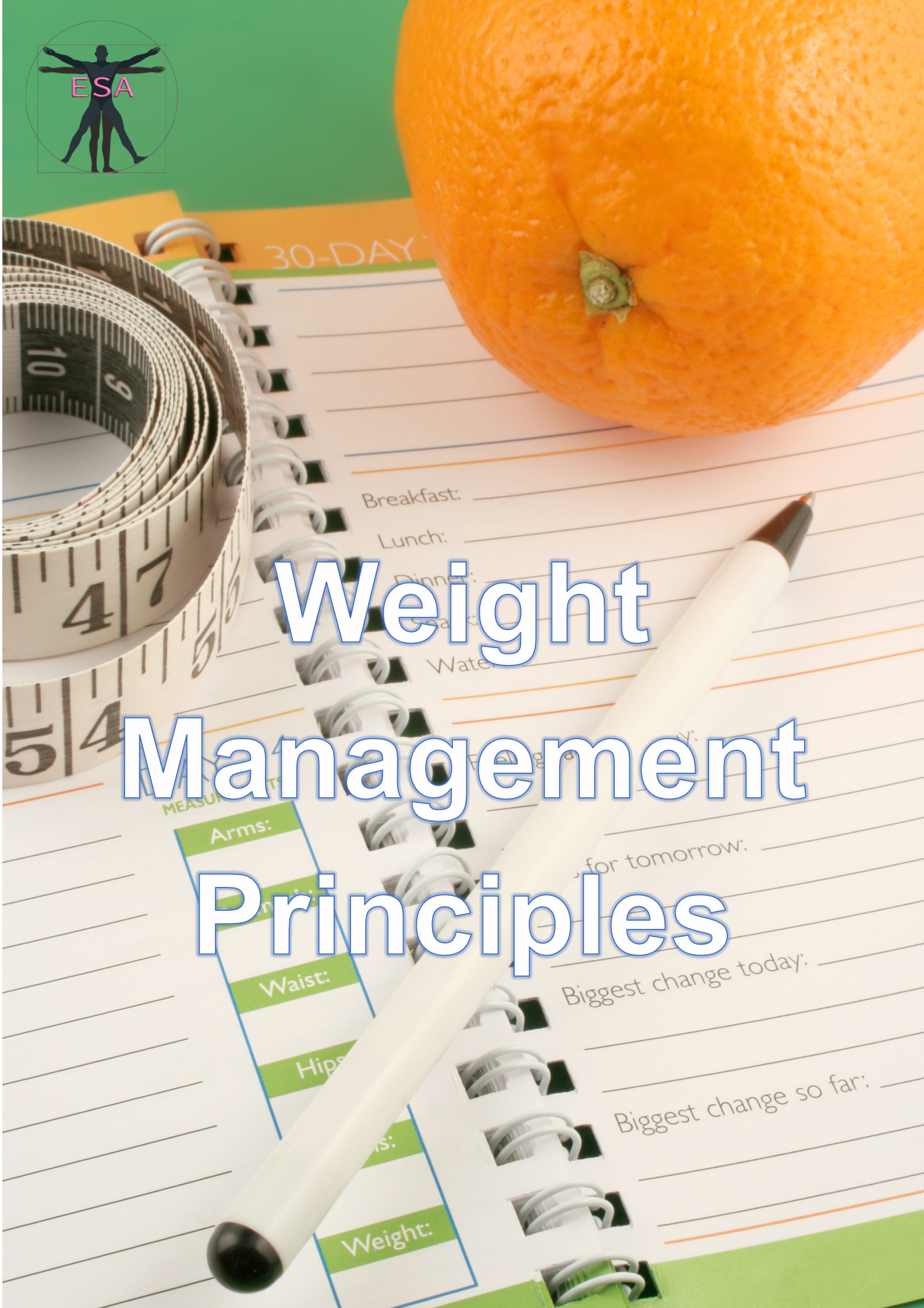

Weight Management Principles

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Introduction

Just as your car travelling 50 miles. d^{-1} needs more fuel than a car travelling 20 miles. d^{-1}, an individual running 5 miles. d^{-1} will require more food than somebody who is inactive. While, unlike a car, an individual can greatly increase their total fuel storage in the form of triglycerides situated inside fat cells identified as adipocytes. This is clear when comparing a regular 'lean' adult with approximately 35 billion adipocytes and around 0.5 µg of triglycerides in contrast to an obese person with nearly 140 billion adipocytes and 1.0 µg of triglycerides (Hall et al., 2012).

Several causal factors determine whether somebody will be at an energy surplus (leading to weight gain) or an energy deficit (resulting in weight loss). Typically, the components that contribute to weight gain or loss can be expressed as follows:

- **Es = EI - (REE + TEF + AEE)**

Where Es = energy balance; EI = energy intake REE = resting energy expenditure; TEF = thermic effect of food AEE = activity energy expenditure.

Almost certainly, you have previously learned that you need to generate a caloric deficit (through reduced food intake, increased activity, or a combination of both) of 3,500 kcal to lose 1 lb (0.45 kg) of fat. Thus, centred on the number of daily calories reduced or expended, you should be able to calculate how long it would take for an 81.6 kg male to lose a pound of fat when his daily caloric requirement is 2,500 Kcal.

If this person above reduced his caloric total to 2,000 Kcal. Based on the 3,500 Kcal estimates, it would take approximately 7 days for him to lose 1lb of fat (i.e.,500-kcal deficit × 7d= 3,500 Kcal). Extending this reasoning further, if he sustained this 500-Kcal deficit, he would lose 23.6 kg in 1 year and therefore disappear from existence in 180 weeks. There are shortcomings in the "3,500 Kcal/1 lb" reasoning, which is why people lose weight at various rates and why the rate of weight loss reduces over time.

Pathophysiology of Obesity

Obesity is a condition characterised by the storage of excessive amounts of fat in adipose tissue beneath the skin and within other organs, including muscles. Central adiposity: fat stored around the central (abdominal) region of the trunk, is more harmful than fat stored in other peripheral areas and is presently considered an independent risk factor for the development of CHD.

The disease process has been researched extensively over the past decades and findings suggest that the influence of hormonal mechanisms in the control of appetite and satiation is central to the development of obesity. Leptin and ghrelin are two of the hormones that have a significant influence on appetite and the regulation of food intake and satisfaction. They, and other appetite-related hormones, act on the hypothalamus. Leptin induces satiation (satisfaction, or sensation of 'fullness'), whereas ghrelin levels increase before meals to stimulate appetite and decrease after meals. In short, a deficiency of leptin - or leptin resistance - leads to overfeeding, whilst too much ghrelin leads to overfeeding, also. Therefore, the balance of these two hormones, in the long-term, is crucial for the maintenance of homeostasis—an imbalance leading to a greater risk of obesity.

Excess intra-abdominal adipose tissue is strongly associated with many health-threatening risk factors involved in an obesity phenotype. Hypertriglyceridemia, hyperglycaemia, pro-inflammatory cytokines, liver insulin resistance and increased very low-density lipoproteins (VLDLs) are among the many metabolic adaptations that occur which could impact a person's mortality (Tchernof and Despres, 2013). As part of the solution to prevent the rise in obesity in the UK, there is a great need to prevent people from becoming overweight, and then obese, as much as help those who are already obese. As exercise specialist, you can help play a part in helping individuals adopt a healthy lifestyle. You can play a role in assessing the risk of obesity to individuals, provide health promotion, and provide individual advice and onward referral to relevant specialists.

Aetiology of Obesity

We all have predetermined beliefs about what causes obesity. Remarkably, many of those views are not founded on scientific research (**Figure 1**). For instance, they are common viewpoints that breastfeeding and eating breakfast reduce the risk of obesity; however, when tested under well-controlled or randomised conditions, both conjectures are not supported (Casazza et al., 2013). What cannot be refuted are the laws of thermodynamics, which state that energy cannot be created or destroyed but rather is changed from one form (food) to another (adipose tissue or heat as a by-product of metabolism). Research by Mayer, Roy and Mitra (1956) demonstrated that humans and animals adjust ad libitum eating founded on the amount of daily activity. The authors reported that most individuals adjusted their caloric intake depending on their profession (i.e., the less physically demanding a job, the less the intake of calories). However, there was one exception to this rule the most sedentary jobs, which consumed more than any other group external of the most active workers. Incidentally, the inactive group also weighed the most.

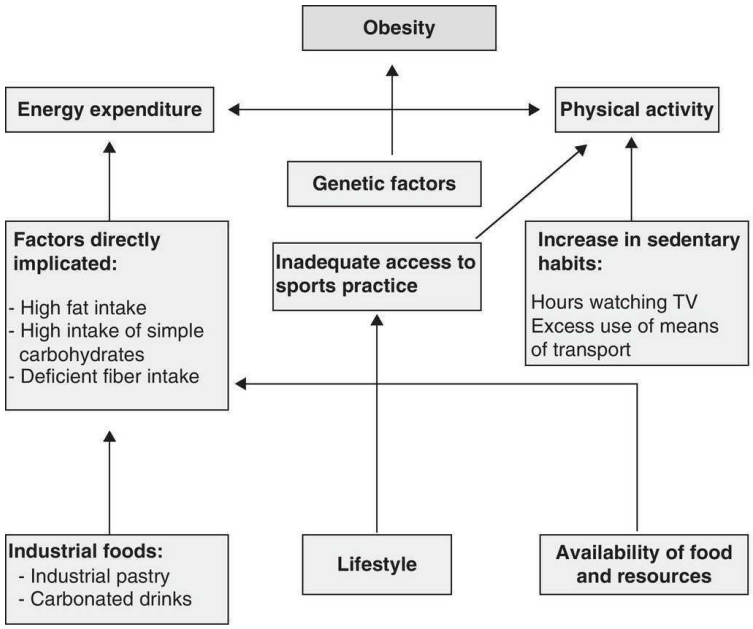

Figure 1. Obesity: Etiologic and Pathophysiology.

Over the last 50 years, much has changed and much of those changes have contributed to an obesogenic setting. For example, food is now comparatively inexpensive and more obtainable than it was previously. Dietary consumption surveys

suggest a per capita increase of 168 Kcal/d for males and 335 Kcal/d for females between the years 1971 and 2000. Jobs are now less physically demanding with the mean daily expenditure during work being 142 Kcal/d less than in 1962. It should therefore be little surprise that the human genome pool, chosen for energy efficiency over centuries of food shortage, should now find itself in this impasse.

Genetic Aetiology of Obesity

The direct cause of obesity in any individual is always an excess of energy intake over energy expenditure. The prevalence of obesity is likely to be due to increasingly sedentary lifestyles combined with the availability of palatable, high-fat, high-sugar, and energy-dense foodstuffs. However, not everyone exposed to this environment becomes obese and there is evidence that the inclination to develop obesity is inherited to some degree. Furthermore, there are a few rare cases of extreme obesity due to a single gene defect.

The rapid increase in obesity levels has occurred in too short a time for there to have been significant genetic changes within the population. It is, therefore, likely that obesity has been brought on by environmental and behavioural changes, which have led to more energy-dense food and a rise in sedentary behaviour. However, this does not mean that genetics or complex physiological mechanisms that control appetite and energy expenditure should be disregarded.

Besides excessive eating, obesity can also be caused by drastically reduced activity, and this often occurs in those who are sedentary or bedridden: a substantial decline in physical activity; for example, people in developed countries are more likely to work in sedentary jobs, use domestic labour-saving devices, and travel by car. However, it is now thought that obesity may be caused by several factors other than overeating or inactivity. It is still not known exactly why some ‘thin’ people eat a lot and exercise little, and why some overweight people eat moderate amounts or reduce food intake, often through repeated attempts at dieting, with no appreciable or sustainable weight loss. The following sub-sections will identify the influences on body composition, which will provide an overall understanding of the aetiology of obesity.

Scientific evidence guided by Xia and Grant (2013) on twin and parent-child studies, indicates that obesity has a strong genetic element. For example, a study by Stunkard et al., (1986) reported a strong relationship between the BMI of adoptees and that of their biological parents but no relationship between the BMI of adoptees and that of their adoptive parents. While twin studies have offered evidence of a

genetic link, detecting the genes specific to the obesity phenotype has mainly remained vague.

One development was the unearthing of the ob/ob gene in rodent models. Mice with mutations to this gene were up to three times heavier than standard mice. Later it was exposed that the ob/ob gene produces a protein that yields the hormone leptin. Leptin is released by adipocytes and controls food intake and metabolism at least partly through its effects on the hypothalamus, which contains a high number of leptin receptors. There was wide interest when leptin was first unearthed in 1994, particularly when the administration of leptin to obese mice with the ob/ob gene displayed a dose-response relationship with weight loss. Though, the administration of leptin in obese individuals who have average leptin levels does not seem to be applicable for weight loss.

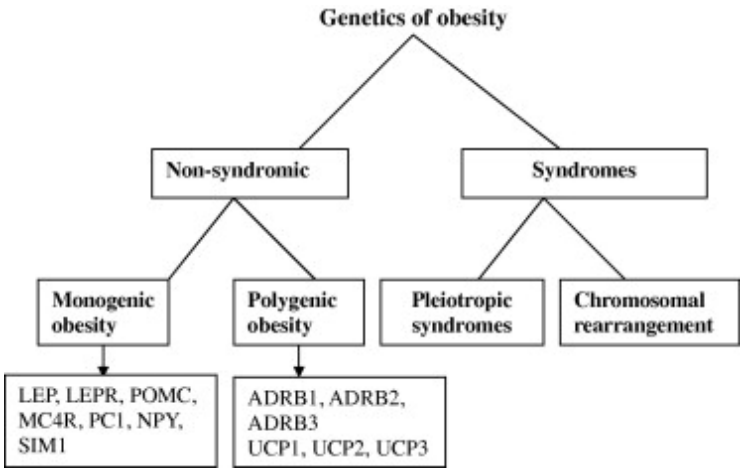

Figure 2. Genetics of obesity (adapted from Shawky and Sadik, 2011).

Recently, genome-wide association research has led to the detection of the FM and obesity-associated gene FTO which is the most robust genetic–obesity association in this period (Fields, Goran, and McCrory, 2002). Intriguingly, the FTO gene was initially assessed as a conceivable type 2 diabetes gene (Xia and Grant, 2013). While the mechanism for how the FTO gene causes obesity is unclear, the allele (i.e., gene variant) linked with obesity has been established within various ethnic populations (e.g., European, and African).

Typically, gene studies try to discover a relationship with the hormones responsible for the regulation of appetite and metabolism. Another regulatory hormone, largely secreted by the stomach, is ghrelin. Ghrelin has been found to directly influence appetite (Zato et al., 2014). Weight loss has a direct association with ghrelin: The greater the weight loss, the greater the quantity of ghrelin. Mason and colleagues (2015) noted that it does not appear to matter whether the weight loss is exercise-induced or diet-induced. Although no clear genetic links have been made between ghrelin and obesity, high levels of ghrelin are found in people with Prader-Willi syndrome. A genetic illness of the 15th chromosome that is related to delays in growth and development as well as a limitless appetite that leads to obesity.

Human obesity is likely to be the result of a multi-gene defect (polygenic) in association with environmental factors. Several gene expressions may, in combination, alter appetite regulation and energy metabolism. Leptin receptors (LEP-R) and the genes POMC and MC4-R are all candidates, and while it is outside the scope of this course to consider these further, you should be aware that there is the potential for polygenic influence in the obese client. Moreover, there appears to be a strong environmental impact on the polygenic profile of a person, so a similar gene profile may not impact different individuals in the same manner.

Weight-Loss Strategies

The public health liability of obesity is boundless. Deaths directly related to obesity were estimated to be around 3 to 4 million globally in 2010 (Ng et al., 2014). Presently, obesity ranks number two as a cause of avoidable death (smoking is the leading cause). Obesity trends have increased suddenly globally over the past few decades. For instance, since the late 1980s, the ratio of obese people in the United States has increased from 23% to 34% (Flegal et al., 2010).

In England, the Health Survey for England (HSE) reported that in 2017, 64% of adults (aged 16 or over) were either overweight or obese. Around a quarter to a third of adults (27% of men and 30% of women) were obese, with the prevalence generally increasing with age. In Scotland, the proportion of adults aged 16-64 who were overweight or obese (BMI of 25 or more) in 2017 was 65%. There has also been a steady upward trend in the prevalence of obesity (BMI of 30 or more) in adults aged 16-64, from 17.2% in 1995 to 28% in 2014 and 29% in 2017. Most of these increases occurred in the 1995-2008 period, as 2008, 2009, 2010, 2014 and 2017 figures were very similar (Scottish Health Survey, 2017). The National Survey for Wales (2017) revealed that 60% of adults were classified as overweight or obese. In Northern Ireland, the Health Survey (2017-2018) reported that 64% of adults were either overweight (37%) or obese (27%). The proportion of adults classed as overweight or obese has increased from the level reported in 1997 (57%) although it has remained at a relatively constant level since 2005/06. The number of overweight and obese people is expected to increase. Indeed, The Foresight report (2007) predicted that by 2025, almost 50% of men and 33% of women will be obese.

Management of obesity methods for weight loss varies in effectiveness and often is used in amalgamation with behavioural strategies that have been reported to increase effectiveness and inhibit relapse (**Table 1**). Regrettably, obesity overall is unmanageable by many conservative weight-loss approaches. Some reports state that by 3 years posttreatment nearly 100% of subjects regain all weight lost (Cooper and Fairburn, 2001). Conversely, several studies, including the Look AHEAD trial, do not report such a negative outlook and suggest that long-term weight maintenance is feasible. According to the National Weight Control Registry, analysts of long-term

success include sustaining leisure-time activities, dietary restriction, frequency of self-weighing, consuming a greater percentage of energy from fat, and disinhibition (Thomas et al., 2014).

Table 1. Treatment Options for Weight Loss.

Treatment Type	Description	Effectiveness	Recidivism
Traditional reduced-calorie diet	Intake is reduced by 500- 1,000 cal/d	Low-moderate	High
Exercise	Calorie expenditure is increased mainly through aerobic exercise (250-300 min/wk).	Low	Moderate-high
Partial meal replacement diet	Pre-packaged foods fortified with vitamins and minerals replace traditional meals.	Moderate	Moderate-high
Very-low-calorie diet	Medically supervised diets use only partial meal replacements and provide ≤ 800 cal/d.	Moderate-high	High
Pharmacotherapy	The mechanism depends on the pharmacological agent (e.g., a sympathomimetic, lipase inhibitor, serotonergic) used	Low-moderate	High
Weight-loss surgery	The stomach volume and/or the absorption of food are reduced through surgery.	High	Low-moderate
Adapted from Casazza et al., (2013)			

Conventional Weight-Loss Approaches

Reduction of food intake alone or increasing exercise alone usually is not effective, particularly in the long term. Though, clinically significant weight loss of greater than 10% can be achievable and maintainable if both are completed together and behavioural approaches are applied. Contemporary recommendations include daily dietary restrictions of 500 to 1,000 Kcal under resting metabolic rate and 60 to 90 minutes of aerobic exercise 5 days per week. Although the model of generating an energy deficit by increasing energy expenditure and decreasing energy intake appears achievable, clients endeavouring to achieve weight loss struggle with various barriers, including their own body's compensatory methods in response to an energy deficit, environmental cues, and an absence of understanding concerning the requirements required to attain weight loss.

When people restrict energy intake, their body takes compensatory actions to decrease energy expenditure and increase appetite. Likewise, as an individual loses more weight, the outcomes of weight-bearing exercise are reduced (e.g., fewer calories are burned while walking because the individual has less weight to support). These alterations in energy requirements are dissimilar to the stationary 3,500 kcal = 1 lb law. This means that instead of a weight loss of approximately 10 lb (4.5 kg)/per year through reductions of 100 kcal/d, the real weight loss would be much less (Hall et al., 2012). The authors produced a computer model to estimate the energy balance variations that happen over time and the expected weight loss based on calories restricted and expended.

These alterations do not reduce the functions of diet and exercise. However, because these compensatory mechanisms can lead to deterrents in people, the exercise specialist instructor (EPI) must provide genuine expectations and effective behavioural approaches. One of the most often theories associated with weight-loss success is self-monitoring (Carels et al., 2005). A study by Guare et al., (1989) found that subjects who kept food diaries more frequently maintained weight loss better (−18 kg) for 1 year compared with those who did not (−5 kg). Supporting evidence from a study by Carels et al., (2005) on 40 middle-aged males and females reported that the use of an exercise diary was linked with less related problems in exercise (r = −.48),

greater weight loss (r = .44), and greater amounts of weekly exercise. An additional mode of self-monitoring is recording a daily weight (Jeffery, 2004). Jefferey (2004) noted that stepping on the weighing scale is one of the strongest predictors of success. Conversely, behaviourists stressed that repeated weighing should be discouraged by those who are concerned that clients will be dejected in weight reduction does not transpire.

Example of Two Lifestyle Weight-Loss Trials

A reproach of lifestyle interventions for weight loss is that long-term adherence is generally poor. Though recidivism and weight regain are challenges for lifestyle interventions, the Diabetes Prevention Trial, and Look Ahead Trial both showed that long-term weight loss and clinical improvements are achievable.

The Diabetes Prevention Trial assessed the incidence of new diabetes incidents by comparing the outcomes of lifestyle changes with those of metformin (diabetic medication) over approximately 3 years. The lifestyle intervention included a 16-week individualised program covering diet, exercise, and behavioural modification to achieve more than 7% weight loss and 150 minutes per week of exercise (Figure 2) This strategy was compared with the metformin group and placebo group, the lifestyle intervention group lost more weight, exercised more, and reported eating fewer calories. Furthermore, the incidence of diabetes was 58% lower in the lifestyle group compared with the placebo group but only 31% lower in the metformin group compared with the placebo group.

The Look Ahead Trial suggested that a lifestyle intervention program involving restricted caloric intake and increased physical activity had significant health benefits in overweight individuals with type 2 diabetes. Particularly, those in the lifestyle intervention group exhibited greater weight loss, greater fitness, and lower levels of glycated haemoglobin compared with the control group. When comparing weight loss, the lifestyle intervention group achieved nearly 6% total weight loss at 4 years, and greater than 40% of subjects maintained at least 10% weight loss. Both trials support the view that alterations in individuals' diet and exercise behaviours can be sustainable and lead to significant improvements in their health.

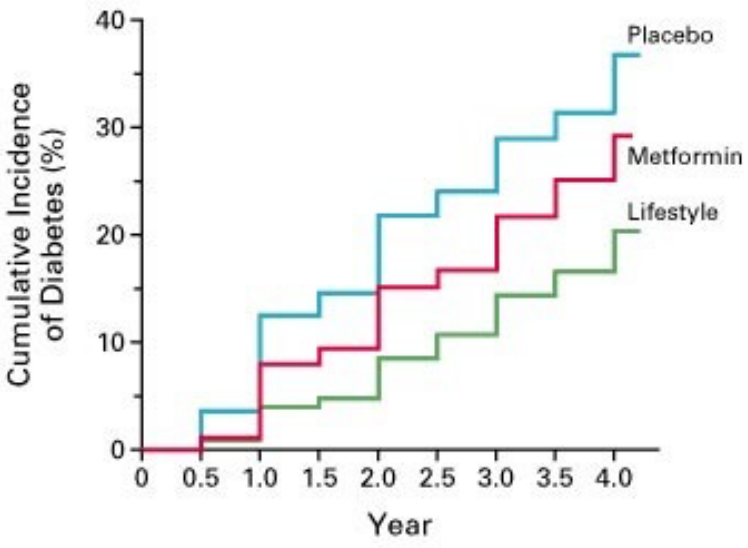

Figure 1. Results from the Diabetes Prevention Trial. Compared with a placebo and the common diabetes drug metformin, lifestyle changes (which included exercise and a weight loss of 7%) most lowered the risk of developing diabetes.

Very-Low-Calorie and Partial Meal Replacement Diets

Very-low-calorie diets (VLCD) usually are administered under medical supervision and comprise supplements fortified with vitamins and minerals (e.g., shakes, and bars). The total daily caloric intake for an individual on a VLCD differs depending on their estimated resting metabolic rate but is approximately 800 Kcal per day (Hall et al., 2012). These supplements usually are high in protein (70-100g per day) and low in carbohydrates (<80 g/d) (Tsai and Wadden, 2006). Detractors of VLCD plans largely involve safety and weight regain.

In the 1970s, there were recounted fatalities of people who experienced significant weight loss while on VLCDs. Features that contributed to these deaths were possibly the poor-quality protein (i.e., hydrolysed collagen) and incomplete supplementation of vitamins and minerals (Thorland et al., 1991). While current VLCD strategies do marginally increase the risk of dehydration, hair loss, and cholelithiasis, the risk of significant side effects is extremely low, especially when people follow the strategy under medical supervision (Tsai and Wadden, 2006). Like conventional low-calorie and exercise methods for weight loss, weight regain is a concern with VLCD procedures. Though, contrary to a commonly held notion, individuals who lose weight rapidly through a VLCD do not end up weighing more (i.e., are heavier than when they started) than those individuals who lose through more conservative means (Casazza et al., 2013).

A meta-analysis performed by Johansson et al., (2014) stated that weight regain can be reduced after a VLCD strategy with partial meal replacements. Fundamentally the same as VLCD supplements, partial meal replacements are supplements that are fortified with vitamins and minerals and normally are high in protein. Though, unlike VCLDs, which are consumed exclusively, partial meal replacements usually are consumed once or twice daily with normal food. Partial meal replacements were consumed during the Look Ahead Trial and were discovered to be a strong predictor of long-term weight-loss attainment (Johansson et al., 2014).

Pharmacological Options for Weight Loss

Despite what media and tv advertisements assert concerning weight-loss products, no 'magic' pill or product existing can yield significant weight loss (i.e., >10%). As discussed previously, trial agents targeting leptin and ghrelin are presently being developed along with other potential treatments, in the pursuit of discovering a more effective and long-term response to obesity. Presently, there is a small quantity of FDA-approved drugs for weight loss. While these agents have indicated modest weight loss (2%-10%), their use normally is suggested in concert with a low-calorie diet and an exercise program for improved weight loss or weight management (Johansson et al., 2014).

An example of an FDA-approved drug is Orlistat, which has been available since 1999 and is a lipase inhibitor. Because Orlistat inhibits the absorption of fat into the intestines, it is consumed with food and does not apply to people who consume a low-fat diet. In comparison with a placebo drug, Orlistat has been shown to produce a 2.9-kg weight loss with 21% of subjects attaining at least a 5% weight loss, and 12% achieving a 10% weight loss (Rueda-Clausen and Padwal, 2014). The reported side effects from taking this drug include malabsorption of fat-soluble vitamins and gastrointestinal symptoms including flatulence, steatorrhea, and faecal incontinence. As a result of the side effects compliance with Orlistat can be reduced.

Table 2. Prescription Medication for Obesity in the United Kingdom.

Names	Desired effect	Potential side effects relating to the exercise
Anti-obesity drugs Orlistat	Reduces the absorption of dietary fat	Liquid oily stools, faecal incontinence, fatigue, anxiety, hypoglycaemia*
*Hypoglycaemia: see diabetes mellitus sections for directives designed to reduce the risk of hypoglycaemia during exercise. Note: An anti-obesity drug is only usually prescribed for those with a BMI of 30kg/m^2 or greater. Appetite suppressants, such as Sibutramine, are now deemed to be unsafe due to serious cardiovascular side effects (March 2010).		

Another agent that has been approved is Phentermine-ER topiramate which is a newer weight-loss drug that contains a sympathomimetic (i.e., phentermine) and an

antiepileptic agent (i.e., topiramate). Though the use of topiramate as a weight-loss drug is new, phentermine has been used as an antiobesity drug since 1959. Phentermine is thought to suppress appetite through direct action on the hypothalamus. A randomised trial of phentermine-ER topiramate showed about a 10-kg weight loss compared with a placebo. However, similar to Orlistat, there was a high rate of attrition for taking on the drug.

Sibutramine is another sympathomimetic drug with mechanisms like those of phentermine. However, in 2010 it was removed from the market because of an elevated risk of stroke and heart attacks. Moreover, because phentermine can perform as a sympathetic agonist and lead to increases in heart rate and blood pressure, further care and attention are suggested for people living with hypertension or potential heart conditions.

Weight-Loss Surgery

When considering weight-loss effectiveness and lasting maintenance, bariatric surgery has the strongest scientific evidence of all currently available treatments. Deitel, Gawdat, and Melissas (2007) performed a meta-analysis of studies reporting long-term follow-up (i.e., ≥2 years) stating that >50% excess weight loss in most bariatric studies. Furthermore, percentage excess weight is the mode of reporting weight loss in surgical subjects and is founded on how much weight an individual has lost compared with their optimum weight.

Percentage Excess Weight Equation

- **[(OPW-FW)/(OPW-IDW)] × 100**

Where OPW = operative weight; FW = follow-up weight; IDW = ideal weight (based on the 1983 Metropolitan Insurance height and weight tables)

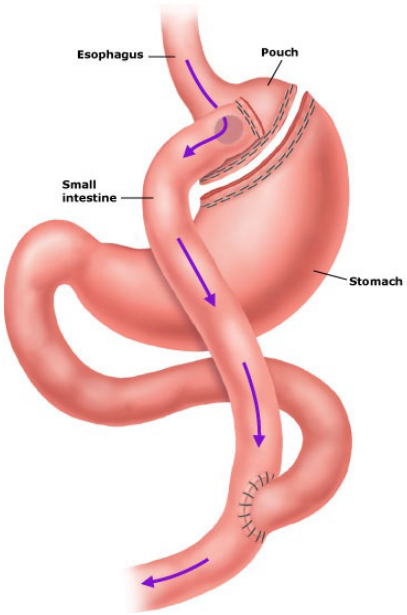

Figure 2. The Roux-en-Y Gastric Bypass.

In the early 1990s, almost 16,000 weight-loss surgeries were implemented annually. This is in contrast, by the mid-2000s the number of surgeries in the United States rose to > 200,000. The most common type of weight-loss is the Roux-en-Y gastric bypass (RYGB) surgery (**Figure 2**), a method that substitutes the stomach

[which has a typical volume of 0.59 litres)] by partitioning a 29.6 mL pouch from the stomach. This helps in limiting the amount of volume and therefore calories one can intake in a single meal. Moreover, the new pouch is enclosed further away from the intestines, which circumvents some digestive enzymes and diminishes total absorption. Another type of surgical operation, known as sleeve gastrectomy, shrinks the stomach to almost the size of a banana (**Figure 3**).

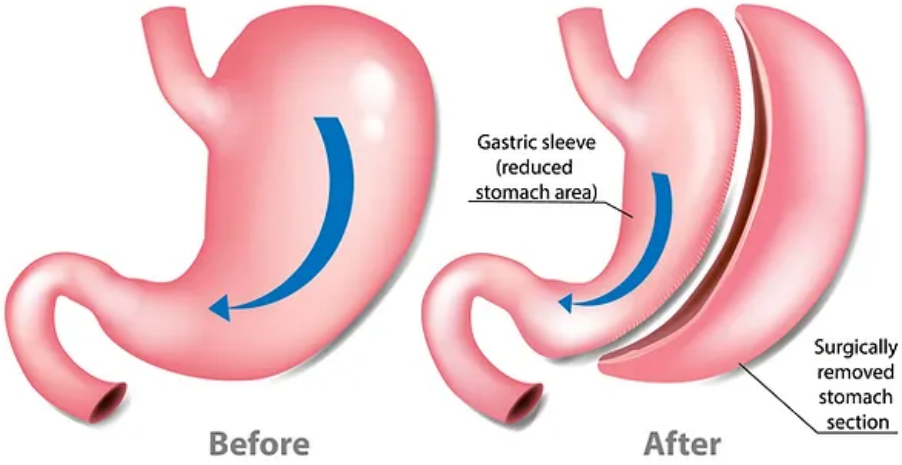

Figure 3. Gastric Sleeve Surgery.

Evidence suggests that bariatric procedures led to superior long-term weight loss and in some cases reduced cardiovascular risk factors (e.g., hypertension, and diabetes). The remission of diabetes, which can be assessed by how many diabetic subjects no longer need insulin or oral diabetes medications, frequently is a stated benefit of weight-loss surgery. Courcoulas et al., (2014) performed a randomised study comparing RYGB surgery against lifestyle interventions for weight loss and reported that subjects with type 2 diabetes had either partial (50% of RYGB subjects) or complete (17% of RYGB subjects) remission of diabetes compared with no remission (partial or complete) in individuals who were randomised to the intensive lifestyle intervention.

Despite the benefits of bariatric surgery, there are associated risks. For instance, changing open surgical methods with less invasive laparoscopic procedures has reduced serious adverse incidents and enhanced recovery time. According to Colquitt et al., (2014), the side effects particular to weight-loss surgery (e.g., leakage,

infection, obstruction, pouch dilatation) differ depending on the type of surgery and the risk profile of the individual undertaking the procedure. Furthermore, a study by McCullough and associates (2006) reported that presurgical fitness (i.e., a peak VO_2 >15.8 $mL \cdot kg^{-1} \cdot min^{-1}$) was linked with fewer problems, reduced hospital stays, and reduced 30-day hospital re-admissions.

It is important to appreciate that undergoing any form of weight-loss surgery does not reduce the significance of exercise, healthy eating, and behavioural modifications because weight regains can happen in this populace. Exercise after bariatric surgery has been shown to improve fat oxidation and inhibit weight regain (Berggren et al., 2008). A conceivable mechanism behind the suggested benefits of exercise in this population group is the loss of muscle that occurs. Research has shown substantial loss of muscle mass and strength after weight-loss surgery. To reduce some of the muscle loss, resistance training 2 to 3 d/ wk. with > 250 minutes of aerobic exercise has been suggested. However, it is unlikely that a well-designed resistance training intervention can entirely counteract this loss.

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30-DAY
DAY 14
MEASUREMENTS:
Arms:
Stomach:
Waist:
Weight:
Breakfast:
Lunch:
Dinner:
Snack:
Water:
Biggest change today:
Biggest change so far: