5–10%) weight loss, which is achievable using a variety of interventions. Long-term studies of ‘weight loss’ are, in reality, combinations of weight loss (usually completed in 1–6 months) followed by variable weight maintenance, set in the context of progressive adult weight gain in an obesogenic environment. Few studies have adopted specific and separate strategies for weight loss and weight maintenance. Meta-analyses conducted by non-expert methodologists have failed to recognize these distinctions, and have criticized the available research without understanding the different needs of studies with weight change as the outcome variable, which require randomized controlled trials (RCT), and those with weight loss as the treatment, intended to improve metabolic or biomedical outcome measures. An RCT design is inapplicable to studies of biomedical end points (e.g. cardiac risk factors) when weight loss is the treatment. Because fixed weight loss cannot be prescribed there is always a range of weight changes in any study, and single-sample studies with regression analysis provide the best design. An RCT study design does not give useful information about clinical value as the control group is always ‘treated’ to some extent. Placebo- (or control)-subtracted differences are misleading because in an RCT all subjects recruited to active treatment, including non-responders, are continued on treatment for the full duration of the study. In routine clinical practice, treatments are changed in the light of early experience as a therapeutic trial to optimize the results for each individual, and audit is required to evaluate ‘long term weight loss’.

Background

The escalating burden of ill-health and costs of obesity on health care (Table 1), recognized in several evidence-based guidelines, has urged a shift in thinking towards a priority for health promotion and health education to develop effective obesity prevention programmes (Thomas, 1995; SIGN, 1996; NIH, 1998). Simultaneously, the same reviews have pointed to major medical benefits from quite modest weight loss of the order 5–10% (Goldstein, 1992) and reduced mortality from intentional and quite modest weight loss, at least from non-insulin-dependent diabetes mellitus (NIDDM) (Lean et al. 1990; Williamson et al. 1995, 1999).

There is confusion over long-term effects of intentional weight loss on health, mainly because of a lack of conventional experimental evidence, partly because of the mismatch between the efficacy of weight loss in improving risks and the efficiency of achieving reliable weight loss in clinical practice. It is obvious that some patients, but not all, can achieve valuable weight loss and maintain that loss for many years (Hakala, 1994; Manning et al. 1998). In fact it is unnecessary to conduct clinical trials to prove the efficacy of an energy-deficit diet, for example 500 kcal/day below energy expenditure will result in weight loss, or that weight loss will be maintained on a subsequent energy-balanced diet. The practical problem which requires evidence is the poor efficiency of management outside the strict confines of a metabolic one.

Uncertainty also arises from confusion as to what obesity is – whether it is a disease or whether it is a risk factor for other diseases, of which the metabolic syndrome is the most obvious. This confusion is manifest in the design of clinical trials, the interpretation of research data, and the terminology used in evidence-based guidelines. A long-term randomized controlled trial (RCT) has recently been proposed to evaluate the effect of weight loss on morbidity and mortality.
weight-loss studies and included 493 in a meta-analyses of diet, exercise or the two combined. Mean programme lengths varied from 15-1 (SEM=0.8, n=224) for diet alone, to 20-9 (SEM=1.8, n=76) for exercise alone, and 13-4 (SEM 0.7, n=119) for exercise alone. Weight loss maintained at 1 year was 6.6 kg (SEM=0.5, n=91) for diet, 8.6 kg (SEM=0.8, n=54) for diet and exercise, 6.1 kg (SEM=2.1, n=7) for exercise alone. Given that not all patients respond well to particular treatment, and 50% or so do better than these mean values, these figures give some reason for optimism.

Little evidence exists from long-term clinical audits, but data from Aberdeen (Lean et al. 1990) showed a weight loss of 6.8 kg at 1 year in 71 unselected obese (BMI > 30 kg/m²) NIDDM patients, given routine dietetic advice. This weight loss was associated with increased survival, so is unlikely to have been the result of worsening disease. This type of evidence clearly indicates that useful long-term weight loss can be achieved in routine practice, and is similar to that achieved in clinical trials (Usitupa et al. 1993; Hakala, 1994; Miller et al. 1997).

This review outlines the range of experimental designs, statistical safeguards and approaches applicable to studies of the biological effects of weight loss. It offers reasons why RCT evidence should not be sought for biomedical benefits.

### Errors and biases in obesity research

A particular pitfall for long-term obesity research is the bias that results from the effects of the passage of time. Ageing and seasonal variations are known to influence a number of metabolic measurements, so systematic changes can be expected over a long observation period. Examples include age-related insulin resistance, increased lipids and BP, and decline in respiratory capacity. These measures will tend to deteriorate over time, and may conceal the effects of weight reduction. The apparently simple solution – to provide a placebo control group – is highly problematic.

In studies of intentional weight loss, subjects are recruited on the basis of high weight or BMI (usually the highest part of the distribution within a population) and weight may tend to fall without treatment, for purely statistical reasons, because high individual measurements will contain an excess of those elevated by peaks of random error or biological variation. Additionally there are likely to be non-random, biological and societal influences on body weight or BMI over time which could lead to some tendency for weight change to systematically. Specifically, volunteers all want to lose weight – i.e. they are at least ‘contemplators’ or intending to make changes (Prochaska & Di Clemente, 1986). On the other hand, weight tends to increase gradually over time for most adults, and body fat accumulates until the 7th decade. Thus weights may tend to drift up in most studies over about 12 months, whatever the treatment, and weight-related metabolic variables likewise (Heitmann & Garby, 1999).

For studies of weight loss, weight measurement is so accurate that a random error is small, and a systematic tendency for the overweight to change weight is more likely to be biological than measurement error. For some studies, subjects are required to be overweight and also to have

<table>
<thead>
<tr>
<th>Problems</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical</td>
<td>Tiredness, Breathlessness, Varicose veins, Back pain, Arthritis, Sweating/intertrigo, Stress incontinence, Oedema-cellulitis</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Hypertension, NIDDM, Hepatic steatosis, Hyperlipidaemia, Hypercoagulation, IHD and stroke</td>
</tr>
<tr>
<td>Social</td>
<td>Isolation, Agoraphobia, Unemployment, Family/marital stress, Discrimination</td>
</tr>
<tr>
<td>Anaesthetic/surgical</td>
<td>Sleep apnoea, Chest infections, Wound dehiscence, Hernia, Venous thrombosis</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Hirsutism, Oligomenorrhea/infertility, Metromenorrhea, Oestrogen dependent cancers (breast, uterus, prostate)</td>
</tr>
<tr>
<td>Psychological</td>
<td>Low self-esteem, Self-deception, Cognitive disturbance, Distorted body image, Depression</td>
</tr>
</tbody>
</table>
Possible sources of error | Possible sources of bias
--- | ---
Few entry criteria for participants | Over-restricting entry criteria
Poor weighing scales, not regularly calibrated | Completing weight measures at different time of day, or day of the week
Poor training of investigators making measurements | Seasonal variation in measurement of body weight and biochemical measures
Failing to complete laboratory analyses in duplicate | Regression to the mean of weight-related measurements in groups selected as overweight
Pre-menstrual fluid accumulation (gender) | Recruiting participants who have already participated in weight-loss studies
Changes in clothing between measures | Recruiting participants who have already recently achieved intentional weight loss
Combining sexes | Subdividing subjects retrospectively on a weight-loss basis to examine metabolic variable
Statistical power may be insufficient to confirm negative conclusions | Too short study duration, effects of acute negative energy balance and of weight loss superimposed
Failure to represent confidence intervals may fail to illustrate the range of the data | Passage of time in long-term studies
Poor matching for age, or unusual age distribution

Table 2. Possible sources of error and bias in studies of weight loss

Elevated coronary heart disease (CHD) risk factor, so there is a risk of regression to the mean on both criteria. Table 2 lists some potential sources of random error and of bias that may affect weight change and its consequences in studies of weight loss.

Specific biases may be introduced to weight loss studies by recruiting subjects who have participated in many other attempts to reduce weight, who have recently lost weight, or who have already intentionally begun to reduce their body weight before participating. Bias arises from participants having already reached a plateau from which further weight loss may be problematic. This is probably the explanation for the frequent ‘poor’ weight loss in NIDDM patients, who have actually already lost what they can (Blonk et al. 1994; Brown et al. 1996; Manning et al. 1998). Recent weight loss can be difficult to maintain during a second intervention, and most weight is lost in the first few weeks, so a diet-only ‘run in’ period minimizes the weight loss from subsequent introduction of a drug or other intervention.

The relationships between metabolic measures, body weight and acute energy balance are complicated. As a broad generalization, measures such as blood pressure, plasma glucose, plasma cholesterol, high density lipoprotein (HDL) or triglycerides all fall rapidly with acutely negative energy balance, but settle slowly to a more modest improvement (including a rise in HDL above baseline) when body weight is reduced and stable. Thus even brief changes in weight or diet prior to baseline or subsequent measurements in a longitudinal study can have seriously confounding effects. A ‘run-in’ period of dietary advice, often recommended for weight-loss studies, intentionally entails some weight loss immediately before introducing the specific intervention of interest. Such a design may sometimes clarify the efficacy of a specific intervention in promoting weight loss, but will confound studies concerned with secondary metabolic consequences of weight change. A paradoxical aggravation of metabolic measures is possible with weight loss if subjects enter a trial with reduced values after a run-in period with negative energy balance (Sjostrom et al. 1998).

Controlled trials of treatment to achieve weight loss

Control subjects are included in clinical trials to quantify and correct for the influence of confounding factors that introduce bias to the results of a specific treatment. Results in the treatment group are thus compared to those in a control group, and a difference between groups is evidence for a treatment effect. Subjects who volunteer for studies of weight loss want to lose weight so are ready to attempt to make lifestyle changes, and their recruitment with overweight as a selection criterion usually results in weight loss in addition to or even without specific treatment or advice. This ‘study effect’ relates to changes in lifestyle, diet and behavioural patterns, which tend to occur simply as a result of overweight patients participating in a research study (Blonk et al. 1994; Svedsen et al. 1994). Therefore all studies to assess the effectiveness of a treatment in achieving weight loss must include a control or comparison group, irrespective of whether they are examining drugs, diet, lifestyle or other treatments. For drug trials, an identical placebo should be given, and this is usually in the context of the same diet and lifestyle advice to both groups. However, it is impossible to have indistinguishable ‘placebo’ lifestyle change for obese subjects recruited to a diet or exercise intervention study, or to have blinded-subject allocation, such as would be mandatory for a drug trial. In practice ‘control groups’ for diet and lifestyle studies may be offered no advice, or usual or routine treatment, but this may confuse the analysis because such subjects recruited as volunteers for weight loss inevitably do make extra changes, thus lose weight to a variable degree and to some extent are effectively ‘treated’. The distributions will be different, but weight changes with diet and lifestyle intervention and non-intervention groups always overlap, and both usually include some subjects who actually gain weight.

There are two main experimental designs to provide control. A crossover design, using subjects as their own controls, is probably the most efficient approach with respect to sample size and power study. Ideally, subjects are randomly allocated to the order of the treatment periods and some form of Latin square design may be employed. A
washout interval may be required between each of the periods to remove any carry-over treatment effects. This approach is impossibly exhausting for comparisons of complicated diet and lifestyle modifications. The simpler alternative is to use parallel group controls. An experimental design which includes parallel controls removes any need for washout periods. Using a parallel control group is a simple design for analysis but variance is increased, reducing study power (or requiring larger numbers). With both designs, overweight subjects are often unwilling to comply with non-intervention arms. Thus this, and the duration of treatment necessary for weight-loss trials, usually a minimum of 12 months, frequently precludes crossover designs. Furthermore, many studies employing diet intervention have shown very limited weight loss when subjects change to the second arm of treatment, irrespective of its type (Lean et al. 1997) even though purely pharmacological actions may still be demonstrable in drug trials (Sjöström even though purely pharmacological actions may still be demonstrable in drug trials (Sjöström et al. 1998). This is probably simply because their willpower and motivation are exhausted in about 3–4 months.

To make a comparison between different styles of management, e.g. intensive versus usual dietary advice, or different behavioural or dietary approaches, control-subtracted results are necessary to prove a real effect, but they have no bearing on results in routine practice. The differences between any measurements made in treatment and control groups are often mistakenly considered to be the effects of the treatment. This is reasonable when the control condition or placebo is inactive and where study effects are close to no treatment. However, the placebo-subtracted results of drug trials for weight loss, essential to prove pharmacological efficacy for regulatory purposes, give a misleading view of treatment outcomes in clinical practice. Firstly, subjects recruited into a double-blind placebo-controlled drug trial with intervention to treat from the full duration are all followed up until the termination of the trial (e.g. 12 or 24 months) whether or not they obtain benefit. In routine clinical practice every prescription is considered a therapeutic trial and the patients who do not respond to a specific drug would have that treatment withdrawn after a few weeks and another started. Second, the aim of drug treatment for obesity in routine practice is always to help patients adhere to better to dietary changes (e.g. by suppressing appetite), and so it is the combined treatment effect of diet and drugs which matters rather than placebo-subtracted differences. If these considerations are applied to the recent 2-year placebo-controlled trial of orlistat (Sjöström et al. 1998), or the trials of sibutramine (Lean, 1997; Bray et al. 1999), it can be seen that the period of weight loss is complete by 3–6 months for most subjects. After that, weight is maintained at a lower level than it would have been without treatment. A flat mean BMI over time means that equal numbers are gaining or losing weight. The placebo group is included to be able to prove that the drug is having an effect (maintained to 2 years shown by difference from treatment curve), but the clinical effect in routine practice will relate to the weight change in relation to the expected result of non-management. The weight loss during the run-in period will have had the effect of reducing weight loss from the drug, but contributes together with diet, etc. to the maintained weight (Fig. 1).

The plot in Fig. 4 shows that ‘success’ in a weight-loss study is relative to a rising baseline in untreated patients (Manning et al. 1998; Heitmann & Garby, 1999), whose trajectory can be considered failure; active weight loss is usually complete in 3–4 months; and the concept of ‘long-term weight loss’ is rather confused. Indefinitely continued weight loss would be disastrous. Completely maintained weight loss (a flat curve) might be ideal, but some degree of subsequent weight regain may still be compatible with major medical benefits compared to the untreated state.

As a general principle, research has concentrated too much on ’weight loss’, and too little on weight maintenance. Patients mostly know they can lose weight, and find the greatest problem is avoiding regain. From first principles, the treatments to achieve weight loss and to prevent (re)-gain need not be the same, but most clinical trials are the same treatment for both, and then become confused as to what is meant by ‘weight loss’ or ‘long-term weight loss’. This is even more confused for trials which have incorporated a diet alone or diet plus placebo run-in period. Attention tends to be focused on the weight loss (after the run-in period). What is important for health is the weight maintenance (in relation to baseline weight before any run-in weight loss). As described earlier, even this is incomplete for a long-term study – e.g. 12–24 months – because without any intervention, weights would have risen at a mean rate of 1–2 kg/year for people with weight problems.

The effectiveness of a treatment (for weight loss or maintenance) can be assessed as (i) comparison of mean weight changes, or (ii) the division of subjects into ‘successes’ and ‘failures’ in terms of achieving pre-set weight loss or BMI. Success criteria achieving e.g. BMI $<30\text{kg/m}^2$ or weight loss $>15\%$ must be defined in relation to time, and at the design stage to avoid introducing bias from post hoc categorization, particularly if secondary metabolic consequences of obesity and weight change are also to be evaluated. As discussed above, it is most sensible to relate weight changes to baseline weight and to randomize subjects directly to treatment and control groups.

Controls and analyses of metabolic studies where weight loss is the treatment

Metabolic studies are frequently conducted to try to improve weight-related factors such as blood pressure, lipids and
glucose tolerance. Such trials could go on to examine morbidity and mortality from the metabolic syndrome. Such trials have not yet been conducted, and at least part of the reason is uncertainty over the design and practicality of such a study (Yanovski et al. 1999). The necessary elements are overweight subjects willing to lose weight and maintain long-term weight reduction, specific interventions (e.g. diet, drugs) to reduce and maintain weight loss, metabolic measures expected to improve with weight loss, and ‘hard end points’ such as myocardial infarction or death.

Theoretically a study could go directly from the intervention (e.g. diet modification) to the ‘hard end points’ without considering the intermediate steps. It can be argued that weight loss may be sufficient as a treatment, but not necessary on a priori grounds, so a double-blind controlled trial of the treatment should be conducted. However this is a reductio ad absurdum of the intention-to-treat principle, neglecting the facts that the subjects must be obese, must want to lose weight to volunteer, and will inevitably lose weight if they follow the treatment. Given these practical considerations, it is clear that the effective treatment is in fact weight loss, not the dietary intervention that caused it.

Difficulties with RCT design arise because weight loss is a rather unusual treatment. Interventions designed to produce weight loss do not, and cannot, introduce a fixed effect on body weight, but lead to a range of individual responses. Weight loss as a ‘treatment’ thus cannot be administered as a pre-set ‘dose’, and instead the ‘dose’ of treatment varies between individuals such that a range of weight losses (and often gains) occur in all groups under study. This situation is very different from a drug trial where a fixed dose of medication allocated to treatment subjects would have a more predictable effect, and placebo has no pharmacological effect.

In an RCT to test the effect of a particular ‘treatment’ against a dummy or ‘placebo’ treatment, the only difference between the two groups should be the treatment, with all other elements remaining the same. In the case of trials of medication efficacy, subjects whose compliance is poor are usually excluded for violation of protocol. In dietary studies there will always be a range in the abilities of subjects to comply with the advice, so some subjects in each study group, treatment and control, may show rises and falls in outcome measures. It has been argued (Yanovski et al. 1999) that a ‘low-intensity intervention’ control group could be included within a study that examines the health effects of moderate weight loss. However, it was proposed that the ‘low-intensity group’ would be still provided with weight loss advice in the form of manuals. Strictly, two interventions (intensive and moderate) are suggested, with comparison in terms of metabolic outcome measures.

The logic behind this proposal is confused. As discussed above, a two-group comparison is logically reasonable if the intermediary of weight loss can be ignored: i.e. if it is sufficient, but not actually necessary, for the treatments to cause weight loss. However the only way to recruit subjects is to select the overweight and to offer weight loss, thus within both intensive and low-intensity interventions there will always be a range of weight changes. The range is usually wide – including some subjects who actually gain weight. It is certainly possible to compare results for intensive and low-intensity groups, and mean or median changes are likely to be different. Such a study could prove only that more intensive intervention has greater effects than a lower intensity one. This is probably self-evident, and irrelevant to a study of biomedical consequences of weight loss.

In short, just as interventions to produce weight loss usually involve more than one modality, different elements of weight management (diet composition, exercise, drugs) may have independent effects on weight-related metabolic variables.

The best way forward for metabolic studies of weight loss

To evaluate the effect of weight loss on secondary metabolic measures or on long-term ‘hard end points’, it would seem most appropriate to recognize, and to make the best use of, the wide variation in ‘treatment doses’ between individual subjects which occur with any intervention. This can be achieved by relating the changes in metabolic and biomedical outcome measures to the amount of weight changes in that individual, using regression analysis in a single sample (Fig. 2). Using this approach, there is no logical need for a control group. The only possible value of a ‘low-intensity’ intervention group would be to increase the range of weight changes which could be assessed. More probably, it would lead to a large clump of subjects with small weight changes. It would be statistically preferable to have graded intervention to ensure an even distribution of weight changes in a single sample.

The single sample approach is the most economical and more ecologically valid, incorporating the range of weight losses (commonly from +2.0 to –10.0 kg) in dietary intervention (Fig. 3) and their consequences across a chosen population outside the artificial constraints of a controlled trial. Another advantage to regression analysis is that it allows adjustment for interacting factors. An example of results from a weight-loss study analysed in two different ways is shown in Fig. 3 from a study by Hankey & Lean (1996). Angina patients with BMI > 30 kg/m² were limited and lost a mean 3.6 kg in 12 weeks with a dietitian-led dietary intervention. There was a significant reduction in angina frequency (Fig. 3a) but with no ‘control group’, scepticism has been expressed over the true relation with weight loss. The same data, plotted as individual weight change against change in angina frequency, make this clear (Fig. 3b). The relationship is weak but real, and weight losses of value for some, but not all patients. A control group would have added nothing.

Attention has recently been given to the possible need for a long-term study of weight loss on morbidity and mortality (Yanovski et al. 1999). The short-term effects of weight loss (< 6 months) are very well established as beneficial, even though the literature is confusing due to the frequent inclusion of both subjects who are still losing weight (i.e. suppressing metabolic variables by acute negative energy balance), and subjects whose weights have stabilized. 
Although they are late and relatively distant complications overshadowed by the host of more immediate symptoms of obesity, 'hard end points' such as myocardial infarction, stroke and cancer are still legitimate outcomes for study. Study power would be increased by choosing high-risk groups (e.g. NIDDM, angina, family history of CHD or breast cancer), but it could still be argued that the results might not be directly applicable to the general population. Conversely, a large study of all overnight subjects might not include enough from these high-risk groups. The single-sample design would, however, allow greater power than the design proposed by Yanovski et al. (1999) with its largely useless control group. Whatever study design is adopted, a long-term weight-management study needs to employ the best-bet methods for initiating weight loss and then for maintenance (reducing weight regain), such as those identified by SIGN (1996) and NIH (1998). The trial will need to recognize that weight losses at age 20 or age 50 are likely to have different effects, and that in observing the natural history of body weights in adults gradual long-term weight gain is to be expected for many subjects, even with successful weight management which causes the weight to be lower than it would have been without intervention (Fig. 2).

A final design point is probably an imponderable one. It is impossible to distinguish with certainty the metabolic or biomedical consequences of weight loss from specific effects of the specific intervention which produced the weight loss. This was recognized by Yanovski et al. (1999), and offering a menu of intervention components seems the most appropriate pragmatic strategy. No single diet or lifestyle is optimal or appropriate for every patient, and some form of triangulation approach would be desirable to show that similar benefits result from similar weight loss achieved by different interventions. To mount single variable long-term RCT clinical trials to test this efficacy of specific single elements for a weight loss is impractical, given the plethora of possible management variables available. A better approach must be to adopt the 'best bet' multi-component management as outlined in evidence-based guidelines (SIGN, 1996; NIH, 1998), and ensure ongoing documentation of methods with regular audit of results and feedback of improvements into the methods to form closed-loop audit cycles. This approach, unlike RCT, can be used to audit biomedical outcome measures as well as weight change, and allows individual patients to follow individualized management courses. The expectations and goals of patients need to be managed to make them reasonable and achievable.

Conclusions

Studies of weight loss are theoretically complex and practically problematic. The literature, including systematic reviews and meta-analyses of weight loss research, has been confused over whether weight loss is the outcome or is the treatment. The issues are summarized in Fig. 5. Treatments designed to produce weight loss as the outcome measure need to be evaluated against controls to prove efficacy: this presents no problem for placebo drug treatments, and control diet composition can be designed to evaluate specific test diets for effects on body weight. 

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Fig. 2. Weight loss as outcome and as treatment. RCT, randomized controlled trial.

1. Treatment \[\rightarrow\] Weight loss

**RCT trial needed to prove treatment effect**

**BUT** Placebo subtracted difference does not equate to treatment effect in routine practice because

(i) some patients in RCT trial do not respond
(ii) treatment effect in routine practice includes adherence to diet and lifestyle measures

2. Treatment \[\rightarrow\] Biomedical effects

**RCT trial needed to prove treatment effect**

**BUT** Confounded by weight change if that is also a consequence of treatment. Compliance affected if weight loss is desired.

3. Treatment \[\rightarrow\] Weight loss \[\rightarrow\] Biomedical effects

**Biomedical effects may include direct effects of the treatment and those of weight loss**

**RCT trial of treatment \[\rightarrow\] biomedical effects would be confounded by the variable weight changes with both treatment and placebo/control, because of variable efficiency**

The true (physiological) cause of biomedical effects is weight loss, but it is impossible to prescribe fixed dose weight loss as a treatment. Therefore maximize the value of the range of weight losses in study design and analyze by correlating weight changes with biomedical changes
However, placebo-subtracted (or control-subtracted) differences do not equate to expected results in routine clinical practice, where every treatment is a therapeutic trial for the individual, and multiple treatments are combined.

The situation is different where the outcome measures are metabolic or other biomedical variables and, effectively, weight loss is the treatment. Firstly, it is not possible to prescribe a fixed ‘treatment’ (weight change/time) for all patients. Secondly, since all patients entering such a study are seeking weight loss, ‘control’ patients will often lose weight and so be ‘treated’. Thirdly, a placebo indistinguishable from weight loss cannot be given.

Experimental designs for ‘slimming studies’ thus need to be absolutely clear whether weight loss is the outcome or the treatment, and systematic reviews of this field need to appreciate this complexity in view of current system of evidence grading. The optimal study design for weight loss as a treatment is a single-strand intervention, employing regression analysis to evaluate the range of weight changes between individuals, and the quest for controlled trial

Fig. 3. Mean data and SD are shown for (a) angina frequency and (b) body weight (Hankey & Lean, 1996). The correlation between change in body weight and angina frequency (c) gives a more complete view of the effects of weight loss. Weight loss varied substantially, and greater weight loss was more likely to improve angina. A ‘placebo’ non-weight-loss group would have added nothing.

Fig. 4. Possible indicators of success in long-term weight management.
evidence for improved metabolic consequences of obesity is misguided.

Questions about long-term benefits of weight management are valid, but weight loss itself is usually a relatively brief component (often completed in 3–4 months). Studies of ‘long-term weight loss’ are in reality studies combining short-term weight loss with longer-term weight maintenance. These components should be evaluated separately, and different strategies or treatments may be appropriate on grounds of efficiency in routine practice. Evidence needs to come from audit, not from controlled trials, and is currently lacking.

References


Svedsen OL, Hassager C & Christiansen C (1994) Six months’ follow up on exercise added to a short term diet in overweight

Fig. 5. A diagrammatic view of the data obtained from a controlled trial of an intervention to produce weight loss. The plots explain why the placebo group (dashed lines) is not necessary to establish the relationships between weight change and metabolic outcomes, and a correlation analysis makes better use of the data. A controlled trial is necessary, however, to identify any weight-independent effect of the active intervention.
is long-term weight loss possible?


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