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Obesity Management

Problems in identifying predictors and correlates of weight loss and maintenance: implications for weight control therapies based on behaviour change

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Summary

Weight management is a dynamic process, with a pre-treatment phase, a treatment (including process) phase and post-treatment maintenance, and where relapse is possible during both the treatment and maintenance.

Variability in the statistical power of the studies concerned, heterogeneity in the definitions, the complexity of obesity and treatment success, the constructs and measures used to predict weight loss and maintenance, and an appreciation of who and how many people achieve it, make prediction difficult.

In models of weight loss or maintenance: (i) predictors explain up to 20–30% of the variance; (ii) many predictors are the sum of several small constituent variables, each accounting for a smaller proportion of the variance; (iii) correlational or predictive relationships differ across study populations; (iv) inter-individual variability in predictors and correlates of outcomes is high and (v) most of the variance remains unexplained.

Greater standardization of predictive constructs and outcome measures, in more clearly defined study populations, tracked longitudinally, is needed to better predict who sustains weight loss.

Treatments need to develop a more individualized approach that is sensitive to patients' needs and individual differences, which requires measuring and predicting patterns of intra-individual behaviour variations associated weight loss and its maintenance. This information will help people shape behaviour change solutions to their own lifestyle needs.

Keywords: Behaviour therapy, obesity, prognosis, weight loss.

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Introduction

The current trends in, and costs and consequences of the obesity pandemic are now well documented and recognized, e.g. (1,2). While prevention is better than cure, the prevalence of obesity is so great in many countries that treatment cannot be ignored (3,4). It is often noted that of those actually studied, and who lose weight in treatment programmes, many regain it to the extent that within *5* years the majority of people have returned to baseline (*5*). Regain is often followed by repeated weight loss attempts. Weight regain is probably a combination of a physiological drive to return body weight to the previous 'settling point' (*6*), and/or the failure to maintain a less obeseogenic lifestyle and eating behaviours. It appears clear from these observations that, for many, weight control is a chronic relapsing condition that has a strikingly similar pattern to conditions characterized by relapse, such as alcohol and

drug dependency, depression and other aspects of lifestyle or behaviour change and mental health (7). This raises certain questions: given this high relapse risk, which treatments and personal characteristics predict success at weight loss and weight loss maintenance? How comprehensive is the evidence base in these areas and how can it be improved? Can subject traits be matched to treatments to improve success? Can better studies be designed that help us match the characteristics of treatment programmes to the needs of individual people, or do we need to develop new research paradigms to reach this goal? This paper focuses on why it is so difficult to use the behavioural or psychological predictors and correlates of weight loss and weight loss maintenance and whether they can be used to improve outcomes for people in the general population. We suggest that recognizing these difficulties may improve outcomes by helping people with self-management of weight control as a chronic relapsing condition.

Definition of successful weight loss

The complexity in every aspect of weight control is heterogeneity. This also applies to the definition of obesity, successful treatments and an appreciation of who and how many people achieve it.

Foster eloquently describes the historical rational behind the shift from aiming for an ideal weight to one where a 'reasonable' target weight was proposed as a definition of successful weight loss (5). Ideal weight is very hard to achieve, is subject to frequent relapse and more moderate goals of 5–10% are still associated with significant improvements in health risk status (5,8–10). Given that 5–10% weight change is now considered a success by many healthcare professionals (5), there is a remarkable paucity of evidence on the normal, within and between subject variability in body weight on a day-to-day, week-to-week, month-to-month and year-on-year basis. Parenthetically, there is a complete dichotomy between apparent medical goals and expectations and those of the patient as the weight loss expectations of patients are usually much higher. Furthermore, the very outcome that we are trying to change as professionals (namely body weight) is one of the least understood or quantified in terms of long-term, frequent longitudinal measures. While it is generally assumed by physiological models of weight control that energy balance is the usual *status quo*, data from 7147 participants of the NHANES (National Health and Nutrition Examination Survey) suggest that American adults gain between 0.2 and 2.0 kg per year of their adult lives (11). This and other data suggest that most US adults at least, are in a state of slight but chronic positive energy balance that leads to weight gain over time (12). The statistical power of these estimates is high and they are corroborated by global trends in obesity (3). Many weight control attempts may achieve simply that – weight control but not weight loss. The impact of these attempts on long-term weight stability should not be overlooked (13).

According to Wing and Phelan, in studies that range in sample size from 225 to ~1000 (14) about 20% of the population do indeed lose and maintain more than 5–10% body weight, but they are the exception rather than the rule. While ostensibly this does not give a lot of weight change to make predictions about, in reality weight losses are characterized by huge intra- and inter-subject variability. This is not the only problem with prediction of successful weight loss and maintenance.

Problems with current estimates and prediction of success

A number of factors make simple prediction of successful weight control difficult. Some of the problems with current estimates and prediction of success can readily be overcome; however, other problems are more difficult to address. Such issues need to be highlighted before considering the pre-treatment predictors and treatment correlates of successful weight loss and correlates of weight maintenance.

Available data are from highly select samples. Brownell and Rodin (15) note that as of 1994 'virtually all data on weight loss and maintenance are from clinical trials conducted in university (or clinical) settings'. Patients seen in speciality obesity clinics appear to differ in important ways from overweight persons in the general population. For example, among individuals seeking clinical obesity treatment 25–50% are binge eaters compared to 5% of the general population, using samples of 455 and 1984, respectively, (16,17). Brownell and Rodin (15) argue that clinical samples may show a conservative bias 'because the participants are more overweight, more likely to be binge eaters, have more psychopathology, and may differ from other overweight persons in other ways not yet studied'. Such individuals probably do not represent those in the general population who are overweight or even those motivated to lose weight (10). They may include a high proportion of people who have failed on their own or in commercial programmes and who thus may be less predisposed to achieve long-term weight maintenance (18). It is also possible there is an over-representation of a range of hormonal and other physical disorders that are associated with weight gain in these populations.

Outside the clinics the majority of people who lose weight do so by themselves and/or by joining commercial diet or self-help groups and exercise programmes. These estimates are derived from two studies, one with a sample size of 4647 and the other only 89, and so their statistical power varies considerably (19,20). Thus it is estimated that 95% of people presenting to lose weight do so outside of

obesity clinics (15). It is likely that those who attend self-help and diet groups differ from the general population also. The majority of those who attend self-help groups, be it for alcohol and substance dependency, mental health or weight loss tend to be characterized by a common trait – they tend to be chronic relapsers (7,15,21). Hence they seek the ongoing support and stability that the safe environment of a self-help group provides. There are also a significant number of people who, by themselves or in professional and commercial programmes, do lose weight and maintain the loss, e.g. (14,22–24). The problem is that they are difficult to quantify either because they develop weight management skills on their own or leave the programme once they have attained the skills to navigate to and stay at a healthy body weight. They are therefore, like dropouts, lost to measurement. This suggests that the most and least successful weight losers are under-represented in such analyses.

The treatments for weight control, the people who are subjected to those treatments, and the measures made of the characteristics of subjects, treatments and outcomes, are all very heterogeneous in nature. This is a major problem that has been the main cause of failure to predict weight loss success. Not only is the condition heterogeneous, but its treatment and outcome measures have all changed dramatically over time (10,25,26). Thus, in most cases, evaluation of behaviour change, diet, exercise, drugs and surgery have been largely made on self-selected or experimenter-selected populations who may or may not represent (i) the general population; (ii) the obese subpopulation – if indeed a continuum could be described as such or (iii) the population most suited to that particular treatment. Therefore, most estimates and predictions of success are likely to be conservative. Completer-only analyses are particularly problematic as they ignore dropouts, who exhibit the greatest barriers to success (25, p. 57). So far, attempts to reduce dropout rates or statistically account for missing data are not entirely satisfactory.

Allison and Engel (27) suggest four main reasons why we cannot reasonably predict treatment outcome. (i) Firstly we may actually be able to predict but do not realize it. They point to Robert Rosenthal's (28) argument that in many cases a specific predictor needs no new studies; it just needs appropriate meta-analysis. The problem with this is that data are often accumulated poorly. Their analysis of *locus of control* shows that many studies do show that it is a predictor of success, but there is large heterogeneity because of differences in the refinement of the measure of the construct itself. Appropriate meta-analytical techniques do yield good significant predictors, but often treatment outcome is still predicted weakly, i.e. the model only explains a small proportion of the variance in outcome. This is a recurring theme in predicting weight loss outcomes. (ii) They advocate multivariate over univariate prediction but caution that in some analyses the number of predictors tried actually exceeds the number of people studied. (iii) They advise the use of dimensional rather than categorical prediction and rail against the categorization of natural continua, which reduces statistical power. The counter argument is that categories are useful for illustrative analyses because they reveal significant contrasts. (iv) Allison and Engel also advocate the use of theory rather than retrospection as an impetus to search for predictors of successful treatment outcomes and the need to consider environmental factors in behaviour change (27). We suggest these are not mutually exclusive and both have useful research potential. For example, there is a need for more qualitative work that explores peoples' own insights and explanations for their relapse and what might have prevented it.

The statistical power of different studies also varies. Estimates of weight change over time are now very well powered with sample sizes now remarkably in the millions, see (3). Measures of behaviours associated with successful weight loss maintenance are also well powered, although they are derived from a specific population that has cumulatively expanded in numbers over time. However, specific predictors and correlates of weight loss and weight loss maintenance are far more variable in the statistical power of the studies that have examined specific issues. For example, in the review of pre-treatment predictors of weight loss by Teixeira *et al*., sample sizes of predictive models range from 42 to 444 (25). Given the other problems associated with predictors/correlates of weight outcomes, this clearly indicates a danger that some studies are underpowered. Tests that are underpowered are likely to miss real predictors by generating false negatives (Type II error). In this sense the power of the test is its sensitivity. Conversely, specificity is 100 minus the significance level (G Horgan, personal communication). Tests that have many predictors and set the significance cut-off at the usual 5% may generate false positive predictors (Type I errors), simply by chance. Alternatively, setting the significance level unusually high will have the same effect.

When considering predictors and correlates of weight loss and maintenance, it is important to recognize that weight management is a dynamic process, with a pre-treatment phase, a treatment phase (involving process) and a post-treatment maintenance or relapse period. Relapse can also occur during treatment. Predictors and correlates of outcomes are not static; they can and do change between phases of treatment. It is useful to consider predictors/correlates of weight outcomes as pre-treatment predictors, treatment/process correlates and post-treatment correlates of weight maintenance. Here treatment variables are defined as factors specific to the treatment (such as duration, type of intervention used), while process relates to variables attributable to or experienced by the individual

during their weight loss history (such as previous dieting attempts and/or early weight loss). The dynamic nature of behaviour change, however, means that changes in behaviour profiles during treatment often carry over into the post-treatment phase and become new weight-maintenance habits.

Pre-treatment predictors of weight loss and weight loss maintenance

Considerable work has characterized pre-treatment predictors of success, but the results are highly variable because there is great heterogeneity in (i) the populations studied and sample sizes; (ii) the treatments administered under the same category (e.g. diet) and (iii) measures of specific constructs (e.g. psychopathology, physical activity, binge eating) (10,25,26).

The main pre-treatment predictors of weight loss are included in Table 1 and have also been summarized by Teixeira *et al*. in studies using samples ranging from 42 to 444 (25). A number of factors would be intuitively expected to predict weight loss, but do not appear to. These include total body fat, fat distribution and body composition, personality and psychopathology test results, dietary restraint and binge eating. Teixeira *et al*. (25) have scrutinized the pre-treatment predictors of success at weight management and categorized them into predictors and non-predictors with consistent evidence, mixed evidence and suggestive evidence (25). It is notable that the majority of predictors fell into the latter two categories and the majority of non-predictors fell into the former category. It is worth considering these factors in detail. Part of these findings may be due to the low power and heterogeneity issues discussed above.

Table 1 Predictors of weight loss

Predictor	Relationship
Patient factors	
Initial body weight or body mass index	+
Gender (men tend to lose more weight)	+ for men
Resting metabolic rate or energy expenditure	+
Adipocyte hyperplasia	+
Self-efficacy	+
Body fat distribution, total fat, body composition	?
Personality/psychopathology test results	?
Dietary restraint	?
Weight cycling	?
Binge eating	?
Process variables	
Early weight loss	+
Attendance	+
Repeated previous attempts at weight loss	–
Experience of perceived stress	–
Treatment factors	
Increased length of treatment	+
Social support	+
Behavioural changes	
Self-monitoring	+
Goal-setting	+
Slowing rate of eating	+
Physical activity	+

Data from Blair, 1993 (150); Foreyt and Goodrick, 1995 (151); Perri *et al*., 1992 (109); Wadden and Letizia, 1992 (77).
Sources: Institute of Medicine, 1995 (10); Brownell and Wadden, 1992 (21); Foreyt and Goodrick, 1991 (111), 1995 (151); O'Neil and Jarrell, 1992 (152); Perri *et al*., 1992 (109); and Wadden and Letizia, 1992 (77).
–, negative correlation; +, positive correlation; ?, mixed findings.

Genetic and physiological predictors of weight loss

The lack of specific quantitatively significant genetic or physiological predictors of successful weight loss (or even obesity predisposition) is striking (see below) (10). There are numerous models based on nutrient partitioning, activity of specific fat depots, nutrient stores, components of energy expenditure (29) and a plethora of putative central and genetic mechanisms, believed to be involved in the development or predisposition to human obesity (30). Indeed, it has been stated that '. . . in the commoner form of obesities, a multitude of polymorphisms located in genes and candidate regions participate in an individual's susceptibility to weight gain in a permissive environment. The effects are often uncertain and the results not always confirmed' (31).

What is certain is that these relationships between putative biomarkers of obesity and obesity itself are not predictors; they are correlates; causation is far from clear. Furthermore, the relative contribution of endogenous (e.g. genes) and exogenous (environmental) determinants is currently speculative. Many genes clearly contribute to obesity in a permissive environment. But how many genes, which environmental contributors and how permissive the environment is, remain unquantified variables. Thus a multitudinous, heterogeneous contribution of many genes and their various alleles appears to have smaller effects on obesity phenotypes than might be expected. Indeed, there is increasing evidence that there are complex interactions of these processes with environmental contingencies. So with few exceptions such as the FTO gene (32) leptin and the MC4 gene variants, e.g. (33), which impact on relatively few people, it is hard to say there are many specific genetic predictors of obesity. At the present time the same arguments can be made not just for the genome but for the proteome and the metabolome. It is likely these models will be greatly refined in the next few years. But dissociating the effects of these causes from each other, let alone from environmental influences of obesity, will be a major challenge for the future (34).

Physiological predictors of short-term weight loss have been found under carefully controlled laboratory conditions, where patients have been subjected to a mandatory energy deficit, and the effects of variability in real-life dietary and activity patterns are removed. For example, Hainer *et al*. (35) studied 67 women as in-patients over 4 weeks and found that in a backward stepwise regression model: age, initial body mass index (BMI) together with baseline levels of growth hormone, peptide YY, neuropeptide Y and C-reactive protein predicted 49.8% of the variability in weight loss (35). Psychological variables did not predict weight loss under these conditions, but they would be unlikely to, as the regimen was imposed on the patients over a short period and food and energy intake, and physical activity were all tightly controlled.

Various studies have found relationships between comorbidity status and weight loss in treatment programmes. For example, in a well-powered study, Janghorbani and Amini investigated patterns of long-term weight changes among 7820 patients with type 2 diabetes mellitus over 9.1 years, and the authors found that '. . . using a stepwise multiple regression model, higher BMI, follow-up fasting plasma glucose, systolic blood pressure, triglyceride level and treatment with insulin increased the per cent weight change, and higher number of follow-ups, cholesterol and smoking significantly decreased it' (36). However, while weight loss outcomes may have been due to the physiological changes observed, the associations detected may have also been influenced by psychological motivation associated with the patients' self-perceived need to reduce their comorbidity status.

There is also some evidence that weight loss by gastric bypass influences the hormones associated with gastrointestinal hunger and satiety (peptide YY [PYY] and to a lesser extent GLP-6) and that this itself may be associated with the increases in satiety associated with the surgery (37). In a separate study, Le Roux *et al*. found that in 16 patients the attenuated appetite after gastric bypass is associated with elevated PYY and GLP-1 concentrations, and appetite returns when the release of gut hormones is inhibited. They suggested that gut hormones have a role to play in the mechanism of weight loss after gastric bypass, through their effects on appetite (38).

At present, surprisingly few metabolic, genetic or hormonal biomarkers specifically predict, to an extent that they can be used as practical tools in weight control programmes, subsequent success or lack of it at weight loss and its maintenance. This is probably due to a situation that is analogous to the psychological and behavioural predictors of weight outcomes. Neuroendocrine predictors and correlates of weight loss are likely to be complex. It is likely that such factors will become more evident as large-scale longitudinal studies begin to analyse the biomarkers of successful weight loss and to construct more complex models of the physiological determinants of weight loss and maintenance (39).

Initial weight or body mass index

Initial weight, initial BMI and a higher initial resting metabolic rate, e.g. (35,40,41) tend to predict greater absolute weight loss (10,25,26,42). However, resting metabolic rate correlates with body size, and the association between initial body size and weight loss is largely an effect of bigger people having a greater energy deficit when on a prescribed fixed-energy diet. In the Sibutramine Trial on Obesity Reduction and Maintenance, resting metabolic rate explained only 1.7% of the variability in weight loss, and was not present in the multiple regression model (41). However, this study is potentially problematic because while the sample size was sufficient to make it sensitive, using a significance cut-off of 15% meant that it was not very specific and may have generated some false positive predictors. In the large multi-European centred DiOGenes study, initial body weight was the strongest predictor of weight loss in a multiple regression analysis of weight loss in overweight or obese adults who followed an 8-week low-calorie diet (43). Initial body weight explained 21% of the weight loss after 8 weeks. Early weight loss, after 1 and 3 weeks, were also significant predictors (see below), increasing the variance explained to 68%. The larger energy deficit of heavier people on fixed energy diets might be expected to result in a positive association between initial body size and dropout rate from weight loss studies, but has not been consistently found (44).

That initial body weight, or BMI, tends to be associated with greater weight loss is also, at least in part, because a disproportionate number of very obese people tend to be treated using more intensive methods, which in the first stages of weight loss at least tend to yield greater success (21).

Gender

Weight loss motivations for men and women appear to differ in nature and intensity (10). Dieting is more prevalent among women than men, and more women enrol in obesity treatment programmes. Women are motivated to improve their appearance while men are motivated more by health and fitness concerns (10, p. 127). However, many studies find that success rates in weight loss programmes vary by gender with women experiencing smaller absolute and relative weight losses than men (10, p. 127).

Age

According to the IOM (Institute of Medicine), 'age potentially confounds many associations of other population

characteristics with weight management variables' (10, p. 128). Indeed, body weight and composition do change across the lifespan. In their characterization of 'the successful weight maintainer', Elfhag and Rössner (26) describe a person who has learned through a variety of coping strategies to manage and avoid relapse, to develop stable patterns of activity and eating behaviour that are conducive to weight maintenance and who has developed a network of support that helps them deal with the stresses of everyday life without resorting to food as a means of comfort. It may be that younger people have not had the time to develop these skills. Conversely, given the relationship between previous dieting attempts and failure to lose weight it may also be that some people fail to develop these skills and are chronic relapsers (7).

Weight cycling

Previous attempts at weight loss or previous weight cycling are consistently reported as predictive of poor success at subsequent weight loss (10,25,26,42). It may well be that previous attempts at weight loss are a separate issue from weight cycling, but they are often treated as similar in the literature (see below). A great deal has been made of the physiological basis of weight cycling and the possibility that previous weight loss makes subsequent weight control harder from a physiological perspective, see (42). However, it is worth bearing in mind that the natural pattern of weight change in subsistence economies is indeed to cycle with the seasons. For example, Gambian subsistence farmers change weight by 10% between the wet and dry seasons (45). Yanovski *et al*. reported, in a prospective study of 195 US adults, that on average they gain 0.5 kg during the Thanksgiving and New Year period, which is not reversed during the spring or summer months. They suggest this gain probably contributes to the tendency for adults to gain weight in US Society (46). Currently, we lack sufficient, frequent measures of body weight in enough people to be able to describe day-to-day, week-to-week and month-to-month patterns of weight change in the whole population or how this varies with degree of obesity. Indeed, one reason why a 5% weight loss may be difficult to sustain is that people vary by this much from month to month and the factors influencing this variation will also influence the outcome of a weight loss within the bounds of that variation. In this context, it is useful to see obesity as a chronic relapsing condition that requires long-term management, control of relapse and recognition of relapse signatures at the level of the individual. In this respect, the stages of change model is highly relevant to weight loss and the prevention of weight regain (47).

Dietary restraint – binge restricted and disinhibited eating

Teixeira *et al*. (25) found little or no association between weight changes during treatment and baseline binge eating in 19 predictive models (most frequently assessed by the binge eating scale). The main reasons they cite for this is heterogeneity of the constructs concerned. For example, in 54 525 subjects it was found that restraint is not a single construct and splits quite neatly into flexible and rigid restraint; flexible restraint being far more predictive of successful weight loss than is rigid restraint (48,49). Secondly, changes in binge eating may only really occur during the course of a lifestyle intervention, and so baseline measures are a poor indicator of change during treatment (25). Some evidence also indicates that individuals with the highest binge eating scores may drop out of treatment earlier (44). However, obtaining reliable measures of binge eating is difficult in such studies and many do not attempt to measure it at all.

Personality profiles

Standardized personality tests are consistent in that they do not predict treatment outcome (25,42). There appear to be two main reasons for this. Firstly, in most people personality is a relatively stable construct; it will not change much in relation to behaviour. However, of perhaps greater significance is the observation that instruments such as the Minnesota Multiphasic Personality Inventory (50) and the Sixteen Personality Factor Questionnaires (51) are in some sense the psychologist's shotgun in that they 'measure a little bit of everything while not necessarily measuring any one construct in an especially focussed manner' (27). In contrast, factors more directly related to eating behaviour, such as the 'food contents' Rorschach variable, may be more strongly related to subsequent weight loss (52,53). The relationship between personality characteristics and weight outcomes are perhaps insufficiently studied at the present time (26).

Psychopathology

Teixeira *et al*. (25) note that in virtually all of the studies they reviewed as pre-treatment predictors of weight loss '. . . measures of psychological well-being or psychopathology (mood, depression, personality disorders, etc.) were not found to significantly predict weight loss'. A major reason why this is so (as for self-efficacy, diet, exercise and binge eating) is that improvement in psychopathological symptoms occur during weight loss treatment (54,55) and tend to covary with weight changes (56,57). However, sample sizes in these studies were small, ranging from 53 to 138. Consequently, baseline assessments may

lack an important or lasting impact. This is discussed further below.

Coping strategies

A more autonomous coping strategy and one that relies more on confronting problems than avoidant strategies (e.g. eating, sleeping or wishing problems would go away) has been found to correlate better with successful weight loss and maintenance than a less autonomous style, in studies of a few hundred people (25,26).

Self-efficacy

Self-efficacy is the conviction that one can successfully execute the behaviour required to produce the outcomes desired (58). Results from studies prior to 1995 (20,47,59) have led to the general conclusion that high self-efficacy towards eating behaviours is perhaps the only reliable predictor of subsequent weight loss (60), but the collective evidence since then does not confirm this (25).

Self-motivation

Self-motivation is predictive of successful weight loss, both for dieting and exercise. However, self-motivation often vacillates, in relation to lapses and relapses. It would be valuable to determine the factors that influence both pre-treatment motivation and changes in this construct during the treatment cycle. It is becoming clear that a major factor undermining self-motivation in relation to small lapses is self-criticism (61).

Attitudes

People who were less likely to attribute their obesity to medical conditions appear more successful in later maintaining weight loss (26). This is similar to having a more internal locus of control and appears to be one of a cluster of factors that correlate with success.

Body image and self-esteem

Given the complexity of these constructs, their interrelationships and the issues that surround them, it is not surprising that body image and self-esteem have yielded mixed results as pre-treatment predictors of subsequent weight loss. As noted by Teixeira *et al*. 'the diversity of assessments for this construct (in fact, the "diversity" of the construct itself) makes a summary conclusion difficult to reach' (25).

Outcome expectancies

There has been considerable debate as to how we should help patients manage their outcome expectancies ever since Foster *et al*. (62) demonstrated that in most cases, of the 60 obese women studied, minimal outcome expectancies exceed the maximal weight lost in a treatment programme. It has been suggested by some authors that unrealistically high outcome expectancies will increase the probability of dropout from any treatment programme. The literature and evidence base is a mixed bag. Teixeira *et al*. (25) felt that the combination of positive and realistic expectations foretell the best results, especially if accompanied by a strong sense of self-assurance. Elfhag and Rössner (26) go much further by suggesting 'unrealistic' optimism can be a healthy and adaptive psychological mechanism which promotes healthier behaviour and cite Taylor *et al*. (63) in support of this controversial point. The bottom line is that despite little hard evidence to support modest weight loss goals, they have become the standard recommendation in many public health policy documents, e.g. (9,60).

Locus of control

A person with an internal locus of control believes that outcomes tend to be under their own control. A person with an external locus of control attributes outcomes to external sources such as chance, the environment, genes or powerful others. Allison and Engel (27) cite locus of control as a clear example of where we may be able to predict outcome but do not realize it. They conducted a mini meta-analysis of 11 studies and found a mean correlation of 0.19 between locus of control and weight loss in 858 pooled subjects. However, a chi-squared test showed a considerable heterogeneity in the results due to some studies using far more focussed measures than others. The more incisive studies yielded higher correlations than the broader measures. Another illustrative and near ubiquitous finding that is generalizable to the whole of weight control prediction is that treatment outcome was weakly predicted so the predictor only accounted for a small proportion of the variance in weight loss.

Diet

There is now a wealth of data from a variety of intervention and survey studies on the role of diet in weight loss. It is uncontroversial that a number of dietary attributes (fat content, energy density, portion size, low protein content, packaging, ease of preparation, low fibre content, low moisture content, etc.) are strongly predictive of weight gain and changes in these factors associate with weight loss, e.g. (64–66). Furthermore, reversing these predictors now forms the cornerstone of dietary and indeed all behavioural approaches to weight management in the first instance. There is still some debate as to the role of specific macronutrients, combinations thereof (e.g. protein and glycaemic index) and certain ingredients in weight control.

Exercise

Baseline exercise behaviour is a poor pre-treatment predictor of weight loss (25). A variety of sources also suggest that exercise is a poor initial means of weight loss (although it is important in weight loss maintenance, see below) (57,67–73). This is largely because, for a variety of reasons, the obese do not want to, or are not able to, exercise enough to have a large impact on energy balance. This is why diet has become the cornerstone of initial weight loss attempts.

Quality of life

There is currently little known of how quality of life predicts subsequent weight loss or its maintenance. As with many predictors of weight loss, quality of life can be confounded and correlated with other variables associated with weight loss.

Social support

Teixeira *et al*. (25) found no evidence that social support is a pre-treatment predictor of weight loss. This is unsurprising since people often use social support to solve behavioural problems during the process of weight loss rather than at the outset.

Process and treatment-based correlates of weight loss

It is easier to distinguish between pre-treatment predictors and weight outcomes than other treatment/process or maintenance correlates of weight loss at subsequent points in the weight loss and maintenance process. Distinguishing between treatment/process correlates and weight maintenance correlates of weight outcomes is less clear. This is because many of the changes that occur during the process of weight loss become the basis for habitual weight maintenance behaviours. Some treatment correlates thus become maintenance strategies, but these processes are variable both within and between people. Table 1 shows that during weight loss treatments, positive personal predictors include high perceived self-efficacy and the fact that men tend to lose weight more than do women. These data are compiled from a variety of sources with varying statistical power, although on aggregate the findings are quite robust. Positive process factors include programme attendance and early weight loss. Positive treatment factors include length of treatment, social support, incorporation of behaviour modification techniques (especially self-monitoring and goal setting) and increased physical activity (10).

Consistent negative behavioural and psychological predictors of weight loss seem to be greater in number. Unsurprisingly, the most consistent negative predictors appear to be the opposite of positive predictors, with the addition of repeated attempts at weight loss and the experience of perceived stress (10,42). These two factors are probably not unrelated. One of the key correlates of relapse is the inability to develop coping strategies that manage various forms of stress (including weight loss) in everyday life (7,25,26,74).

Attendance

Attendance at a programme is one of the most consistent process-based correlates of weight loss (10,26,75,76). The key issue, however, is whether attendance predicts weight loss, or weight loss predicts attendance. It has been noted that both factors may be influenced by a third one, such as increased motivation or fear (10). The Institute of Medicine's 1995 review summarized predictors of attendance as positive (closed group classes, refundable deposits, perceived health improvements), negative (stress, small initial loss, low perceived energy) and non-predictors (age, start weight and BMI, mood, age of obesity onset and hunger susceptibility) (10, p. 123).

Early weight loss

Early weight loss in a programme is consistently and significantly associated with subsequent weight loss (77). In the DiOGenes study, weight loss after the first and third weeks were predictive of final weight loss (43). First week's weight loss, but not weight loss at weeks 2–8, predicts weight loss at 12 weeks and explains some 20% of the variance in a supervised commercial weight loss programme (78). Early weight loss also correlates with subsequent attendance (10).

Self-monitoring

It has now been found in a number of studies that self-monitoring behaviour in terms of weight, diet and activity are cardinal behaviours of successful weight controllers, e.g. (10,22–24,42,75). Given the difficulty even scientists currently have with monitoring food intake and energy expenditure it seems likely that successful weight maintainers are successful at monitoring the behaviours that are associated with weight stability rather than directly measuring their intake or expenditure. Conversely, it has long been known that those with refractory obesity underestimate their intake and overestimate their energy expenditure, e.g. (79). Self-monitoring is crucial if relapse is the key mechanism by which weight regain occurs. This is because in order to avoid relapse one must learn to cope with, and

navigate around, a lapse. To do this most effectively it is valuable if people can become aware of their own relapse signatures through some form of self-monitoring. This is an exciting area for the future use of hand-held technology, especially in the areas of dietary and physical activity behaviours.

Social support

While social support is a poor pre-treatment predictor of weight loss, social support during weight loss is a correlate of weight loss (26,75). Also it is known that group support tends to be better than go-it-alone approaches or even one-to-one approaches (10,75,76). One such study was a well-powered randomized controlled trial (76). However, an individual's degree of success is likely to be heavily influenced by their capacity to construct linkages and alliances with people sharing the same problem, and the quality of the social support network available (80). Williams and colleagues noted that perceived type of support from the intervention team in 128 subjects, measured 5–10 weeks into the programme, was important for later success (81). Specifically, when support was viewed as fostering autonomy as opposed to being controlling, participants obtained significantly better results (see below) (25). It is likely that the self-help group dynamic of weight loss groups acts as a catalysing environment for the adoption of coping skills and strategies (80). Humans are intensely social animals and often find it far easier to solve specific problems in a social context provided they consider that environment to be safe and secure (61,80).

Dietary restraint, binge eating, restricted and disinhibited eating

It was argued above that restraint, disinhibition and binge eating are poor pre-treatment predictors of weight loss and that the behavioural changes that correlate with success may only occur during the course of treatment. Evidence is mixed in relation to weight loss maintenance. Earlier clinical studies, according to Wilson (42), show that binge eating is not a reliable predictor of outcome in obese patients. This contrasts with the findings of the National Weight Control Registry (NWCR) who noted that members of the registry who gained weight over a 12-month period reported greater decreases in restraint and increases in hunger, dietary disinhibition and binge eating (23). While the NWCR has large numbers and high statistical power they may be a specific sub-population in that they are characterized by successful weight loss maintenance. Thus the current fragmentary evidence suggests that these traits (with the exception of flexible and rigid restraint) do not reliably predict treatment outcome but are significantly associated with post-treatment maintenance of weight loss and perhaps compliance with treatment programmes.

Personality profiles and psychopathology

There is some (weak) suggestive evidence of personality in relation to weight maintenance. Elfhag and Rössner (26) note that the Karolinska Scale of Personality (a scale of socialization) has been related to weight loss maintenance in two studies (82,83), while others have not replicated the findings (84). Earlier work seemed to suggest that a more developed personality with regard to relating to other people (85) is associated with success. Conversely, more perceived initial dysfunctions in social interactions has predicted weight relapse (86), although evidence for a relationship between psychopathology and weight maintenance is sparse (26). There are weak indications that a trend towards psychopathology is associated with poor weight loss in obesity treatments (53,87,88). This issue is complicated by medication usage as use of many mood stabilizers and antipsychotics is associated with weight gain. Other authors have sounded a note of caution with current measures of psychopathology in obesity research since they may be tools designed to detect a range of psychopathology that covers the full clinical spectrum and may be too broad to detect some of the subclinical manifestations of these disorders in the obese population (74). Here is another example of where more specific and incisive tools may help us to better focus on the predictors we are looking for. The same arguments hold for depression. Some of the analyses appear counter-intuitive, in which greater depression at baseline predicts greater weight loss (89,90); however, these are relatively small studies with low statistical power. It may well be that a degree of dissatisfaction and unhappiness with the current state of one's life is part of the stage of change model required to muster the motivation to change in the first place. For others, however, bouts of depression may be the triggers to lapse which lead to relapse and regain. This is again a heterogeneous predictor in an equally heterogeneous population. Furthermore, many studies (especially clinical trials) initially screen out subjects with clinical depression or clinically diagnosable psychopathology, which may render some analyses inappropriate because of lack of variance in the trait of interest.

Coping strategies

In general, active problem solving and coping is more adaptive than passive forms (42). Kayman *et al.* (91) found that problem-focussed rather than emotion-focussed coping strategies were associated with weight loss maintenance over 2 years. Sample sizes were, however, quite low (44 relapsers, 30 weight maintainers and 34 controls). The

former involves developing problem solving skills aimed at directly resolving challenging situations, and the latter involves ways to reduce emotional stress such as eating, sleeping more or simply wishing a problem would go away. Elfhag and Rössner (26) note that research findings on relapsers (weight regainers) indicate that they tend to eat in response to stressful or negative life events and negative emotions that can be evoked by stressors in everyday life (91,92), and have a tendency to use eating to regulate mood rather than directly confronting life's problems. Again, sample sizes were not large and did not exceed 100. Relapsers have also been shown to report more help seeking behaviour as a means of coping with a dietary relapse. It may be that they exhibit a more external locus of control and/or a lack of self-efficacy. However, it may be that these are people who seek, but do not attain, the social support they need to develop problem solving skills that cope with the vagaries of a lapse, whereas, successful lapse-managers establish a robust support network upon which they can rely, in addition to their own resources.

Self-efficacy

Changes in self-efficacy are predictive of success, at least in groups ranging from $n = 54$ to $n = 113$ (86,93–95). Some of the more general measures of self-efficacy may be more predictive of success than more specific measures (25).

Body image and self-esteem

Retrospective studies show that those with more concern for their shape and appearance successfully maintain a lower body weight. Elfhag and Rössner (26) term this a 'healthy narcissism' about their appearance and physical condition. Given that the majority of women in the general population want to lose weight for reasons related to self-worth, self-esteem and appearance, it is not surprising that pride in appearance has been found to be one of the four main factors facilitating weight maintenance in a sample of 109 subjects (96). Nonetheless, this is again a heterogeneous and complex issue. Of most concern is the point at which so-called healthy narcissism becomes unhealthy. A tendency to evaluate one's self-worth in terms of body image (weight and shape) has been associated with weight gain in 76 women (97), and some regainers do not see themselves as just heavier but can view themselves as ugly (91). This implies that body shame could be an import factor (98).

Many obese women have low esteem and lack confidence, both of which improve following weight loss; they also feel that they are discriminated against, and are stigmatized by their weight (99). Perceived discrimination may actually increase following bariatric surgery, as surgery is often seen by others as an easy or lazy method of weight loss (99).

Many obese people see themselves as a failure, ugly, disgusting, ashamed, guilty and at fault. In one commercial slimming organization, associated with one of the authors, this is called 'the burden of shame and guilt' that needs to be lifted before 'the burden of body weight' can be addressed. Until recently these dimensions of the participant's perspective on their own weight and ability to control it have received little attention from the majority of the clinical or scientific community.

It is becoming clear that successful weight loss does improve a patient's perception of self-image and enhances self-esteem (99,100). In particular, a meta-analysis of 117 weight loss treatment tests showed that weight loss treatment was associated with lowered depression and increased self-esteem (100). How these improvements relate to the maintenance of weight loss is less clear. It seems, however, that the factors relating to body image and self-esteem that empower a person to cope with a lapse situation should be predictive of weight loss maintenance as they would buffer that person from the gravitational force of their previous lifestyle, to which they will feel pulled as they try to establish new lifestyle habits that maintain them at a new lower body weight.

Diet

A regular dietary pattern involving a low fat diet, of low energy density, containing a large proportion of fruit and vegetables and breakfast, is associated with weight loss maintenance, e.g. (10,22–24,42,75). It seems that at the individual level establishing an individual adaptive pattern of behaviour for weight maintenance is rather more important than rigid adherence to specific recommendations. Thus, 45% of the NWCR studied by Klem *et al.* (22) lost weight on their own. Many people who are successful work it out for themselves and find their own solutions that map onto their own lifestyle patterns (91). In this way they are able to adapt their diets to their individual needs and make it a habit.

Exercise

Exercise becomes increasingly important as weight loss proceeds, and is of overwhelming importance in maintaining weight loss. However, the amount of exercise required to maintain weight loss is quite considerable as has been documented (101–103). Increases in physical activity and decreases in sedentary behaviours are key correlates of weight loss maintenance (104,105). The statements made about diet are also largely applicable to exercise. The key criterion by which exercise helps weight maintenance is that the person does enough of it (101–103) in a manner that can be adapted to their habitual lifestyle. Recently, Teixeira *et al.* have documented, in 225 women, that

success in long-term weight maintenance interventions are associated with increases in both exercise self-efficacy and exercise intrinsic motivation (106).

Quality of life

It is now clear that increased BMI is associated with reduced quality of life in a sample of 3353 US adults (107), and that several approaches to weight loss improve quality of life. Also the improvements in quality of life are related largely, but not entirely, to the degree of weight loss in a meta-analysis of 117 weight loss treatment tests (100). Other factors such as psychotherapeutic approaches that do not produce so much weight loss also positively impact on quality of life. While it would seem intuitively obvious that improved quality of life would increase maintenance of weight loss, this is yet to be clearly demonstrated.

Duration of treatment

Given the issues of attendance discussed above it is not surprising that duration of treatment predicts weight loss (108). That is at least for those who remain in treatment. The problem is those who do not, as they are often lost to study and it is difficult to know what exactly happened to them. Another problem is the law of diminishing returns. Weight loss is not directly proportional to length of treatment but tends to fall off or decelerate in a curvilinear fashion as time progresses, e.g. (109).

Type of treatment

It is reasonable to say that, with the possible exception of surgery, all are limited in efficacy and duration. Complex interventions are more effective than monotherapies, as illustrated by extensive reviews (10,21,110). Hence the call by some for lifelong approaches (111). It also seems that the more dimensions or modules of behaviour change that a person can fit into their lives the greater the chance of success, e.g. (10,21,112). In discussing whether we can better match subjects to treatments, Allison and Engel (27) note that the study of attribute–treatment interactions is at an early stage of its infancy. They note that there is virtually no research that tries to target specific treatments to different types of individuals using randomized protocols. In fact, where such attempts have been made all too often an attribute–treatment interaction is not found. For example, randomizing people who preferred group therapy over individual therapy produced no attribute–treatment interaction, just a main effect of group over individual therapy (113). Another interesting methodological point here is that while patients are rarely allotted to the treatment they prefer, they are often prescribed a treatment the physician deems most suitable for them or as recommended by some guide such as the Obesity Treatment Pyramid. Thus, comparisons of treatment are often done using patients specifically if not appropriately selected for that treatment.

The key features of psychological and behavioural models that attempt to predict weight trajectory in the real world are that: (i) many predictors explain a small percentage of the variance; (ii) the more closely analyses focus on a given variable, the more likely it is to be accounted for by several other small variables, each accounting for a smaller proportion of the variance; (iii) correlative or predictive relationships are different in different populations studied, partly because of the attributes of the populations, and partly because of the characteristics of the treatments; (iv) most of the variance remains unexplained. Physiological models are likely to show very similar patterns.

Correlates of weight maintenance

It is logical to assume that many of the behaviour changes initiated during the treatment period of weight loss will carry over into the post weight loss, maintenance phase to form the basis of a new pattern of behaviour that is associated with weight loss maintenance (8). Wing *et al.* and Hill *et al.* characterized the attributes of successful weight losers in the NWCR and these analyses tell a reasonably similar and statistically robust story (22–24,114,115). Positive predictors of weight loss maintenance include physical activity (which is a poor predictor of weight loss itself), self-monitoring, a positive coping style, continued social support, normalization of eating patterns and reduction of comorbidities (10, p. 123). The NWCR data show that flexible coping strategies and eating breakfast are also positive predictors of weight loss maintenance (22–24,114,115). Similar results have recently been reported in a smaller ($n = 225$) sample of Portuguese women by Teixeira *et al.* with the inclusion of lower emotional eating, flexible cognitive restraint, body image dissatisfaction, exercise self-efficacy and exercise motivation (106). Negative predictors of weight loss maintenance include negative life events and family dysfunction, while higher levels of depression, dietary disinhibition, small lapses and binge eating were predictive of weight regain (23). It is interesting that the NWCR is likely to be more representative of people in the general population who have succeeded in maintaining a significant (at least ~15 kg) weight loss over a sustained period of time than many of the groups studied in clinical trials or research interventions. While the sample size is large (now several thousands), they are still a very specific self-selected subgroup, who are mainly middle aged, female, college educated and Caucasians, and it is not yet clear how generalizable the findings from this group are. Having said this, a general overview of the behaviours of successful weight loss maintainers in the NWCR is interesting and illuminating. Figure 1 shows the approximate

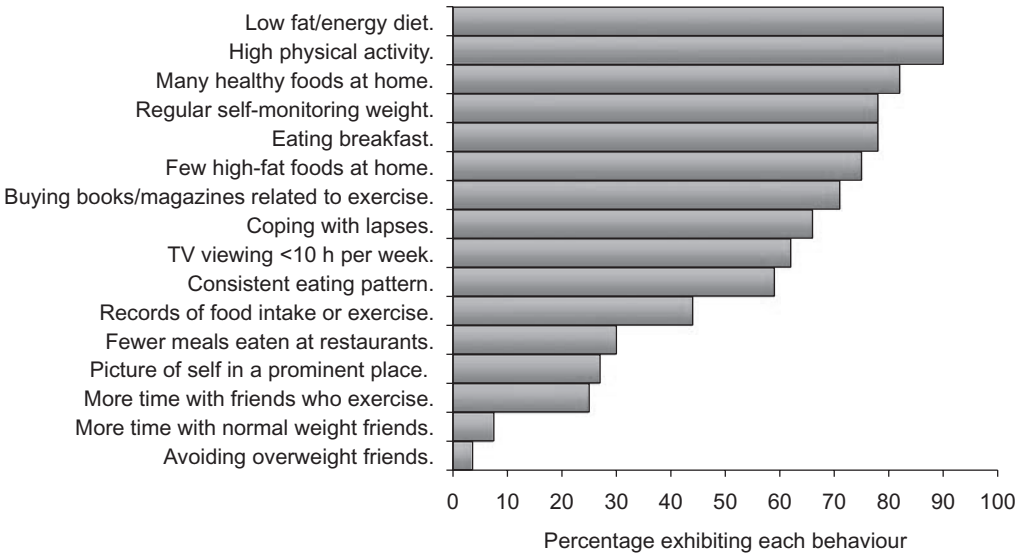

Figure 1 League table of weight control behaviours of successful weight loss maintainers in the National Weight Control Registry. Values are approximate because measures and numbers of registrants vary between publications over the 10 years of the registry's existence.

rank order of specific behaviours practised by registrants. This is approximate because measures and numbers of registrants vary between publications over the 10 years of the registry's existence. It is, however, clear that the behavioural correlates of successful weight loss maintenance in the NWCR are individual and heterogeneous. The range of behaviours exhibited indicates that different people achieve weight loss maintenance in different ways. While many registrants engage in the behaviours depicted, individuals tend to choose a behavioural profile that presumably best meets their own lifestyle needs, to control their weight (14).

Correlates of poor weight maintenance or relapse

It is clear that weight loss is difficult and relapse is frequent. Indeed, it has been robustly argued and observed that obesity, like substance dependence and certain mental health conditions (e.g. bipolar disorder), is a chronic relapsing condition (7). This is supported by numerous trials of behaviour modification, diet, exercise, drugs and surgical approaches to obesity treatment (7). In the NWCR – who are the most successful long-term weight loss maintainers longitudinally studied on earth – 2400 people were followed to scrutinize the issue of relapse. They had lost an average of 32.1 kg and kept it off for 6.5 years. The mean reported weight change from entry into the registry until 2 years later was 3.8 ± 7.6 kg. At year 2, 96.4% of the sample remained at or more than 10% below the maximum lifetime weight. Small regains were common, and few people were able to re-lose weight after any weight regain. Of the participants who gained any weight between baseline and 1 year ($n = 1483$; 65.7%) only 11% returned to baseline weight or below by year 2. Of participants who relapsed, which was defined as weight regain of 5% or more at year 1 ($n = 575$ or 25.5% of the sample), only 4.7% returned to their baseline weight or below by year 2, and only 12.9% re-lost at least half of their year 1 gain by year 2 (116). Thus, even the best of the best weight loss maintainers experience frequent small lapses, from which they find it difficult to recover. The crucial thing about chronic relapsing conditions is that they cannot be cured by a simple medical intervention. They are conditions that you are either born with or have a susceptibility to and they need to be managed over the course of the lifespan.

There is some quite convincing evidence that there is quite a strong physiological basis to weight relapse. Leibel *et al.* have shown in well-designed laboratory studies how maintenance of a reduced body weight lowers energy requirements, which opposes the maintenance of weight loss (and vice versa) (117). They examined the effects of altering body weight on energy expenditure and its components (resting and non-resting energy expenditure, and the thermic effect of feeding) in 18 obese and 23 never-obese subjects. Subjects were studied at their usual body weight, after losing 10 to 20% or gaining 10% of their usual weight by underfeeding or overfeeding, respectively. Maintenance of a body weight 10% or more below normal was associated with a mean (±SD) reduction in total energy expenditure of 25 ± 12.6 kJ kg^{-1} fat-free-mass d^{-1} in the subjects who had never been obese ($P < 0.001$) and 34 ± 21.0 kJ kg^{-1} fat-free-mass d^{-1} in the obese subjects ($P < 0.001$). This is equivalent to ~1.25 MJ d^{-1} and 2.4 MJ d^{-1} assuming a fat-free-mass of 50 kg and 70 kg in

the lean and obese subjects, respectively. Resting and non-resting energy expenditure each decreased 13 to 17 kJ kg^{-1} fat-free-mass d^{-1} in both groups of subjects (~0.65 and 1.2 MJ d^{-1}). Thus, a decrease in weight of 10–20% decreased total energy expenditure by 1.85 MJ d^{-1}. Maintenance of body weight at a level 10% above the usual weight was associated with an increase in total energy expenditure of 38 ± 29 kJ kg^{-1} fat-free-mass d^{-1} (~1.9 MJ d^{-1}) in the subjects who had never been obese ($P < 0.001$) and 33.6 ± 17 kJ kg^{-1} fat-free-mass d^{-1} (~2.4 MJ d^{-1}) in the obese subjects ($P < 0.001$). The thermic effect of feeding and non-resting energy expenditure increased by approximately 4 to 8 and 34 to 38 kJ kg^{-1} fat-free-mass d^{-1}, respectively, after weight gain. Clearly, the greatest change in energy expenditure occurred through non-resting energy expenditure. Furthermore, changes in the hormones, leptin and ghrelin associated with these changes in body size and composition indicated a suite of weight loss-induced physiological changes place a neuroendocrine 'pull' on behaviour and motivation, which opposes further weight loss and may promote relapse (118–122). There is also evidence that administration of low-dose leptin ameliorates the strength of some of these signals (123–125). Thus, it is possible that in the future neuroendocrine predictors of relapse risk may be of value, although they may have greater capacity to predict weight relapse or failure at weight maintenance, than weight loss itself. Throughout this paper, we have touched on a number of areas that are predictive of relapse.

It appears from the above discussion that there is a strong biological basis to weight relapse and its high frequency is uncontroversial. A consideration of the habits of successful weight loss maintainers in the NWCR is highly illuminating in this regard, because the NWCR are largely people who have learned to develop behavioural strategies to avoid or deal with relapse, albeit with difficulty (see above) (22–24,114,115). Even then, relapse is frequent and coping with even small lapses requires considerable effort. It appears that the simplest predictor of relapse is a failure to build on and maintain the patterns of behaviours that led to weight loss in the first place. Thus, in a small study of five men and 35 women, Schlundt *et al.* found that the main categories of high-risk situation for relapse were positive social interactions, negative emotions and physiological craving as well as impulsive eating (126). Similarly, Kayman *et al.*'s analysis of weight maintainers, relapsers and controls found that of relapsers, few (34%) exercised regularly, most ate unconsciously in response to emotions (70%), few used available social support (38%) and few confronted problems directly (10,91). These data along with those of the NWCR illustrate that to avoid relapses, one has to develop skills and coping strategies in the domains of behaviour (e.g. diet and exercise), and emotion (e.g. structure vs. impulsiveness, social support, food as a means to cope with emotional perturbations). Furthermore, it seems these changes need to become practised until they become autonomic, default and habitual. Even then, as illustrated by the discussion of the NWCR above, small lapses do occur and require considerable effort to overcome. Turner *et al.* emphasize the importance of low self-esteem as a trigger for relapse (127). A survey by Cachelin *et al.* of 3394 men and women found that women in particular rated depression, stress, low self-esteem and need to avoid situations as the more important reasons for weight gain, and women were more likely to feel terrible and regain as response to a relapse (128). Similarly, Byrne *et al.* identified certain characteristics associated with weight regain but not maintenance (97). These were failure to achieve weight goals and dissatisfaction with weight achieved, tendency to evaluate worth in terms of weight and shape, a lack of vigilance with regards to weight control, dichotomous thinking and a tendency to use food to regulate mood. In addition to this a large examination of a group of weight controllers ($n = 286$) and subsequently 3345 members of the NWCR, extracted two factors from the disinhibition scale: 'internal' factors such as feeling, thoughts and affect; 'external factors' such as social events and the sight of food. Internal disinhibition significantly predicted weight change at 1 year while external disinhibition did not. It has recently been articulated that feelings of failure, stigma, self-criticism and shame may be key factors underlying relapse in people who have a tendency to relapse, and this is an important area of research for the future (98).

Thus, while the predictive capacity of individual factors that may lead to relapse is not yet well documented, there is emerging a cluster of traits, which when taken on aggregate do indicate a tendency to fail to resist the physiological pull induced by an imposed energy deficit.

Attrition

Attrition is a major problem in most if not all obesity trials. A recent systematic review of 121 articles estimates that the mean dropout rate is ~37% (129). In clinical trails dropout can actually increase in treatment groups, relative to controls, e.g. (129). Attrition is also influenced by initial weight loss (78,130). However, attendance also influences weight loss. The exact relationship is not yet clear. Weight loss begins rapidly and starts to slow as weight loss proceeds. It is clear from a number of other studies in the literature that attendance is a major correlate of weight lost (10,131–134). The exact mechanisms by which attendance translates into weight loss is not clear and may well differ for different people. It may be that those who lose a greater per cent of baseline weight in the first week are more motivated (either before they attend or as a consequence of their experiences in the first week), they may have familiarized

themselves with the treatment programme to a greater extent, are simply on a consistent trajectory of greater weight loss or a combination of these variables.

In other studies, the predictors of attendance are not dissimilar to those for relapse. Again, it is likely that the two are related. It is pertinent to point out here, however, that this is what we capture in studies that have a beginning and an end, arbitrarily set by the investigator. In real life it seems that a significant proportion of the overweight population make numerous attempts at weight control and so perhaps the whole concept of an 'obesity treatment programme' is as much reflective of the intentions of the experimenter or clinician as they are the subject's. Given that obesity is a chronic relapsing condition, we should perhaps evolve models that take account of the more dynamic nature of attempted weight control patterns, both within and between people and over time.

Thus, in a study of 6943 people using Internet weight control interventions, non-usage attrition at 12 weeks was associated with higher age, lower exercise, emotional eating and skipping meals. However, for the 2656 remaining at 52 weeks, eating breakfast and avoidance of sugared beverages were the only pre-treatment predictors of non-usage attrition (135). In another examination by Garaulet *et al*. in 454 participants greater obesity and barriers to weight loss, lower planning of eating behaviour and experience of stress all predicted attrition (136). Elfhag and Rössner's analysis of 247 obese participants of a weight loss programme suggested that lower education, being an immigrant, lack of occupation, fewer previous weight loss experiences and body dissatisfaction were all associated with attrition (130). Fabricatore *et al*. found in 224 obese adults that after adjusting for other variables, younger age and greater baseline depressive symptoms were associated with attrition (137). In an analysis of 24 randomized controlled trails, Fabricatore *et al*. found that gender was related to attrition (higher for women) and that designs with a lead-in period lowered attrition rates (129). Dalle Grave *et al*. found that unrealistic weight loss expectation predicted attrition in 2978 Italians (138), which has implications for the discussion of healthy Nacissism above. Teixeira *et al*. found in 158 overweight and obese women pre-treatment predictors of attrition were previous weight loss attempts, poorer quality of life, more stringent weight outcome evaluations, initial weight, exercise minutes, fibre intake, binge eating, psychological health, body image and lower reported carbohydrate intake at baseline (44). The interrelatedness of many of these predictors is demonstrated by Sherwood *et al*.'s analysis of binge eating status as a predictor of weight loss outcomes in 444 women. They found that binge eating was related to dieting history, weight cycling, depressive symptoms, perceived barriers to weight loss and attrition (139). They also found that assessment of binge status covaried with weight loss and regain. Herein lies another problem of heterogeneity. The above studies cited were as different in design and execution as were many of their outcomes. Nevertheless, there is an emergent pattern. As with predictors of weight loss outcomes, predictors of relapse and attrition are complex, heterogeneous, multifactorial and interrelated.

An illuminating perspective of the complexity of attrition in the treatment of obesity has been given by Grossi *et al*. who conducted structured telephone interviews on 940 obese patients involved in an 18-month treatment programme. They noted that 'After a median observation period of 41 months (range, 25–50), 766 of 940 patients (81.5%) discontinued treatment. Sixty-two per cent of total dropout occurred in the first year of follow-up. Seventy-four per cent of dropouts reported a single primary reason for treatment interruption. Two primary reasons were reported by 22.4% of patients, and three reasons by 3.4%. Practical difficulties, alone or in combination, were reported by more than half of dropouts (55%), and were the leading cause of attrition followed by perceived failure of treatment. Among practical difficulties, family problems or problems at work and logistics, coupled with health problems other than obesity, were the most frequent reasons of attrition, but also a perceived sense of abandonment or a bad interaction with therapists were frequently reported' (140). Their conclusion that practical difficulties and psychological problems are the most important reasons of attrition reported by patients seems reasonable.

Environmental correlates of weight loss and weight loss maintenance

Rössner (141) has elegantly outlined how successful weight loss and its maintenance can be readily achieved when the environmental changes are largely out of the control of the people concerned and involuntarily imposed. Within Western society the environmental correlates of weight loss and maintenance are far less clear. Indeed, we are remarkably short of direct, clear empirical measures of the environment that correlate with or predict weight loss or its maintenance. Furthermore, there are a number of agents in the environment who hold much of the information on this topic as confidential proprietary data on the behaviour of consumers – namely the retail sector. They appear far more able to predict our habits than most of us are as individuals. This is a critical issue because we are creatures of habit, and when measurable (as in the case of activity patterns) those habits can be described and predicted as demonstrated in a sample of 100 000 anonymized mobile phone users (142). There are currently some very exciting developments in mobile technology to monitor patterns of human movement and even energy expenditure (143–148). The key point here is that in order to help people change their habits we need more measurement, modelling and prediction of

intra-subject patterns of behaviour. Prediction of intra-subject variables in eating and activity behaviour would assist people in maintaining the new habits associated with weight loss by recognizing relapse signatures and departures from their new 'healthy' habits.

When predictive/correlative measures are made and how they relate to weight outcomes

Table 2 shows the dynamic phases of behaviour change people progress through to successfully lose weight and maintain the loss. The table can readily be reconfigured for unsuccessful weight loss by substituting negative outcomes at various points during the treatment and post-treatment phase. The table is a product of the above discussion and its salient features help explain why so few key predictors explain much of the variability in weight loss outcomes that can be generalized to the whole population.

Pre-treatment predictors of weight loss are baseline measures. Little has yet changed and if it is eating and activity behaviour that brings about changes in energy balance, or indeed if these changes are attended by alterations in attitudes, affect and psychopathology, we should not expect baseline measures to strongly predict outcomes. This is unless certain traits are consistently associated with specific outcomes. Even then, the traits themselves are numerous and so should not be expected to predict much of the variance in outcomes.

During treatment, it is increasingly clear that different people change various traits to differing degrees over time, and so the correlates of successful weight loss are complex, heterogeneous and dispersed over time. While this is the point at which most change actually occurs, changes are not uniform across individuals or time, making causal relations difficult to establish. Furthermore, during this phase many people experiment with different behavioural solutions – they chop and change between options until they find pathways of behaviour change that best suit their lifestyles. Under these conditions variability in both predictors and outcomes is inconsistent and high.

In the post weight loss or weight maintenance phase there is evidence that successful weight loss maintainers settle into a new profile of behaviours, attitudes and psychological profiles that essentially keep them in a state of constant vigilance (14,26). Many, however, relapse and either regain weight or revert to strategies of attempted weight loss. Under these conditions variability in both correlates and outcomes is inconsistent and high for relapsers but less so for maintainers. However, for weight maintainers, there is enormous heterogeneity in the specific degrees

Table 2 The time windows in which measures are made in relation to the dynamic changes in psychological traits and behaviour during the pre-treatment, treatment and post-treatment phases of a weight loss journey. The table indicates changes associated with success but could readily be reconfigured to illustrate points of lapse and relapse

	Pre-treatment	Weight loss	Maintenance
Physiology: comorbidity	Baseline, elevated	Comorbidities improve	Improves motivation?
Physiology: energy balance mechanisms	Chronic positive energy balance	Compensatory mechanisms (e.g. increased appetite, decreased resting metabolic rate)	Compensation is counteracted by behaviour
Personality	Baseline	Stable	Stable
Psychopathology	Baseline indications but measurement tools may be insensitive	Improves	Improves
Coping strategy	Baseline poor	Develops	Maintains
Social support	Baseline often poor	Develops	Maintains
Self-monitoring	Baseline undeveloped	Develops	Maintain & refine
Diet	Baseline, indicative of risk for weight gain	Changes and improves	Maintain as new habits
Exercise	Baseline, sedentary, little or no exercise	May not change initially	Increase and maintain new habits
Locus of control	Baseline, often but not always external	Greater internalization	Greater internalization and autonomy
Dietary restraint	Baseline, often but not always lower	More flexible	Restrained but flexible
Body image	Baseline, often but not always negative	Improves	Improves but self-monitored
Self-compassion	Baseline, often but not always low	Improves	Greater and maintained
Self-esteem	Baseline, often but not always low	Improves	Greater and maintained
Social support	Baseline, often but not always low	Develops	Improved and maintained
Quality of life	Baseline, sometimes but not always low	Improvements may be gradual	Greater and maintained
Attitudes to obesity	Ambivalent	Develops	Vigilant
Binge eating	May be under-represented	May improve	Decreased frequency

to which individuals exhibit specific profiles of behaviour change and psychological profiles. There is also variability in the extent to which they relapse, at least in the large sample of the NWCR (14). While a range of behaviours and psychological traits change significantly in weight loss maintainers, as a consequence of weight loss, few specific traits explain much of the variance for the whole population; intra-individual variability is too great. It is therefore to be expected that models do not explain a large proportion of the variance in weight loss outcomes, because they are population models, which attempt to elucidate common pathways for groups of people. In reality those groups of people appear to be exhibiting a range of behavioural and psychological changes but to differing degrees. Hence, we need more process models, which acknowledge that for any one individual, obesity is like headaches or depression; it is an end state arising from multiple interacting processes. It is the fine grain research at the level of individual variations that will help us to understand and design better interventions. So for some, obesity might be linked to poor knowledge of diet or poverty, to difficult emotions or history of childhood trauma, to poor impulsive control, to alcohol use when eating at night, to a work style of business lunches and dinners. Factors that are linked to vulnerability might be different to those that are linked to triggering onset, and vary from those that make engagement, help seeking or 'treatment' difficult, which vary from those that help recovery, and which vary from those that maintain recovery. As a simple example, a problematic relationship and divorce might trigger emotional difficulties leading to overeating, loneliness may maintain that pattern, but meeting a new partner or gaining a social support through a slimming group, or gaining a new job might enhance self-esteem, improve motivation and impact positively on diet.

To date the majority of measures have been made as pre-treatment predictors of weight loss outcomes, e.g. (25) or post-treatment correlates of weigh loss maintenance (14). As discussed throughout this paper, these measures are highly variable, in different sub-populations of the obesity spectrum and studies use sample sizes that range from many thousands to a small handful. Few studies of sufficient size, scale or complexity track people through the dynamic, relapsing process of weight change. Even if they did it is likely that predictive or correlative models would not account for much of the variance, for the reasons discussed above.

Summary and recommendations

At the present time, the pre-treatment predictors of weight loss and its maintenance are relatively few, the evidence is that predictors are heterogeneous and predictive models are weak. Most models and indeed constructs explain no more than 25–30% of the variance in weight that is lost (25). The same is true for the maintenance of weight loss. Many of the factors that might seem intuitively good pre-treatment predictors of weight loss (self-esteem, motivation, binge eating, dietary behaviour and exercise) do not turn out to be so. However, many of these factors are important correlates of success, although the amount of variance they explain is either small or highly variable between different groups. It is likely that combinations of these types of process are important. Other reasons for poor prediction are populations, treatments, constructs and measures of constructs; these are all highly heterogeneous, usually in small groups of subjects studied. Consequently, the evidence base in these areas is limited. It is reasonably certain that it is not going to be possible to use the information we currently have to match the needs of individuals with the characteristics of treatment programmes. We need to develop new research programmes to reach the goal of matching treatments to patients' needs to improve success at weight loss maintenance. We also need to recognize that people will need different things at different points in their treatment cycle. What might help them in a chronic or acute state may be quite different from what is helpful in a maintenance stage. Helping people to be more attentive and mindful of their relapse signatures and having specific coping behaviours for those relapse signatures – as is increasingly important in mental health – is likely to be important for this population too. But that requires a highly individualized analysis.

It may be more valuable to develop flexible weight loss programmes that allow people to tailor them to their own lifestyle rather than trying to crowbar different 'types' of people into discrete types of treatment. Indeed, given that most behaviour constructs are dimensional, they are better described as continua rather than categorical. If this is indeed the case, the idea of matching type of subject to type of treatment in a categorical way is likely to fail. Instead, we should be trying harder to measure and predict patterns in individual behaviours that are associated with weight stability and those that are at risk for weight gain. We should use this information to help people shape behaviour change solutions to their own lifestyle needs and monitor lapse and relapse signatures when they start to drift back towards weight regain. It may be that for some specific measures (e.g. diet, exercise, self-efficacy) people do cluster, but that variability between specific measures will be very heterogeneous. Hence, types of people do not match to treatments, but modular treatments can be adapted to meet the needs of specific behaviour patterns.

We also need to think if we are targeting the right psychological processes. For example, there is a lot of evidence now that shame, self-criticism and experiences of

stigma have major impacts on people's mental health and abilities to cope (98). Self-criticism in particular is highly linked to emotional difficulties (149). For the most part, these have not been systematically studied in obese populations (although we are now beginning such studies). Nor has targeting them as treatment interventions been explored.

It is worth bearing in mind that the various psychological constructs discussed in this paper often overlap and may be simply glimpsing the same phenomenon (ability to control weight) by viewing it from different perspectives. This phenomenon can be called the ability to navigate to a healthier body weight and remain at that point. In order to do so one needs to alter behaviour enough to alter energy balance. Having done so, the person then needs to maintain a new pattern of behaviour until it becomes habitual, and resist the pull of physiological, psychological, lifestyle, economic and environmental factors that draw them back from whence they came. So strong are these factors that without constant self-monitoring, weight regain for many is almost inevitable. Thus, obesity is a chronic relapsing condition that requires lifelong management in the same way that addictions, affective disorders and some metabolic disorders require constant self-monitoring and strategies for coping with lapses, to avoid total relapse.

We need to develop models that account for complexity and heterogeneity. In order to achieve this it will be necessary to modify protocols and research designs to overcome some of the problems of previous attempts as suggested by Teixeira *et al*. (25). This includes using larger sample sizes and different populations, including cross-cultural studies; standardized definitions of constructs, predictors, success, lapse and attrition; the use of multiple and where possible, longitudinal measures of psychological and behavioural predictors/correlates of weight change, including intake and expenditure behaviours and frequent longitudinal measures of weight; more robust approaches to statistical modelling and analysis incorporating the statistician at the outset of studies; a greater focus on intra-individual variability in behavioural pathways to and from body weight gain; and an appreciation of what characterizes the behaviours of successful weight maintainers.

Conflict of Interest Statement

No conflict of interest was declared.

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References

1. Royal College of Physicians. *Storing up Problems – The Medical Case for A Slimmer Nation: Report of A Working Party*. Royal College of Physicians: London, 2004.
2. Zaninotto P, Wardle H, Stamatakis E, Mindell J, Head J. *Forecasting Obesity to 2010*. Department of Health: London, 2006.
3. Finucane MM, Stevens GA, Cowan MJ, Danaei G, Lin JK, Paciorek CJ, Singh GM, Gutierrez HR, Lu Y, Bahalim AN, Farzadfar F, Riley LM, Ezzati M. National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9·1 million participants. *Lancet* 2011; **377**: 578–586.
4. World Health Organization. *Obesity: Preventing and Managing the Global Epidemic*. World Health Organization: Geneva, 2000.
5. Foster GD. Reasonable weights: determinants, definitions and directions. In: Allison DB, Pi-Sunyer FX (eds). *Obesity Treatment: Establishing Goals, Improving Outcomes, and Reviewing the Research Agenda*. Plenum Publishing Corporation: New York, 1995, pp. 35–44.
6. Muller MJ, Bosy-Westphal A, Heymsfield SB. Is there evidence for a set point that regulates human body weight? *F1000 Med Rep* 2010; **2**: 59.
7. Brownell KD, Marlatt GA, Lichtenstein E, Wilson GT. Understanding and preventing relapse. *Am Psychol* 1986; **41**: 765–782.
8. Heymsfield SB. What is the weight required for substantial health gains? In: Pi-Sunyer FX, Allison DB (eds). *Obesity Treatment: Establishing Goals, Improving Outcomes, and Reviewing the Research Agenda*. Plenum Publishing Corporation: New York, 1995, pp. 21–27.
9. National Institute for Health and Clinical Excellence. *Obesity: Guidance on the Prevention, Identification, Assessment and Management of Overweight and Obesity in Adults and Children*. Department of Health, UK: London, 2006.
10. Institute of Medicine. *Weighing the Options: Criteria for Evaluating Weight Management Programmes*. National Academy Press: Washington DC, 1995.
11. Kant AK, Schatzkin A, Graubard BI, Ballard Barbash R. Frequency of eating occasions and weight change in the NHANES I Epidemiologic Follow-up Study. *Int J Obes* 1995; **19**: 468–474.
12. Shah M, Hannan PJ, Jeffery RW. Secular trend in body mass index in the adult population of three communities from the upper mid-western part of the USA: the Minnesota Heart Health Program. *Int J Obes* 1991; **15**: 499–503.
13. Rössner S. Factors determining the long term outcome of obesity treatment. In: Bjorntorp P, Brodoff BD (eds). *Obesity*. JB Lippincott Company: Philadelphia, PA, 1992, pp. 712–719.
14. Wing RR, Phelan S. Long-term weight loss maintenance. *Am J Clin Nutr* 2005; **82**(Suppl.): 222S–225S.
15. Brownell KD, Rodin J. The dieting maelstrom. Is it possible and advisable to lose weight? *Am Psychol* 1994; **49**: 781–791.
16. Bruce B, Agras WS. Binge eating in females: a population-based investigation. *Int J Eating Disorders* 1992; **12**: 365–373.
17. Spitzer RL, Devlin MJ, Walsh BT, Hasin D, Wing R, Marcus M, Stunkard A, Wadden T, Yanovsky S, Agras S, Mirchell J, Nonas C. Binge eating disorder: a multisite field trial of the diagnostic criteria. *Int J Eat Disord* 1992; **11**: 191–204.
18. Brownell KD. Whether obesity should be treated. *Health Psychol* 1993; **12**: 339–341.
19. Jeffery RW, Adlis SA, Forster JL. Prevalence of dieting among working men and women: the healthy worker project. *Health Psychol* 1991; **10**: 274–281.

20. Jeffery RW, Bjornson-Benson WM, Rosenthal BS, Lindquist RA, Kurth CL, Johnson SL. Correlates of weight loss and its maintenance over two years of follow-up among middle-aged men. *Prev Med* 1984; **13**: 155–168.
21. Brownell KD, Wadden TA. The heterogeneity of obesity: fitting treatments to individuals. *Behav Ther* 1991; **22**: 153–177.
22. Klem ML, Wing RR, McGuire MT, Seagle HM, Hill JO. A descriptive study of individuals successful at long-term maintenance of substantial weight loss. *Am J Clin Nutr* 1997; **66**: 239–246.
23. McGuire MT, Wing RR, Klem ML, Seagle HM, Hill JO. Long-term maintenance of weight loss: do people who lose weight through various weight loss methods use different behaviors to maintain their weight? *Int J Obes Relat Metab Disord* 1998; **22**: 572–577.
24. Wyatt HR, Grunwald GK, Mosca CL, Klem ML, Wing RR, Hill JO. Long-term weight loss and breakfast in subjects in the National Weight Control Registry. *Obes Res* 2002; **10**: 78–82.
25. Teixeira PJ, Going SB, Sardinha LB, Lohman TG. A review of psychosocial pre-treatment predictors of weight control. *Obes Rev* 2005; **6**: 43–65.
26. Elfhag K, Rössner S. Who succeeds in maintaining weight loss? A conceptual review of factors associated with weight loss maintenance and weight regain. *Obes Rev* 2005; **6**: 67–85.
27. Allison DB, Engel CN. Predicting treatment outcome: why have we been so unsuccessful? In: Pi-Sunyer FX, Allison DB (eds). *Obesity Treatment: Establishing Goals, Improving Outcomes, and Reviewing the Research Agenda*. Plenum Publishing Corporation: New York, 1995, pp. 191–198.
28. Rosenthal R. *Meta-Analatic Procedures for Social Research*. Sage: Newbury Park, CA, 1991.
29. Stubbs RJ. Peripheral signals affecting food intake. *Nutrition* 1990; **15**: 614–625.
30. Rankinen T, Zuberi A, Chagnon YC, Weisnagel SJ, Argyropoulos G, Walts B, Perusse L, Bouchard C. The human obesity gene map: the 2005 update. *Obesity* 2006; **14**: 529–644.
31. Clement K. Genetics of human obesity. *C R Biol* 2006; **329**: 608–622.
32. Loos RJ, Bouchard C. FTO: the first gene contributing to common forms of human obesity. *Obes Rev* 2008; **9**: 246–250.
33. Mutch DM, Clement K. Genetics of human obesity. *Best Pract Res Clin Endocrinol Metab* 2006; **20**: 647–664.
34. Lubrano-Berthelier C, Clement K. [Genetics of human obesity]. *Rev Med Interne* 2005; **26**: 802–811.
35. Hainer V, Hlavata K, Gojova M, Kunesova M, Wagenknecht M, Kopsky V, Parizkova J, Hill M, Nedvidkova J. Hormonal and psychobehavioral predictors of weight loss in response to a short-term weight reduction program in obese women. *Physiol Res* 2008; **57**(Suppl. 1): S17–S27.
36. Janghorbani M, Amini M. Patterns and predictors of long-term weight change in patients with type 2 diabetes mellitus. *Ann Nutr Metab* 2009; **54**: 111–118.
37. le Roux CW, Aylwin SJ, Batterham RL, Borg CM, Coyle F, Prasad V, Shurey S, Ghatei MA, Patel AG, Bloom SR. Gut hormone profiles following bariatric surgery favor an anorectic state, facilitate weight loss, and improve metabolic parameters. *Ann Surg* 2006; **243**: 108–114.
38. le Roux CW, Welbourn R, Werling M, Osborne A, Kokkinos A, Laurenius A, Lönroth H, Fändriks L, Ghatei MA, Bloom SR, Olbers T. Gut hormones as mediators of appetite and weight loss after Roux-en-Y gastric bypass. *Ann Surg* 2007; **246**: 780–785. 10.1097/SLA.0b013e3180caa3e3.
39. Weyer C. Hormones in concert. *The Scientist* 2009; **23**: 34.
40. Vogels N, Diepvens K, Westerterp-Plantenga MS. Predictors of long-term weight maintenance. *Obes Res* 2005; **13**: 2162–2168.
41. Hansen DL, Astrup A, Toubro S, Finer N, Kopelman P, Hilsted J, Rössner S, Saris WHM, Van Gaal LF, James WPT, Goulder M. Predictors of weight loss and maintenance during 2 years of treatment by sibutramine in obesity. Results from the European multi-centre STORM trial. *Int J Obes* 2001; **25**: 496–501.
42. Wilson GT. Behavioural and psychological predictors of treatment outcome in obesity. In: Pi-Sunyer FX, Allison DB (eds). *Obesity Treatment: Establishing Goals, Improving Outcomes, and Reviewing the Research Agenda*. Plenum Publishing Corporation: New York, 1995, pp. 183–190.
43. Handjieva-Darlenska T, Handjiev S, Larsen TM, van Baak MA, Jebb S, Papadaki A, Pfeiffer AFH, Martinez JA, Kunesova M, Holst C, Saris WHM, Astrup A. Initial weight loss on an 800-kcal diet as a predictor of weight loss success after 8 weeks: the Diogenes study. *Eur J Clin Nutr* 2010; **64**: 994–999.
44. Teixeira PJ, Going SB, Houtkooper LB, Cussler EC, Metcalfe LL, Blew RM, Sardinha LB, Lohman TG. Pretreatment predictors of attrition and successful weight management in women. *Int J Obes* 2004; **28**: 1124–1133.
45. Lawrence MF, Cole TJ, Coward WA, Singh J, Whitehead RG. Seasonal pattern of activity and its nutritional consequences in Gambia. In: Sahn DE (ed.). *Seasonal Variability in Third World Agriculture: The Consequences for Security*. Johns Hopkins University Press: Baltimore, MD, 1989, pp. 47–56.
46. Yanovski JA, Yanovski SZ, Sovik KN, Nguyen TT, O'Neil PM, Sebring NG. A prospective study of holiday weight gain. *N Engl J Med* 2000; **342**: 861–867.
47. Prochaska JO, Norcross JC, Fowler JL, Follick MJ, Abrams DB. Attendance and outcome in a work site weight control program: processes and stages of change as process and predictor variables. *Addict Behav* 1992; **17**: 35–45.
48. Westenhoefer J. Dietary restraint and disinhibition: is restraint a homogeneous construct? *Appetite* 1991; **16**: 45–55.
49. Westenhoefer J, Pudel V, Maus N. Some restrictions on dietary restraint. *Appetite* 1990; **14**: 137–141.
50. Mullen FA. The Minnesota multiphasic personality inventory; an extension of the Davis scoring method. *J Clin Psychol* 1948; **4**: 86–88.
51. Johnson SF, Swenson WM, Gastineau CF. Personality characteristics in obesity: relation of MMPI profile and age of onset of obesity to success in weight reduction. *Am J Clin Nutr* 1976; **29**: 626–632.
52. Elfhag K, Rossner S, Carlsson AM, Barkeling B. Sibutramine treatment in obesity: predictors of weight loss including rorschach personality data. *Obesity* 2003; **11**: 1391–1399.
53. Elfhag K, Rössner S, Lindgren T, Andersson I, Carlsson AM. Rorschach personality predictors of weight loss with behavior modification in obesity treatment. *J Pers Asses* 2004; **83**: 293–305.
54. Gladis MM, Wadden TA, Vogt R, Foster G, Kuehnel RH, Bartlett SJ. Behavioral treatment of obese binge eaters: do they need different care? *J Psychosom Res* 1998; **44**: 375–384.
55. Rapoport L, Clark M, Wardle J. Evaluation of a modified cognitive-behavioural programme for weight management. *Int J Obes Relat Metab Disord* 2000; **24**: 1756–1737.
56. Cuntz U, Leibbrand R, Ehrig C, Shaw R, Fichter MM. Predictors of post-treatment weight reduction after in-patient behavioral therapy. *Int J Obes* 2001; **25**: S99–S101.
57. Wadden TA, Vogt RA, Andersen RE, Bartlett SJ, Foster GD, Kuehnel RH, Wilk J, Weinstock R, Buckenmeyer P, Berkowitz RI, Steen SN. Exercise in the treatment of obesity: effects of four

interventions on body composition, resting energy expenditure, appetite, and mood. *J Consult Clin Psychol* 1997; **65**: 269–277.
58. Bandura A. Self-efficacy: toward a unifying theory of behavioral change. *Psychol Rev* 1977; **84**: 191–215.
59. Bernier M, Avard J. Self-efficacy, outcome, and attrition in a weight-reduction program. *Cognit Ther Res* 1986; **10**: 319–338.
60. National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults – the evidence report. national institutes of health. *Obes Res* 1998; **6**: 51S–209S.
61. Gilbert P. *The Compassionate Mind*. Constable and Robinson: London, 2009.
62. Foster GD, Wadden TA, Vogt RA, Brewer G. What is a reasonable weight loss? Patients' expectations and evaluations of obesity treatment outcomes. *J Consult Clin Psychol* 1997; **65**: 79–85.
63. Taylor SE, Kemeny ME, Reed GM, Bower JE, Gruenewald TL. Psychological resources, positive illusions, and health. *Am Psychol* 2000; **55**: 99–109.
64. Blundell JE, Stubbs RJ. Diet composition and the control of food intake in humans. In: Bray GE, Bouchard C, James WPT (eds). *Handbook of Obesity*. Marcel Dekker: New York, 1998, pp. 243–272.
65. Stubbs RJ. Appetite, feeding behaviour and energy balance in human subjects. *Proc Nutr Soc* 1998; **57**: 341–356.
66. Stubbs J, Ferres S, Horgan G. Energy density of foods: effects on energy intake. *Crit Rev Food Sci Nutr* 2000; **40**: 481–515.
67. Wing RR. Physical activity in the treatment of the adulthood overweight and obesity: current evidence and research issues. *Med Sci Sports Exerc* 1999; **31**: S547–SS52.
68. Bertram SR, Evnter I, Stewart RI. Weight loss in obese women – exercise vs. dietary education. *S Afr Med J* 1990; **78**: 15–18.
69. Blonk MC, Jacobs MA, Biesheuvel EH, Weeda-Mannak WL, Heine RJ. Influences on weight loss in type 2 diabetic patients: little long-term benefit from group behaviour therapy and exercise training. *Diabet Med* 1994; **11**: 449–457.
70. Marks BL, Ward A, Morris DH, Castellani J, Rippe JM. Fat-free mass is maintained in women following a moderate diet and exercise program. *Med Sci Sports Exerc* 1995; **27**: 1243–1251.
71. Ross R, Pedwell H, Rissanen J. Effects of energy restriction and exercise on skeletal muscle and adipose tissue in women as measured by magnetic resonance imaging. *Am J Clin Nutr* 1995; **61**: 1179–1185.
72. Ross R, Rissanen J, Pedwell H, Clifford J, Shragge P. Influence of diet and exercise on skeletal muscle and visceral adipose tissue in men. *J Appl Physiol* 1996; **81**: 2445–2455.
73. Sweeney ME, Hill JO, Heller PA, Baney R, DiGirolamo M. Severe vs moderate energy restriction with and without exercise in the treatment of obesity: efficiency of weight loss. *Am J Clin Nutr* 1993; **57**: 127–134.
74. Friedman MA, Brownell KD. Psychological correlates of obesity: moving to the next research generation. *Psychol Bull* 1995; **117**: 3–20.
75. Foreyt JP, Goodrick GK. Prediction in weight management outcome: implcations for practice. In: Pi-Sunyer FX, Allison DB (eds). *Obesity Treatment: Establishing Goals, Improving Outcomes, and Reviewing the Research Agenda*. Plenum Publishing Corporation: New York, 1995, pp. 199–208.
76. Heshka S, Anderson JW, Atkinson RL, Greenway FL, Hill JO, Phinney SD, Kolotkin RL, Miller-Kovach K, Pi-Sunyer FX. Weight loss with self-help compared with a structured commercial program: a randomized trial. *JAMA* 2003; **289**: 1792–1798.
77. Wadden TA, Letizia KA. Predictors of attrition and weight loss in patients treated by moderate and severe caloric restriction. In: Wadden TA, Van Itallie TB (eds). *Treatment of the Seriously Obese Patient*. The Guilford Press: New York, 1992, pp. 383–410.
78. Stubbs RJ, Pallister C, Whybrow S, Avery A, Lavin JH. Weight outcomes for 34,271 participants in a commercial/primary care weight management partnership scheme. *Obes Facts* 2011; in press.
79. Lichtman SW, Pisarska K, Berman ER, Pestone M, Dowling H, Offenbacher E, Weisel H, Heshka S, Matthews DE. Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. *N Engl J Med* 1992; **327**: 1893–1898.
80. Humphreys K, Ribisl KM. The case for a partnership with self-help groups. *Public Health Reports* 1999; **114**: 322–329.
81. Williams GC, Grow VM, Freedman ZR, Ryan RM, Deci EL. Motivational predictors of weight loss and weight-loss maintenance. *J Pers Soc Psychol* 1996; **70**: 115–126.
82. Jönsson B, Björvell H, Levander S, Rössner S. Personality traits predicting weight loss outcome in obese patients. *Acta Psychiatr Scand* 1986; **74**: 384–387.
83. Rydén O, Hedenbro JL, Frederiksen SG. Weight loss after vertical banded gastroplasty can be predicted: a prospective psychological study. *Obes Surg* 1996; **6**: 237–243.
84. Poston WS, Ericsson M, Linder J, Nilsson T, Goodrick GK, Foreyt JP. Personality and the prediction of weight loss and relapse in the treatment of obesity. *Int J Eat Disord* 1999; **25**: 301–309.
85. Guntrip H. *Schizoid Phenomena, Object Relations, and the Self*. International Universities Press: New York, 1969.
86. Karlsson J, Hallgren P, Kral J, Lindroos AK, Sjostrom L, Sullivan M. Predictors and effects of long-term dieting on mental well-being and weight-loss in obese women. *Appetite* 1994; **23**: 15–26.
87. Barrash J, Rodriguez EM, Scott DH, Mason EE, Sines JO. The utility of MMPI subtypes for the prediction of weight loss after bariatric surgery. Minnesota Multiphasic Personality Inventory. *Int J Obes* 1987; **11**: 115–128.
88. Rowe JL, Downey JE, Faust M, Horn MJ. Psychological and demographic predictors of successful weight loss following silastic ring vertical stapled gastroplasty. *Psychol Rep* 2000; **86**: 1028–1036.
89. Averbukh Y, Heshka S, El-Shoreya H, Flancbaum L, Geliebter A, Kamel S, Pi-Sunyer F, Laferrere B. Depression score predicts weight loss following Roux-en-Y gastric bypass. *Obes Surg* 2003; **13**: 833–836.
90. Dubovsky SL, Haddenhorst A, Murphy J, Liechty RD, Coyle DA. A preliminary study of the relationship between preoperative depression and weight loss following surgery for morbid obesity. *Int J Psychiatry Med* 1985; **15**: 185–196.
91. Kayman S, Bruvold W, Stern JS. Maintenance and relapse after weight loss in women: behavioral aspects. *Am J Clin Nutr* 1990; **52**: 800–807.
92. Gormally J, Rardin D. Weight loss and maintenance and changes in diet and exercise for behavioral counseling and nutrition education. *J Cous Psychol* 1981; **28**: 295–304.
93. Forster JL, Jeffery RW. Gender differences related to weight history, eating patterns, efficacy expectations, self-esteem, and weight loss among participants in a weight reduction program. *Addict Behav* 1986; **11**: 141–147.
94. Glynn SM, Ruderman AJ. The development and validation of an Eating Self-Efficacy Scale. *Cognit Ther Res* 1986; **10**: 403–420.
95. Dennis KE, Goldbery AP. Weight control self-efficacy types and transitions affect weight-loss outcomes in obese women. *Addict Behav* 1996; **21**: 103–116.

96. De Pue JD, Clark MM, Ruggiero L, Medeiros ML, Pera VJ. Maintenance of weight loss: a needs assessment. *Obes Res* 1995; **3**: 241–248.
97. Byrne S, Cooper Z, Fairburn C. Weight maintenance and relapse in obesity: a qualitative study. *Int J Obes* 2003; **27**: 955–962.
98. Gilbert P, Miles JM. *Body Shame; Conceptualistion Research and Treatment*. Routledge: London, 2002.
99. Hayden MJ, Dixon ME, Dixon JB, Playfair J, O'Brien PE. Perceived discrimination and stigmatisation against severely obese women: age and weight loss make a difference. *Obes Facts* 2010; **3**: 7–14.
100. Blaine BE, Rodman J, Newman JM. Weight loss treatment and psychological well-being. *J Health Psychol* 2007; **12**: 66–82.
101. Schoeller DA. But how much physical activity? *Am J Clin Nutr* 2003; **78**: 669–670.
102. Saris WHM, Blair SN, van Baak MA, Eaton SB, Davies PSW, Di Pietro L, Fogelholm M, Rissanen A, Schoeller D, Swinburn B, Tremblay A, Westerterp KR, Wyatt H. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. *Obes Rev* 2003; **4**: 101–114.
103. Jakicic JM, Marcus BH, Lang W, Janney C. Effect of exercise on 24-month weight loss maintenance in overweight women. *Arch Intern Med* 2008; **168**: 1550–1559.
104. Donnelly JE, Blair SN, Jakicic JM, Manore MM, Rankin JW, Smith BK, American College of Sports Medicine Position Stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Med Sci Sports Exerc* 2009; **41**: 459–471.
105. Physical Activity Guidelines Advisory Committee. *Physical Activity Guidelines Advisory Committee Report, 2008*. U.S. Department of Health and Human Services: Washington DC, 2008.
106. Teixeira PJ, Silva MN, Coutinho SR, Palmeira AL, Mata J, Vieira PN, Carraca EV, Santos TC, Sardinha LB. Mediators of weight loss and weight loss maintenance in middle-aged women. *Obesity* 2010; **18**: 725–735.
107. Kolotkin RL, Crosby RD, Williams GR. Health-related quality of life varies among obese subgroups. *Obes Res* 2008; **10**: 748–756.
108. Wadden TA, Bartlett SJ. Very low calorie diets and overview and appraisal. In: Wadden TA, Van Itallie TB (eds). *Treatment of the Seriously Obese Patient*. The Guilford Press: New York, 1992, pp. 44–79.
109. Perri MG, Nezu AM, Viegener BJ. *Improving the Long-Term Management of Obesity: Theory, Research, and Clinical Guidelines*. Wiley: New York, 1992.
110. Glenny A-M, O'Meara S, Melville A, Sheldon TA, Wilson C. The treatment and prevention of obesity: a systematic review of the literature. *Int J Obes* 1997; **21**: 715–737.
111. Foreyt JP, Goodrick GK. Factors common to successful therapy for the obese patient. *Med Sci Sports Exerc* 1991; **23**: 292–297.
112. Bray GA. Obesity and the heart. *Mod Concepts Cardiovasc Dis* 1987; **56**: 67–71.
113. Dance KA, Neufeld RWJ. Aptitude-treatment interaction research in the clinical setting: a review of attempts to dispel the patient uniformity myth. *Psychol Bull* 1988; **104**: 192–213.
114. Phelan S, Wyatt HR, Hill JO, Wing RR. Are the eating and exercise habits of successful weight losers changing? *Obesity* 2006; **14**: 710–716.
115. Wing RR, Hill JO. Successful weight loss maintenance. *Annu Rev Nutr* 2001; **21**: 323–341.
116. Phelan S, Hill JO, Lang W, Dibello JR, Wing RR. Recovery from relapse among successful weight maintainers. *Am J Clin Nutr* 2003; **78**: 1079–1084.
117. Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. *N Engl J Med* 1995; **332**: 621–628.
118. Crujeiras AB, Goyenechea E, Abete I, Lage M, Carreira MC, Martinez JA *et al*. Weight regain after a diet-induced loss is predicted by higher baseline leptin and lower ghrelin plasma levels. *J Clin Endocrinol Metab* 2010; **95**: 5037–5044.
119. Pardina E, Lopez-Tejero MD, Llamas R, Catalan R, Galard R, Allende H *et al*. Ghrelin and apolipoprotein AIV levels show opposite trends to leptin levels during weight loss in morbidly obese patients. *Obes Surg* 2009; **19**: 1414–1423.
120. Maestu J, Jurimae J, Valter I, Jurimae T. Increases in ghrelin and decreases in leptin without altering adiponectin during extreme weight loss in male competitive bodybuilders. *Metabolism* 2008; **57**: 221–225.
121. Kotidis EV, Koliakos GG, Baltzopoulos VG, Ioannidis KN, Yovos JG, Papavramidis ST. Serum ghrelin, leptin and adiponectin levels before and after weight loss: comparison of three methods of treatment – a prospective study. *Obes Surg* 2006; **16**: 1425–1432.
122. Weigle DS, Cummings DE, Newby PD, Breen PA, Frayo RS, Matthys CC *et al*. Roles of leptin and ghrelin in the loss of body weight caused by a low fat, high carbohydrate diet. *J Clin Endocrinol Metab* 2003; **88**: 1577–1586.
123. Rosenbaum M, Murphy EM, Heymsfield SB, Matthews DE, Leibel RL. Low dose leptin administration reverses effects of sustained weight-reduction on energy expenditure and circulating concentrations of thyroid hormones. *J Clin Endocrinol Metab* 2002; **87**: 2391–2394.
124. Rosenbaum M, Goldsmith R, Bloomfield D, Magnano A, Weimer L, Heymsfield S, Gallagher D, Mayer L, Murphy E, Leibel RL. Low-dose leptin reverses skeletal muscle, autonomic, and neuroendocrine adaptations to maintenance of reduced weight. *J Clin Invest* 2005; **115**: 3579–3586.
125. Rosenbaum M, Sy M, Pavlovich K, Leibel RL, Hirsch J. Leptin reverses weight loss–induced changes in regional neural activity responses to visual food stimuli. *J Clin Invest* 2008; **118**: 2583–2591.
126. Schlundt DG, Sbrocco T, Bell C. Identification of high-risk situations in a behavioral weight loss program: application of the relapse prevention model. *Int J Obes* 1989; **13**: 223–234.
127. Turner LW, Wang MQ, Westerfield RC. Preventing relapse in weight control: a discussion of cognitive and behavioral strategies. *Psychol Rep* 1995; **77**: 651–656.
128. Cachelin FM, Striegel-Moore RH, Brownell KD. Beliefs about weight gain and attitudes toward relapse in a sample of women and men with obesity. *Obes Res* 1998; **6**: 231–237.
129. Fabricatore AN, Wadden TA, Moore RH, Butryn ML, Gravallese EA, Erondu NE, Heymsfield SB, Nguyen AM. Attrition from randomized controlled trials of pharmacological weight loss agents: a systematic review and analysis. *Obes Rev* 2009; **10**: 333–341.
130. Elfhag K, Rössner S. Initial weight loss is the best predictor for success in obesity treatment and sociodemographic liabilities increase risk for drop-out. *Patient Educ Couns* 2010; **79**: 361–366.
131. Fontaine KR, Cheskin LJ. Self-efficacy, attendance, and weight loss in obesity treatment. *Addict Behav* 1997; **22**: 567–570.
132. Orth W, Madan A, Taddeucci R, Coday M, Tichansky D. Support group meeting attendance is associated with better weight loss. *Obes Surg* 2008; **18**: 391–394.

133. Hickson M, Macqueen C, Frost G. Evaluation of attendance and weight loss in an intensive weight management clinic compared to standard dietetic care. *J Hum Nutr Diet* 2009; **22**: 72–76.
134. Kaiser KA, Franks SF, Smith AB. Positive relationship between support group attendance and one-year postoperative weight loss in gastric banding patients. *Surg Obes Relat Dis* 2011; **7**: 89–93.
135. Neve MJ, Collins CE, Morgan PJ. Dropout, nonusage attrition, and pretreatment predictors of nonusage attrition in a commercial web-based weight loss program. *J Med Internet Res* 2010; **12**: e69.
136. Garaulet M, Corbalán-Tutau MD, Madrid JA, Baraza JC, Parnell LD, Lee Y-C, Ordovas JM. PERIOD2 variants are associated with abdominal obesity, psycho-behavioral factors, and attrition in the dietary treatment of obesity. *J Am Diet Assoc* 2010; **110**: 917–921.
137. Fabricatore AN, Wadden TA, Moore RH, Butryn ML, Heymsfield SB, Nguyen AM. Predictors of attrition and weight loss success: results from a randomized controlled trial. *Behav Res Ther* 2009; **47**: 685–691.
138. Dalle Grave R, Calugi S, Molinari E, Petroni ML, Bondi M, Compare A, Marchesini G. Weight loss expectations in obese patients and treatment attrition: an observational multicenter study. *Obesity* 2005; **13**: 1961–1969.
139. Sherwood NE, Jeffery RW, Wing RR. Binge status as a predictor of weight loss treatment outcome. *Int J Obes Relat Metab Disord* 1999; **23**: 485–493.
140. Grossi E, Dalle Grave R, Mannucci E, Molinari E, Compare A, Cuzzolaro M, Marchesini G. Complexity of attrition in the treatment of obesity: clues from a structured telephone interview. *Int J Obes* 2006; **30**: 1132–1137.
141. Rössner S. Relapse management. *Int J Obes* 2008; **31**: S10.
142. Gonzalez MC, Hidalgo CA, Barabasi A-L. Understanding individual human mobility patterns. *Nature* 2008; **453**: 779–782.
143. Luinge HJ, Veltink PH. Inclination measurement of human movement using a 3-D accelerometer with autocalibration. *IEEE Trans Biomed Eng* 2004; **12**: 112–121.
144. Godfrey A, Conway R, Meagher D, OLaighin G. Direct measurement of human movement by accelerometry. *Med Eng Phys* 2008; **30**: 1364–1386.
145. Mackey AH, Stott NS, Walt SE. Reliability and validity of an activity monitor (IDEEA) in the determination of temporal-spatial gait parameters in individuals with cerebral palsy. *Gait Posture* 2008; **28**: 634–639.
146. Gorelick ML, Bizzini M, Maffiuletti NA, Munzinger JP, Munzinger U. Test-retest reliability of the IDEEA system in the quantification of step parameters during walking and stair climbing. *Clin Physiol Funct Imaging* 2009; **29**: 271–276.
147. Welk GJ, McClain JJ, Eisenmann JC, Wickel EE. Field validation of the MTI Actigraph and BodyMedia armband monitor using the IDEEA monitor. *Obesity* 2007; **15**: 918–928.
148. Maffiuletti NA, Gorelick M, Kramers-de Quervain I, Bizzini M, Munzinger JP, Tomasetti S, Stacoff A. Concurrent validity and intrasession reliability of the IDEEA accelerometry system for the quantification of spatiotemporal gait parameters. *Gait Posture* 2008; **27**: 160–163.
149. Gilbert P, Clarke M, Hempel S, Miles JN, Irons C. Criticizing and reassuring oneself: an exploration of forms, styles and reasons in female students. *Br J Clin Psychol* 2004; **43**: 31–50.
150. Blair SN. Evidence for success of exercise in weight loss and control. *Ann Intern Med* 1993; **119**(7 Pt 2): 702–706.
151. Foreyt JP, Goodrick GK. Prediction in weight management outcome: implications for practice. In: Allison DB, Pi-Sunyer FX (eds). *Obesity Treatment: Establishing Goals, Improving Outcomes, and Reviewing the Research Agenda*. Plenum Publishing Corporation: New York, 1995, pp. 199–206.
152. O'Neil PM, Jarell MP. Psychological effects of obesity and dieting. In: Wadden TA, VanItallie, TB (eds). *Treatment of the Seriously Obese Patient*. Guildford Press: New York, 1992, pp. 252–270.