

FACTS ABOUT OBESITY

Theories of weight gain

Experts subscribe to one or other of two theories of weight increase, which are the *Push* or the *Pull* theories. The *Pull* Theory suggests that weight is regulated by *internal* factors which pulls food in - for example, there are lots of hungry fat cells waiting to be filled - *or* the *Push* Theory suggests there is something *external* in the family or the culture which pushes food in.

There is a lot of evidence for the Push Theory. After all, we live in a culture which celebrates food. Holidays and many other social occasions are centered on eating. If you want to celebrate something you don't take your family to a salad bar. In our culture there are countless opportunities to eat, together with countless messages telling you that you will be lacking something important - happiness even - if you do not eat. This environment fosters fat.

Exposing rats to what he described as a *supermarket environment* - plenty of energy rich food with great variety - Kelly Brownell found that rats became 300% fatter than their natural weight. In addition, we are much more sedentary than we used to be. Researchers in Britain measured the effect of our sedentary lifestyle compared to that of 30 years ago. Despite the fact that we eat marginally fewer calories than we used to - energy saving appliances account for a potential gain of 5-10 lbs each year if we were to eat as much as we used to.

There is an evolutionary benefit conferred on people who are able to store fat. In olden times when food was scarce, it was fatter people - those who were able to store fat - who would survive illness and shortages of food. Obesity was rare in those times, since periodic shortages plus a lack of energy saving devices made it impossible for weight to be gained progressively. It would seem that our bodies are still adapted to a hunger-gatherer environment; we cannot cope with an environment laden with food in which we need expend little energy to obtain it. Weight gain *must* consequently be the result.

Push Theory may operate on the family level. Some families foster overeating, for emotional or cultural reasons, or simply from ignorance. Parents might teach bad habits, like forcing children to clear everything on their plate, eating quickly, or they provide food when everyone sits down to watch TV. Research indicates that eating in front of the TV may foster overweight. Metabolism slows down when people are in a mild trance state and energy from the diet is less likely to be converted to heat.

Stress is another factor in the external world which might lead to weight gain. Fatter people appear to eat more than thin people in response to stress, loneliness or anger. This suggests a *personality-led vulnerability* to Push factors in the environment. From a physiological perspective, it is now believed that stress leads to a heightened cortisol response, which in turn favours the deposition of central fat. Central fat is a risk for the Metabolic Syndrome, which facilitates weight gain overall. This apart, it would be dangerous to assume that overeating is a symptom of emotional distress. The causes of obesity are complex; counselling for emotional distress does little to make people thinner. Obviously, something else is going on.

Evidence for the Pull Theory

Fat Cell Biology

A lot of work is being done on the biology of fat cells. Researchers in Sweden recently discovered that people tend to drop out of weight loss programmes - not when they had attained their target weight - but when their fat cells had reached normal size. For two people of the same height, this could occur at greatly different weights. One person may have more fat cells than the other.

Some overweight children and morbidly obese adults are known to have more fat cells than usual. This is called *hyperplastic obesity*. Fat cells are usually formed at two critical periods of a person's life - in early childhood and at puberty. We now know that new fat cells can be formed at any time of life if weight is gained rapidly or if fat cells grow to over 50% of their normal size.

Similarly, if people try to reduce their weight to the point where their fat cells shrink below normal size, the organism starts to behave as if starving (even if they eat fairly well). People with fat cells below normal size display all the usual symptoms of people with eating disorders (craving food, being obsessional and ritualistic). So there is obviously some kind of biological pressure to keep fat cells at approximately their normal size, even if technically this means that a person may be culturally "overweight. This provides evidence for the **Set Point Theory** which is described below.

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Set Point Theory

Further support for the Pull Effect comes from Set Weight Theory. Question - do we all have a biological mechanism which determines our natural weight - and will this mechanism *pull* food into our mouths until we have attained that weight? There is a lot of evidence for a “set weight” which is personal to each of us:

- Rats given as much normal rat food as they can eat are good at keeping a stable weight. It is only in the “supermarket environment” where this ability to manage weight breaks down
- 97% of all people who lose weight on a diet regain all the weight they have lost; magically settling to more or less what they were before the diet. Note: we must be cautious interpreting this evidence. People can and do lose weight on diets and many people are thinner than their heaviest-ever weight. But people who lose weight generally have to work quite hard to keep it off. It is not really possible to lose weight and trust that weight will stay off by itself.
- People who gain weight in the short term, such as during festive events, generally lose weight naturally as they return to normal life and eating habits.

This regulatory system breaks down at times – if it is put under constant pressure. Satiety signals are always weaker than the strong biological signals which drive us to eat, and to eat to surfeit when the food tastes good.

Whilst the evidence for a biological set-weight is strong, experts now feel that it is not an exact level of weight but rather a broader range, within plus or minus 10 pounds either way of a central point. The theory is that we will naturally fluctuate within this weight range; the position at any one time depending on our lifestyle, food choices, exercise levels, our age and gender.

Metabolism

Overweight people often claim that they have a slow metabolism, which we could ascribe to the Pull Theory. Food is pulled in but it cannot be burned. Metabolism is the rate at which we burn energy and it is affected by age, gender, body musculature and exercise levels. The components of metabolism include our *Resting Metabolic Rate* (RMR); the energy we need simply to stay alive and fulfil our vital processes. The second component of metabolism is influenced by the action of digesting and absorbing our dietary food, which we call *the thermic effect of food*. The third component of metabolism is the energy we expend by day to day activity and this will vary from one person to another in a number of important ways.

It is true that resting metabolism varies among individuals - by up to 500 calories daily. And there is evidence to the effect that overweight children burn energy at a slower level than slim children - although this could be because slim children run around more easily. Overweight adults do *not* generally have slow metabolism relative to people of normal weight. Indeed, metabolic rate is likely to be heightened because they have more weight to carry around, together with more muscle tissue to support the extra weight.

Activity has a large effect on metabolism; this being mostly due to sensitisation of cell membranes to insulin. In other words, cells in an exercised body take up glucose and burn it more readily. Dieting, conversely, reduces metabolic rate - at least while the individual is short of food.

Genes

Another possible Pull factor is our genes. Is overweight hereditary? Well yes, it does run in families. Only 20% of children with no obese parent become overweight. 60% of children with one parent are likely to be overweight, rising to 80% of children who have two overweight parents. Could it just be bad eating habits being passed on?

In adoption studies in Denmark in the 1980s, the weight of adoptees was compared to that of their natural and their adoptive parents. Children’s weight corresponded most closely to the weight of their natural parents. Even more interesting were the studies done with identical twins raised apart. Thin separated twins grew up identically thin. Overweight separated twins grew up overweight, but there

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was much more variability in their body size. This seems to prove that genes play a powerful role in obesity, by the manner in which they sensitise individuals to the environment. From these studies, we can estimate that genes account for up to 70% of the influence on the weight of any individual. Still, genes will not condemn a person to be overweight. It just gives some people a greater challenge with respect to weight control than others.

Other factors

Even sleep deprivation and over-sufficiency are associated with weight gain in adults and in children. Poor sleep causes a decrease in circulating leptin and an increase in ghrelin which causes appetite to rise. This may hark back to our evolutionary past when food was scarce. Humans would forage more for food when there was more daylight and needed a high appetite to eat more and store it as fat in the winter months.

Glands

Are they a Pull factor? Glandular problems such as Hypothyroidism, account for less than 5% of obesity cases.

Myths

After looking at all these factors, while we cannot be specific about the causes of weight gain, we can at least dismiss some of the myths about fat people, namely:-

➤ ***Fat people are greedy?***

There is no evidence that fat people are greedy. Fat people need to eat more than thin people to support higher energy needs due to their additional weight. What is true is that fat people are more likely than normal weight people to turn to food in times of stress. It may be due to intrinsic personality differences but there is no evidence for this either. Obese binge eaters, however, are more likely to have a history of early personal trauma, lifetime incidence of psychiatric and emotional problems, and a higher incidence of substance abuse.

➤ ***I was born to be fat?***

Twin studies and recent gene studies proposing a 70% heritability of obesity in an obesogenic society, might lead people to despair - *what's the use; I can't do anything about it!* It is unfair, but it doesn't mean that weight cannot be controlled. All we inherit is a tendency to put on weight, although careful eating and exercise habits lower the probability of obesity.

➤ ***Fat people are lazy?***

Many fat people do not exercise due to embarrassment or because they find it uncomfortable, but many people do not exercise for all sorts of different reasons.

➤ ***Fat people are responsible for their condition?***

This kind of thinking is unfair and misplaced.

Who Needs to Lose Weight?

So who does need to lose weight? Certainly not as many people as are concerned about it. Recent surveys show that almost 70% of people are concerned about their weight and half as many are trying to do something about it right now, including large numbers of children, and people who acknowledge that they are not strictly overweight. There is a massive obesity phobia in the culture.

4 out of 10 women say that they are terrified about the prospect of gaining weight and children as young as 6 choose as a potential playmate a child who is disabled rather than one who is fat. The amount of distress experienced by some women who are marginally over their ideal weight bears no relationship to the effect it has on their looks. Perhaps these biases arise from the importance we place in our culture on self-denial and self-control.

Experts generally agree that health risks set in at about 30lbs excess weight or a waist measurement over 95 cms in males and 85 cms in females. The risks of overweight are as follows, and rise with degree of adiposity:-

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- Hypertension risk, 6x normal
- Coronary artery disease, 3x normal
- Diabetes, 4x normal
- Cancer - obese men are significantly at risk with higher rates of colon prostate and rectal cancer. Women have higher risk of bowel gallbladder and breast cancer at 30% overweight.

The health risks set in sooner for people with upper body fat.

Weight Control Strategies

Reducing Calories

Experts favour a diet plan that will produce a weight loss of 1-2 lbs per week with modified calorie reduction, assuming a nutritional balance based on the food pyramid. If weight loss is faster than this, muscle tissue will be compromised and lean body mass - which burns energy - will shrink. Dieting has a short term effect on metabolism. Also, few people realise that by the end of a weight loss programme fewer calories are needed to maintain body weight.

The BBC Diet Trials at the University in Surrey in 2004 went a long way to proving that there is no one diet that is superior to others. Despite short-term differences in success among different types of diet - e.g. Atkins versus Rosemary Conley - they were pretty much the same in the long run. Most of the dieters regained all the weight that they had lost.

These studies did show however, that some people are more suited to some diets than to others, with variables being type of food plan, solo versus group approaches and even dieting milieu, (village hall versus health club).

The problem of fitting programmes to individuals. Despite a long analysis by NICE (*Obesity Guideline 2006*), it is still clear that health professionals are challenged to provide an holistic weight loss plan that best fits the individual, their lifestyle and values. There is however much less variability among people when it comes to the strategies which best maintain weight loss (such as planning, increasing activity, keeping a nutritionally sound diet, targeting weak spots, and being vigilant in responding to small changes in weight).

Liquid Diets

Liquid diets (Very Low Calorie Diets-VLCDs) are a recent development in weight control, and are heavily marketed in the form of meal supplements that are taken by many thousands of people. VLCDs were associated in the early days with a few deaths, attributed to poor quality protein. People are asked to take their meals in the form of specially prepared drinks which provide 300 to 500 calories daily in the form of protein with nutritional supplements. Weight loss is rapid; but in the early days this consists of water and sodium because the body experiences shock. There are side effects, weakness and sickness. Long-term outcomes are very poor, and VLCDs are associated with rapid post-diet weight gain. Although VLCDs are on open sale, experts agree that they are unsuitable for people with only moderate amounts of weight to lose, or for people with disordered eating habits. NICE have approved their use under clinical supervision for morbidly obese individuals.

Exercise

This is recommended for dieters and is strongly associated with maintenance of weight loss. Among people who succeed in keeping their weight down over a 5 year period, over 70% exercise regularly, compared to only 20% of people who have regained their weight. (*National Weight Control Study, USA, ongoing*)

Some individuals use exercise as a compensatory behaviour for overeating, which may be counter-productive because it may not achieve very much. While it takes only 5 minutes to eat a Mars Bar, one would have to run eleven miles to burn it off.

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Different forms of exercise have differing effects on calorie output, strength, fitness and stamina. The weight loss counsellor must be selective in guiding people to do the right kind of activity for their age and physical status. Activity is helpful in many ways; it does change our physiology; it helps reduce appetite in the long run, it aids in relaxation and well-being, boosts motivation and it has a profound effect on glucose transfer; i.e. cells burn glucose more readily.

Drugs

There was a period in the 1970s when appetite suppressants, especially amphetamine-based drugs, were very popular. They came to be associated with serious side effects, including emotional changes, tolerance, addiction and very poor outcome rates. It was usual for weight to pile back on when drugs were ceased. Amphetamines are not now prescribed for weight loss.

Neurological targets of weight loss drugs have historically included the adrenergic system and the serotonin system. There are also chemicals which block the absorption of dietary fat.

In some private slimming clinics, pills are dispensed irrespective of body weight and without proper medical evaluation. Drugs are often given out for cosmetic rather than medical reasons.

Scientists are interested now in the hedonic *opioidergic* brain, which is involved in *liking* rather than *wanting* food. There is also interest in a compound named *Hoodia*, a cactus plant which is widely used in Africa by natives who wish to control their appetite during hunting expeditions.

At the time of writing, and as scientists become more familiar with the mechanisms affecting appetite, hunger, satiety and set weight mechanisms, there are many attempts to find a universal drug for weight control. But at present there is nothing that is right for all of the people, all of the time.

Surgical Techniques

The NICE Bariatric Surgery Guideline 2002 recommended surgery for obesity where it is a serious threat to life. Jaw wiring and gastric balloons have gone out of favour, but there is increasing competency among surgeons in a range of surgical procedures which restrict food intake, affect absorption of calories, or both.

The gold standard variants of bariatric operations include the following

- **Restrictive Surgery**, with gastric banding to reduce the effective size of the stomach.
- **Restrictive Malabsorptive Surgery** such as the Roux En Y operation. Food intake is reduced by reducing the size of the stomach through a combination of stapling and resection. The digestive circuit can then be anastomosed further down the intestinal tract.
- **Malabsorptive-Restrictive Surgery**, as in Bilio Pancreatic Diversion. The size of the stomach is radically reduced by resection and the digestive circuit is joined almost at the level of the colon, so that very little food will be absorbed.

In more radical surgeries, weight loss is dramatic and guaranteed. Gastric banding has more variable outcomes; it is safer and reversible, but it is also associated with excessive hunger in some people, who may out-eat the band and fail to lose weight.

Liposuction is increasingly popular. It involves the mechanical removal of fat which is liquefied and then sucked out through a pipette. But this method is only useful for selected areas of the body where fat has accumulated. It is possible that fat may return to the area.

Behavioural Approaches

Most diets contain simple behavioural advice such as *keep naughty foods out of the house or pin a photo of yourself when fat to the fridge door*. The value of these motivators is unproven. A true behavioural modification programme employs a variety of techniques which are designed to:-

- Reward positive behaviour

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- Help people become their own problem solvers through record keeping so that they can identify the attitudes, emotions, situations and habits which encourage overeating
- Help people apply **stimulus control** - the way they cook food, store it, buy it or expose themselves to it generally
- Help control relapses
- Change the automatic thinking processes which influence behaviour, which is a deeper influence on day to day eating choices

Behaviour therapy aims for gradual but permanent changes in behaviour and can be successful when used in conjunction with a sensible eating plan.

Which Approach is Best?

There are many weight loss techniques and lots of different approaches, ranging from Weight Watchers to Homeopathy and Hypnosis. No doubt there are some people who are helped by any of these approaches. Cognitive Behavioural Therapy is viewed as the gold standard for obesity management, but even this approach offers success in less than 1 in 3 people, maintaining an average loss of 10% of their pre-morbid weight. This is not enough for people who are seriously overweight, although even small weight losses may lead to significant benefits to health.

Behavioural approaches work best when *psychological readiness* is built into the programme. In other words, people need to be emotionally prepared and appropriately motivated, not have other major life issues to wrestle with, (including an active eating disorder), know how to elicit support from significant others; be trained in the methods of maintaining commitment, and have skills for dealing with lapses.

Fitting the individual to the best personalised weight-loss approach is a challenge for which we currently have no answers. People tend to blame themselves when they fail to lose weight and serious emotional harm can be done by giving an overweight person an unsuitable regime. Also, there is evidence that repeated cycles of weight gain and weight loss can make it progressively harder for weight to be lost at all.

In general, moderate obesity responds best to a behavioural programme. Severe obesity responds better to more aggressive approaches such as drugs, VLCDs or surgery, always under medical and psychological supervision.

Cultural Factors Affecting Obesity

Today's culture is defined as *obesogenic*. Our lifestyle has changed, along with the availability of food, and changes in the ways in which food is delivered, presented and eaten. These changes combine to provide a powerful drive toward eating more, moving less and promoting physiological adaptations which favour storage of fat.

Where *lifestyle* is concerned, we note:

Energy Saving Devices

The motor car, television, selling off school playing fields, the growth of sedentary activities such as home computing, one-stop shopping or shopping by telephone or internet, remote control devices for appliances - are making us move less. Subtle changes in energy expenditure have a great impact on weight. One researcher in the States (*LEARN 1998*) calculated that by using a remote device for changing TV channels we conserve the equivalent of 7lbs weight in a single year.

Lifestyle Change

Certain changes in life circumstances can trigger weight gain (or loss) often due to changes in eating opportunities, emotional eating or changes in activity levels. These may include leaving home and having one's own money to spend for the first time, use of alcohol, retirement, job change or redundancy, being in a relationship or breaking up.

Availability of Food

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Growth of eating areas in shopping malls providing largely calorie and fat laden choices for consumption on family outings. Very few works canteens, school meals, cafes and even sports centres; very few cinemas or other family entertainment venues such as football grounds or fairgrounds offer healthy alternatives to sweets, ice cream and other fast foods.

Change in Food Choices

The disappearance of the traditional family meal has introduced a culture of snacking, and snack foods are often energy-dense and high in fat.

There is a move to convenience foods as a consequence of a change in working practices, perceived occupational stress, single parent families and a change in women's roles. Fruit and vegetables are often not a part of these meals which makes the food more energy dense. Parents are no longer acting as the gatekeeper for foods, which promotes obesity in childhood. Obesity in children heightens the risk of obesity in adult life.

The growth in awareness of the importance of healthy eating is undermined by misleading food labeling, further provoking fattening food choices. Consumers are seduced by labels such as "*low fat*" as in low fat crisps which contain 60% pure fat, while they are made anxious about drinking full fat milk which is 96% fat free. Labels such as *lite* or *healthy* or *natural* do not reflect the nutritional value of food.

Consumers are misled into thinking that a consumable is low in fat when it is not. In a sandwich advertised as 10% fat (by weight), 30% of its calories are fat calories since fat by weight is more calorie-dense (9 calories/gm) than proteins or carbohydrate (4 calories/gm.)

The British diet is low in fruit and vegetables, and this is exacerbated by large subsidies given to farmers for producing dairy products and meat. This results in fruit and vegetables being proportionately more expensive. In the Midlands and North of England, there is high consumption of pies, chips, fried food and cakes. This was not a problem when most of the family was occupied manually, and when children walked to school and played outside.

Children are *neophobic* (suspicious of new tastes) and do not traditionally like fruit and vegetables. They will eschew these when other more tasty choices are available or obtainable. Again, this was less of a problem when children moved more and fewer unhealthy choices were in their day-to-day environment.

Disordered Eating

Disordered eating may be directly caused by pressures to be thin and inducements to control body size by dieting. According to the restraint model of binge eating, dieting will lead to reactive binge eating and this will affect some people more than others; depending on personal resources and other psychological or social factors. We estimate that up to 1 in 3 people with weight problems suffer from compulsive eating which is clinically significant, accounting for the intake of thousands of unwanted calories.

Medical Factors

Some medical factors predispose to weight gain. For example, Polycystic Ovary Syndrome and Hypothyroidism. Medication such as steroids for control of auto-immune conditions, and some antidepressants cause weight gain. Any demobilizing illness may lead to weight gain due to inactivity.

More About Metabolism- Resting Metabolic Rate

It is possible that one of the inherited pre-dispositions to obesity is reduced energy expenditure. Total energy expenditure is the sum of all the energy our body requires for rest, digestion and physical activity. Before reviewing what we know about energy expenditure and overweight, let's review some basics of energy balance.

The Energy Balance Equation

Body weight is determined by the energy balance equation

If energy in = energy out, weight maintenance occurs.

If energy in exceeds energy out, weight gain occurs.

If energy in is less than energy out, weight loss occurs

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Weight gain is the result of more energy coming into the body than the body is burning or expending. Energy comes into our body through the food we eat. Each food contains a certain amount of energy measured in calories.

Energy is used or expended by our bodies in three principal ways. The first is *resting metabolic rate (RMR)* which is the energy needed to keep our body functioning at rest, in breathing, heart pumping, digestion etc, and it accounts for 60-70% of all the energy we expend in one day. The energy required *digesting and processing the food we eat* is approx.10% of our total daily energy expenditure, and is called the *thermic effect of food*. The last and most variable component of energy expenditure is *physical activity*. Even persons who are not physically active spend 15 - 30% of their expenditure on every day activities. *So energy expended = activity plus physiology.*

Highly trained athletes may spend more energy on physical activity than their RMR.

Although all 3 components of energy expenditure may play important roles in the development of obesity, RMR is the primary focus of this discussion.

RMR in Lean and Overweight Individuals

Since most of the energy we expend is in RMR, a number of researchers have explained the relationship between RMR and overweight. One approach has been to examine differences in RMR between average weight and overweight individuals. Studies have shown that overweight people as a group have a higher RMR than their lean counterparts of the same age, sex and height; because these individuals weigh more, more energy is required to sustain the extra weight. The increased RMR is generally in proportion to the increased weight. The comparisons of RMR of lean and overweight individuals, however, are of limited use in understanding why obesity develops, because they describe the RMR of people *after* they are overweight.

RMR in the Development of Overweight

A better way to determine the role of RMR in the development of obesity is to follow people over time and determine whether low energy expenditure is associated with weight gain. Two studies have done just that. One study by Dr Susan Roberts at the Massachusetts Institute of Technology, and her colleagues at the Dunn Nutrition unit in England, examined infants born to 6 lean and 12 overweight mothers. 50% of the infants born to the overweight mothers became overweight while none of the infants born to lean mothers became overweight.

The researchers found that the children who had become overweight at the end of one year had a 21% lower energy expenditure at 3 months than those children who had not become overweight.

Dr Eric Revussin and his colleagues at the National Institute of Health examined the contribution of increased energy expenditure to weight gain in a group of 286 S.W. American Indians. The researchers studied 3 groups; those with a low, medium or high RMR.

Of 89 subjects followed for 2 years, less than 5% of those with a high RMR gained <20 lbs, while over 25% of those with a low RMR gained <20 lbs. People with a low RMR gained an average of 6 lbs a year compared to a 0.2 lb increase for those with a high RMR.

These studies suggest that a low **total** energy expenditure and / or a low RMR may contribute to weight gain and subsequent obesity. Both of the studies indicate that decreased RMR or total energy expenditure does not account for all of the weight gain - suggesting that environmental factors (calorie intake) and / or biological factors (fat cell size and number) may also contribute to increased weight.

Relevance of RMR to Weight Control

In addition to its role in weight gain, RMR certainly plays an important role in long term weight control; in fact, studies have shown that RMR is the single most important factor determining weight lost, where a specific number of calories are consumed. Is there any way to know what your RMR is? Conventional wisdom based on the pioneering studies of Harris and Benedict, 1990; suggest that age, weight, height and sex were the key determinants of RMR. Specifically, male individuals who are younger, heavier and taller would have a higher RMR than older, lighter and shorter females, and therefore would be expected to lose more weight given the same calorie intake.

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Recent studies have shown that these original assumptions about age, weight, height and sex may not be true, particularly for the overweight.

In a study done at the University of Pennsylvania in Columbia State University, Dr Waceen compared the **predicted** RMR based on age, weight, height and sex, to **actual** RMRs in 80 overweight women. He found that the predictions were accurate for less than 60% of the women. A more striking finding was the marked variability of RMRs among the overweight.

He compared the measured RMR of 5 women who were the same age, weight and height. Theoretically, these 5 women would have the same RMR - approx. 1740 calories per day. The RMR of these 5 women varied by almost 1000 calories per day. If they consumed a 1200 calorie daily diet for 3 months, they would all lose different amounts of weight - between 11 and 40 lbs. Based on their predicted RMR, all 5 women would be expected to lose 27 lbs.

This study suggests that estimates of RMR are not useful in overweight people. This study also reveals that the RMR of individuals varies greatly; suggesting that weight losses among people will be very different even when they consume the same number of calories.

What Determines This Difference?

Several researchers have shown that RMR has a strong genetic component. Bogardus and his colleagues in Phoenix showed a high similarity in RMR within families, while Dr Bouchard and others at Laval University in Canada showed that genetic factors accounted for 40% of the differences in RMR when he examined parent-child groups, identical twins and fraternal twins. These studies do not suggest that genetics is the *only* determinant of RMR, but it does play *some* role in addition to age, height and sex.

Muscle also influences RMR - the more muscle you have the higher your RMR. Thus exercises that increase muscle mass - e.g. weight training - may increase your RMR.

Effects of Dieting on RMR

It is well documented that the body adapts to calorie restriction by decreasing RMR. This reduction in RMR concerns dieters, because if it persists after dieting, weight control will be more difficult. In any event RMR will decrease after dieting because a person weighs less - there is less body weight to sustain. For example it should take approx. 10% less calories to maintain weight after a 10% weight loss. The critical question is, whether the decrease in RMR is more than that expected from the decrease in weight?

Recent studies have provided good news in this regard. Although the short term drop in RMR in response to calorie restriction is dramatic, the long term change is *what would be expected from the decreased weight*. This seems to be true whether the diet is moderate or more severe. Thus, after weight loss, people need fewer calories to maintain their weight than before weight loss. Lighter bodies need less energy. Exercise combined with dieting may be more helpful in preserving RMR, although this is still a matter of controversy.

Summary

Although overweight people have a higher RMR than their average weight counterparts, a low RMR seems to contribute to weight gain and subsequent obesity. RMR is extremely variable in the overweight, and this variability will result in different weight loss even when people consume the same diet. There is some evidence that dieting has *no long term* effects of RMR other than that predicted from weight loss. Although RMR may be under the influence of genetic factors and age, sex and height, choosing diets that minimise muscle loss and choosing activities that increase muscle mass, may increase RMR.

More about Fat Cells

What is a Fat Cell?

Fat cells are the body's storage depots for fat. Persons of average weight have 25 - 35 billion fat cells. Persons with severe obesity may have as many as 100-150 billion cells. Fat cells expand and contract as weight is gained and lost. There appear to be two critical periods for fat cell development; the second year of life and, for females, in adolescence. Although these are the times when the number of fat cells increases most dramatically, new fat cells may be formed at any time a significant weight gain occurs.

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Weight Gain and Fat Cells

The causes of overweight are multiple and varied. Independent of the cause, however, the effect is an excess of triglycerides, or fat. This excess fat is stored in fat cells distributed throughout the body. The initial response to weight gain is to increase the size of fat cells. They do so in a manner similar to a balloon expanding to accommodate increase in air or water. As weight gain continues, fat cells reach their limit in capacity for storing fat. At this point, new fat cells are formed.

The degree of overweight at which new fat cells are formed is not exactly known, but it is estimated to be approximately 50 - 60% above ideal weight. Studies which have examined fat cell size across a wide range of body weights indicate that individuals with mild obesity have increased fat cell size called *hypertrophic* obesity. Moderate obesity is characterized by both increased size and an increased number of cells. Severe obesity results in no further increase in size, but a marked increase in number - known as *hyperplastic* obesity.

Weight Loss and Fat Cells

During weight loss, fat is mobilised from fat cells to provide energy. Since less fat is in the cell, the cell size is reduced. There are limits however on the degree to which the size of a fat cell can be reduced. Just as fat cells have an upper limit for storage before new cells are formed, they also have a lower limit. An average fat cell weighs 0.4 to 0.6 micrograms. Extreme cases, such as anorexia can result in fat cells being reduced lower than this, but in general fat cells are resistant to shrinking below their normal range.

One example of this biological defence is a study by Per Bjorntorp and his colleagues in Sweden. They studied 26 patients who were losing weight and measured the fat cell size of the patients before weight loss and again when they stopped losing weight. Despite the fact that the patients varied greatly in their fat cell size before weight loss, 23 out of the 26 patients stopped losing weight when their fat cells reached between 0.4 and 0.6 micrograms. Some of these people were still overweight, however, because they had an excess number of fat cells. This study suggests that fat cell size may determine how much weight can be lost.

An equally interesting finding from this study was that when fat cells were reduced through dieting to below normal size, *irrespective of the actual weight of the individual*, there was a significant increase in symptoms of eating disordered behaviour; cravings, depression, and ritualised behaviours.

Fat cell size is reduced by weight loss, but what about fat cell number? Unfortunately a fat cell is a friend for life. Although some cases of reduced fat cell number have been reported with extreme weight loss, fat cell number does not decrease with weight loss.

Implications for Weight Loss Goals

Since fat cell number can not be reduced with weight loss, the number of cells may identify how much weight a person can lose. If Mary has 40 billion fat cells and Joan has 60 billion, and both are enlarged to the same degree, if they diet they will both lose weight but Joan is destined to remain at above average weight. Her cells may be normal in size but excess in number and she will carry extra weight.

How are Fat Cell Size and Number Determined?

Fat cell size and number are assessed by first determining the total amount of body fat. The most reliable method is underwater weighing. The difference between the weight in water and the weight outside can be used to calculate the density of the body, and from this measure of density the percentage of fat and muscle can be determined.

Once total body fat is determined, fat cells are removed from various parts of the body including buttocks and thighs. These cells are examined microscopically and measured for cell size. Total body fat is then divided by fat cell size to determine number. In general, anyone who is overweight has increased fat cell size. Increased fat cell number is likely in people with childhood-onset obesity or who have had significant weight gain in adulthood.

Distribution of Fat

Fat cells are distributed throughout the body. Where the excess fat is stored has implications for health. Upper body or *android* obesity is characterised by fat in the upper body - abdomen chest and arms. Lower

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body or *gynoid* obesity is where fat is distributed below the waist; on hips, thighs and legs. Upper body obesity is more prevalent in men and lower in women, although there are exceptions.

Waist to hip ratio gives an indication of lower body obesity. Over 0.8 and 1.0 in men indicates upper body obesity, which is associated with increased risk of heart disease, stroke and diabetes.

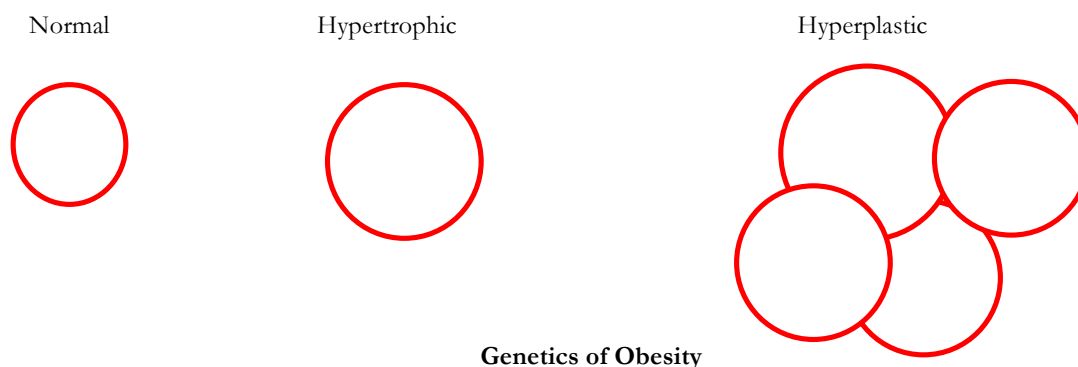
Summary

Fat cell size and number have important implications for weight control. Significant increases in weight are associated with increase in fat cell number. Increased fat cell number can prevent the attainment of an ideal weight because fat cells will resist being reduced below normal levels.

Increased fat cell number is associated with childhood onset obesity or severe weight gain in adult life. People with increased fat cell numbers should set realistic weight-loss goals and recognise the difficulty they may have in reaching an ideal weight.

Upper body obesity is more dangerous than lower body obesity.

Types of Obesity – Fat cells



Genetics of Obesity

The question of genes vs. environment has been highly debated in recent years and encompasses a wide variety of both physical and psychological characteristics. These characteristics include intelligence, athletic ability, heart disease and schizophrenia. Such traits are often examined within families for their genetic or environmental influences. For example, do you prefer certain foods because everyone in your family ate them as you were growing up (environment), or are the shared taste preferences in your family a result of heredity (genes)?

As you might suspect, body weight is one trait that has attracted a considerable amount of attention. It is well known that obesity runs in families. For example, children with two obese parents have an 80% chance of being overweight, compared to 40% for children with one obese parent and 20% when neither parent is obese. One scientist even went to the extreme of documenting that obese pets are almost twice as prevalent in obese households as non-obese households. These findings have been used to argue that environment is the key.

Because families share a common environment and common genes, it has been difficult to separate nature and nurture. However, studies in the last few years have greatly clarified the role of environmental and genetic factors. We will review what is known about genetics and what it means for weight control.

Adoption Studies

An intriguing method for assessing the genetic influence on body weight is to study adoptees. They are ideal for two interesting reasons. First, studying this group who are not biologically related to their adoptive parents allows scientists to attribute any similarities between adoptive parents and their children to environmental factors. On the other hand, similarities between adoptees and their biological parents can be attributed to genetic factors.

Dr Albert Stunkard at the University of Pennsylvania, with other colleagues, has recently conducted several studies on the role of heredity in determining body weight. In 1986, he and Dr Sorenson studied 504 adoptees and their biological and adoptive parents using the Adoption Register in Denmark. Denmark was

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chosen for this study because its adoption records are unusually comprehensive and include information about weight and height for adoptees as well as for both sets of parents. The adoptees were separated from their biological parents early in life, so they were not influenced by their biological parents' eating or exercise habits.

The results were striking. The weights of the adoptees more closely resembled the weights of their biological mothers. The next strong resemblance was with the weight of the biological fathers. There was no relationship between the weights of adoptees and their adoptive parents. These findings were the first to document a strong genetic component in human obesity.

Twin Studies

Another method of separating genetic traits from environmental traits is the study of twins. In twin studies researchers study both identical and fraternal twins. Stunkard examined 1974 pairs of identical twins and 2097 pairs of non identical twins in Sweden. Most of the variability in body weight was explained by genetic factors, which confirmed the heredity study of adoptees.

An even more interesting way of studying twins is to compare identical twins who have been raised apart. Twins raised together share the same genes and the same environment. Making the distinction between genes and environment is difficult. Twins raised apart have only their genes in common.

Stunkard and colleagues examined 673 pairs of twins, of whom 93 were raised apart. The body weights of the twins was the same irrespective of whether they had been raised together or apart, suggesting that environment plays almost no role in the development of weight!

Dr Claude Bouchard in Canada has done much work on the power of genes on the ability of the body to gain weight. They took 12 pairs of identical twins and overfed them by 100 calories for 100 days. The results were remarkable. There was high variability of weight gain between different sets of twins but great similarity within each pair. One pair gained 9 lbs and one pair gained 29 lbs. Even though people vary in the way they gain weight (even when given the same calorie intake) these gains are largely under the influence of genes. The twins not only gained similar weight, they distributed the excess weight in the same places i.e. some on the stomach and some on the thighs.

The OB Gene, the DB Gene and Leptin

Comuzzie AG and Allison DB: The search for human obesity genes. Science 280:1374, 1998

Farmers have known for centuries that they could selectively breed animals for fatness or leanness. Scientists also know that selective breeding indicates a genetic influence in laboratory rats and mice. Recently, research has shown that certain strains of lab animals become massively obese because of the effects of a single gene, i.e. there is one gene among the many in animals that seems to account for its obesity. Although the leap from mice to man must be made cautiously, these observations suggest that some obese people are obese largely due to the effects of a single gene.

The **OB-gene** encodes the hormone Leptin, which is produced by fat cells when they expand and which has the dual activity of decreasing food intake and increasing metabolic rate. Faulty expression of this gene leads to faulty encoding of Leptin and a grossly obese mouse.



The **DB-gene** encodes the Leptin receptor in the brain. Insensitivity of the receptor means that Leptin fails to counter the appetite hormone NPY and satiety is similarly impaired. DB mice are also overweight.

Melanocortins

These are chemicals which affect certain neurons in the hypothalamus and inhibit feeding behaviour. Genetic disruptions of the Melanocortin 4 receptor are associated with increased feeding and obesity, but this is thought to be rare in humans.

Other Genes

Research is discovering other genes which are thought to be determinants of eating behaviour. Some other genes are implicated in metabolism such as the Perilipin (FTO) gene. People who inherit one version of the gene are 70% more likely to be overweight. About 1 in 6 persons have the most vulnerable genetic make up with this variant.

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Genes and Metabolism

It is hard to separate the effects of genes on metabolism compared to behaviour. However Dr John Castro carefully monitored fraternal vs. identical twins' eating habits, and found that identical twins were significantly more similar in what they chose and how they ate, leading us to assume that eating habits and exercise patterns *are* influenced by genetic makeup.

There is renewed interest in genetic influences on “brown fat” which is a metabolic engine. **Beta-adrenergic receptors** are present on brown fat and perhaps white fat. Binding of norepinephrine to this receptor on fat cells leads to increased transcription of the mitochondrial uncoupling protein, allowing increased heat production via hydrolysis of fatty acids. It was recently reported that mutations in this gene predispose people to become obese and develop diabetes before middle age.

Genes versus Environment

Genetic studies have taught us two important things about the effect of environment on obesity. First, most environmental influences on weight gain are *unique* rather than *common* environmental influences. *Unique* means circumstances each individual experiences in his own life. *Common* refers to factors shared by members of a group - like all living in the same house. Secondly, environmental influences are transient in nature. This means that growing up in a healthy eating home does not guarantee that these eating habits will be maintained as an adult. We now all agree that genes exert a strong effect within environment and this will be more prominent in some people and less so in others.

What does it all Mean?

You may be thinking what's the point of fighting genes! Genes *do* influence body weight as well as the ability to gain weight and store fat. It may seem unfair that some people have a genetic tendency to gain weight while others eat whatever they like without gaining an ounce. These facts however do not mean that weight is completely out of our control.

Obesity is not a trait like eye colour, which is determined at the moment of conception and does not change. A tendency for obesity is inherited. This tendency needs to have an environment that will nurture its development before it becomes a reality. All of the studies above were conducted in western industrialised societies that consume a large percentage of their calories as fat.

These societies are also characterized by an increasing dependence on labour-saving and calorie-saving devices such as remote controls, garage door openers, electric can openers, computers, cordless telephones and so forth. It takes an effort to oppose the effects of the environment. However, changing eating and exercise habits can make a difference, even if an individual has been born with the genetic tendency to store energy.

Modifying the environment to decrease high-fat food and to increase physical activity will lower the chance that a genetic tendency will become a reality. Many medical disorders such as diabetes and high blood pressure are influenced by genetic factors but can be controlled by diet and exercise.

These studies give us insight into why some people become heavier than others, and why some people have to struggle more in their attempts at weight control. We hope these findings also provide some relief. There is now scientific evidence for what you may have suspected for years. Overweight people are not weak-willed or lazy. Losing weight and keeping it off may not be as easy for some as it is for others, if a tendency to be overweight is inherited.

As a society, the only way we can reduce obesity is through environmental changes, not just target specific families or persons. These changes must be pervasive and enduring. Weight control may not be easy, particularly if a person has inherited the tendency to be overweight but it can be done. Providing an environment of low fat foods and increased physical activity can stifle even the most stubborn genes.

Conclusion

Obesity is a complex issue. There are different types of obesity with different causes. It has different physiological and psychological manifestations and cannot be explained by a single label such as laziness or

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gluttony. The regulation of body weight is at least as complex as any other biological regulatory system. It involves neurochemical, gastrointestinal and hepatic signals, mechanical and humoral processes, genetic factors and an autonomic nervous system. Some of the time, the control can be overridden by deliberate behaviour in the context of psychological, social and situational factors, emotions and stress, the person's thoughts about what they have eaten and their anticipation of the consequences.

These forces are orchestrated to sustain a symphony in normal weight people and often chaotic dissonance in people with weight problems.

There are no single, guaranteed treatment programmes for obesity. Individuals must be matched to treatments and any programme undertaken must be sensitive to motivation and to personal factors which affect long-term commitment. The search is still on for a psychological treatment, safe drug therapy or even a surgery which will reduce body fat; it being recognised that adjustments will continue for life.