

The safety of dieting

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The safety of a reducing diet depends on the composition of the diet, the nutritional status of the recipient, and the time-period for which it is given. Possible dangers of dieting may be classified as nutritional or psycho-social, which will be considered in turn.

NUTRITIONAL DANGERS

The ultimate low-energy diet is total starvation: on this regimen the subject relies entirely on his body stores for nutrition. The magnitude of these stores in an average adult is shown in Table 1. If a rough estimate is made of the daily requirement for a nutrient, and of the proportion of the store of this nutrient which can be lost without severe dysfunction, it is possible to calculate how long it will take for a deficiency of the nutrient to become manifest if input is zero. This calculation is not very reliable, because in states of depletion most nutrients are conserved, so the stores last longer than the calculation suggests. However, it is evident from Table 1 that for nutrients such as water, sodium and potassium the stores last only for a few days, for energy and protein a few months, and for elements such as calcium they would last for several years. This crude calculation agrees well with clinical experience: a person totally deprived of water will die of dehydration in a few days; the number of days is greatly influenced by the ambient temperature and humidity. If given access to water a starving man of normal body composition will die in about 10 weeks, but a severely obese man has survived 315 d of therapeutic starvation. The cause of death is probably something to do with loss of

Table 1. *The approximate magnitude of the body stores of some nutrients in a normal adult, and the time these would last with zero input*

(This calculation does not take into account metabolic conservation which occurs in depleted individuals)

Nutrient	Body contains	Dispensable store	Daily turnover	Time-period stores would last when starving (d)
Water (kg)	42	4	1	4
Sodium (mmol)	2500	800	200	4
Potassium (mmol)	3000	300	70	4
Energy (MJ)	700	400	10	40
Protein (g)	12 000	2000	40	50
Vitamin C (mg)	1500	1200	20	60
Thiamin (mg)	25	25	0.3	75
Iron (mg)	4000	3000	20	150
Calcium (g)	1500	500	0.1	14 years
Vitamin B ₁₂ (µg)	5000	5000	1	14 years

essential protein. In a starved rat the liver loses 20% of its protein in the first 2 d while other tissues of the body have lost only 4% of their protein, but then there is an adaptation and visceral proteins are defended at the expense of muscle protein (Addis *et al.* 1936*a,b*). However, muscle cannot go on indefinitely making good the inexorable nitrogen loss in urine, and collagenous tissues such as skin and bone have a slow rate of protein turnover and an amino acid composition which has a low nutritional value, so they cannot make much of a contribution. The time must come when the essential enzyme systems, which are themselves proteins, can no longer be maintained, and then death is inevitable. Analyses of cadavers of children who have died of undernutrition show extreme loss of protein, and of the protein which remains a large proportion is collagen (Picou *et al.* 1966).

Total starvation is no longer used as a treatment for obesity, because it is recognized that losses of lean tissue are excessive, and some unexpected deaths have occurred in patients undergoing starvation (Garrow, 1988). The effects of prolonged semi-starvation have been examined in the classic studies of Benedict *et al.* (1919) and Keys *et al.* (1950). These were experiments in which normal male volunteers were semi-starved in order to reduce their body-weight by 10% in 2 months (in the Benedict *et al.* (1919) study) or by 25% in 6 months in the Minnesota study of Keys *et al.* (1950). The intention was to reproduce, under controlled conditions, the undernutrition which was prevalent in Europe after two World Wars, and to compare the efficacy of different diets in the rehabilitation of these malnourished people. At the start of the Minnesota study the average weight of the volunteers was 69.4 kg and height 1.79 m, giving a Quetelet's index (QI) of 21.7. After 24 weeks they weighed 52.6 kg, with a QI of 16.6. (The limits of survival in undernutrition in males is QI of about 13 ± 1 according to Henry (1990).) After this severe undernutrition the subjects showed a significant reduction in haemoglobin concentration (by 22%), of basal metabolism (by 31%), of basal pulse rate (by 32%), of handgrip strength (by 28%), of endurance running time (by 79%) and an increase in the Minnesota multiphasic personality inventory depression score (by 36%). After 12 weeks of refeeding the subjects had made good these losses to the extent of 37% for body-weight, 31% for haemoglobin concentration, 49% for basal metabolism, 69% for basal pulse rate, 33% for handgrip strength, 34% for running endurance and 42% for depression score. It seems, therefore, that over a period of 6 months initially fit young men can withstand a loss of 25% in body-weight, and 69% in body fat, without permanent harm. The rehabilitation diets differed in their energy, protein and vitamin content, but the rate of recovery of function was related to the energy content of the rehabilitation diet but not significantly to its protein or vitamin content.

Semi-starvation in young women and children is a rather different matter. In the winter of 1944–5 there was famine in the towns of north-west Holland, due to a transport blockade. The official daily food ration fell from about 6 MJ/d to less than 3 MJ/d, and this was associated with a sharp decrease in birth rate in the affected population. However, after a study of children born during or after the famine months Stein *et al.* (1975) conclude that above a threshold value of food intake, a mother afforded the fetus protection from nutritional deprivation. The lesson to be learned concerning reducing diets is that it is necessary to be cautious before reducing greatly the food intake of pregnant women. The available evidence suggests that it is reasonable to aim for zero weight gain (but not weight loss) during pregnancy in women who are obese at the outset (Garrow, 1988).

SPECIFIC NUTRIENT DEFICIENCIES INDUCED BY DIETING

So far we have considered the effect of starvation, or of a restricted energy intake, on the nutritional status of normal subjects. However, slimming diets are normally taken by obese people: what effect has a low-energy diet on the protein, vitamin and mineral stores of obese people? As before, the answer depends on the nature of the diet and the length of time for which it is taken. If the diet consists largely of a protein-free energy source, such as sugar or alcohol, then the subject will become protein depleted. However, we can note that for a given low-energy diet the weight lost by obese people has a lower proportion of body protein than when the same diet is given to lean people (Forbes & Drenick, 1979; Garrow *et al.* 1981). The loss of protein relative to weight loss is greater at the beginning of the diet than later on, if the diet has a low protein content, and if it is taken in few large meals rather than several small ones (Garrow, 1988).

The danger of vitamin and mineral deficiency arises mainly when obese people take semi-synthetic diets for long periods. If the protein source in the diet is a protein food (meat, milk, egg, cereal) then the vitamins and minerals associated with these foods will be taken as well. It is not a coincidence that these foods provide roughly the correct mixture of minerals for human nutrition: the animal or plant which produced the protein used similar enzyme systems, with similar trace metals as co-enzymes, as those in man. If this were not true man could not have evolved to live on his present diet.

Vitamin deficiencies rarely appear in obese patients even when the diet fails to meet the recommended dietary allowance of some vitamins (Garrow *et al.* 1989).

METABOLIC RATE AFTER WEIGHT LOSS

It is commonly feared that after weight loss metabolic rate will be so depressed that it will be virtually impossible for the reduced person to maintain a constant weight. It is true that after substantial weight loss metabolic rate decreases: if it were not so that would imply that the weight which had been lost involved no metabolic cost for its maintenance, which is nonsense. The important question is if the post-obese person has a lower metabolic rate than that of a person of the same age, sex, weight and body composition who has not lost weight. To answer this question it is necessary to derive an equation to predict metabolic rate in a given person, and then to compare the observed metabolic rate of individuals with the predicted value both before and after weight loss. This was done by Dore *et al.* (1982) with results which are illustrated in Fig. 1. The study was conducted on nineteen obese women who initially weighed 104.5 (SD 9.1) kg. Their observed metabolic rate was 290 (SD 32) ml oxygen/min, which agreed well with the rate of 278 (SD 17) ml O₂/min which was predicted from a regression equation using weight, age and fat-free mass. After weight loss their average weight was 73.7 (SD 10.5) kg, and their metabolic rate was 234 (SD 26) ml O₂/min, compared with a predicted rate of 232 (SD 16) ml O₂/min. It can be seen from Fig. 1 that the change in metabolic rate was in general parallel to the line of identity: the ranking of each individual in observed *v.* predicted metabolic rate was preserved after weight loss. The only marked exception to this rule was the one subject who achieved by far the greatest and most rapid weight loss (from 112.1 kg to 63.7 kg in 10 months, see Fig. 1). She was involved in a national slimming competition (which she won) and unwisely resorted to spells of total starvation to hasten her weight loss. This study shows that after weight loss metabolic rate is not

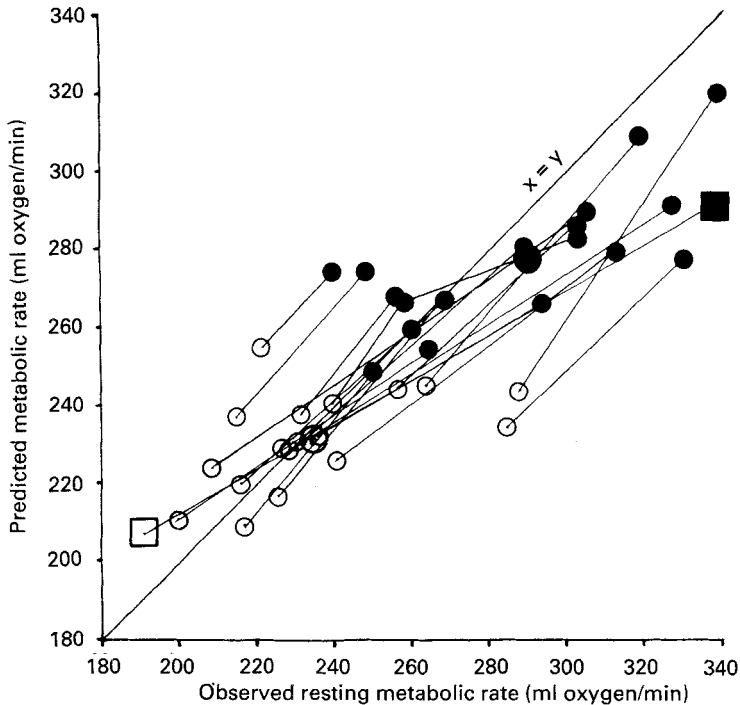


Fig. 1. Relationship between observed resting metabolic rate and that predicted by the formula of Dore *et al.* (1982) among nineteen obese women before (○) and after (●) they lost on average 30.8 kg (from Dore *et al.* 1982). (□, ■), Subject who achieved greatest and most rapid weight loss (112.1 kg to 63.7 kg in 10 months), resorting to spells of total starvation to hasten weight loss.

adversely affected relative to normal people of similar body composition provided weight loss is not too rapid.

We may conclude, therefore, that although total starvation will certainly cause death eventually, a reduction of food intake by up to 50% of habitual intake, until fat stores are reduced to normal levels, does no physiological harm to obese non-pregnant adults. In those populations in wartime when food supply was rationed the death rate from many diseases actually decreased (James, 1988).

PSYCHOLOGICAL DANGERS OF DIETING

Much has been written about the neuroses associated with dieting. Orbach (1978) maintains that the typically female anxiety about slimness is a manifestation of low self-esteem which has been promoted by the Media in a male-dominated society. Her solution is that women should reject the slim image promoted by the fashion industry and value themselves for what they are, rather than striving hopelessly to achieve an unattainable ideal shape. This is very sensible advice. Any physician who deals with obesity has experience of young women who are well within the range which is associated with longevity and good health (QI 20–25) who are trying to find some rapid method

which will reduce their weight to unphysiologically low levels, and who may well be using laxatives and self-induced vomiting to achieve this end. These are exactly the people in whom weight loss can bring no health benefit, and in whom sudden weight loss is particularly liable to lead to excessive loss of lean tissue. Every health professional should try to help such people to see that their intention to lose weight is misguided, and that their dissatisfaction with their weight is obscuring another problem to which their energies might be more usefully directed.

Unfortunately the problem mentioned previously, which concerns mainly young women, has caused some psychiatrists to condemn all dieting as misguided, and to maintain that it is the cause of eating disorders. Undoubtedly severe prolonged food restriction does cause eating disorders: Keys *et al.* (1950) review many reports of people who were half-starved because they were shipwrecked or imprisoned, and who overate to an extent which was sometimes fatal when abundant food became available. It is also true that some people who are depressed or discontented with their personal relationships may blame this on real or imagined obesity, and they may compound their difficulties by dramatic dieting efforts which have no hope of success. The problem is to know if dieting caused the neurosis, or if the neurosis caused the dieting.

Selection bias is very likely to affect the opinion of professionals in this field: the patients who are referred to psychiatrists with an interest in eating disorders are likely to have these disorders, and are probably unsuccessful dieters also. Furthermore, psychiatric patients who are on treatment with neuroleptic drugs such as flupenthixol or fluphenazine are very likely to be obese (Silverstone & Goodall, 1987), so psychiatrists have good reason to believe that obesity and dieting is strongly related to neurosis and psychosis.

The key question is: Are obese people more likely than normal to be anxious or depressed, and does dieting make their anxiety better or worse?

In a survey of 393 girls in a local school (average age 15·8 years) Wadden *et al.* (1989) found no association between measures of anxiety or depression and weight category, although the overweight girls were significantly more dissatisfied with their figures, and more of them were trying to lose weight.

Rothschild *et al.* (1989) recruited by advertisement sixty-three men aged 25–36 years, who ranged from very thin to very fat. They found that there was a significant positive association between obesity and depression (measured by the Beck depression inventory), but that the average scores for the obese, overweight and normal-weight subjects were all within the normal ranges.

Wadden *et al.* (1985) monitored the psychological state of seventeen severely obese women before, during and after a 6-month period of dieting during which the average weight loss was 20·5 (SE 9·2) kg. They administered both the Beck depression inventory and the Minnesota multiphasic personality inventory. The results during the control period, and at 6 weeks and 25 weeks of dieting are shown in Table 2. The scores on all the scales were within normal ranges at all times, but there was a significant improvement in all scales except anxiety at 6 weeks, and in all the ratings including anxiety at 6 months.

It is, therefore, not true to say in general that obese people, or people on diets, are significantly depressed or anxious, but of course these traits are frequently seen in patients referred to hospital. When it has been carefully monitored the effect of dieting in obese people has been to improve psychological function rather than to include neurosis.

Table 2. *Changes in rating on Beck depression inventory (BDI), the State-trait anxiety inventory (STAI) and Minnesota multiphasic personality inventory (MMPI) among seventeen obese women before starting a diet, and at 6 weeks and 25 weeks of dieting (from Wadden et al. 1985)*

Scale	Measure	Baseline	Week 6	Week 25
BDI	Depression	10.6	5.0**	5.7**
STAI	Trait anxiety	49.2	41.2**	43.1**
	State-anxiety	47.3	44.8	41.3**
MMPI	Hypochondriasis	56.2	50.7**	
	Depression	62.0	57.3**	
	Psychopathic	64.7	60.8*	
	Psychaesthesia	54.9	49.6**	
	Mania	48.5	53.5*	

Values showed a significant improvement from baseline values: * $P < 0.05$, ** $P < 0.01$.

SOCIAL PENALTIES OF DIETING

Anyone who has tried it can confirm that it is a social handicap to be on a diet. In our society it is a sign of good fellowship to be able to eat and drink freely with friends, and reluctance to do so may be interpreted as a slight. However, this stigmatism of the dieter is not necessary: diabetics and patients with coeliac disease or inborn errors of metabolism are known by their friends to be intolerant of certain foods, and this is respected and not made a cause for offence. The trouble about weight-reducing diets is that they are generally considered rather a joke, which people try for 1 week or so and then give up. It is astonishing that even severely obese and incapacitated patients who are referred to hospital may have difficulty in getting members of their family to be helpful towards their dieting efforts, rather than seeking to ridicule or undermine them. Sometimes the explanation for this attitude may be envy: dieters in the family who have failed to lose weight may not relish the idea of another member of the family succeeding.

The solution to this problem must be public education by health professionals, and the provision of slimming groups where people with a common goal can draw social support. It is no longer considered civilized behaviour to laugh at people with physical deformities, which is an advance on the days when Mr. Punch, the universal clown, was portrayed as a hunchback. It is to be hoped that obesity will not be a matter for mockery, and that obese people who are taking the appropriate dietary steps will be given proper respect and support for their efforts.

THE DANGERS OF NOT DIETING

For the obese person the medical, psychological and social dangers of dieting must be set against the corresponding dangers of not dieting. The medical dangers of obesity have been reviewed elsewhere in the present symposium, and it is obvious that the medical dangers of dieting are far less than those of remaining obese for anyone whose *QI* is greater than 30. It is also true that obesity carries social penalties (Wadden & Stunkard, 1985), and for a given level of intelligence and education obese people show a lower

social achievement than people of normal weight (Sonne-Holm *et al.* 1986). In principle these social disadvantages could be eliminated by proper public education, but on balance it is very much safer for obese people to diet than not to diet. The dangers lie with normal weight people who try to achieve unphysiological thinness by extreme dieting, since these are candidates both for the physical disadvantages of excessive lean tissue loss and also the psychological disorders which go with extreme food deprivation.

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