Options in obesity management

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The management of obesity requires acknowledgment of its heterogeneity. This derives from differences in pathogenesis, in genetic and psychological background, in physical activity, in food intake, and in aspects of lifestyle like cigarette smoking; as well as in degree, fat distribution, stability, and in consequences and associated disease. Pivotal to management is an appreciation that negative energy balance can be achieved at various levels of energy intake, depending on physical activity or on the degree of inefficiency of energy utilization. Reduced food intake can help people start with reduction in body fatness and is sometimes necessary for extended periods, but in the long run, an emphasis on increased levels of physical activity is preferred. Management endpoints need careful consideration since a great deal can be done to help the obese without necessarily changing weight. It is important to document changes in body fat and its distribution particularly by way of abdominal girth. Setting other healthful endpoints, such as food intake itself, self-esteem, physical fitness, risk profile for non-communicable disease, and self-care, are equally important. Management options – social, behavioural, exercise, pharmacotherapeutics and surgical – can be considered singly, sequentially or in combination. There are risks of management and these will include social, psychological (sense of failure and alteration in body image), economic, nutritional and physical (eg injury), and the more specific side-effects of pharmacotherapeutics and of surgery. Finally, the prevention of obesity requires the early detection of risk, eg the emergence of abdominal fatness with little change in total body fatness, and attention to health education, regular physical activity and the use of food with little fat.

Heterogeneity of obesity

No one approach to the management of obesity is possible because of its heterogeneity which is evident from several considerations (Table 1).

Pathogenesis

As far as pathogenesis is concerned, *genetics* is undoubtedly important and underscores individual difference in susceptibility[17]. We do not know how his difference operates, but mechanisms may include different thermogenic responses to food or exercise, recourse to physical activity, or even differences in food choice or preference. The genetic difference may not be in evidence unless the food supply or sedentary lifestyle allows.

Understanding the particular *social* circumstances which predispose to positive energy balance can pave the way for more successful management. These may include economics which limit food choice; the eating environment; occupational factors including workload, the canteen, peer pressure; attitudes of friends; needs which eating occasions serve; sense of loneliness and role of food and exercise to offset it; education and skills which enable preferred lifestyle to develop[21,55].

Psychological factors which contribute to obesity are often a significant barrier to progress with its management. These include self-esteem, mood and approach to crisis resolution with resort to food. Rationalization and justification of detrimental behaviours, along with illogical explanations of changes in weight or even fatness, do not help.

Arguably, the most important factor contributing to the increased prevalence of obesity in practically all countries in the latter part of the 20th century has been a decrease in *physical activity*, or not enough to match the increased energy density of the human diet (which makes it more difficult to maintain energy balance). Many individuals now seek to incorporate daily activity by way of walking, jogging, swimming, cycling, gymnasium work-outs, as substitutes for the ordinary activities formerly required for personal transportation, domestic activities, food production and preparation, and physical exertion in the course of occupation. Despite the evidence that this is now underway in society, it may not be enough, especially for those at genetic or socio-economic risk.

Food intake itself, often perceived as the most pathogenetic factor in obesity, may operate in several ways (Table 1). *Choice* may be limited, dependent on the food supply, even with an apparently abundant food supply. For example, once dairy fat as butter, cream and cheese are added to all manner of meals, snacks and dishes, energy-dense food (kiloJoules/gram or kiloJoules/ml) become the order of the day – even low-energy dense items like cereals, vegetables (cooked or salad), and fruit, become a problem[56]. *Amount* of food assessed as mass or volume is rarely a problem, but its energy density is. Indeed, if bulky foods are satiating or distending of the abdomen, then the signals for regulating intake are working well and must not be misunderstood.

There is evidence that *texture, taste and sound of chewing* may be more difficult to satisfy in those prone to

Table 1. Heterogeneity of obesity.

Pathogenesis
- Genetics
- Social: Situational
  Need
  Education
- Psychological
  Self-esteem
  Mood
  Crisis Resolution
  Illogicalities & rationalizations for detrimental behaviours
- Physical activity
- Food: Choice
  Appetite
  Amount
  Frequency/pattern
  Texture, taste, sound
  Variety
  Thermogenicity

Obesity – degree, distribution, stability
- Gender
- Age
- Other risk factors

Consequences
MVD
Diabetes
Neoplastic disease
Accident proneness
Biliary tract disease
Osteoarthritis
Reflux oesophagitis

Associated disease (not necessarily consequential on obesity):
- Physical handicap (CVD, osteoarthritis, chronic
- neurological disease)
- Chronic inflammatory bowel disease
- Chronic obstructive lung disease
- Cardiac decomposition

obesity[23]. The food advertising industry does not help the obese-prone here, at least where, for example, the flavour, creaminess and crunch are to be had from fatty, crunchy confectionery. On the other hand, nor does help come from nutritionists who encourage endless salad consumption in preference to cooked vegetables, where the volatile aromas of food can help achieve flavour satisfaction.

If small, frequent *snacks* are non-fatty (low in energy density) then they may decrease coronary risk profiles, are unlikely to contribute to obesity, and may actually help avoid it[25].

The question of *food variety* is an interesting one for obesity. For a while, bland food may be sufficiently monotonous so as not to stimulate appetite, which may work as long as other food is not around to be eaten. But once it is, managing a wider range of foods without excessive energy consumption is an important attribute. Again, if this wider array of foods is low in energy density (low in fat, refined carbohydrate and alcohol) then it is likely to be manageable. Indeed, in an acculturating community, Chinese women in Melbourne, Australia, it has been found that the greater the variety of foods ingested, the less the abdominal obesity[10,55].

Table 2. Factors that were predictive of body fatness in food intake models[a], by gender. Source: Hage, 1992.

	Food intake	
Risk factor	Positive	Negative
BMI)		
Men	Citrus/apples pears/bananas, light snacks, tropical fruit, poultry	Mushrooms
Women	Wine, nuts potatoes	Rice
WHR (Waist-to-hip ratio)		
Men	Seaweeds	Citrus/apples/ pears/bananas
Women	Processed seafoods, melon, carrots	Wine, pastry, biscuits

[a], Taking into account the residual effect of WHR in the total fatness (BMI) model and the residual effect of BMI in the abdominal fatness (WHR) model; ED, education level ('2', 0–6 yrs; '3', 7–9 yrs; '4', 10–12 yrs; '5', 13 plus yrs schooling); LOSIA, the length of stay in Australia.

We are now beginning to identify differential effects of particular foods and food patterns on total fatness as opposed to abdominal fatness[57]. In the Melbourne Chinese study, consumption (citrus, apples, pears and bananas) allowed a greater total body fatness (for most this was within the acceptable BMI range of 19-25 kg/m^2) whilst protecting against abdominal fatness which could exceed acceptable limits even for an acceptable BMI (Table 2).

There is now evidence for factors in food, like capsaicins in chillis, which increase the background thermogenic response to food[8,26]. This may possibly account for different proneness to obesity in different food cultures.

Degree of obesity

The stratification of overfatness most commonly used clinically and for public health purposes is based on body mass index (BMI) in kg.m^{-2} where:

<18.5 is chronic energy undernutrition
<20 is underweight
25–29.9 is acceptable
30–39.9 is obesity
>40 is morbid obesity.

However, commonly in North America, obesity is taken as a BMI ≥27.5 kg.m^{-}2, roughly equivalent to the Metropolitan Life Insurance Company of New York desirable weight criterion of 120% for obesity (130% roughly equates to a BMI of 30). These categorizations are arbitrary, and based on level of acceptable risk of continuing with the degree of fatness from prospective studies. The cut-off points clearly affect prevalence rates, up from 6–7% in 1980–1983 to 10–12% in 1989 in Australia for BMI ≥30 (National Heart Foundation of Australia), whereas in the USA at the beginning of the 1980s it was about 12%[30]. Bray has suggested that the level of fatness is a principal consideration in choice of therapy[29,47].

Fat distribution

Much of the predictive power of total body fatness for

adverse health outcomes resides in abdominal fatness, at least as expressed in ratios of abdominal circumference to hip circumference[11,11a]. *Abdominal circumferences* used include the narrowest ('waist'), 12 cm below the xiphisternal notch, that of the umbilicus, or at the lowest rib margin when standing. *Hip circumference* has been variously recorded at the iliac crest (which may be above the umbilicus), level of the greater trochanter, or of the greatest gluteal protuberance (these latter two circumferences are very similar). WHR (waist to hip ratio, or more correctly, abdomen to hip ratio) embraces several tissue compartments; these are, mainly, abdominal fat (both subcutaneous and omental), hip fat and hip muscle. It is interesting and remarkable that, so simply measured, WHR should be so predictive of health whilst having a complex tissue basis. Various metabolic studies suggest that too much omental fat is adverse to health whilst hip fat in women is important for reproductive function and defended as such; and muscle wasting at the hips is not a sign of health. With more appropriate clinical methodology, probably each of these compartments will be assessed in its own right.

Stability

The directional change of body fatness at the time seen, is at least as important as the degree and distribution of fatness. Is it decreasing or increasing, how often and when has it done so? This kind of understanding provides a greater opportunity for effective intervention and prevention. For example, if after each pregnancy, with each bout of depression, after each vacation, at the end of winter, on marriage, body fatness has increased or is increasing, the underlying basis can be targeted.

There is some evidence that repeated losses and gains of weight may not be conducive to health and longevity[32,32a]. What we do not know is where people who have repeatedly lost weight would have been with their weight had they not made these efforts. Anecdotal actuarial analysis suggests they might have been more obese. For example, a patient at worst weight now may be no more than worst weight 10 years ago, yet during this time the community as a whole has become progressively more fat. The study of this problem is particularly important. Weight cycling also does not necessarily mean increasing metabolic efficiency, unless lean mass has been inappropriately lost[32,33,48]. Lean mass is a major determinant of basal metabolic rate (BMR) or of basal energy expenditure (BEE) and it, in turn, of total energy expenditure.

Consequences of obesity

These in themselves create additional heterogeneity for the obese and their management (Table 1). The consequences include macrovascular disease, diabetes, neoplastic disease (probably breast cancer, possibly prostatic cancer and colorectal cancer) accident proneness, cholelithiasis, osteoarthritis, reflux oesophagitis.

The likelihood of adverse consequences will also differ amongst the obese depending on gender, age, and other risk factors for the disease in question. In general the risk of myocardial infarction in women is deferred a decade, and the advent of the menopause changes cardiovascular risk status, although some of this may still be age-related. The health consequences of obesity have been judged prospectively beginning mainly in younger adult life. Available evidence suggests that, for a similar level of fatness, older people may be at less risk[27]; this is reflected in recent US Dietary Guidelines (from 1990 Guidelines, Baltimore)[28,58].

The cigarette smoker who is also obese is at greatly (2–3 fold) increased risk of premature death[30]. The MONICA study indicates that the obese with lipoproteins containing relatively more apo B48 of the 'hypervariable region' kind are at greater risk of premature coronary mortality than those who are negative for this genetic trait[34].

Associated diseases

It is also necessary to take into account disease associated with obesity, whether or not a consequence of it (Table 1).

This is especially so with physical handicap which limits physical activity, critical for long-term management of excessive body fatness. Here it may be envisaged that selective exercise and increased nutrient density in the diet (possibly with nutritionally-complete supplements or formula foods) may be required[37-39].

Altering energy balance

It is possible to enter *negative energy balance* at various levels of energy intake, depending on physical activity or on the degree of inefficiency of energy utilization (Table 3). As far as possible, it is better to maintain a higher than a lower intake so as to obtain the associated food factors, both nutrient and non-nutrient. This is most easily achieved by being physically active. For a negative energy balance of about 500 kcal (2100 kJ) per day, in 2 weeks about 1 kg body fat is lost. Below about 800 kcal (3400 kJ) per day, it is virtually impossible to derive a nutritionally adequate diet from food alone, and preferable for it to be above 1000 kcal (4200 kJ) per day.

Table 3. Altering energy balance.

- Physical activity
- Food intake
  - Availability
  - Choice
  - Appetite and Hunger
    - (A) Activity
    - (B) Food energy density
    - (C) Pharmacotherapeutics
- Efficiency of utilization
  - Lean mass
  - Thermogenic factors
  - Absorption

Randomised studies which have looked at the relative merits of *exercise* versus energy restriction[7] show similar weight losses at 1 year, but exercise allows better preservation of lean mass. The combination of techniques can be additive. Exercise alone continues to be effective beyond 6 months. It also reduces abdominal fatness which may be in more evidence than change in weight (and of greater health consequence).

Longer term prospective studies show that those who have a higher plane of energy nutrition ('more in and more out') live longer[13] and have lower coronary mortality in men[12,14] and women[11]. Prospective studies of physical activity support this view[35,36].

Reduced food intake can help people get started with reduction in body fatness and sometimes it is necessary for extended periods of time. The element of foods to reduce are fat, alcohol and refined carbohydrates (especially with fat). A *food history or diary* will help locate possible areas for change. Extra attention may need to be paid to cooking techniques including frying with fat, use of spreadable fat, food choices when eating away from home (fast food and restaurants where fatty sauces, batters and pastries are common), misleading food labels ('low cholesterol', but fatty foods; 'baked not fried', but fatty cheese and vegetable oils as ingredients), and beverages positioned as healthful (flavoured, sweetened mineral waters; fruit juices and cordials; sugared hot beverages had many times a day).

The use of *non-nutritive or minimally-nutritive substitutes* for sweeteners (sugar) and fat in food has the potential for allowing a reduction in energy intake. This remains contingent on compensation for reduced intake not taking place elsewhere in the diet[59,60]. A range of such sweeteners is now available including cyclamate, saccharin, aspartame, alitame, acesulfame K and sucralose. Fat substitutes include the micro particulate protein, Simplesse, and the sucrose ester, Olestra. The evidence is increasing that these approaches can be helpful[24,44–46].

Guidelines for use of VLED (very low energy diets) are given in Table 4[47,59].

Table 4. Guidelines for use of VLED (very low energy diets).

1. Where urgent weight reduction is required:
   - (A) For life threatening or severe complications of obesity, including intractable ischaemic heart disease or cardiac failure, the sleep apnoea syndrome, ulcerative reflux oesophagitis, and refractory non-insulin-dependent diabetes.
   - (B) Prior to elective surgery including hip and knee replacements, coronary bypass/ angiography, cholecystectomy, hernia repair.
2. Where nutritionally complete, but low energy, feeding is required for obese patients:
   - (A) Who are ill.
   - (B) After post-gastric partition surgery.
   - (C) Who are elderly.
3. Where weight reduction will benefit obese patients who are unable to increase their physical activities, with:
   - (A) Severe osteoarthritis.
   - (B) Neurological disease including stroke and Parkinson's disease.
4. Where dietary restriction of food intake and increased exercise appear to have failed.
5. Where motivation and/or early demonstrable weight loss is required.

Appetite may be stimulated where *hunger* is not present. The important strategy here is to arrange that appetising foods are not fatty or will not be accompanied by energy containing beverages (having plenty of water or unsweetened tea around, for example). Regular exercise helps achieve an appropriate appetite[9].

Finally, medications which help reduce appetite or include satiation (like dexfenfluramine) may be appropriate where other measures are inadequate[37–39]. Although studies are available for such agents for up to a year, and obesity and its consequences are appreciated as a long-term problem, caution would have us prefer to use these agents for about 3 months whilst recruiting diet and exercise strategies for the long-term.

It may be possible to alter the *efficiency of energy utilization* (Table 3) as already indicated by increasing lean mass and taking advantage of thermogenic factors in food. Various efforts have been made to reduce the absorption of macronutrients like fat, protein or carbohydrate by way, for example, of enzyme inhibition or by making these compounds less digestible. Short of these food technologies or pharmacological approaches, it is possible to take advantage of the physico-chemistry of food. Particle size confers indigestibility[40,41] as does the development of resistant starch (notably through cooking rice, noodles and pasta and then partially cooling prior to ingestion)[44].

Management

End-points

A great deal can be done to help the obese individual without necessarily changing weight (Table 5). Firstly, it is important to document changes in *body fat* (e.g. by a simple skin fold like that above the iliac crest, which should be less than 25 mm or 1 inch) or by *abdominal girth* (sometimes best judged by belt size or dress size). Impedance techniques (as simple as an ECG) are likely to become more a part of regular clinical practice to assess total body fatness.

Table 5. Management end-points.

- Body fatness
  - Amount
  - Distribution (WHR)
- Non-fatness
  - Food intake
  - Self-esteem
  - Physical fitness
  - Risk profile for non-communicable disease (MVD), diabetes, cancer, accident proneness, cholelithiasis, osteoarthritis, reflux oesophagitis)
  - Self-care

Secondly, setting other healthful endpoints is of value. A repeat *food* record can demonstrate achievement in this domain. If variety has increased, fat consumption decreased and fish consumption increased to 2 or 3 meals a week, then the prospects for total mortality are likely to have improved irrespective of a change in body fatness (Table 5).

Options

With the background already adduced, the options for

management of obesity will be largely self-evident (Table 6). The question is how the options might be decided upon and applied.

Table 6. Management options.

Single:

Social (esp. education)
Behavioural
Exercise
Pharmacotherapeutics
Surgical

Sequential:

Social → Behavioural → Diet → Exercise → Pharmaco → Surgical

Combined:

Social
+
Behavioural
+
Diet
+
Exercise

It is theoretically possible to use any one of them in isolation, but usually undesirable to do so. For example, several studies show that the value of food intake and exercise approaches is enhanced and made more durable by the application of *behavioural techniques*[47]. These techniques, as developed by Stunkard, are shown in Table 7.

Table 7. Obesity management behavioural techniques.

1. Self-monitoring-description of the behaviour to be controlled
2. Control of the stimuli that precede eating
3. Development of techniques to control the act of eating
4. Reinforcement of the prescribed behaviours
5. Cognitive restructuring
6. Diet
7. Exercise

If one had to single out a preferred strategy it would be regular *physical activity* (with behavioural therapy helping to build it into regular daily routines whether or not advantage is taken of specific exercise programmes). This might be avoiding lifts at one's place of employment and climbing several flights of stairs, several times a day; or walking 6 days out of 7 for an hour or so a day after evening meals with one's partner.

Physical activity has the several values of:

(a) expending energy for the duration of the activity, and more so if weight increases and one covers the same distance,
(b) maintaining or building lean mass with its contribution to BEE (basal energy expenditure),
(c) setting appetite more correctly,
(d) providing feedback signals about body shape through movement (somewhat analogous to John Garrow's where he has tied nylon cord around the abdomen once abdominal girth has been reduced[48], and
(e) improving self-esteem and ability to manage other areas of life including food choice.

The time for *pharmacotherapeutic* intervention is where motivation is required or where accelerated weight loss is sought. This can still be with ordinary healthful food. Apart from food, the same is true for VLED, except that their added advantage is that they can provide nutritionally-complete meal substitutes where energy expenditure is low and low energy intake mandatory for fat loss.

Surgery, principally gastric reduction surgery, is reserved for the morbidly obese (BMI >40 $kg.m^{-2}$) or those with BMI ≥35, where obesity is disabling or incapacitating and a serious threat to life in the shorter term[49]. Surgery has the best results of any strategy in this group. Long-term follow-up is required contractually with the patient to avoid mechanical problems or deal with them, and avoid nutrient deficiencies[49].

It may be that the several options are used sequentially to ensure that a sound foundation of behavioural, nutritional and physical education skills is acquired by the patient, but various combinations of options are what will ultimately prevail (Table 6).

Coronary risk factors in the obese

Particular problems arise in relation to the co-management of certain coronary risk factors in the obese (Table 8). Fortunately, once negative energy balance is in place, serum lipoproteins, glucose intolerance, and hypertension begin to improve. Even left ventricular hypertrophy (LVH) progressively decreases[19]. Obese people who have normal serum lipids may be falsely reassured that they can eat fatty foods without difficulty. They may have low HDL cholesterol concentrations and slightly elevated triglycerides with a relatively satisfactory total cholesterol, but an overall lipid profile that is risky for macrovascular disease (MVD). They need to be reminded that food intake is important in its own right, as is physical fitness.

Table 8. Managing coronary risk factors in the obese.

- Cigarette smoking
- Serum lipoproteins
- Glucose intolerance
- Hypertension
- Left ventricular hypertrophy

Cigarette smokers who are obese are at great risk from premature death. Cigarette smoking increases the risk of abdominal fatness[20]. Smoking increases metabolic rate, decreases taste, and mechanically replaces food. Thus, its cessation can lead to an increase in energy intake and increased efficiency of energy utilization. What is confusing is that the increase in total fatness may increase abdominal fatness – exacerbating the problem of smoking itself.

Thus one of the best approaches is to have an exercise

programme in place for the obese smoker. This will help with smoking cessation and offset adverse effects of its cessation.

Risks of management

As with all areas of clinical work, one must weigh the risks and benefits. Some of the risks of managing obesity are shown in Table 9. The most risk-free intervention is physical activity. Modest food changes consistent with the patients' food culture are also relatively risk free.

Table 9. Obesity management risks.

Social
- Interpersonal relationships

Psychological
- Sense of failure

Economic
- Costs of various measures

Nutritional risks
- Precarious essential nutrient intake with energy restriction
- Undue preoccupation with food
- Inappropriate loss of lean mass

Physical activity
- Injury

Pharmacotherapeutics
- CNS side-effects
- Other systems

Surgical
- Of surgery itself
- Nutrient deficiency
- Mechanical
- Failure

Clinical practicalities

How to be of help in a problem-solving way to the obese is not easy and requires considerable intelligence, empathy and deftness. Some practicalities are identified in Table 10.

Table 10. Clinical tips.

- Understand how patient developed obesity
- Consider importance of
  - Food
  - Physical activity
- Document degree and distribution of fatness
- Define how risky the fatness is for the patient in question
- Identify 'non-fatness endpoints'
- Indicate that several, sequential, and combined options exist for a long-term management approach
- Consider family

Prevention

Opportunities for prevention frequently arise in the course of clinical practice[50,51]. Identifying at-risk families for obesity, or those where obesity has led to complications or where osteoarthritis is a problem, could be of special value.

Early detection of weight increase at risk periods in a patient's life (marriage, pregnancy, change of job) will make for better health, achieved with much less difficulty (Table 11).

Table 11. Prevention.

- Education
- Regular physical activity
- Food with little fat
  - Variety, especially of vegetables, fish, crustaceans and shellfish
  - Flavoursome, spicy, texturally interesting
  - Low-energy-density snacks

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Options in obesity management

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摘要

肥胖症處理方法的選擇

肥胖症處理需要承認它的复雜性。這種复雜性是由於不同的病理，遺傳和心理因素；不同的体育活動，進食和吸煙；不同的肥胖程度；脂肪分布；穩定性；后果和有關疾病而引起的。關鍵性的處理是根据不同的体育活動或能量利用失效程度，使病人在不同能量進食水平下達到能量負平衡的目的。減少食物進食可幫助人們減少体脂，但從長遠來説，不如強調增加体育活動爲好。處理的目的需要小心考慮，因爲有大量的方法可以幫助肥胖者而無需改變他的体重。記錄体脂及其分布的改變，特別是腹圍的改變是重要的。制定其它有益健康的措施，如病人食物進食量，自我感覺，体育健身，非傳染病的危險因素和自我護理也同樣重要。處理方法有：社會的，行爲的，運動的，藥物治療的和外科的選擇，可考慮單獨，連續或合併使用。處理過程中的危險因素包括社會的，心理的(体型改變和身體失靈的感覺)，經濟的，營養的，体育活動的(如損傷)和藥物治療和外科手術的副作用。最后，預防肥胖症需要對其危險因素的早期探索，注意隨全身脂肪微少改變而出現的腹部脂肪，注意健康教育，經常体育活動和進食少脂肪的食物。