College of Family Physicians Singapore

The Singapore Family Physician

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Basic Obesity Management Accreditation 6



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GLP-1 RA, glucagon-like peptide-1 receptor agonist; MASH, metabolic dysfunction-associated steatohepatitis; NAS, NAFLD activity score; NAFLD, non-alcoholic fatty liver disease; SoC, standard of care; Wk, week.

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EDITORIAL
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Basic Obesity Management Accreditation 6

Dr Chiang Shu Hui Grace

SFP2026; 52(5)

Obesity is a chronic multisystem disease and serious global public health threat associated with increased morbidity and premature mortality.[1,2] Obesity and its determinants are risk factors for three of the four leading causes of non-communicable diseases (NCDs) worldwide, including cardiovascular diseases, type 2 diabetes, and certain cancers.[3,4] The prevalence of overweight and obesity has nearly tripled over the past 40 years, reaching epidemic proportions globally.[3] By 2025, the Global Burden of Disease data suggests that nearly 268 million children and adolescents across 200 countries will be overweight, while 124 million will have obesity.[7] Obesity has now emerged as a major chronic disease and societal burden[4] that has detrimental consequences on population health and well-being, with at least 2.8 million people dying each year as a result of being overweight or obese.[3] The annual cost of obesity is estimated to be about US$2 trillion,[5] representing 2.8 percent of the world's GDP.[6]

Obesity is broadly defined as excess body weight for a given height. The pathogenesis of obesity is complex, involving a combination of environmental, sociocultural, physiological, medical, behavioural, genetic, epigenetic, and numerous other factors contributing to causation and persistence.[8]

Effective and adequate obesity management requires a multidisciplinary and transdisciplinary approach.[9] While individual and medical management is important, multiple sectors of society will have to be engaged and involved to effect change in the multiple external factors that influence obesity such as food systems and food environments, access to health services, education, and public policies.[4]

This issue will provide an update on the latest evidence-based treatment options in basic obesity management.

In Unit 1, Drs Tham Kwang Wei and Benjamin Lam offer a concise explanation about the biology of weight regulation as a basis for understanding obesity as a disease and detail the complex and multifactorial pathogenesis of obesity.

In Unit 2, Drs Amanda Lim and Benjamin Lam elaborate on how to approach a patient with obesity through a practical 5As framework (Ask, Assess, Advise, Agree, and Assist) for obesity counselling.

In Unit 3, Ms Izabela Kerner discusses the various evidence-based dietary interventions for clinical practice.

In Unit 4, Mr Muhammad Jazimin Bin Haron provides a review of the weight management and health benefits of physical activity and exercise on obesity.

In Unit 5, Dr Tham Kwang Wei writes about the general approach to pharmacotherapy in obesity management and the various anti-obesity medications currently approved.

In Unit 6, Dr Shanker Pasupathy discusses the indications of bariatric surgery and provides a case for bariatric surgery as a viable treatment option in obesity.

In Unit 7, Drs Elaine Chew and Chin Xinyi provide a comprehensive approach to understanding and managing childhood and adolescent obesity.

In Unit 8, Mr Adrian Toh addresses the barriers to lifestyle intervention and how cognitive behavioural therapy can be effective in addressing unhealthy eating habits, lack of physical activity, and obesity.

In this issue, A/Prof Goh Lee Gan has selected 10 current readings on topics related to basic obesity management. These readings include articles on managing obesity in individuals with underlying comorbidities.

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- Unit 8: Understanding Matters of the Mind in Obesity

BASIC OBESITY MANAGEMENT ACCREDITATION 6
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Unit No. 1

UNDERSTANDING OBESITY: HOW AND WHY?

Dr Tham Kwang Wei, Dr Lam Chih Chiang Benjamin

ABSTRACT

Obesity is currently recognised as a chronic disease that is often relapsing and progressive. This requires attention for long-term monitoring to treat or prevent obesity-related complications. This article discusses the biology of weight regulation as a basis for understanding obesity as a disease, and to appreciate the complex and multifactorial pathogenesis of obesity. With this understanding of the factors involved in perpetuating obesity in different individuals, the approach to patients with obesity should be as comprehensive and systematic as with other chronic diseases. Management strategies and treatment plans, which need to have long-term weight maintenance and weight regain prevention in mind, can be more individualised for patients with obesity.

Keywords: Obesity, chronic disease, pathogenesis, body weight regulation, weight maintenance

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INTRODUCTION

Over the past 40 years, the prevalence of obesity has risen substantially in almost all world regions, such that there are now more than 600 million obese adults and 100 million obese children worldwide.[1] This increasing burden of obesity has been identified in Singapore and other Asia-Pacific countries.[2] From 1992 to 2010, the prevalence of obesity more than doubled from 5.1 percent to 10.8 percent. A parallel rise in the prevalence of diabetes mellitus has also been seen, reflecting the burden of obesity.[3] Based on body mass index (BMI) categories that indicate increased health risks in Asian populations, 52.5 percent of Singaporeans have a BMI in the moderate- to high-risk groups (≥23.0 kg/m^2), heralding the potential for a greater burden of adiposity-related comorbidities in Singapore.[3]

Obesity has often been viewed as a result of a simple lack of self-discipline and willpower to "eat less and move more" or plain laziness on the part of the person with obesity.[4] This oversimplified perception results from the lack of understanding that beyond the passive accumulation of excess energy as adiposity, obesity is a chronic, often relapsing, and progressive disease resulting from a multitude of factors. It has also led to weight bias and obesity stigma against people living with obesity (PwO), including by healthcare professionals (HCPs).[4] Despite being at further risk of many obesity-related diseases, PwO often do not seek medical attention early, and the weight bias by HCPs also impedes them from providing quality care.[4]

As with many chronic diseases, the pathogenesis of obesity involves many roots (aetiologies and associated factors). We now understand that there is a complex interplay of physiologic, genetic, epigenetic, and developmental factors with variables of behavioural, psychological, socioeconomic, medications, and environmental ones, both intrinsic and extrinsic, leading to the pathogenesis and perpetuation of obesity.[5] Only by addressing these roots and understanding obesity as a chronic condition can we adequately manage and prevent obesity and its multitude of associated costly comorbidities.[4,5]

BIOLOGY OF WEIGHT REGULATION

The body's adipose tissue represents energy stores to survive energy-scarce conditions. Hence, it would not be surprising that body weight (or more accurately, adipose tissue in the body) is tightly regulated by an extremely complex neuroendocrine energy balance circuitry. This circuitry is composed of specific nuclei in various brain regions, most prominently the hypothalamic arcuate nucleus (ARC), the paraventricular nucleus, the lateral hypothalamic area, and the nucleus of the solitary tract of the hindbrain (refer to **Figure 1**).[6,7]

Under relatively constant environmental conditions, this regulatory system senses and processes various metabolic signals regarding the current energetic status and adjusts the metabolic responses to maintain a stable weight without conscious control.[6] This homeostatic regulation of body weight is similar to that of other physiologic parameters, such as body temperature, blood pressure, or blood glucose, where a "set point" seems to exist, and deviation from this "set point" elicits a compensatory response in the opposite direction to restore this "set point". Therefore, weight regains after weight loss is actually physiological[8,9] and not necessarily due to a failure of conscious efforts (to lose weight). For example, energy expenditure is reduced in response to weight loss, in an effort to resist further weight loss so that the "set point" can eventually be restored. However, in PwO, it has been observed that such responses can go beyond what is expected of the weight loss, and based on experimental data, these responses can even persist for years despite weight regain, further predisposing the individual to further weight (re)gain.[5,8,9]

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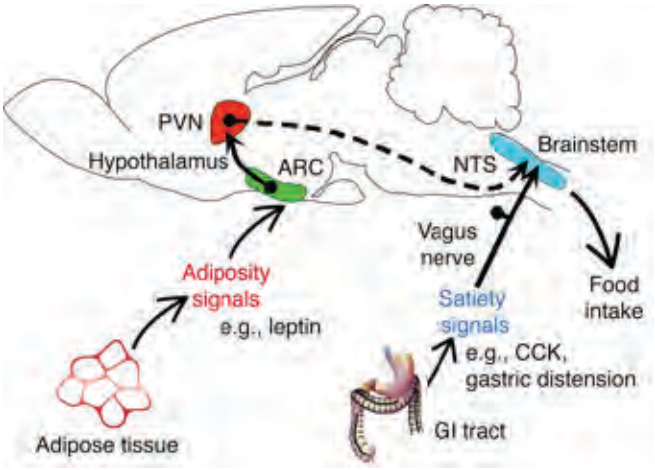
Figure 1. Model for regulation of the hindbrain response



Additionally, a different set of neuroendocrine signals guide food intake based upon the reward value of the food, also known as the reward or "hedonic" system.[6,10] The brain regions responsible for this reward system are dispersed in the corticolimbic structures. A primary characteristic of this system is its ability to override the signals from the homeostatic circuits as described.[6] Hence, the reward system is non-homeostatic regarding energy balance. This system integrates basic midbrain and hindbrain functions with more complex cortical functions involving arousal at the sight of palatable food items and the procurement of food, mediating the "liking" (level of pleasure or reward) and "wanting" (the motivation or drive to consume food), which are subconscious processes.[6] In human studies, functional MRI (fMRI) studies have shown overactivation of reward-encoding brain regions and/or deficiency in cortical inhibitory networks in PwO.[6,10]

OBESITY AS A DISEASE: ABNORMAL PHYSIOLOGY AND ROOTS OF OBESITY

With the understanding of the biology of weight regulation, obesity is now understood to signify abnormal physiology whereby there has been a surplus intake of energy and an elevated body weight set point is now defended.[5,8,9] A mismatch of as little as 3 percent can lead to a weight gain of 1–2 kg per year and, if persistent over the years, can ultimately result in severe obesity.[5] The factors are known to cause this are complex and multiple. They range from genetic to socioeconomic to environmental and emotional factors that are well-known to be potent modulators of appetite and energy expenditure.[7] Twin, family, and adoption studies show that the rate of heritability of BMI is high, ranging from 40 to 70 percent,[11] demonstrating a major genetic component.

In addition to syndromic and monogenic forms of obesity (e.g., MC4R mutations, leptin deficiency), which account for less than 5 percent of general obesity in adults, genome-wide association studies (GWAS) have identified more than 700 independent loci associated with BMI and/or obesity without.[12–14] These, however, attribute to less than 5 percent of the inter-individual variation in BMI and traits linked to obesity. It is more likely that the presence of a combination of various gene alleles (giving rise to a high polygenic risk score) and epigenetic factors make one susceptible to weight gain in a conducive obesogenic environment (gene-environment interaction).[11,14]

Maternal obesity, malnutrition, gestational weight gain, or weight loss, especially in early pregnancy, have been found to result in childhood adiposity, adverse cardiometabolic profiles, and insulin resistance, which may persist into adulthood.[15] The adverse effect of maternal obesity and metabolic ill-health on offspring have been postulated to be mediated through epigenetic modifications, which alter gene function without any DNA defects.[11] In the Growing Up in Singapore Towards healthy Outcomes (GUSTO) study involving a cohort of nearly 1,000 mother-offspring sets, greater maternal adiposity and higher polygenic risk scores linked to maternal obesity were associated with higher birth weights and early childhood adiposity. This suggests that prenatal genetic influences and epigenetic factors can influence childhood adiposity, which can be lasting.[16] Intra-uterine exposure to endocrine-disrupting chemicals has also been postulated to be associated with childhood obesity.[5]

Environmental and lifestyle factors favouring a positive energy balance and weight gain include increasing per capita food supplies and consumption, particularly of highly processed, energy-dense, and palatable foods that are often served in large portions. These factors are influenced by one's socioeconomic situation, such as decreasing time spent in occupational physical activities, displacement of leisure-time physical activities with sedentary activities such as television watching, use of electronic devices, growing use of medicines that have weight gain as a side effect, stress, and inadequate sleep.[11] More recent studies have identified a potential role for the gut's microbial content in determining a broad range of metabolic abnormalities, including obesity.[17,18] The evidence supporting causation includes animal studies that show that obesity, as a phenotype, is transmittable via the transfer of gut microbiota from the obese (mice/humans) to germ-free mice,[19,20] and mechanistic studies that demonstrate the possible mechanisms linking the gut microbiota with obesity.[17,21]

Apart from the mentioned factors mentioned that are linked to the pathogenesis of obesity, numerous other factors contribute to or exacerbate obesity and may lead to the attenuation of obesity treatment (refer to **Figure 2**). These should also be considered and adequately addressed when assessing and managing patients with obesity.[22]

OBESITY AS A DISEASE: HEALTH CONSEQUENCES

Obesity is not benign. The failure of adipose tissues to continually expand lead to pathological changes in the adipose tissue, which is characterised by macrophage invasion and/or increased release of pro-inflammatory adipokines and decreased release of anti-inflammatory adipokines such

Figure 2. ROOTS of Obesity Fact Sheet. World Obesity Federation 2021[22]



as adiponectin (refer to **Figure 3**).[23] In addition, this failure to further expand and act as a "metabolic sink" results in harmful ectopic fat deposition in lean tissues such as the heart, liver, pancreas, and kidneys.[23] These two phenomena contribute to a pro-inflammatory and insulin-resistant milieu, giving rise to metabolic complications such as type 2 diabetes mellitus (T2DM), non-alcoholic fatty liver disease (NAFLD), and cardiovascular disease (CVD).[23,24] Additionally, the mechanical forces resulting from excessive adipose tissue can give rise to biomechanical consequences (such as Obstructive Sleep Apnoea (OSA) and low back pain), and obesity as a condition has been associated with various psychosocial issues, impacting mental health.[25]

All these adverse consequences affect the quality of life, increase healthcare costs, and increase mortality.[26] Therefore, based on the current knowledge that the development of obesity results from abnormal physiology, with attending health consequences (complications, morbidities, and mortality), obesity fulfils the criteria for a disease state and is now determined to be a disease[27] rather than just a lifestyle risk factor. Several associations and organisations, including the World Health Organisation (WHO), have now declared obesity as a disease (refer to **Figure 4**). This is an important first step to tackling the problem of obesity, which has emerged as an epidemic that poses an unprecedented public health challenge.[27]

Figure 3. Pathological changes in adipose tissue

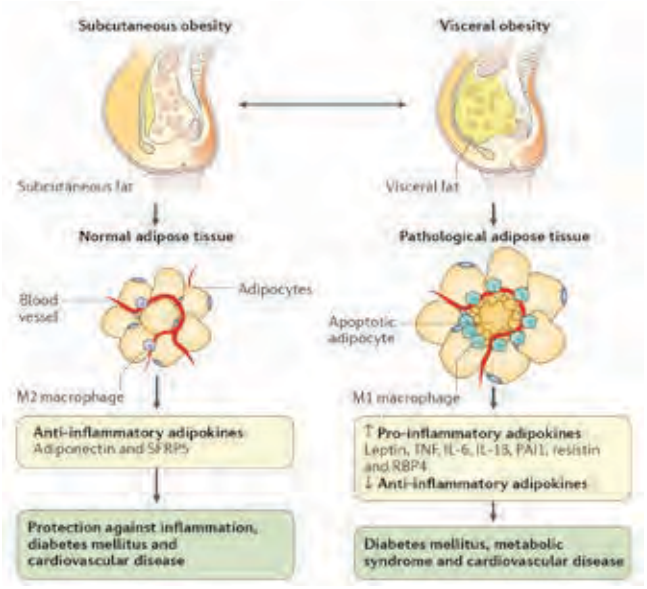


Figure 4. Organisations that have declared obesity as a disease



Box 1
Associations or organizations that have declared obesity is a disease

- National Institutes of Health
- US Food and Drug Administration
- Federal Trade Commission
- American Medical Association
- World Health Organization
- American College of Physicians
- American Association of Clinical Endocrinologists
- American College of Cardiology
- The Endocrine Society
- American Academy of Family Physicians
- Institute of Medicine
- The Obesity Society
- World Obesity Federation
- American Heart Association
- American Diabetes Association
- American Academy of Family Physicians
- American Society for Reproductive Medicine
- American Urologic Association
- American College of Surgeons

Data from Kahan S, Zvenyach T. Obesity as a disease: current policies and implications for the future. Curr Obes Rep 2016;5(2):291–7, and Bray GA, Kim KK, Wilding JPH. Obesity: a chronic relapsing progressive disease process. A position statement of the World Obesity Federation. Obes Rev 2017;18(7):715–23.

APPROACH TO MANAGEMENT OF OBESITY AS A DISEASE AND ITS COMPLICATIONS

Recognising obesity as a disease is a pertinent first step in the management of PwO. This will aid assessment using a systematic approach similar to how we approach any chronic disease and devise management plans from an aetiologic perspective.[22,27] As with any disease state, its management requires an understanding of how severe the disease is.[28]

For obesity, management guidelines have slowly moved from a BMI-centric approach, where the goal of therapy is to lose a given amount of weight (e.g., 5–10%), to a complications-centric approach, where weight is no longer the major determinant of appropriate treatment, but now based on the risk, presence, and severity of obesity-related complications.[28,29] For example, at least 10 percent weight loss is needed to improve NAFLD and OSA significantly.[28,29] Hence, for a person with multiple complications, including NAFLD and OSA, modest weight loss (defined as 5–10% weight loss) may be inadequate, and more aggressive treatment options effecting more than modest weight loss need to be considered. Although more aggressive treatment may involve higher risk, the benefit of treating the various obesity-related complications should outweigh this risk. Therefore, the main goal of therapy now is to treat or prevent obesity-related complications, rather than to purely lose weight per se.[28]

IMPORTANCE OF A MULTI-LEVEL AND INDIVIDUALISED MULTI-PRONGED APPROACH TO TREAT OBESITY

It is now known that the simple calculations underlying the traditional adage of "eat less, exercise more" are fatally flawed.[30] Aiming for a 500 kcal deficit (energy expenditure more than energy intake) per day, cumulating to 3,500 kcal per week (equivalent to ~0.5 kg of fat) will not result in a 0.5 kg/week weight loss indefinitely, because this calculation does not consider the homeostatic mechanisms that will resist further weight loss, and in fact will conspire to regain weight to restore the original "set point".[8,9,30] It is also important to note that the same diet and exercise plan (often prescribed once in the beginning) will not suffice to maintain that 500 kcal deficit per day as a declining weight will mean declining energy expenditure.[5,30] Nonetheless, the point here is that asking all obese people to just "eat less and exercise more" overly simplifies the obesity problem.[4,27]

Understanding the biology of weight regulation and the appreciation of the complex and multifactorial nature of how this regulation can go wrong resulting in obesity would indicate that there is no one-size-fits-all intervention or solution.[31] Considering the different (roots) factors that lead to weight gain in different individuals (e.g., sleep disruption in one patient, stress-eating or medications causing weight gain in another) would necessitate a multi-level and individualised multi-pronged approach to treating obesity. Multi-level, apart from the individual, would include the social and community, physical (environment), and economic levels of interventions,[31] while a multi-pronged approach at the individual level would encompass not just the lifestyle and behavioural modifications but also the possible combination with pharmacologic and even bariatric surgical procedures based on individualised risk-benefit assessment.[28,32,33]

Since obesity is a chronic, often relapsing, and progressive disease, weight regain ("relapse") after weight loss is common, in part consequent to the physiologic counter-response to negative energy balance. Long-term follow-up for monitoring of weight regains and obesity-related comorbidities is necessary and prudent.[28,32] Management strategies for weight maintenance/weight regain prevention of at least 6–12 months should be considered during weight loss treatment, understanding that there is also reduced adherence to lifestyle changes with time.[30,33] Some of these measures might involve staying in frequent contact with the patient even after weight loss is attained (e.g., once a month), long-term use of anti-obesity medications, initiation of anti-obesity medications after weight plateau with lifestyle changes, and/or intermittent use of very low- or low-calorie diets.[30,32,33]

Obesity stigma and discrimination that PwO face can be pervasive and poses an often unrecognised detrimental effect on their mental and physical health. This extends to the workplace, schools, healthcare settings, and social circles. As HCPs, understanding obesity as a disease and its causes (roots) and refraining from using language and narratives that unfairly stereotype our patients with obesity as unmotivated and lazy is a form of addressing the obesity stigma and helping our patients overcome this discrimination. Educating patients and their families on the nature of obesity as a disease will also help to fight weight bias and stigma. Compositely, such measures can help PwO obtain the quality healthcare they need.[4,27]

Lastly, obesity prevention remains key to reducing the burden of disease associated with obesity as a population moving forward.[31] Primary care practitioners are often the first point of patient contact. Initiating the conversation in obesity with patients and addressing childhood obesity are important roles primary care practitioners play in tackling the obesity epidemic. With obesity being prevalent in women in their reproductive years, the potential impact of maternal obesity on the adiposity and metabolic health of future generations may be colossal and under-addressed. While major public health measures to drastically reduce maternal obesity for a downstream impact seem unlikely at the present moment, HCPs can play an important role in educating women in their reproductive years to maintain a healthy weight to improve pregnancy outcomes and potentially the health of future generations.[15]

CONCLUSION

Obesity is today recognised as a disease and has been described as a complex, chronic medical condition with a major negative impact on human health.[27] A number of associations and organisations, including the World Health Organisation (WHO), have declared obesity as a disease, and this is an important first step in tackling the problem of obesity. Understanding the biology of weight regulation and the appreciation of the complex and multifactorial nature of how this regulation can go wrong resulting in obesity would indicate that there is no one-size-fits-all intervention or solution[31] and would necessitate a multi-level and individualised multi-pronged approach to treating obesity and its related conditions. Strategies for long-term weight maintenance, chronic follow-up with monitoring of weight regain and obesity-related diseases, and addressing the stigma and bias that PwO face are pertinent for successful obesity management. Primary care practitioners play a pivotal role in initiating conversations on obesity and addressing obesity prevention.

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LEARNING POINTS

- **Obesity is today recognised as a chronic disease that is complex and of a multifactorial nature.**
- **Understanding the various roots in the individual will allow interventions to be tailored to address these aetiologies and aggravating factors. There is no one-size-fits-all solution, and management necessitates a multi-level and individualised multi-pronged approach to treating obesity and its related conditions.**
- **Long-term follow-up is required for assessment of treatment, and monitoring of weight regain and obesity-related diseases. Alongside addressing the obesity stigma and bias, these practices are pertinent in managing and preventing obesity and its related complications.**

BASIC OBESITY MANAGEMENT ACCREDITATION 6
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Unit No. 2

A PRACTICAL APPROACH TO THE PATIENT WITH OBESITY

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ABSTRACT

The prevalence of obesity and obesity-related comorbidities is rising in Singapore and across the globe. Primary care physicians are at the frontline of healthcare and play a central role in the management of obesity. In this article, we discuss the 5As framework (Ask, Assess, Advise, Agree, and Assist) as a practical framework for obesity counselling, focusing on how to initiate the conversation and assess the person with obesity. The assessment includes taking a weight history, excluding secondary causes, understanding lifestyle factors contributing to weight gain, and assessing for complications of obesity. This assessment then makes possible subsequent patient engagement, which would include advising, agreeing (goal setting), and assisting the patient on an individualised care plan.

Keywords: Obesity, weight management, clinical examination, patient-centred care

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INTRODUCTION

The global prevalence of obesity more than doubled between 1990 and 2022, with 16 percent of adults with obesity in 2022.[1] In Singapore, the prevalence of obesity in adults aged 18 to 74 has risen from 10.5 percent in 2020 to 12.7 percent in 2024.[2] The impact of obesity is multisystemic and it is linked to many other diseases, including type 2 diabetes, cardiovascular disease, mood disorders, and certain cancers, resulting in reduced quality of life, higher healthcare costs, and increased mortality.[3]

Despite the serious multisystemic impact of obesity on health, according to international surveys and interviews, people with obesity might not perceive their weight to be a significant problem.[4,5] Even if they do, it might take several years of struggling with excess weight before they finally consult a healthcare professional.[4] Therefore, the primary care physician, being at the frontline of healthcare, has a key role in identifying the person with obesity and broaching the topic of weight management. Additionally, counselling delivered by primary care providers can positively impact their patients.[5] In a meta-analysis of 12 studies involving 207,226 individuals, those who were provided with weight loss advice by their primary care provider were nearly four times more likely to attempt weight loss.[6] However, observational and self-report evidence suggests that less than half of patients with obesity are advised by their physicians to lose weight.[4,7] Primary care physicians often cite lack of motivation by the patient as the main reason for not discussing weight during consultations.[4,5] Other barriers described include limited understanding of obesity care, concern about negative consequences of raising a sensitive topic, and limited time and resources.[7]

On the other hand, patients with obesity do believe that it is the responsibility of their primary care physicians to initiate the conversation about weight management and would like them to do so.[4] Only 3 percent were offended by such a conversation, according to an online survey conducted in 11 countries involving 14,502 participants.[4] This survey also showed that nearly half of them said they were motivated to lose weight, with >80 percent of the total participants saying they had made at least one serious weight loss effort in the past.[4] These results should reassure primary care physicians that a sizeable population with obesity is willing to receive help for their weight.

Hence, this article aims to reiterate the central role of the primary care physician in the management of obesity and discuss how to initiate the conversation and assess the person with obesity.

5AS FRAMEWORK FOR OBESITY COUNSELLING

A practical framework for obesity counselling is the 5As framework (*Ask, Assess, Advise, Agree, and Assist*), a universal approach to encouraging behavioural change, which was initially developed to support smoking cessation (refer to **Figure 1**).[8] This intervention strategy takes the individual's perceived need as the starting point, which then makes it possible to implement a process of care that is individualised and patient-centred.[9]

A 2014 study found that this framework was easy to implement, doubled the initiation of obesity management in primary care clinics, and resulted in positive behavioural health changes for patients.[10] Another study found that patients managed by internal medicine residents trained in the 5As framework lost a mean of 1.53 kg at 12 months while those managed by non-trained residents gained a mean of 0.30 kg. This difference was statistically significant.[11]

However, while this approach seems simple, it should not be applied simplistically. The success of this framework rests on a strong therapeutic relationship between the physician and patient, where the physician can guide the patient through this journey of behavioural change at a pace that is suited to the patient's situation.[9]

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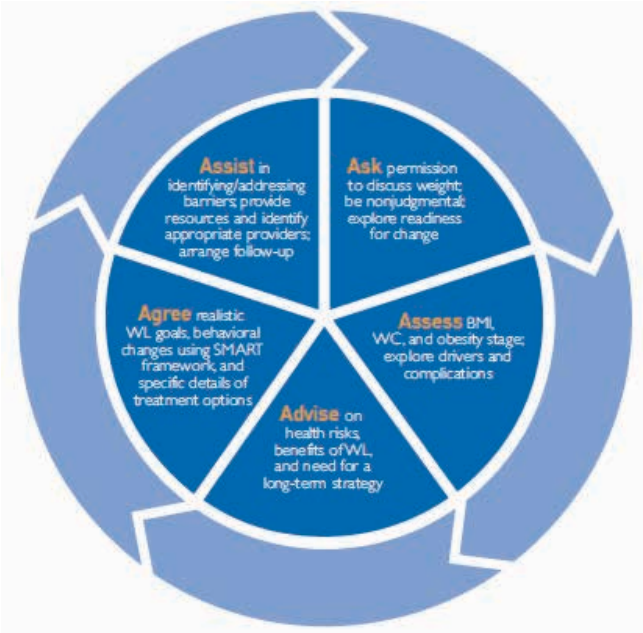
Figure 1: The 5As framework for obesity counselling[8]



BMI = body mass index
SMART = specific, measurable, achievable, relevant, time-based
WC = waist circumference
WL = weight loss

Ask: Initiating the Conversation

Communication is a fundamental aspect of obesity management. The first A (Ask) involves seeking permission to talk about obesity and its management, especially if the "reason for encounter" is not related to weight. Based on a local qualitative study, primary care physicians tend to do this by presenting the patient's anthropometric measurements and getting them to reflect on the weight instead of directly asking for permission.[12] When a patient presents with a problem that could be attributed to weight, primary care physicians usually have no issues initiating the conversation.[5] However, the physician should be cautious about focusing on the patient's weight at the cost of neglecting to explore other possible causes of the problem.[13,14]

Regardless, there are two main points to highlight regarding a successful conversation about obesity. First, the physician should communicate in a non-judgemental, respectful manner, with empathy, and without any bias, especially since evidence now suggests that obesity has a complex aetiology that may not fully be under voluntary control. Patients with obesity have been described as lazy, non-compliant, weak-willed, unintelligent, and even dishonest by healthcare professionals, and this stigmatisation only exacerbates the problem.[15] Hence, the physician should take care not to use inappropriate or hurtful words and use people-first language ("patient with obesity") as opposed to condition-first language ("the obese patient"). This is recommended for other medical conditions as well.[16]

Second, in order to have a motivating conversation so that behavioural change can be elicited, the physician should employ motivational interviewing (MI).[17,18] MI is a patient-centred, goal-directed counselling technique aimed at increasing intrinsic motivation for change by exploring and resolving ambivalence to change. MI's core principles include expressing empathy, developing discrepancy (between patient's goals and current behaviour), rolling with resistance, avoiding argumentation, and supporting the patient's self-efficacy (empowerment); it involves **o**pen questions, **a**ffirming the patient's perspectives, **r**eflecting what was heard to ensure understanding, and **s**ummarising shared understanding to set specific goals (acronym OARS). In a meta-analysis of 11 randomised control trials involving 1,448 participants, MI significantly enhanced weight loss in overweight and obese patients.[17] Hence, MI is an effective counselling approach, which can be used within the 5As framework for obesity counselling.

Assess: History

History—An Obesity-Focused History

Once the patient is ready to engage in conversation about his or her weight, the physician can initiate an obesity-focused history taking. An obesity-focused history taking has the following aims:

- Assessing for secondary obesity and contributing factors of obesity
- Assessing for obesity-related comorbidities
- Gathering information to formulate a treatment plan

Weight History

In taking a weight history, the mnemonic "OPQRST" may be used to evaluate weight. This includes **o**nset, **p**recipitating events, **q**uality of life, **r**emedy, **s**etting, and **t**emporal pattern (refer to **Table 1**).[19,20] In addition, comprehensive history-taking also includes delving into the pattern of weight changes, duration of obesity, highest weight, previous weight loss attempts, and response to these attempts. If time permits, graphing the patient's weight over time and inserting events or treatments that they feel were temporally related to weight changes may be useful. This weight chart may reveal significant events that cause changes in weight trajectory, such as physiological changes (e.g., puberty, pregnancy, menopause), medical factors (e.g., diagnosis of medical disorders, initiation of medications), and social factors (e.g., change of job, marriage, divorce, grief, etc).

By using open-ended questions, the physician permits the patient to express their own weight journey and challenges. This also enables the physician to understand the patient's knowledge, attitude, and motivation, which would provide a basis for formulating a treatment plan.

Table 1: Using the mnemonic OPQRST to take a weight history[19,20]

	Sample Questions
Onset	"When did you first begin to gain weight?" "Have you struggled with your weight since childhood?" "What did you weigh in school, in college, in your early 20s, 30s, 40s?" "Did the weight gain begin when you started taking a certain medication?"
Precipitating	"What life events might have led to your weight gain—such as work stress, marriage, divorce, financial loss, a period of depression, the onset of an illness?" "How much weight did you gain with pregnancy?" "How much weight did you gain when you stopped smoking?" "How much additional weight did you gain when you started insulin?"
Quality of life	"At what weight did you feel your best?" "What is hard to do at your current weight?" "How does your weight affect how you feel and function?"
Remedy	"What have you tried in the past to control your weight?" "Which medications, if any, have you taken to help control your weight?" "What have you found to be particularly helpful when trying to lose weight?"
Setting	"What was going on in your life when you last felt in control of your weight?" "What was going on when you gained your weight?" "What role has stress played in your weight gain?" "Which people in your life, if any, have been helpful in supporting your efforts to control your weight?"
Temporal pattern	"What is the pattern of your weight gain?" "Did you gradually gain your weight over time?" "Are there large swings in your weight, and if so, what is the weight change?" "What was your lightest and heaviest weight as an adult?"

Assessing for Secondary Obesity and Contributing Factors

Secondary Causes

The physician should be aware of secondary causes of obesity. Secondary causes can be elicited in the history, physical examination, and initial investigations. Certain clinical features such as the early or sudden onset of obesity, temporal relationship to an event, or associated signs and symptoms of another medical condition (e.g., Cushing's syndrome, hypothyroidism) may suggest an underlying medical disorder. The onset of weight gain after initiating a medication may suggest drug-induced weight gain. Paediatric patients with extreme early-onset obesity (<5 years of age), severe hyperphagia, developmental delay, and/or dysmorphism will warrant a referral to a paediatrician.[21] The causes of obesity are summarised in **Table 2**.[22]

Table 2: Causes of obesity[22]

Causes of Obesity
Primary Genetic and epigenetic causes Monogenic disorders (e.g., MC4R mutation, Leptin deficiency, POMC deficiency) Syndromes (e.g., Prader-Willi syndrome, Bardet-Biedl syndrome)
Secondary Neurological Brain injury Brain tumour Cranial irradiation Hypothalamic obesity Endocrine Hypothyroidism[a] Cushing's syndrome Hypogonadism Insulinoma Psychological Depression[b] Eating disorders Drug-induced Anti-depressants Anti-psychotics Anticonvulsants Sulphonylureas Insulin Thiazolidinediones Steroids Oral contraceptives
[a]It is controversial whether hypothyroidism causes obesity or exacerbates obesity [b]Depression may be associated with overeating or binging

Table 3: STOP-BANG Questionnaire[27]

Snoring	Do you SNORE loudly (louder than talking or loud enough to be heard through closed doors)?	☐ Yes	☐ No
Tired	Do you often feel TIRED, fatigued, or sleepy during the daytime?	☐ Yes	☐ No
Observed	Has anyone OBSERVED you stop breathing during your sleep?	☐ Yes	☐ No
Pressure	Do you have, or are you being treated for high blood PRESSURE?	☐ Yes	☐ No
BMI	BMI of more than 35 kg/m^2?	☐ Yes	☐ No
Age	AGE over 50 years old?	☐ Yes	☐ No
Neck	NECK circumference >40 cm?	☐ Yes	☐ No
Gender	GENDER: Male?	☐ Yes	☐ No
Scoring criteria: Low risk of OSA: Yes to 0–2 questions Intermediate risk of OSA: Yes to 3–4 questions High risk of OSA: Yes to 5–8 questions			

Lifestyle and Social Factors

Taking a dietary and physical activity history will enable the physician and patient to identify areas where changes can be made to improve health. Energy intake from self-reported dietary history is frequently underestimated, but it is an important starting point for awareness and self-reflection. A 24-hour diet recall may be used. Assessing the type of food, portion size, the timing of meals, and location of meals is useful in dietary assessment since disordered eating patterns such as night eating or emotional eating may be uncovered.

Physical activity is an important factor in weight loss maintenance. Furthermore, physical activity is also associated with decreased mortality independent of weight.[23] During the assessment of a person's baseline physical activity, the physician should also assess for any medical limitations for exercise (e.g., cardiovascular disease, respiratory disease, musculoskeletal problems) as this will influence exercise prescription. The use of smartphone apps and fitness devices for food and physical activity tracking might help patients monitor their dietary intake and physical activity, and their usage should be noted so that an individualised plan can be formulated.[24]

Disordered sleep patterns are associated with weight gain. Shift workers who have disrupted circadian rhythms have an increased risk of weight gain.[25] Disrupted sleep or poor sleep quality may also be due to obstructive sleep apnoea (OSA), which has a high prevalence in patients with obesity.[26] A useful tool in assessing for OSA is the STOP-BANG questionnaire to screen for OSA (refer to **Table 3**).[27]

Past Medical History

Many medical conditions are caused or contributed to by obesity. These include type 2 diabetes mellitus (T2DM), hypertension, dyslipidaemia, metabolic dysfunction associated steatotic liver disease (MASLD), polyendocrine metabolic ovarian syndrome, OSA, ischaemic heart disease, etc. The presence and control of these obesity-related complications (refer to **Table 4**) will influence treatment options and urgency of treatment of obesity. Many medications used to manage obesity-related comorbidities might contribute to weight gain. Psychological history including history of depression, anxiety, eating disorders, and the use of psychiatric medications are important to note and would impact the management of obesity.

Table 4: Complications of obesity

Metabolic
Diabetes mellitus
Hypertension
Dyslipidaemia
Gout
Metabolic dysfunction-associated steatotic liver disease
Polyendocrine metabolic ovarian syndrome (formerly polycystic ovarian syndrome)
Coronary artery disease
Cancer—endometrial, breast, colon, cervical, oesophageal, kidney, prostate, etc.
Mechanical
Obstructive sleep apnoea
Gastroesophageal reflux disease
Osteoarthritis
Venous stasis
Mental or Psychological
Depression
Eating disorders
Stigma and discrimination

Assess: Examination and Further Investigations

Assessment of Obesity

Obesity is abnormal or excessive fat accumulation that presents a risk to health. The most utilised marker for classifying weight is the body mass index (BMI), which is calculated from weight (kg) divided by height (m) squared. Overweight is defined by a BMI of ≥25 kg/m^2 and obesity is defined by a BMI of ≥30 kg/m^2, where the severity of obesity increases with increasing BMI (refer to **Table 5**). In Asians, a BMI of >23.0 kg/m^2 is considered overweight and the BMI cutoffs for intervention for obesity categories are lowered by 2.5 kg/m^2 as Asians have higher adiposity for a given BMI and metabolic risk.[28]

In the recent 2025 Lancet commission on clinical obesity, the diagnosis of clinical obesity is moving away from using solely BMI to include additional anthropometric measurements such as waist circumference, waist-hip ratio or waist-height ratio, or body fat measurements. Additionally, the commission distinguishes between "pre-clinical obesity", in which there is excess body fat but normal organ function, from "clinical obesity", in which there is excess body fat causing organ dysfunction, physical limitation, or illness.[29]

Waist circumference is a useful and simple clinical measurement of obesity. An increased waist circumference is associated with increased visceral adiposity and increased risk of cardiometabolic disease.[29,30] The waist circumference is measured at the midpoint between the iliac crest and lower border of the ribs. A waist circumference of >80 cm and >90 cm in Asian females and males, respectively, indicates increased metabolic risk.[31] Another marker is the waist-hip ratio, which is calculated from waist circumference divided by hip circumference. A ratio of >0.90 in men and >0.85 in women is indicative of an increased risk of metabolic complications.[32] Neck circumference has also been associated with visceral adiposity and is used in the STOP-BANG score for OSA.[27,33] If available, physicians may utilise bioimpedance to measure body fat percentage; however, the thresholds of body fat percentage for defining obesity are not defined.[29]

The rest of the physical examination and investigations should be guided by the patient's history, suspicion of secondary causes, and the screening for obesity-related complications. Acanthosis nigricans, a dark velvety discolouration of the skin on the neck and armpits, may be seen as and is a sign of insulin resistance. If secondary causes of obesity are suspected (refer to **Table 2**), further investigations or appropriate referrals may be required. The physician should screen for obesity-related complications (refer to **Table 4**) in the clinical examination and investigations. The presence of complications portends greater risk to health than BMI alone. **Table 6** suggests investigations to consider in patients with obesity. Of note, the calculation of the FIB-4 index is useful in the assessment of the risk of fibrosis in MASLD.[34]

In the assessment of obesity, there are staging systems for obesity that are more comprehensive and predictive of outcomes than BMI alone. One staging system is the Edmonton Obesity Staging Scale (EOSS).[35] The EOSS has five stages from Stage 0 to Stage 4, depending on the presence of obesity-related comorbidities, their severity, and functional limitations (refer to **Table 7**). It is superior to BMI classification in predicting mortality for people who are overweight and with obesity.[35,36] The presence of obesity-related comorbidities and a higher EOSS stage suggest greater urgency in addressing obesity. For example, a patient with obesity with T2DM, MASLD, and OSA would require more urgent and aggressive weight management than a patient with similar BMI without any obesity-related comorbidities. Thus, it is useful to consider the staging and the impact of obesity on health when assessing a patient.

Table 5. BMI classification for obesity and waist circumference threshold for increased metabolic risk

BMI Classification	**WHO cutoff for BMI (kg/m^2)**	**BMI cutoff for action in Asians* (kg/m^2)[28]**
Obese	>30.0	
Class I obesity	30.0–34.9	27.5–32.4
Class II obesity	35.0–39.9	32.5–37.4
Class III obesity	>40.0	>37.5
*BMI threshold for action is lowered by 2.5 kg/m^2 in Asians		
Waist circumference (WC) for increased metabolic risk	**WC threshold (cm)[32]**	**WC threshold in Asians (cm)[31]**
Female	>88	>80
Male	>108	>90

Table 6. Investigations in a patient with obesity

<u>To investigate underlying causes</u> Thyroid function test	 Exclude hypothyroidism
Cushing's work up if clinically indicated (overnight dexamethasone suppression test, 24-hour urinary free cortisol, late-night salivary cortisol, or referral to an endocrinologist)	Exclude Cushing's syndrome
<u>Screening for complications of obesity</u> *Clinical* Blood pressure STOP-BANG score	 Screen for hypertension Screen for OSA
Laboratory investigations Fasting glucose and HbA1C +/- 75 g OGTT Lipid panel Renal panel, urine albumin:creatinine ratio Liver panel Full blood count	 Screen for DM, IFG, and IGT Screen for dyslipidaemia Screen for abnormal renal function Screen for MASLD [Calculation of FIB-4 index utilises age, AST, ALT and platelet count]
Others (e.g., Mammogram, pap smear, referrals as required)	Screen for cancers
OSA: Obstructive sleep apnoea DM: Diabetes mellitus IFG: Impaired fasting glucose IGT: Impaired glucose tolerance OGTT: Oral glucose tolerance test MASLD: Metabolic dysfunction associated fatty liver disease AST: Aspartate Aminotransferase ALT: Alanine Aminotransferase FIB-4: Fibrosis-4 index = [age (years) x AST (u/L)] / [platelet count (x10^9/L) x √ALT (u/L)] *If Fibrosis-4 (FIB-4) index ≥1.3 or >2 if age above 65 years old for further assessment, consider further fibrosis assessment or referral to Hepatology[34]	

Table 7. Edmonton obesity staging scale (EOSS)[35]

Stage 0	No signs of obesity-related risk factors No physical symptoms No psychological symptoms or functional limitations
Stage 1	Subclinical obesity-related risk factors (e.g., Borderline hypertension, impaired fasting glucose, impaired glucose tolerance, elevated liver enzymes, etc.) Mild physical symptoms Mild obesity-related psychopathology, functional limitations, and/or mild impairment in well-being
Stage 2	Established obesity-related comorbidities (e.g., Type 2 diabetes mellitus, hypertension, OSA, PMOS) Moderate obesity-related functional limitations and/or moderate impairment of well-being
Stage 3	Significant obesity-related comorbidities (e.g., Myocardial infarction, heart failure, diabetes complications, incapacitating osteoarthritis) Significant obesity-related psychopathology, functional limitations, and/or impairment of well-being
Stage 4	Severe (potentially end-stage) disabilities from obesity-related comorbidities Severe disabling obesity-related psychopathology, functional limitations, and/or severe impairment of well-being

Advise

The next step would be to provide advice. This entails informing the patient about the results of the assessment and treatment options. Advice should include the health risks of obesity, weight loss benefits, and discussion of strategies for weight loss.

Agree: Discussing a Management Plan and Goal-Setting

Assessing readiness for change is important before discussing a management plan with the patient. When confronted with any potential change, ambivalence is expected. The physician can help the patient to resolve any ambivalence through MI techniques, before progressing further in the discussion.

Discussing a management plan should be patient-centred and collaborative. Patient-centred care encourages active collaboration and shared decision-making between patients and providers. Apart from clinical considerations, the patient and provider would need to consider emotional, mental, social, and financial perspectives.[37] Patients who share their perspectives achieve better outcomes.[20,38] As lifestyle modification requires changes in a person's daily behaviours, each person's treatment needs to be tailored and personalised. To facilitate this process, the physician may need to start by setting realistic weight loss expectations and clarifying misconceptions. A modest target of 5–10 percent weight loss can have significant benefits on obesity-related comorbidities.[39] For some patients, preventing further weight gain may be a realistic initial target.

In formulating a management plan, focusing on behavioural goals where patients have a higher locus of control may be more beneficial, rather than only concentrating on weight loss outcomes. These behavioural goals may be related to healthy eating, physical activity, or other behavioural goals. Examples include intake of fruits and vegetables, reducing sugar-sweetened beverages, self-monitoring of dietary intake, self-monitoring of weight, reducing sedentary time, increasing steps, or exercise sessions per week. Patients should choose their own behavioural goal to work on. Ideally, this behaviour should be sustainable and one that the patient believes is important and is confident in carrying out. In collaborating with the patient to form behavioural goals, one may use the SMART (**s**pecific, **m**easurable, **a**chievable, **r**elevant, **t**ime-based) goals as a guide.

Assist

To conclude the consultation for obesity, the patient should be assisted in further management. This may include additional education, support programmes, referrals to the dietitian, exercise physiologist or community resources for physical activity, or referrals to other specialists as appropriate. Adjustment of medications that contribute to weight gain by adjusting doses or using weight-favourable alternatives would also facilitate weight management. Depending on the assessment and health goals, adjuncts such as obesity pharmacotherapy or referral to a bariatric surgery unit might be suitable and should be discussed in a patient-centred manner.

CONCLUSION

The role of the primary care physician is central to the management of obesity. The 5As framework (Ask, Assess, Advise, Agree, Assist) is a useful tool for guiding consultations. The clinical assessment of obesity is an important skill to learn. It is important to exclude secondary medical causes and understand lifestyle factors contributing to weight gain. The use of motivational interviewing and focusing on behavioural goals may be useful during the consultation. Lastly, the management of patients with obesity should be patient-centred and collaborative.

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LEARNING POINTS

- **Initiate a collaborative conversation about weight management with patients in a non-judgemental, empathetic, and respectful manner.**
- **The 5As framework is a useful and practical framework for obesity counselling.**
- **Motivational interviewing is an effective counselling approach that can be used within this framework.**
- **A comprehensive clinical assessment should be performed for the patient with obesity, aiming to assess for secondary causes, contributing factors of obesity, and obesity-related comorbidities.**

BASIC OBESITY MANAGEMENT ACCREDITATION 6
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Unit No. 3

DIETARY INTERVENTIONS FOR WEIGHT LOSS

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ABSTRACT

Obesity is a complex and chronic condition that requires continuing care. A variety of diet plans are available for use in the clinical setting. Exactly what type of diet is the most beneficial remains up for debate. Numerous clinical trials have been carried out over the years comparing an array of dietary interventions for weight loss, including calorie-restricted diets, altered macronutrient composition diets, or specific dietary patterns. This paper will provide an overview of some of the evidence-based dietary interventions for clinical practice.

Keywords: Obesity, weight loss, dietary interventions, low-carbohydrate diet, energy restriction

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INTRODUCTION

Obesity is a serious global epidemic associated with numerous metabolic complications, including type 2 diabetes (T2DM), hypertension, cardiovascular disorders, and several cancers. The aetiology of obesity is multifactorial, involving an interplay of genetic, biological, environmental, social, cultural, and behavioural factors. Even though a successful weight loss strategy should be individualised and address all the underlying causes of obesity, dietary and lifestyle interventions remain the cornerstone of treatment. However, the optimal dietary approach to weight loss is still a subject of debate amongst experts, healthcare professionals, and the public, as studies have failed to demonstrate the superiority of one diet plan over another in the long term. Comprehensive assessment of an individual's dietary habits and lifestyle should be the first step in deciding on the best dietary intervention for weight loss and avoiding a one-size-fits-all approach. There is evidence that even modest weight loss of 5–8 percent body weight achieved with diet and lifestyle interventions improves glycaemia, blood pressure, lipid profile, mobility, and quality of life.[1–2] Greater weight loss might be needed to produce health benefits in individuals with morbid obesity or multiple comorbidities.

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ENERGY RESTRICTION DIET INTERVENTIONS

Energy-restricted diets range from continuous or intermittent diet plans with moderate to severe caloric deficit depending on the health status and weight loss goals of a patient or client. A calorie deficit of at least 500–750 per day is recommended to achieve a weight loss of 0.5–1.0 kg per week, which is the standard approach in clinical practice. These continuous energy restriction diet plans are formulated using healthy eating and exercise principles incorporating individual food preferences and behaviour modification to increase compliance. However, specially formulated meals or partial liquid meal replacements have also been employed to help achieve the necessary caloric deficit to drive weight loss in certain individuals after considering their health status, dietary preferences, and cost.

On the other hand, Low and Very Low Energy Diets (VLED) based on total meal replacements restrict energy intake to about 800 calories per day or less, which results in a more rapid weight loss and improvement of obesity related comorbidities including remission of type 2 diabetes.[3–5] The use of total meal replacements helps to deliver adequate levels of essential nutrients and improves adherence. VLEDs have been underutilised in clinical practice in view of concerns over the potential loss of lean body mass, risk of precipitating eating disorder, overall safety, and subsequent weight regain due to rapid weight loss. However, when clinically supervised, there is no evidence of VLED causing eating disorders or resulting in worse outcomes on knee strength, handgrip strength, or bone density compared to moderate energy-restricted diets in short-term studies lasting 3–6 months.[6–8] The use of VLEDs in supervised conditions for up to three months in patients who fail to meet weight or metabolic targets with a standard approach is gaining support from institutions such as the National Institute for Health and Clinical Excellence and clinicians. A structured, individualised weight maintenance programme with gradual readjustment to normal eating after VLED is necessary to maintain weight loss and metabolic benefits.[5,9,10] Further research is needed to assess the long-term impact of VLED plans on body composition, weight maintenance, and metabolic outcomes.

Intermittent energy restriction (IER) is another dietary approach for weight loss that involves periods of fasting and eating or Intermittent Fasting (IF) with or without calorie restriction. Popular types of IF include alternate-day fasting (ADF), the 5:2 fast (five days of normal eating and two days of restricted eating per week), and time-restricted eating (TRE).[11] In the TRE trials, the fasting window varies from 12 to 20 hours with an *ad libitum* diet during the eating period. The key appeal of IF is high compliance, easy sustainability compared to continuous daily caloric restriction, and other

potential health benefits such as improvements in cognitive function, metabolic health, and longevity demonstrated by animal studies.[12] However, feelings of hunger may be more pronounced during IER.

The evidence from human trials suggests that IF is safe for most healthy adults and achieves comparable weight loss and metabolic improvements to continuous energy restriction.[13,14] In recent meta-analyses, both intermittent and continuous energy restriction resulted in a similar weight loss, weight maintenance, and improvements in cardiovascular risk factors.[15,16] However IF may be more effective as a weight loss intervention for people with a higher BMI.[17] Many studies on IF in humans are short-term and involve a small number of subjects. Furthermore, with the focus of IER on the timing of eating rather than on food choices, some individuals may not achieve a desirable diet quality. It is therefore important to counsel patients about making healthy food choices when they are not fasting to improve their health further or prevent potential nutrient deficiencies. Longer-term trials are needed to further address the safety and efficacy of IF for different patient groups to help build confidence in recommending these IER plans.

ALTERED MACRONUTRIENT COMPOSITION DIETS

Low and very low carbohydrate diets, often referred to as "keto" diets, have gained popularity amongst healthcare professionals and the public as an effective tool for weight loss and a means to reduce metabolic complications associated with overweight and obesity. However, the terms "low carbohydrate diet", "very low carbohydrate diet", and "ketogenic diet" are often used interchangeably with lack of consensus in the literature on their definition. Low carbohydrate diets are defined as providing less than 26 percent of calories from carbohydrates (50–130 g per day) while very low carbohydrate diets limit carbohydrate calories to less than 10 percent (20–50 g per day).[18]

The ketogenic diet was initially developed to treat severe epilepsy in infants and children under medical supervision. It is a very low carbohydrate high-fat diet plan, resulting in a state of ketosis where fat instead of glucose is being burnt for fuel. True ketogenic diets used in clinical settings can limit carbohydrates to as little as 5 percent of calories, primarily from non-starchy vegetables, and provide up to 85 percent calories from fat with enough protein to preserve lean body mass but maintain ketosis.

However, when used as a tool for weight loss, these "keto" diet plans vary in the proportion of fat, protein, and carbohydrates they provide. The standard ketogenic diet usually provides 60–70 percent calories from fat, 20–30 percent from protein and up to 10 percent from carbohydrates. However, there is an individual variation in the level of carbohydrate and protein intake that is compatible with ketosis thus diet personalisation is required. "Keto flu" is a frequent side effect of a keto diet, which can include light-headedness, fatigue, headaches, nausea, and constipation, in particular during the adaptation phase. Multivitamin, mineral, and fibre supplements can be considered in some individuals to reduce side-effects. The nutritional adequacy of "keto" diet plans will depend on the overall diet composition and the nutrient sources.

A recent review of evidence on low and very low carbohydrate diets found them to be effective but not superior to other weight-loss diets.[19] There was no difference in weight loss between lower carbohydrate (4–45 percent calories)/higher fat (30–75 percent calories) diets compared to higher carbohydrate (50–65 percent calories)/lower fat (20–25 percent calories) diets when protein and energy levels were kept the same.[18,20] However, studies in patients who were overweight and diabetic following a low carbohydrate diet showed improvements in triglycerides and HDL cholesterol levels, insulin sensitivity, and glycaemic control with mixed effects on LDL cholesterol.[21,22]

One advantage that "keto diets" may offer is controlling the cravings and hunger often reported with other diet plans. A review published in 2015 found that individuals adhering to a ketogenic diet reported significantly less hunger and desire to eat compared with baseline.[23] Even though well-formulated ketogenic diets may offer short-term health benefits in some individuals, they are difficult to sustain, and long-term risks and benefits are not fully understood in the absence of long-term studies.

SPECIFIC DIETARY PATTERNS

Diets focusing on dietary patterns such as the Dietary Approaches to Stop Hypertension (DASH) diet, Mediterranean diets, and Plant-Based Diets (PBD) have also been studied in weight loss trials.

The DASH diet recommends specific servings of different food groups depending on daily caloric needs. It focuses on whole grains, fruit and vegetables, fat-free and low-fat dairy, lean meat, fish, and poultry. A meta-analysis revealed that overweight and obese adults on the DASH diet lose more weight than controls following a standard diet in studies ranging from 8–24 weeks.[24] Calorie-restricted DASH diets led to even greater weight loss when compared to other low energy diets.

Mediterranean diets emphasise the intake of vegetables, fruit, legumes, nuts, whole grains, and olive oil as the main source of fat, with moderate amounts of fish and poultry, low intake of red meat, and moderate consumption of wine. Meta-analysis of RCTs found that energy-restricted Mediterranean diets achieve as much or more weight loss than low-carbohydrate and low-fat diets with or without energy restriction among overweight and obese adults when followed for at least six months.[25]

A variety of PBDs including vegan and vegetarian diets have been investigated for their beneficial effects on weight loss and associated comorbidities. In a recent systematic review, PBDs were found to be effective in reducing weight and

waist circumference in individuals with T2DM, especially in studies with a duration of at least 16 weeks.[26] PBDs are characterised by high intake of dietary fibre from wholegrains, vegetables, and legumes, and low glycaemic index, which may enhance satiety and improve glycaemic control.

The DASH, Mediterranean diet, and PBDs can be safe, effective, and sustainable weight loss eating plans that also improve metabolic complications.

CONCLUSION

A variety of dietary approaches, with sufficient reduction in energy intake and high level of dietary adherence, can produce weight loss. IER and ketogenic diets are gaining popularity and have shown superiority in reducing body fat and improving obesity-related metabolic complications in the short term. However, studies have failed to establish the superiority of one diet plan over another in the long term. Weight loss interventions must address the underlying causes of overweight and obesity and facilitate sustainable behavioural and lifestyle changes to prevent and manage relapse.

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LEARNING POINTS

- **To date, studies have failed to demonstrate the superiority of one diet plan over another as patients can lose weight on any diet in the short term. Long-term effectiveness studies are lacking.**
- **Any dietary approach to weight loss should be individualised and consider the health status, personal preferences, and ability of the person to sustain the recommendations in the plan.**
- **As healthcare professionals, we should be realistic when discussing treatment expectations with our patients and provide ongoing support to ensure long-term weight loss maintenance and manage relapse.**

BASIC OBESITY MANAGEMENT ACCREDITATION 6
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Unit No. 4

MOVING FOR WEIGHT LOSS AND HEALTH GAIN IN OBESITY

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ABSTRACT

Physical activity and exercise are essential to obesity management, with benefits extending beyond weight loss. This narrative review summarises their roles in weight management and health improvement in adults who are overweight and obese. Aerobic exercise produces modest weight loss, with greater benefits at higher exercise volumes and when combined with dietary energy restriction. Resistance exercise improves strength and helps preserve lean mass, particularly during weight reduction and among older adults. Even without weight loss, exercise can reduce visceral and liver fat, improve cardiometabolic health, and enhance physical function and quality of life. Sustainable obesity care should combine individualised aerobic and resistance exercise, reduced sedentary time, and gradual progression, recognising health improvements alongside changes in body weight.

Keywords: obesity; physical activity; aerobic exercise; resistance training; weight management; cardiometabolic health; body composition

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INTRODUCTION

Physical activity and exercise are core components of obesity care, with benefits that extend beyond changes in body weight.[1] Physical activity includes any skeletal muscle movement that increases energy expenditure above rest. This includes commuting, housework, and leisure activities. Exercise is a subset of physical activity. It is planned, structured, repetitive, and is undertaken to improve or maintain health and fitness.[2] Health improvements include reducing visceral adipose tissue in adults with overweight and obesity,[3,4] reducing ectopic fat including hepatic and cardiac fat deposits, which are important contributors to cardiometabolic risk,[5] and improvements to cognition, memory, sleep, and overall quality of life.[6] Exercise should also be individualised and aim to address both health-related and skill-related components (refer to **Table 1**).[3] While skill-related components of fitness may not have direct impact on health, they support daily function and movement safety, and may lower the risk of injury.

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Table 1. Components of physical fitness

Health-related components of fitness
1. Cardiovascular endurance 2. Muscular endurance 3. Muscular strength 4. Flexibility 5. Body composition
Skill-related components of fitness
1. Power 2. Speed 3. Agility 4. Reaction time 5. Coordination 6. Balance

EXERCISE FOR WEIGHT LOSS AND WEIGHT MAINTENANCE

Aerobic Exercise

Aerobic exercise is the most extensively studied exercise modality in obesity management and contributes meaningfully to reductions in body weight, body fat, and cardiometabolic risk factors. It improves cardiovascular endurance and increases energy expenditure. However, average weight loss from aerobic exercise alone is modest. An overview of 12 systematic reviews covering 149 studies reported approximately 1.5 to 3.5 kg greater weight loss than non-exercising controls.[7] The European Association for the Study of Obesity similarly advises an expected average reduction of about 2 to 3 kg.[8] Individual responses vary with exercise dose, adherence, and compensatory changes in energy intake and other activity. Combining exercise with an appropriate dietary energy deficit is therefore more effective than relying on exercise alone.[7,8]

In 2024, a dose response meta-analysis of 116 randomised trials involving 6,880 adults estimated weight reductions of 2.79 kg (95% CI 2.29–3.29) at 150 minutes, and 4.19 kg (95% CI 2.41–5.98) at 300 minutes of aerobic exercise weekly.[9] Waist circumference and body fat also improved as exercise volume increased. The minimum recommendation of 150 minutes of moderate intensity exercise per week should therefore be viewed as foundation for health improvement, with progression towards 250 to 300 minutes where feasible and when greater weight reduction is the goal.[8,10] Aerobic exercise should also be performed on most days of the week to support adherence, energy expenditure and cardiometabolic health.

Long-term physical activity is recommended after weight loss. The American College of Sports Medicine (ACSM) associates more than 250 minutes of moderate intensity exercise per week with better maintenance,[10] while NICE

states that some people may need 60 to 90 minutes of moderate intensity aerobic exercise daily.[11] NB: these are individualised goals rather than starting prescriptions.

Resistance Exercise

Resistance exercise occurs when there is muscle contraction against internal or external load with sufficient effort and time under tension.[12] It is used to improve muscular strength and muscular endurance, and when performed quickly, can improve power. Resistance exercise has a modest impact on overall body mass through reducing fat and increasing or preserving lean mass.[13] This is particularly relevant during energy restriction and in older adults at risk of sarcopenia and functional decline. Reviews support including resistance exercise to attenuate diet-related muscle loss, although complete preservation is not assured.[14]

Among older adults with obesity undergoing diet therapy, combined aerobic and resistance exercise produced greater functional improvement and strength gains than either modality alone.[15] Strength gains for the combined aerobic and resistance exercise group (272 to 320 kg [18% increase]) were similar to resistance exercise-only group (288 to 337 kg [19% increase]), but more than aerobic exercise-only group (265 to 270 kg [4% increase]) (P<0.001) for both comparisons. In addition, there was limited lean-mass loss in the combined aerobic and resistance exercise group, compared to the aerobic exercise-only group. Thus, aerobic and resistance exercise have complementary roles in a comprehensive programme.

The ACSM recommends engaging in resistance exercise at moderately heavy loads at least twice a week, covering the major muscle groups. A practical approach to prescribing exercise is to include "upper" and "lower" body regions for "push" and "pull" exercises in "horizontal" and "vertical" directions for upper body exercises.[16]

HEALTH BENEFITS WITHOUT WEIGHT LOSS

Stable body weight does not imply an ineffective intervention. Aerobic exercise can reduce visceral adipose tissue without prescribed caloric restriction. Early meta-analyses have found stronger evidence for aerobic than resistance training for this outcome.[4,17] Exercise can also reduce liver fat in people with type 2 diabetes, although effects vary across ectopic fat deposits.[5] Improvements in fat distribution may therefore occur despite little change in overall weight. Regular physical activity without weight loss can also improve hyperglycaemia and insulin sensitivity, high blood pressure, and dyslipidaemia.[18]

CONCLUSION

Exercise contributes to modest weight loss, favourable changes in body composition, healthier fat distribution, and preservation of physical capacity and improved cardiometabolic health. Its value remains substantial when body weight is unchanged. Sustainable obesity care should combine aerobic and resistance exercise, reducing sedentary time, and gradual progression shaped by the individual's goals, abilities and clinical status.

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LEARNING POINTS

- **Exercise supports obesity management with greater benefits when combined with dietary energy restriction.**
- **Health benefits extend beyond weight loss. Regular exercise can reduce visceral and liver fat and improve cardiometabolic health, physical function, and quality of life, even in the absence of weight loss.**
- **Aerobic and resistance exercise are complementary and can help improve fitness and strength while helping preserve lean mass during weight reduction.**

Help people with obesity

LIVE LIGHTER™

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ONCE-WEEKLY wegovy® semaglutide injection

In adults with obesity, along with diet and exercise,

Wegovy® delivered weight loss from fat mass while preserving muscle function[1-3]

Total body fat

84%

FAT MASS LOSS

from total weight loss[1*^]

Visceral fat

-31%

REDUCTION

in mean visceral fat[2^]

Muscle function

4.1kg

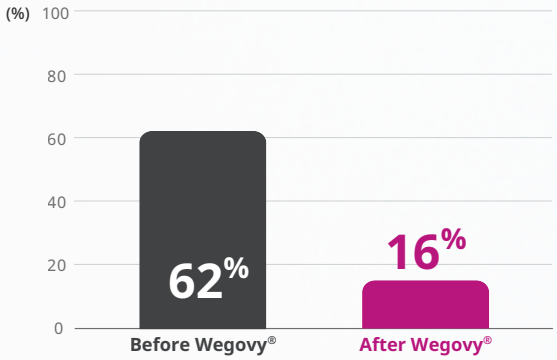
INCREASE

in handgrip strength from baseline[3]

Significant improvements in body composition seen from 7 months of treatment[3]

Wegovy® reduced the mental burden of constant food noise and emotional eating[4]

Percentage of people living with obesity (PwO) who found themselves constantly thinking about food[4†]



-74%

REDUCTION

in constant thoughts about food[4†]

* The change in adipose tissue mass as a proportion of mass reduction was calculated as: {[(absolute volume change [L] in adipose tissue x 0.92)/[(absolute volume change [L] in adipose tissue x 0.92) + (absolute volume change [L] in lean tissue x 1.1)]} x 100 = {(11.0 x 0.92)/[(11.0 x 0.92) + (1.7 x 1.1)]} x 100 = 84.4%.

^ Results shown are from a pooled analysis including Wegovy® 2.4 mg and 7.2 mg doses.

† Result from survey question 'I found/find myself constantly thinking about food throughout the day' after initiating semaglutide for weight management.

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BASIC OBESITY MANAGEMENT ACCREDITATION 6
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Unit No. 5

USE OF PHARMACOTHERAPY IN OBESITY MANAGEMENT

Dr Tham Kwang Wei

ABSTRACT

Obesity is a chronic disease that is relapsing and progressive due to a disruption in energy homeostasis, rendering people with obesity the challenge of attaining adequate weight loss and/or weight maintenance after successful weight loss. Depending on the presence, type, and severity of the obesity-related comorbidities and complications (ORC), patients may require an amount of weight loss beyond what lifestyle and behavioural modification can attain to improve/treat the ORC. Hence, obesity medications are required to attain clinically meaningful weight loss. After metabolic bariatric surgery (MBS), some patients may not attain their target weight loss or may experience weight regain. Obesity medications may be required to achieve their target weight loss and metabolic control. The use of pharmacotherapy in obesity management remains a vital adjunct to lifestyle and behavioural modifications and even to MBS, particularly in those with multiple or severe ORC and severe stages of obesity. This article discusses the general approach to pharmacotherapy in obesity management, the various obesity medications currently approved, and novel and pipeline medications for obesity treatment.

Keywords: Obesity, obesity medications, pharmacotherapy, weight loss

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INTRODUCTION

The global burden of obesity has increased substantially over the last four decades with obesity prevalence still projected to rise. By 2035, 51 percent of the world's population will be estimated to have overweight or obesity, with about one in four persons having obesity.[1] Obesity is now established as a chronic, progressive disease and often relapsing[2–4] with a complex host of pathogenic and perpetuating factors.[5] These factors, along with the underpinning biologic responses to weight loss, often present people with obesity (PwO) the challenge of attaining adequate and/or maintaining weight loss to improve health,[5–6] often necessitating the use of multiple modalities including pharmacotherapy in obesity management.

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Despite this clinical need, the use of obesity medications in the United States remains low at an estimated <5 percent among those in whom there is a medical indication. This is clearly much lower than the usage of pharmacotherapy in other chronic diseases like type 2 diabetes.[7,8] In Singapore, people living with overweight and obesity view that weight loss medications are dangerous (65%) and only 20 percent feel that medications are effective in weight loss. Coupled with the belief that the responsibility to manage obesity and weight issues (90%) lies solely with PwO, this may contribute to PwO not seeking medical attention as they should.[9]

Inadequate healthcare coverage for obesity treatments stemming from misconceptions about PwO and about the disease itself results in high out-of-pocket costs and contributes to the poor uptake of obesity pharmacotherapy despite the need for treatment.[10,11] Weight bias and stigma by healthcare professionals from a variety of reasons has resulted in healthcare professionals not adequately addressing obesity in patients.[12]

Over the years, several approved weight loss medications (e.g., fenfluramine, sibutramine, rimonabant, lorcaserin) were withdrawn from the market due to serious adverse events.[11] This may have eroded the confidence in obesity medications in not just the general public but among prescribers. Despite studies proving that weight loss of 5–10 percent improves ORC and cardiovascular risk, the absolute difference may be deemed as insignificant to patients (or even physicians) and might contribute to the low uptake and prescription of obesity medications. Instead, many resort to over-the-counter (OTC) products or unlicensed interventions with undetermined efficacy and safety profile. In recent years, there have been multiple reports of such OTC products being adulterated with obesity medications already withdrawn from the market causing serious side effects to consumers.

To tackle the increasing burden of obesity associated with serious health sequelae, there is clearly a need to address these issues. This paper aims to address the rationale for the use of obesity medications, discuss the currently-approved obesity medications, and the approach physicians can adopt when utilising pharmacotherapy to treat obesity. A brief review of novel and pipeline medications for obesity treatment is also included.

RATIONALE AND CLINICAL REASONING FOR THE USE OF OBESITY MEDICATIONS

Weight Loss Needed for Health Improvement and ORC Control

Lifestyle changes, mainly through instituting a reduction in caloric intake and increased physical activity, and behavioural

modification, remain the cornerstone in obesity treatment. Clinically meaningful weight loss of 5–10 percent of initial weight can significantly reduce cardiovascular risk factors and improve obesity-related comorbidities or complications (ORC) such as obstructive sleep apnoea, metabolic dysfunction-associated steatotic liver disease (MASLD; commonly known as fatty liver disease), and the prevention or delay in the development of type 2 diabetes.[13,14] However, some ORC require weight loss beyond 5–10 percent for benefit. For example, improvement in symptomatology and function in osteoarthritis and improvement in ovulation and pregnancy outcomes in female infertility generally require weight loss of ≥10 percent. Weight loss quantum of up to 40 percent might be needed to improve or reverse fibrosis in steatohepatitis. For improvement in the severity of obstructive sleep apnoea (OSA), weight loss of at least 7–11 percent is needed.[15] Reduction in cardiovascular events and mortality is typically seen with greater weight loss (>15 percent). This has been observed after sustained weight loss over 8–15 years after metabolic bariatric surgery.[16,17]

Weight Loss Attainable with Lifestyle and Behavioural Interventions

Intensive lifestyle and behavioural therapy (ILBT) in the most rigorous clinical trials for weight loss can achieve a weight loss of 6.1–6.6 percent[16,18] at one year, which can be maintained at 6 percent over 10 years in the Look AHEAD study.[19] However, as in most weight-loss clinical trials involving lifestyle modification, weight regain is inevitable over time. Real-world data from a Canadian multidisciplinary practice using lifestyle and behavioural interventions in routine clinical practice showed that over a follow-up period of 7.5 years, 64 percent of patients lost ≤3 percent of initial weight, with only 32 percent of patients losing significant amounts of weight of ≥7.5 percent.[20] Hence, adjunctive pharmacotherapy is often necessary for clinically meaningful weight loss, especially in patients who require greater amounts of weight loss to treat their ORC.

Nonetheless, obesity medications should always be used in addition to best efforts in lifestyle and behavioural modification tailored for the patient and never as a substitute. The effect of obesity medications will then be further enhanced and patients can derive the best benefit of obesity medications as demonstrated repeatedly in clinical trials. In the STEP 3 trial, a mean weight loss of 17.6 percent with once-weekly semaglutide 2.4 mg was seen when used in addition to ILBT (6 percent).[18,21]

Counteracting the Physiologic Adaptive Response to Weight Loss

The negative energy balance created during weight loss evokes a robust physiologic adaptive response effected to restore energy homeostasis. This leads to increased food intake (due to reduced satiety and satiation coupled with increased hunger) and decreased energy expenditure with resultant weight regain.[22,23] Hence, obesity treatment should include therapies that can counteract these adaptive responses for enhanced weight loss and weight maintenance. Obesity medications play a crucial role here as all but one obesity medication act centrally to increase satiety and/or to reduce hunger and food cravings, with the aim being to counteract these adaptive responses via multiple pathways.[10,11]

IN WHOM SHOULD OBESITY MEDICATIONS BE INITIATED?

In Singapore, the use of obesity medications is recommended for those with a body-mass index (BMI) of ≥30 kg/m² or BMI ≥27 kg/m² in the presence of at least one ORC.[24] While the BMI cutoff appears to be the indicator for the initiation of obesity medications, a complications-centric approach assessing the severity of obesity or the extent to which obesity has impacted the patients' health should guide physicians on the need for and choice of obesity medication.[14]

Before considering the use of obesity medications, a thorough assessment to gauge the severity of obesity based on the presence and severity of ORC is warranted. The AACE/ACE Adiposity-Based Chronic Disease (ABCD) model and Edmonton Obesity Staging System can be used for this purpose.[15,25] This will guide the decision on the urgency of treatment and, if ORC are present, how much weight loss is needed to ameliorate or prevent progression of the ORC. Therefore, in the presence of ORC, the treatment of overweight and obesity should be prioritised especially if the ORC are either not well-controlled despite maximum medical therapy (severe) or in which treatment of obesity is fundamental to its management, e.g., type 2 diabetes mellitus, dyslipidaemia, steatohepatitis (metabolic dysfunction-associated steatotic hepatitis, MASH) with fibrosis. In these patients, pharmacotherapy should be initiated early as an adjunct to ILBT to treat these moderate to severe ORC and reduce their cardiovascular risks.[15]

WHEN AND WHAT OBESITY MEDICATION TO INITIATE?

When to Initiate?

In the following situations, the initiation of obesity medications should be considered:

- Concurrent/From the outset: Presence of ORC that are moderate or severe, especially if lifestyle and behavioural interventions alone will not achieve the weight loss required to improve the ORC (e.g., in severe OSA, MASH fibrosis)
- Sequential: When initial implemented lifestyle interventions result in inadequate weight loss to achieve improvement or resolution of ORC, or greater weight loss is desired to meet patient's goals
- Weight regain after lifestyle interventions
- Weight regain or inadequate weight loss after bariatric surgery

There are often differing opinions on the optimal timing of initiation of obesity medications. However, it has been shown that early weight reduction is a key predictor of long-term weight loss success. For this reason, the initiation of adjunctive treatments or intensification of treatment should not be met with inertia.[26]

What to Initiate?

There are currently five obesity medications approved for the adjunctive treatment of obesity in Singapore: Phentermine, approved for short-term use; and orlistat, phentermine, liraglutide 3.0 mg, naltrexone/buproprion ER, and subcutaneous semaglutide 2.4 mg approved for long-term use. Weight loss of 3–13 percent over placebo can be seen with these obesity medications.[10,11,27–29] As in any chronic disease, the ultimate choice of obesity medication needs to take into consideration the cost (affordability) of the medication(s), contraindications for use, weight loss efficacy and additional benefits of the medication (in treating ORC), patient's phenotype (e.g., tendency for cravings, poor satiety), and most importantly, the patient's choice and preference including on mode of administration (e.g., oral vs parenteral).

Orlistat

Orlistat is a gastrointestinal lipase inhibitor administered as 120 mg TDS prior to meals, which reduces intestinal dietary fat absorption by 30 percent. It is one of two medications approved for use in adolescents in Singapore. It is also the most well-studied obesity medication approved with the longest study duration (of four years). Due to its safety record, it is available in some countries over the counter, administered as 60 mg TDS.[27]

Its effect on weight loss is modest albeit significant with weight loss of 3.4 kg (3.1%) and 3.6 kg (3.3%) over placebo at 12 and 24 months respectively. Of note, in the XENDOS study, which saw a weight loss of 2.7 kg (2.4 percent) over placebo maintained over four years, there was a significant risk reduction of nearly 40 percent in DM development.[30]

Despite having the longest safety profile, its use is often limited by the common undesirable side effects of steatorrhea, faecal urgency, and oil spotting. Long-term use can result in deficiencies in fat-soluble vitamins, hence supplementation with a multivitamin is recommended. Patients should be warned of drug interactions with warfarin, anti-epileptics, cyclosporine, and levothyroxine with proper administration advised.[10,11]

Phentermine

An amphetamine-derivative deemed to have low potential for abuse, phentermine is a sympathomimetic agent that acts centrally in the hypothalamus to stimulate release of norepinephrine. Approved in the US in 1959 for short-term use (≤12 weeks), it is the most commonly prescribed obesity medication in the US. In Singapore, phentermine is available as 15 mg and 30 mg once daily and is approved for short-term use of up to 6–12 months.[24] It should be initiated at the lowest possible dose and increased for efficacy as needed to minimise its side effects.[31,32]

Most studies of phentermine are carried out for 12–28 weeks. At a dosage of 15 mg/day, total weight loss of 6.1 percent (or 4.4% above placebo) can be seen while total weight loss of 6.3–8.1 kg (~4-6 kg above placebo) can be expected with 30 mg/day.[11,31] A 36-week study showed that intermittent (alternate month) use of phentermine is as effective as continuous use of phentermine. When used in conjunction with a low-calorie diet (1,000 kcal/day), total weight loss of ~13 kg was seen, although the very high attrition rate of ~40 percent could have augmented its effect.[33]

Common side effects include palpitations, dry mouth, insomnia, and constipation. Phentermine can increase nervousness and should be avoided in those with anxiety disorder. Increases in blood pressure and heart rate observed with phentermine use may have implications for adverse cardiovascular effects in the long term. However, to date, there are no long-term cardiovascular outcome studies for obesity medication used in patients with obesity. Using electronic health record data of a cohort of nearly 14,000 adults who have used phentermine in several US health systems, it was observed that off-label use of phentermine of more than three months in patients with low risk of cardiovascular disease (CVD) was associated with greater weight loss without increased risk of incident CVD or death, up to three years after initiating phentermine.[34] In general, phentermine as monotherapy is still restricted to short-term use with need to closely monitor the blood pressure and heart rates and it is contraindicated in those with uncontrolled hypertension, active cardiovascular disease, and glaucoma.[15,32]

Liraglutide

An injectable glucagon-like peptide-1 receptor agonist (GLP1-RA), liraglutide enhances satiety and reduces appetite. Liraglutide is initiated at 0.6 mg daily with weekly dose escalation of 0.6 mg/day as tolerated. It was initially approved for the treatment of T2DM at doses of up to 1.8 mg daily. Used for the treatment of obesity, it can be titrated up to a maximum dose of 3.0 mg daily.[35] In December 2020, the US FDA approved liraglutide for the treatment of obesity in adolescents.

Weight loss of 6–8 percent (4–5.6% over placebo) at one year is seen[35,36] and this can be maintained up to three years with continued use,[37] with weight loss ≥10 percent occurring in up to 25 percent of individuals on liraglutide 3 mg/day.[35] When used as an adjunct to ILBT or used after a 12-week course of very-low calorie diets, liraglutide can result in total weight loss of up to 12 percent (6% over placebo) in one year.[18,38] Such adjunctive treatments are feasible in the primary care setting (total weight loss of 7.5% in one year).[39] Increasing liraglutide from 1.8 mg/day to 3.0 mg/day in a person with diabetes will provide additional weight loss without further lowering the HbA_{1c}.[36]

Although an increase in heart rate of 2–3 bpm over placebo is associated with liraglutide, when used in people with T2DM at a maximum of 1.8 mg/day, liraglutide was shown to reduce cardiovascular risk in individuals with T2DM in the LEADER trial.[40] Gastrointestinal side effects (most commonly nausea, vomiting, and diarrhoea) can occur in up to 65 percent of people using liraglutide for weight loss but these are usually mild and improve with time.[35] There is a potential risk of pancreatitis and medullary thyroid cancer though in clinical trials of longer duration, the risk of gallbladder disease was of a greater concern.[37]

In general, when weight loss is <4 percent after 16 weeks from initiation, cessation should be considered. In clinical practice, maximally tolerated doses should be used and monitored for effect for at least 12 weeks before considering stopping the medication.[10]

Naltrexone/Bupropion ER

Commonly known as CONTRAVE, the combination of naltrexone, an opoid antagonist, and bupropion, inhibitor of the neuronal reuptake of dopamine and norepinephrine, was approved for the treatment of obesity by the FDA in 2014 and by the Health Science Authorities in Singapore in January 2022. Formulated as an extended-release tablet, each tablet contains 8 mg naltrexone and 90 mg bupropion, titrated weekly to a maximum dose of 32 mg/360 mg (two tablets twice) daily. Although the exact mechanisms leading to weight loss are not fully understood, the central effect of naltrexone and bupropion on appetite regulatory centre (hypothalamus) and the reward system (mesolimbic dopamine circuit) can lead to appetite suppression and reduction in food cravings.[11,15]

At one year of treatment, weight loss of 4.2–5.2 percent above placebo is seen.[40] The most common side effects associated with naltrexone/bupropion are nausea, constipation, headache, vomiting, dizziness, insomnia, anxiety, dry mouth, and diarrhoea.[40,41] The use of naltrexone/bupropion is contraindicated in pregnancy, uncontrolled hypertension, those with a past and current history of seizures (buproprion reduces seizure threshold), bulimia or anorexia nervosa, severe depression, chronic opioid use, and acute alcohol and substance withdrawal. Caution is needed for use in those with a history of depression, anxiety, bipolar disorder, and migraines, with special assessment for suicidal ideation during use. The safety of naltrexone/bupropion has not been studied in those with cardiovascular disease and with its impact on blood pressure and heart rate, patients should be closely monitored for increase in these two parameters after initiation.[15,32]

Semaglutide 2.4 mg

Semaglutide is a once-weekly subcutaneous GLP1-RA approved for the long-term treatment of obesity and T2DM. At a dose of 2.4 mg weekly, semaglutide can result in placebo-substracted average weight loss of 12.4 percent at 68 weeks in people with obesity without T2DM[28] with maintenance of the weight loss (12.6 percent above placebo) up to 104 weeks after initiation. In people with T2DM, a mean total weight loss of ~10 percent at 68 weeks is observed.[43] At present, oral semaglutide (up to 14 mg once daily) is approved only for the treatment of type 2 diabetes mellitus in Singapore although a recent study of oral semaglutide 50 mg once daily in the OASIS 1 study resulted in 15.1 percent weight loss at 68 weeks, with 85 percent of subjects losing ≥5 percent of body weight.[44] The side effects of semaglutide are similar to that seen in liraglutide 3.0 mg with caution to monitor for suicidal behaviour, gastroparesis, pancreatitis, and ileus.[29]

Semaglutide 2.4 mg once weekly is the first obesity medication shown to confer cardiovascular benefits in patients with obesity (without diabetes). The SELECT cardiovascular outcome trial followed 17,604 patients who were overweight or obese with established cardiovascular disease and no history of diabetes over a period of five years.[45] Treatment with semaglutide 2.4 mg was associated with a statistically significant 20 percent reduction in major adverse cardiovascular events (MACE), defined as cardiovascular death, nonfatal myocardial infarction, or nonfatal stroke compared with placebo.

In a separate RCT, the STEP-HFpEF trial, patients with heart failure with preserved ejection fraction (HFpEF) and obesity who were treated with semaglutide 2.4 mg experienced greater (10.7%) weight loss, larger reductions in heart failure symptoms and physical limitations, and greater improvements in exercise function than placebo.[46]

In a recent RCT of biopsy-proven metabolic dysfunction-associated steatohepatitis (MASH) and liver fibrosis (ESSENCE trial), 72-week treatment with semaglutide 2.4 mg compared to placebo resulted in greater resolution of MASH (62.9% vs 34.1%) and improvement in fibrosis (37% vs 22.5%).[47,48] This was on a background of significant body weight reduction of 10.5 percent with semaglutide compared to 2 percent with placebo, demonstrating a weight-independent effect of semaglutide on MASH and MASH fibrosis.

There is also emerging data on the benefits of semaglutide beyond weight loss including in cognitive impairment (dementia), psoriatic arthritis, and possibly in (substance) addictions, although more rigorous trials are still needed to demonstrate its full clinical efficacy in treating these specific conditions.

Combination Treatments

In Singapore, the fixed combination drugs of phentermine/topiramate-ER is neither available nor approved for use and will not be discussed here. Combination therapy of orlistat, phentermine, and liraglutide and other approved obesity medications has not been well-studied and should not be considered as routine clinical practice.[27]

WHEN TO STOP OBESITY MEDICATIONS?

Obesity medications should be stopped if weight loss of 4–5 percent is not attained after 12–16 weeks on the highest-tolerated dose.[10] Obesity is a chronic disease, with a relapsing nature due to biological reasons as discussed above. As with other chronic diseases like hypertension and T2DM, pharmacotherapy should not be planned only for the short term (1–3 months) but for chronic weight management and/or control of ORC. Just because the parameters are controlled in chronic diseases does not imply that treatment needs to be stopped. The goal of therapy is for the long term, to prevent weight regain or weight maintenance and prevent/manage the ORC. Hence if an obesity medication is efficacious, long-term use at the lowest and safest possible doses should be considered, particularly to maintain the weight loss.

OBESITY MEDICATIONS ON THE HORIZON

A deeper understanding of the role of gut-based and nutrient-stimulated hormones in the regulation of appetite and energy homeostasis, and the metabolism of glucose and lipids, has led to the development of targeted therapeutics in obesity and T2DM. Many analogues of these hormones have either been approved for use in T2DM treatment or have undergone phase II trial or undergoing phase III evaluation for the treatment of obesity and ORC including for MASLD, T2D, OSA, and HFpEF.

Tirzepatide is a dual agonist of GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) administered as a once-weekly subcutaneous injection, which has been approved in Singapore for the treatment of T2DM and also for the chronic treatment of obesity in the USA in 2023. A 72-week RCT in participants with obesity without T2DM (SURMOUNT-1) showed a weight loss of 20.9 percent with the highest dose (15 mg) of tirzepatide versus 3.1 percent with placebo.[49] Most common adverse events observed are mostly mild to moderate and transient gastrointestinal symptoms. In the SURMOUNT series of trials, tirzepatide has not only been shown to be a potent weight loss medication across different patient populations and clinical settings, it has displayed significant benefit in the improvement of ORC such as OSA (SURMOUNT-OSA trial)[50] and in HFpEF (SUMMIT Trial),[51] once again demonstrating the potential therapeutic benefits of novel incretin-based therapies beyond weight loss.

In a 32-week phase 2 trial in adults with T2DM and a BMI ≥27 kg/m², co-administration of once-weekly subcutaneous cagrilintide 2.4 mg, an amylin analougue, and semaglutide 2.4 mg (CagriSema) was shown to be more efficacious in reducing HbA_{1c} (2.2%) compared to semaglutide 2.4 mg (1.9%) and cagrilintide (0.9%) alone. Mean weight loss at week 32 was also significantly greater with CagriSema (15.6%) compared to semaglutide (5.1%) and cagrilintide (8.1%),[52] with a potential for weight loss exceeding 20 percent if continued longer. A series of phase 3 trials with CagriSema (known as the REDEFINE Programme) will evaluate its impact on weight loss in a larger patient population with overweight and obesity, with or without T2DM, over a longer period of time.

Retatrutide, a single-molecule triple-hormone (GLP1, GIP, glucagon) receptor agonist administered subcutaneously once weekly, demonstrated an average 2–2.2 percent reduction in HbA_{1c} at 24 weeks and a mean 16–17 percent reduction in body weight (with its highest dose, 12 mg) at 32–36 weeks in those with obesity and T2DM.[53] In people with obesity and without T2DM, the highest dose of retatrutide resulted in a mean 24 percent reduction in body weight, with 83 percent of participants achieving a weight loss of 15 percent or more, and 26 percent of participants achieving a weight loss of 30 percent or more after 48 weeks of treatment.[54] Improvements in blood pressure resulted in discontinuation of at least one antihypertensive medication in 30–41 percent of the participants using the higher doses (8 and 12 mg). In a prespecified subgroup analysis of the same cohort of patients at 24 weeks, the higher two doses (8 and 12 mg) of retatrutide produced complete resolution of hepatic steatosis in 79 percent and 86 percent of participants respectively, with a similar percentage of liver fat reduction in the same period.[55] The TRIUMPH Programme, a series of phase 3 trials, will examine retatrutide in chronic weight management, obstructive sleep apnoea, and knee osteoarthritis in people with overweight and obesity.

Survodutide, a GLP-1/glucagon receptor dual-agonist, has shown promising results in people with biopsy-proven MASH and fibrosis stage F1 through F3. After 48 weeks of treatment, 75 percent of participants treated with survodutide experienced resolution of MASH with no worsening of fibrosis compared with 15 percent of patients on placebo. At the highest dose of survodutide (6.0 mg), two-thirds of patients showed evidence of fibrosis regression (based on available biopsy data) within 48 weeks.[56]

AMG133 (maridebart cafraglutide), which combines GLP1 agonism with GIP antagonism, has shown promising results after 12 weeks of a 4-weekly injection regime. Weight loss ranged from 7.2 percent at the lowest dose to 14.5 percent at the highest dose by day 85, with weight maintained even 150 days after the last dose.[57]

Although once-weekly administration of incretin-based therapies is efficacious in the treatment of T2DM and obesity, injections may serve as a barrier to treatment uptake. The once-daily oral formulation of semaglutide, approved for the treatment of T2D, requires strict administration due to the use of an absorption enhancer to enable absorption in the stomach, which might prove cumbersome for some patients. Further development of oral and non-peptide formulations will serve to overcome these barriers somewhat and hopefully improve uptake. In a phase 2 trial in overweight and obesity without T2DM, over 36 weeks, Orfloglipron, an oral non-peptide small molecule potent partial agonist of the GLP-1 receptor administered once-daily, resulted in mean weight loss of 9.4 to 14.7 percent (across the different doses) compared to 2.3 percent with

placebo, with nearly 50 percent of participants losing ≥15 percent body weight.[58] Several other oral GLP-1 receptor agnoists are currently under investigation in phase 1 and 2 trials such as GSBR-1290 and ecnoglutide (XW004).

VK2735 is a dual agonist of GLP and GIP receptors currently under investigation. In a 28-day phase 1 multiple ascending dose study, subjects on the oral formulation of VK2735 had dose-dependent body weight reductions, ranging up to 5.3 percent.[59] Oral amycretin, a novel protein-based unimolecular amylin combined with a GLP1-RA, showed promising results with a mean weight loss of 13.1 percent after 12 weeks of treatment on its twice-daily dosage compared with placebo (1.1%) in a phase 1 study.[60]

There are many more compounds such as oxyntomodulin with dual GLP-1 and glucacon receptor agonism, and PYY agonists that have either shown promising weight loss results or are still undergoing phase 1 or 2 studies, potentially offering patients with obesity a wider armamentarium of treatment options.

However, with obesity medications now able to result in magnitude of non-surgical weight loss, it must be cautioned that the fundamentals of obesity management comprising of lifestyle and behavioural modification and holistic care remain unchanged. Monitoring for the side effects of medications and the risk of malnutrition and sarcopenia will need to be concurrently carried out by adequately trained healthcare professionals experienced in the management of obesity.

CONCLUSION

The use of obesity medications is pivotal as an adjunct to lifestyle and behavioural therapy to augment the effect of weight loss needed to treat obesity and its ORC. Newer obesity medications can now effect weight loss of up to more than 20 percent with additional benefits on ORC. Despite the clear benefits and efficacy of obesity medication, numerous barriers remain in the appropriate use of pharmacotherapy in obesity treatment, creating a gap. Proper physician and patient education, and improved access to affordable obesity medication can help to improve uptake of obesity medication and bridge these gaps. Regardless of choice of obesity medication, the management of obesity must be in the context of a chronic disease, under the supervision of trained healthcare professionals of multi-disciplines to concurrently address the multiple facets of obesity.

Table 1. Efficacy, usage, common side effects, contraindications, and precautions to be considered with the obesity medication approved for long-term use.[15,29]

Obesity pharmacotherapy, indication/use[a]	Mechanism of action, study name, study duration: % TBWL greater than placebo or mean kg weight loss over placebo	Dose	Common side effects	Contraindications, cautions, and safety concerns	Monitoring and comments
Orlistat Chronic weight management FDA-approved for children ≥12 years old	Lipase inhibitor XENDOS **1 year: 4.0%** **4 years: 2.6%**	120 mg PO TID (before meals) OTC: 60 mg PO TID (before meals)	• Steatorrhea • Fecal urgency • Incontinence • Flatulence • Oily spotting • Frequent bowel movements • Abdominal pain • Headache	√ Pregnancy and breastfeeding √ Chronic malabsorption syndrome √ Cholestasis √ Oxalate nephrolithiasis • Rare severe liver injury • Cholelithiasis • Malabsorption of fat-soluble vitamins • Effects on other medications: - Warfarin (enhance) - Anti-epileptics (decrease) - Levothyroxine (decrease) - Cyclosporine (decrease)	**Monitor for:** • Cholelithiasis • Nephrolithiasis - Recommend standard multivitamin (to include vitamins A, D, E, and K) at bedtime or 2 hours after orlistat dose - Eating >30% kcal from fat results in greater GI side effects - Administer levothyroxine and orlistat 4 hours apart
Phentermine Short-term use (<12 weeks) for the management of obesity	NE-releasing agent **2–24 weeks: 3.6 kg**	15–37.5 mg (HCI) PO once daily 15–30 mg (ion-exchange resin complex) PO once daily	• Headache, elevated BP, elevated HR, insomnia, dry mouth, constipation, anxiety • Cardiovascular: palpitation, tachycardia, elevated BP, ischemic events • Central nervous system: overstimulation, restlessness, dizziness, insomnia, euphoria, dysphoria, tremor, headache, psychosis • GI: dryness of the mouth, unpleasant taste, diarrhea, constipation, other GI disturbances • Allergic: urticaria • Endocrine: impotence, changes in libido	• Anxiety disorders (agitated states) • History of heart disease, uncontrolled hypertension • Seizure • MAOIs • Pregnancy and breastfeeding • Hyperthyroidism • Glaucoma • History of drug abuse • Sympathomimetic amines	• Long-term use may lead to pharmacological tolerance, dependence, and withdrawal symptoms

Phentermine/topiramate ER Chronic weight management FDA-approved for adolescents ≥12 years	NE-releasing agent (phentermine) GABA receptor modulation (topiramate) EQUIP CONQUER SEQUEL **1 year: 8.6–9.3% on high dose; 6.6% on treatment dose** **2 years: 8.7% on high dose; 7.5% on treatment dose**	**Starting dose:** 3.75/23 mg PO QD for 2 weeks **Recommended dose:** 7.5/46 mg PO QD **Escalation dose:** 11.25/69 mg PO QD **Maximum dose:** 15/92 mg PO QD	• Headache • Paresthesia • Insomnia • Decreased bicarbonate • Xerostomia • Constipation • Nasopharyngitis • Anxiety • Depression • Cognitive impairment (concentration and memory) • Dizziness • Nausea • Dysgeusia	√ Pregnancy and breastfeeding (topiramate teratogenicity) √ Hyperthyroidism √ Acute angle-closure glaucoma √ Concomitant MAOI use (within 14 days) • Tachyarrhythmia • Decreased cognition • Seizure disorder • Anxiety and panic attacks • Nephrolithiasis • Hyperchloremic metabolic acidosis • Dose adjustment with hepatic or renal impairment • Concern for abuse potential • Combined use with alcohol or depressant drugs can worsen cognitive impairment	**Monitor for:** • Increased heart rate • Depressive symptomatology or worsening depression especially on maximum dose • Hypokalaemia (especially with HCTZ or furosemide) • Acute myopia and/or ocular pain • Acute kidney stone formation • Hypoglycaemia in patients having T2DM treated with insulin and/or sulfonylureas - Potential for lactic acidosis (hyperchloremic non-anion gap) in combination with metformin - MAOI (allow ≥14 days between discontinuation) - 15 mg/92 mg dose should not be discontinued abruptly (increased risk of seizure); taper over at least 1 week - Healthcare professional should check βHCG before initiating, followed by monthly self-testing at home - Monitor electrolytes and creatinine before and during treatment - Can cause menstrual spotting in women taking birth control pills owing to altered metabolism of estrogen and progestins

Naltrexone ER/bupropion ER Chronic weight management	Opiate antagonist (naltrexone) Reuptake inhibitor of DA and NE (bupropion) COR-I COR-II COR-BMOD **1 year: 4.2–5.2%**	**Titrate dose:** **Week 1:** 1 tab (8/90 mg) PO QAM **Week 2:** 1 tab (8/90 mg) PO BID **Week 3:** 2 tabs (total 16/180 mg) PO QAM and 1 tab 8/90 mg) PO QHS **Week 4:** 2 tabs (total 16/180 mg) PO QHS	• Nausea • Headache • Insomnia • Vomiting • Constipation • Diarrhoea • Dizziness • Anxiety • Xerostomia	√ Pregnancy and breastfeeding √ Uncontrolled hypertension √ Seizure disorder √ Anorexia nervosa √ Bulimia nervosa √ Severe depression √ Drug or alcohol withdrawal √ Concomitant MAOI (within 14 days) √ Chronic opioid use • Cardiac arrhythmia • Dose adjustment for liver or kidney impairment • Narrow-angle glaucoma • Uncontrolled migraine disorder • Generalized anxiety disorder • Bipolar disorder • Safety data lacking in patients who have depression • Seizures (bupropion lowers seizure threshold	**Monitor for:** • Increased heart rate and blood pressure • Worsening depression or suicidal ideation • Worsening of migraines • Liver injury (naltrexone) • Hypoglycemia in patients having T2DM treated with insulin and/or sulfonylureas • Seizures (bupropion lowers seizure threshold) - MAOI (allow ≥14 days between discontinuation) - Dose adjustment for patients with renal and hepatic impairment - Avoid taking medication with a high-fat meal - Can cause false positive urine test for amphetamine - Bupropion inhibits CYP2D6
Liraglutide 3.0 mg Chronic weight management FDA-approved for adolescents ≥12 years	GLP-1 receptor analogue SCALE Obesity & Prediabetes **1 year: 5.6%** **3 years: 4.3%**	**Titrate dose weekly by 0.6 mg as tolerated by patient (side effects):** 0.6 mg SC QD→ 1.2 mg SC QD→ 1.8 mg SC QD→ 2.4 mg SC QD→ 3.0 mg SC QD	• Nausea • Vomiting • Diarrhea • Constipation • Headache • Dyspepsia • Increased heart rate	√ Pregnancy and breastfeeding √ Personal or family history of medullary thyroid cancer or MEN2 √ Pancreatitis √ Acute gallbladder disease • Gastroparesis • Severe renal impairment can result from vomiting and dehydration • Use caution in patients with history of pancreatitis • Use caution in patients with cholelithiasis • Suicidal ideation and behaviour • Injection site reactions	**Monitor for:** • Pancreatitis • Cholelithiasis and cholecystitis • Hypoglycemia in patients having T2DM treated with insulin and/or sulfonylureas • Increased heart rate • Dehydration from nausea/vomiting • Injection site reactions - Titrate dose based on tolerability (nausea and GI side effects)

Semaglutide 2.4 mg Chronic weight management	GLP-1 receptor analogue STEP Obesity Adults without T2DM **68 weeks: 10.3–12.4%** **104 weeks: 12.6%** Adults with T2DM **68 weeks: 6.2%**	**Titrate dose every 4 weeks as tolerated by patient (side effects):** 0.25 mg SC QD→ 0.5 mg SC QD→ 1.0 mg SC QD→ 1.7 mg SC QD→ 2.4 mg SC QD	• Nausea • Vomiting • Diarrhea • Constipation • Headache • Fatigue • Dyspepsia • Dizziness • Abdominal distension • Eructation • Gastroenteritis • Gastroesophageal reflux disease	√ Pregnancy and breastfeeding √ Personal or family history of medullary thyroid cancer or MEN2 √ Pancreatitis √ Acute gallbladder disease • Gastroparesis • Ileus • Severe renal impairment can result from vomiting and dehydration • Use caution in patients with history of pancreatitis • Use caution in patients with cholelithiasis • Suicidal ideation and behaviour • Injection site reactions	**Monitor for:** • Pancreatitis • Cholelithiasis and cholecystitis • Hypoglycaemia in patients having T2DM treated with insulin and/or sulfonylureas • Diabetic retinopathy in patients with T2DM • Increased heart rate • Dehydration from nausea/vomiting • Injection site reactions • Ileus • Titrate dose based on tolerability (nausea and GI side effects)

Adapted from the AACE/ACE Comprehensive Clinical Practice Guidelines for Medical Care of Patients with Obesity[15] and Obesity in South and Southeast Asia—A new consensus on care and management[29]

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LEARNING POINTS

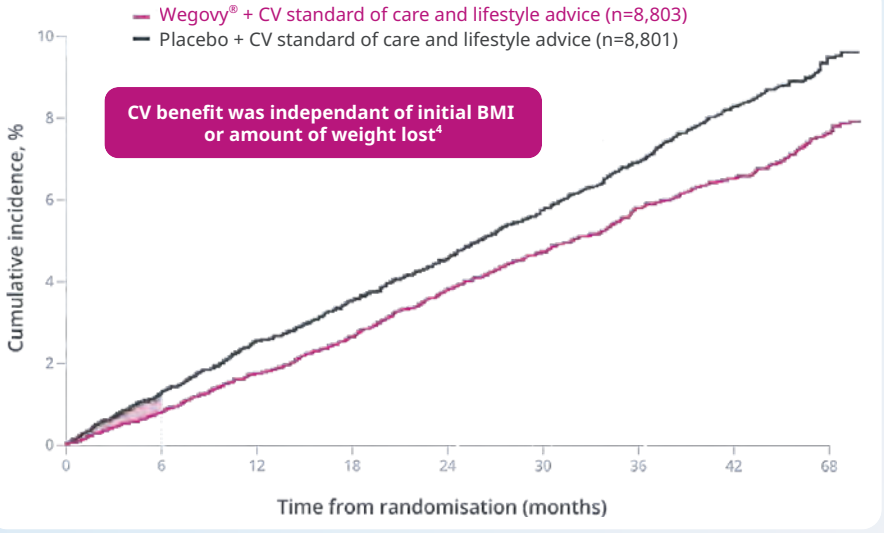
- **Pharmacotherapy in obesity management plays a crucial role as an adjunct to lifestyle and behavioural modification and metabolic bariatric surgery.**
- **Assessment of the stage/severity of obesity is a necessary initial step as more severe stages of obesity (usually in the presence of ORC) will warrant more urgent treatment with consideration of obesity medication at the outset.**
- **There are now safe and effective obesity medications approved for long-term use in obesity management with more in the pipelines. Understanding the indications, efficacy, and side-effect profile of each obesity medication along with patient phenotype and risk profile will help to match the most suitable treatment to the patient. This will improve compliance to the treatment and harness the best benefits for treating obesity and its ORC.**

ONCE-WEEKLY wegovy® semaglutide injection

Cardiovascular disease is the #1 cause of death for adults with obesity[1]

Only Wegovy® is proven to reduce the risk of MACE and treat obesity[2,3]

SELECT trial primary composite end point: Time to first occurrence of 3-point MACE (CV death, non-fatal MI, or non-fatal stroke)[3*]



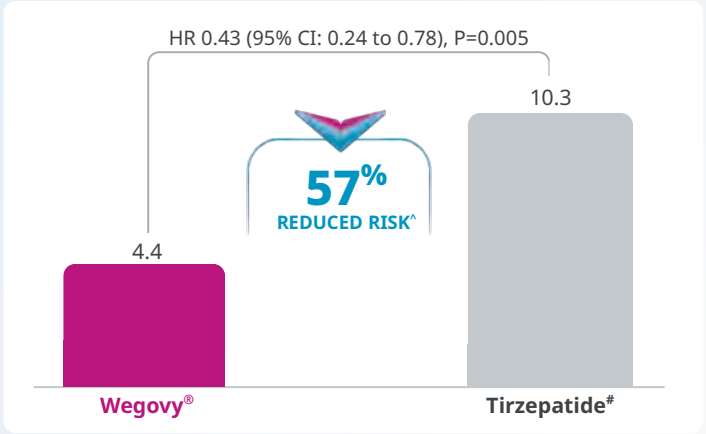
20%
RISK REDUCTION IN MACE
vs placebo when added to SOC†

*Data from SELECT: 3-point MACE included CV death, non-fatal MI or non-fatal stroke. Mean follow-up period of ~40 months (cumulative incidence of 6.5% with Wegovy® vs 8.0% with placebo). Mean change in body weight over the 104 weeks after randomisation was -9.4% with Wegovy® and 0-9% with placebo. Mean baseline body weight and BMI was 96.7 kg and 33.1 kg-m^2, respectively.

†Standard of care for CV risk included medical treatment that could be adjusted at the investigator's discretion for continued participation in the study and health lifestyle counselling.

Wegovy® reduces the risk of MACE in real world - 57% stronger effect than tirzepatide[4^]

Revised 3-point MACE# incidence per 1,000 patient-years[4^]



- Wegovy® is associated with **greater reductions in the risk of MACE** compared with tirzepatide in people with obesity.[4]
- Wegovy® patients experienced a lower rate of MACE per 1,000 patient-years vs tirzepatide (4.4 vs 10.3, respectively).[4]

^Per-protocol analysis (patients who stayed on treatment).
#Tirzepatide is not approved for MACE risk reduction in people with obesity.

CV, cardiovascular; MACE, major adverse cardiovascular events; SOC, standard of care.

#Data from STEER: Revised 3-point MACE included myocardial infarction, stroke, and all-cause mortality. 3-point MACE included myocardial infarction, stroke, and CV-related mortality.

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BASIC OBESITY MANAGEMENT ACCREDITATION 6
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Unit No. 6

INTENSIFYING TREATMENT: BARIATRIC SURGERY

Dr Shanker Pasupathy

ABSTRACT

Obesity is a chronic disease that is increasing at epidemic rates worldwide. Diet and lifestyle intervention form the basis of healthy weight management but are not effective in promoting substantial weight loss in morbidly obese individuals. Bariatric surgery can provide not only profound and sustainable weight loss but also excellent control of attendant comorbidities, particularly type 2 diabetes mellitus. Despite the high quality of evidence from numerous prospective randomised trials and cohort studies, fears and concerns regarding the safety and aggressiveness of surgery is limiting the acceptance of bariatric surgery as a viable treatment option. Newer, less invasive procedures hold promise in filling the gap between conservative and surgical therapies for weight loss.

Keywords: Obesity, Weight loss, Bariatric surgery, Metabolic surgery, Gastric bypass, Gastric band, Gastric sleeve, Gastric balloon, Endoscopic sleeve gastroplasty

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INTRODUCTION

Bariatric surgery traditionally refers to surgical procedures carried out on the gastro-intestinal tract to reduce the amount of food consumed and/or nutrients absorbed by the body. This has given rise to the concept that bariatric surgery induces "restriction" and "malabsorption". The principal indication for bariatric surgery is to enable significant and sustained weight loss in morbidly obese individuals. The resulting benefit to obesity-related medical and physical comorbidities is not insignificant.[1] As published in a recent Cochrane review, "surgery results in greater improvement in weight loss outcomes and weight associated comorbidities compared with non-surgical interventions, regardless of the type of procedures used."[2]

Despite its effectiveness, the uptake of bariatric surgery remains low. The reasons for this may lie with incomplete understanding of the underlying physiology of obesity and limited awareness of treatment options, including risks and outcomes. This article looks at how bariatric procedures have evolved over the past 50 years to minimise risk by reducing invasiveness and morbidity, thereby promoting acceptance.

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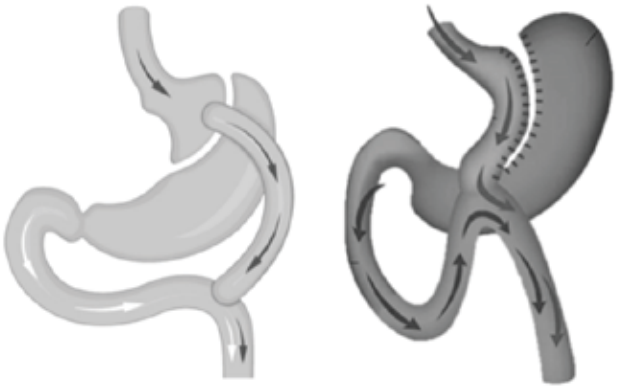
TYPES OF PROCEDURES

Gastric Bypass

The first safe and effective weight loss surgery to be performed on a large scale was the gastric bypass introduced in the 1960s, which involved stapling the stomach to create a small gastric "pouch" and creating a gastro-jejunostomy in order to divert nutrient passage further down the alimentary tract into the mid-jejunum, bypassing the duodenum and proximal jejunum. The advent of laparoscopy led to a dramatic rise in gastric bypass surgery in the USA and worldwide. Today, two types of gastric bypass are typically performed: the Roux-en-Y, and the one-anastomosis gastric bypass[3,4] (refer to **Figure 1**). Notwithstanding the different types of gastro-intestinal anastomoses, weight loss results and improvement in co-morbidities (especially diabetes) following the two procedures are largely similar.

Early morbidity of gastric bypass is approximately 4 percent and usually related to bleeding, perforation, and leakage.[5] Late complications include intestinal obstruction, marginal ulceration, and anastomotic stenosis. Vitamin and mineral supplementation for life is mandatory after all types of gastric bypass surgery. More aggressive procedures include the "distal gastric bypass", "bilio-pancreatic diversion", and "duodenal switch". These make up less than 5 percent of bariatric procedures because they are associated with higher surgical and nutritional morbidity in the long term.

Figure 1. Roux-en-Y gastric bypass (left) and one-anastomosis gastric bypass (right)



Gastric Band

Adjustable gastric banding was introduced in 1993 and saw a dramatic uptake worldwide.[6] The gastric band is deemed adjustable due to an expandable bladder on the inside. The bladder is connected by a tubing to a port, which is implanted subcutaneously. Fluid is added or removed from the band by cannulation of this port in the doctor's office every 4–6 weeks.

The "reversibility" and "no-cutting or stapling" appeal of the procedure was soon supplanted by complications relating to the placement of a silicone ring constricting the upper part of the stomach (refer to **Figure 2**). Despite sutures placed to fix the band at the level of the gastric cardia, slippage remained a troublesome complication. Rapid swallowing invariably led to choking, retching, and vomiting. Inadequately masticated pieces of meat and other firm chunks of food might get stuck above the band and require a late-night visit to the emergency room. Most patients had their bands removed within 10 years of placement as they could not tolerate the mantra of "small bites" and "chew slowly".

In the past decade, gastric band insertions have plummeted worldwide. In Singapore, the lap band™ is not currently available, although there remains a sizeable minority of patients who have successfully lost weight and return to the clinic for band adjustments from time to time. The vast majority of gastric bands have been removed, with some patients electing to convert to a different bariatric procedure such as gastric bypass or sleeve to improve or maintain their weight loss.[7]

Figure 2. Gastric Band



Gastric Sleeve

The procedure that emerged to take the place of the band was the laparoscopic vertical sleeve gastrectomy, or simply, the "gastric sleeve".[8] During this procedure, a bougie or calibration tube is placed along the lesser curvature and about 70–80 percent of the stomach is removed (refer to **Figure 3**). This changes the stomach into a tubular shape. Most of the body and fundus along with the greater curvature of the stomach is resected using a stapler, providing a smooth and haemostatic cut line.

Gastric sleeve numbers have overtaken all other procedures as the most popular bariatric surgery worldwide.[9] The main reason for this lies with the simplicity and efficacy of the sleeve: reduction of meal portions without requiring any foreign body placement or diversion of food passage. The main problem with this procedure is the potential for narrowing or twisting of the gastric tube, obstructing food passage, and causing pain, reflux, and vomiting. Bleeding and leakage from the staple line have also been reported. Post-operative management following the sleeve procedure is simpler than after gastric bypass as there is no intestinal component to the surgery.

Figure 3. Gastric Sleeve



Gastric Balloon

First approved by the FDA in 1985, there are several types of gastric balloons available in Singapore today. The aim of the gastric balloon is to fill up the stomach and reduce hunger and cravings (refer to **Figure 4**). Gastric balloons are typically placed and removed by endoscopy and can be kept in the stomach for up to a year. The Spatz balloon is adjustable, so that an initial smaller volume can be placed to avoid nausea and cramps. Once the stomach is adapted to the balloon, it can be inflated further to occupy more space with the expectation of driving weight loss. Expected weight loss is in the range from 10–15 percent.[10]

Figure 4. Gastric Balloon



NEWER PROCEDURES

Over the past decade, there has been a clear trend of developing less invasive weight loss interventions that provide better weight loss than diet, lifestyle, and pharmacotherapy, but lower risk for adverse events when compared to bariatric surgery. Two new products are disrupting the bariatric landscape in Singapore.

Gastric Pill Balloon

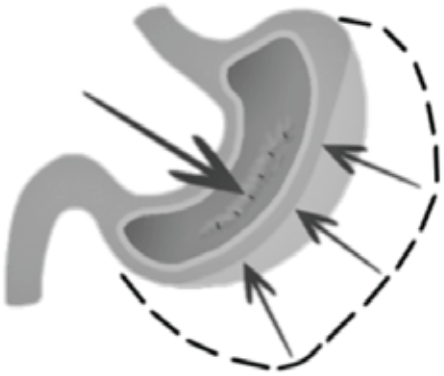
The first is the Elipse pill balloon. The balloon is tightly packed into a capsule that can be swallowed in the clinic. No anaesthesia or endoscopy is required. An x-ray is done to check that the capsule has entered the stomach prior to inflation. The entire placement can be completed in 20 minutes with the patient actively participating.

Balloon removal is even more efficient. The Elipse has a biodegradable control valve that opens and empties after 16 weeks, and the fully deflated balloon passes out naturally with stool. Weight loss after Elipse is comparable to that of other intra-gastric balloons despite remaining in the body for only four months.[11]

Endoscopic Sleeve Gastroplasty

The other product that received full HSA licensing in 2021 is the Overstitch device, a remarkable convergence of concept and technology. It does exactly what its name suggests. The Overstitch is a suturing tool that is mounted on an endoscope. It can be used to place sutures during endoscopy, for example, to close defects after removal of polyps, suture bleeding ulcers, etc. For obese patients, the Overstitch can be used to perform a stomach plication procedure, restricting gastric capacity and distensibility. The stomach is sutured from within during a gastroscopy, reducing its volume by 70 percent, similar to that achieved by a gastric sleeve surgery (refer to **Figure 5**). This new procedure is called an endoscopic sleeve gastroplasty (ESG) or "endo-sleeve".[12,13] A recent multi-centre RCT demonstrated 13.6 percent total body weight loss after one year for individuals with class 1 and 2 obesity following ESG.[14]

Figure 5. Endoscopic Sleeve Gastroplasty



WEIGHT LOSS AND METABOLIC IMPROVEMENT

Bariatric surgical interventions produce 10-30 percent weight loss, maintained for at least 20 years.[1] Glycaemic control is dramatically improved in up to 90 percent of patients, so much so that bariatric surgery has been renamed "metabolic" surgery and added to the treatment algorithm for diabetes management in most countries.[15] The name is indeed apt because of its powerful impact on hypertension, dyslipidaemia, fatty liver, and polycystic ovarian syndrome. The physical effects of weight loss leading to improvement in obstructive sleep apnoea, musculoskeletal pains, chronic leg swelling, and overall quality of life cannot be overstated.[16–18]

HOW BARIATRIC SURGERY WORKS

The goal of bariatric surgery is to enable individuals to feel satisfied with smaller meal portions. Limiting gastric capacity directly impacts the size of a meal one can consume. This effect alone is responsible for most of the weight loss one experiences after bariatric surgery. Furthermore, diversion of nutrient flow distally into the gut will reduce the total amount of food that can be digested and absorbed. In the early days of bariatric surgery, extensive intestinal bypasses were commonly performed. They were overly effective in reducing nutrient absorption, to the point of inducing diarrhoea, malnutrition, and potentially liver failure. That is why many of those procedures are obsolete and gastric bypass procedures carried out today generally retain an adequate length of small intestine (referred to as the common channel) for nutrient absorption.[19]

Gut-Brain Feedback

The sense of satiety and regulation of appetite is complex, with recent evidence pointing to fundamental roles for genetic, homeostatic, and hedonic mechanisms.[20] "Restriction" of food intake and "malabsorption" of nutrients were long believed to be the fundamental effects of bariatric surgery. However, critical analysis of eating behaviour and gut-brain signalling after bariatric surgery suggests a far more complex relationship between digestive and metabolic processes in the body. It is likely that alteration of nutrient passage through the stomach and intestine influences appetite control centres in the brain via a variety of neuro-biological feedback pathways.[22] Several candidates identified to contribute to weight loss after gastro-intestinal manipulation include altered secretion of the incretin hormones ghrelin, PYY, oxyntomodulin and GLP-1, gut microbes, and bile acids.[23]

The ultimate control of what and how much we consume appears to rest not in the stomach or adipose tissue but in the brain. To simply "eat less and move more" as is often advocated by many self-styled diet gurus and obesity experts fails to recognise the ongoing struggle between nutrient sensors, endocrine factors, and neural and emotional signals.[21]

PROCEDURE CHOICE AND INFORMED CONSENT

It is ultimately the patient who must live with the procedure, hence it is imperative that an informed choice is made. Most of the fears and concerns that patients express are related to the surgery itself. In modern bariatric surgery, all procedures are done by laparoscopy or keyhole technique with a typical hospital stay of 1–2 days. Adverse event rates in experienced centres are below 5 percent.[24] Pain and general side effects of surgery are similar to that of other GI procedures such as appendectomy and cholecystectomy. Newer alternatives such as the Elipse balloon and endoscopic sleeve gastroplasty are appealing because they are even less invasive, are reversible, and can be done on an outpatient basis.

Pure gastric restrictive procedures rarely bear any long-term nutritional risk, although women may be at risk of iron deficiency. It is different for gastric bypass procedures where vitamin and mineral supplementation must continue throughout the patient's life.[25] This is usually acceptable to a patient who has been on treatment for diabetes, hypertension, etc because they will be swapping out their medications for supplements.

In Singapore, bariatric surgery is regarded as a medical treatment for obesity if the body mass index is >32.5 kg/m^2 and if related medical and physical comorbidities are present.[26] Medisave may be used for reimbursement and many of the integrated shield plans have permitted the inclusion of these conditions, with type 2 diabetes mellitus, fatty liver, polycystic ovarian syndrome, metabolic syndrome, and obstructive sleep apnoea in particular among their approved indications for bariatric surgery.

FOLLOW-UP

Adapting to new dietary and lifestyle habits can often be a challenge. Many patients do not realise at the outset that despite having bariatric surgery, these daily habits are the key to shed weight and keep it off. That is why close clinical follow-up is important and positively correlated with greater weight loss, regardless of procedure type.[27] Visits should be scheduled with not just doctors but also the dietitian and exercise therapist. Some patients may benefit from psychological supervision during the early adjustment period.

During follow-up, it is important for clinicians to identify any problems or complications early. For example, vomiting may sometimes occur if a patient eats too quickly or drinks fluids at mealtimes. However, if pain or vomiting persist, it may be a symptom of an underlying stenosis or ulcer, which may need investigation and treatment. On the other hand, it is rare for a patient to regret their decision to undergo bariatric surgery.[28] When questioned, most patients say that they wish they had surgery earlier.

CONCLUSION

With proper planning, consideration, and procedure selection, bariatric surgery can be a powerful tool to tackle obesity and its related comorbidities. Successful weight loss after intervention requires close monitoring and follow-up by a multidisciplinary team. In 2022, a wide variety of bariatric procedures are available in Singapore, from office-based treatments to outpatient endoscopic therapy and finally laparoscopic surgery.

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LEARNING POINTS

- **Obesity is a chronic neuro-biological disorder that is an important risk factor for cardio-metabolic disorders and cancer.**
- **Bariatric surgery is an effective treatment for obesity and its comorbidities.**
- **Bariatric procedures alter the amount and timing of nutrient passage through the digestive system.**
- **Gastro-intestinal alterations after bariatric surgery lead to changes in gut hormones and feedback signalling to appetite control centres in the brain.**
- **Close multi-disciplinary follow-up is essential to promote successful weight loss and avoid nutritional complications in the long term.**

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Unit No. 7

APPROACH TO CHILDHOOD AND ADOLESCENT OBESITY

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ABSTRACT

The prevalence of child and adolescent obesity is increasing both globally and in Singapore, and primary care providers play an important role in stemming this rise. Accurate measurement of Body Mass Index (BMI) and monitoring of growth across time can enable early diagnosis and intervention. Addressing childhood and adolescent obesity and its consequences should be done in a sensitive and non-judgemental manner. Childhood and adolescent obesity have adverse medical and psychosocial consequences and early screening for these complications is important in assessment. Adopting a family-based approach for healthier lifestyle changes is important in the prevention of childhood and adolescent obesity. Pharmacotherapy may be considered as an adjunct to lifestyle changes in selected cases.

Keywords: childhood obesity, adolescent obesity, obesity prevention, body mass index

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INTRODUCTION

Epidemiology and Causes of Childhood and Adolescent Obesity

According to the World Health Organisation (WHO), the prevalence of overweight and obesity among children and adolescents aged 5–19 has increased from 4 percent in 1975 to 18 percent in 2016.[1] Similarly in Singapore, obesity among children aged 6–18 rose from 11 percent in 2013 to 16 percent in 2021,[2] exacerbated by the COVID-19 pandemic. A study conducted by Health Promotion Board found that 70 percent of children who were overweight at age seven continued to be overweight as adults.[3]

Obesity is a complex disease with numerous contributory factors. Genetic, behavioural, environmental, and social factors all contribute to obesity.[4]

ROLE OF PRIMARY HEALTHCARE PROVIDERS

Children and adolescents with obesity present to clinical services more frequently than their healthy weight peers.[5] However, overweight is seldom addressed due to barriers such as lack of recognition of overweight status and lack of training to address the issue of obesity.[6] The frequent touchpoints to primary care such as well-child visits and acute conditions provide the opportunities to discuss a child's growth, identify weight issues early, and provide anticipatory guidance.

Addressing childhood and adolescent obesity can be a sensitive situation for families as the need for intervention to address the complications intersects with the social determinants of obesity and the normal development of adolescents. This can result in an emotionally sensitive situation for families in a healthcare setting. An overweight child might be viewed as acceptable and reflective of the caregiver's caregiving abilities. On the other hand, adolescents with obesity commonly experience criticism about their weight and lifestyle habits from their families; bringing up health consequences of obesity can lead to emotionally charged situations where the adolescents are blamed for the issue.[7] Broaching the topic of obesity will thus need to be done in a sensitive, non-judgemental, and non-stigmatising manner, while highlighting the risks of future health consequences. Having a family-based approach is important so that the child/adolescent will not feel targeted and other family members can also benefit from healthier lifestyle changes. This is especially important as obesity is an intergenerational condition and primary care providers commonly care for the family. They are thus well-positioned to address the health needs of the family and to provide family-based guidance across their life course.

ASSESSMENT OF OBESITY

Visual assessment of the weight status of children is known to be inaccurate, hence objective measures are important for growth assessment.[8] Accurate weight and height measurement using well-calibrated instruments and proper measurement techniques as recommended by WHO is important to calculate Body Mass Index (BMI).[9] Careful plotting of BMI on age- and gender-appropriate BMI percentile charts is important in diagnosing obesity and for monitoring of changes in obesity status longitudinally. For children in Singapore aged 6–18 years, the Health Promotion Board (HPB) defines a BMI of more than the 90th percentile as overweight, while a BMI of more than the 97th percentile is considered severely overweight (obesity equivalent).[10]

After assessing that the child/adolescent's BMI is in the overweight/obesity range, family history of obesity and obesity-related diseases, dietary habits, and physical activity should also be assessed to evaluate the risk factors for obesity (refer to **Table I**). History and physical examination should also look out for possible secondary causes of obesity such as genetic, endocrine abnormalities, or medication use that can lead to weight gain and assess for potential obesity-related co-morbidities[11] (refer to **Table II**).

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Table I: Assessment of risk factors and health behaviours

Risk factors
• Obesity in parents and first-degree relatives • History of metabolic diseases in first- and second-degree relatives • Maternal history of diabetes or gestational diabetes • Small for gestational age
Diet
• Main caregiver for child • Consumption of sweetened beverages, fruits, and vegetables • Frequency of eating out and family meals
Activities
• School-based activities and after-school physical activities • Screen time use and duration • Sleep time and duration

Table II: Systems review of child/adolescent with obesity

System	Symptoms or signs	Possible causes
General inspection	Dysmorphic features or developmental delay	Genetic syndromes such as Prader-Willi
	Cushingoid	Hypocortisolism or use of glucocorticoids
	Presenting affect	Depression or anxiety
	Height and Weight	Assess severity of overweight
	Binge-eating, purging or use of laxatives	Eating disorder
	School-avoidance or anxiety or low mood	Depression or anxiety
Skin	Acanthosis nigricans (especially on neck, axilla, and antecubital fossa)	Type 2 Diabetes Mellitus
	Acne and hirsutism (females)	Polycystic Ovarian Syndrome
	Skin folds infection (especially in lower abdomen and genital area)	Intertrigo Hyperhidrosis
Eye	Papilloedema	Pseudotumour cerebri
	Recurrent headaches	Pseudotumour cerebri
Oral	Tonsillar hypertrophy	Obstructive sleep apnoea
	Dental hygiene	Dental caries
	Snoring and daytime somnolence	Obstructive sleep apnoea
Neck	Thyroid enlargement	Hypothyroidism
Respiratory	Dyspnoea and wheezing	Asthma (which may lead to exercise intolerance)
	Poor exercise tolerance	Asthma or lack of physical conditioning
Cardiometabolic	Blood pressure	Hypertension
	Urinary frequency, polydipsia and polyuria, and recent weight loss	Type 2 Diabetes Mellitus
Gastrointestinal system	Abdominal pain or tenderness	Gastro-oesophageal reflux or cholelithiasis or functional abdominal disorders
	Hepatomegaly	Non-alcoholic fatty liver disease
Musculoskeletal	Abnormal gait	Slipped capital femoral epiphysis or joint strain
	Bowing of tibia	Blount disease
	Foot pain	Pes planus
Reproductive system	Tanner staging	Altered onset of puberty
	Breast development (in males)	To evaluate height potential
		Gynaecomastia
	Irregular periods or amenorrhoea	Polycystic Ovarian Syndrome

COMPLICATIONS OF CHILDHOOD AND ADOLESCENT OBESITY

Childhood and adolescent obesity can adversely affect almost every organ of the body[12] as discussed in **Tables I** and **II**. In the audit of our paediatric weight management clinic, 68 percent of those enrolled had already developed at least one obesity-related co-morbidity at presentation. The most common obesity-related co-morbidity was dyslipidaemia followed by hypertension.[13]

Child and adolescent obesity also increase the risk of adult obesity with development of obesity-related co-morbidities and increased cardiovascular mortality.[14] Early prevention and management of childhood obesity is thus important to avoid these complications.

Initial tests to consider include fasting lipids, glycated haemoglobin, and alanine amino transferase (+/- liver function tests) as recommended by various international expert guidelines.[15–17] Risk factors for Type 2 diabetes include a maternal history of diabetes (including gestational diabetes), type 2 diabetes in a first-degree relative, clinical signs of insulin resistance—acanthosis nigricans, other conditions associated with obesity—hypertension, dyslipidaemia, fatty liver disease, polycystic ovary syndrome, small for gestational age, and use of psychotropic medications.[18] As with all investigations, it is important to consider the various risk factors and whether testing will alter the course of treatment.

MANAGEMENT OF CHILDHOOD AND ADOLESCENT OBESITY

Lifestyle Management

Comprehensive dietary and physical assessments are impractical in a busy clinical setting, hence it is generally recommended to focus on common dietary and physical activity behaviours that have the strongest evidence and are more easily modifiable as a family.[15] These recommendations are in line with the 24-hour activity guidelines for children and adolescents and suitable for healthy children and adolescents (refer to **Table III**).[19] These guidelines can also support parents in making family-wide changes, especially if they have both overweight and normal weight children to avoid stigmatising the overweight children. These health behaviours are helpful in both the reduction of obesity and the promotion of other health benefits to promote the holistic physical and psychosocial development of healthy children and adolescents. Other healthcare providers such as dietitians or nurses, trained in child health, can also help to assess and monitor health behaviours.

Comprehensive, intensive behavioural interventions with at least 26 contact hours or more that include supervised physical activity sessions for up to 1-year result are effective for weight loss in children and adolescents with improvements in cardiometabolic risk factors. These behavioural interventions commonly consist of multiple components and include sessions for both parent and child (separately, together, or both) and sessions may be group or individual session.[31] While such comprehensive and intensive behavioural interventions are available mostly in a hospital setting,[32] identifying and addressing high BMI are important steps in helping children and families get the support they need.

Use of a patient-centric communication technique, such as motivational interviewing, has been found to be effective in behaviour change.[20] Motivational interviewing takes into consideration patients' readiness to change, use of non-judgemental questions, and reflective listening to understand one's beliefs and values. This can help patients formulate a plan that is consistent with their values rather than physicians imposing their plans, reducing the stigma associated with obesity.

Providing a close follow-up in the subsequent few weeks or months after the initial visit will be important in monitoring changes in weight and health behaviours and providing the required support for families.[15] While weight management does not lower self-esteem or increase the risk of eating disorder, it is important to note that children and adolescents with obesity are at risk of eating disorder.[31] Thus it is important to follow up on the child's growth to ensure that dietary changes aren't too drastic, which can lead to excessive weight loss affecting normal growth or the early onset of eating disorder.

Pharmacotherapy

Pharmacotherapy, though very limited, may be considered as an adjunct to lifestyle interventions in selected children with extreme obesity, especially if they face significant physical or psychological comorbidities.

Orlistat, for more than a decade and up till recently, was the only licenced drug by the FDA for the treatment of obesity in adolescent patients over 12 years of age. It is an intestinal lipase inhibitor, thereby decreasing hydrolysis of ingested triglycerides and reducing gastrointestinal absorption of fat. A series of randomised trials demonstrated that orlistat reduced BMI by 0.5–4.2 kg/m² compared to placebo.[21] The side effects include diarrhoea, flatulence, and abdominal pain. These understandably can result in a high discontinuation rate; reportedly 75 percent by the end of three months.[22] As orlistat reduces adsorption of dietary fat-soluble vitamins, supplementation of vitamins A, D, E, and K is recommended.[23]

Glucagon-like peptide-1 analogues (GLP-1) is a gut peptide that increases the postprandial insulin level in a glucose-dependent manner, reduces glucagon secretion, slows gut motility, and suppresses appetite.

Subcutaneous Liraglutide is approved by the Health Science Authority (HSA) for paediatric patients with Type 2 Diabetes Mellitus (T2DM) above age of 10 years old (maximum dose 1.8 mg) and has expanded to the treatment of obesity in patients above 12 years of age (maximum dose of 3 mg). Over a 3-month period, one study showed that patients'

BMI dropped by 2.1 kg/m^2,[24] and another showed that half lost 5–10 percent of their body weight and one-quarter lost >10 percent.[25] There was also significant improvement of other metabolic indices.[25] After a trial of liraglutide for 12 weeks, if patients have not lost at least 4 percent of their BMI or BMI z score on the 3.0 mg/day or maximum tolerated dose, it should be discontinued. Kelly et al[26] demonstrated that after discontinuation of liraglutide after one year of administration, weight gain ensued, suggesting that longer-term treatment may be required as part of management of obesity as a chronic disease. A recent publication on the use of subcutaneous Liraglutide (maximum dose of 3 mg) in patients aged six to below 12 years old found similar improvement in BMI percentage of 7.4 percent compared to placebo, and 46 percent of participants on liraglutide had at least 5 percent reduction in BMI.[33]

Subcutaneous Semaglutide has recently been approved by Health Science Authority for paediatric patients (≥12 years old) with obesity. Weghuber et al[34] reported a significant improvement in BMI of participants on semaglutide (-16.1% versus 0.6%) compared to those on placebo over a 68 weeks' trial period. There was improvement in cardiometabolic risk factors (waist circumference, glycated haemoglobin, total cholesterol, LDL cholesterol, VLDL cholesterol, triglycerides, and alanine aminotransferase). A recent meta-analysis of 18 randomised trials in children and adolescents (<18 years) confirmed the efficacy of GLP-1 RAs in lowering BMI and improving glycaemic control across multiple populations, including those with T2DM, prediabetes, and obesity. Adverse effects were largely gastrointestinal, and tolerability was similar across agents.[35]

Nonetheless, important gaps remain: data on long-term use beyond 68 weeks, impact on growth and neurodevelopment, and risk of weight regain upon cessation are limited. Data in children <6 years are also sparse, and ongoing surveillance for rare but serious adverse events (e.g., hypersensitivity reactions, thyroid tumours in animal models, renal complications) is warranted.

Topiramate/Phentermine combination therapy has recently been approved by the FDA for treatment of obesity in adolescents above 12 years of age, but this combination therapy is currently unavailable in Singapore. The treatment is however associated with suicidal behaviours and impulsivity, hence further trials examining long-term safety profile and efficacy are needed.

Metformin is an attractive drug of choice given its various desirable effects on insulin resistance and cardiovascular risk profile. Unfortunately, its effect on weight reduction is consistently statistically insignificant.[27] As such, its licenced use is limited to the treatment of children with T2DM from 10 years of age.

Setmelanotide (melanocortin 4 receptor agonist) and Metreleptin (a synthetic recombinant leptin analogue) are approved for specific monogenic obesity conditions (refer to **Table IV**).

Table III: Recommended health behaviours for children and adolescents

Dietary
• Avoid sugar-sweetened beverages inclusive of fruit juices and malt drinks. These can reduce the risk of obesity and formation of dental caries. • Consume two servings and fruits and vegetables per day. Fruits and vegetables contain vitamins, minerals, and fibre, which are important for healthy growth and development and prevention of chronic diseases. Fibre can also keep children fuller for a longer time. • Encourage regular family mealtimes. Parents are important role models for healthy eating habits and family meals allow parents to influence the portions and quality of the meals for their children.
Physical Activity
• Achieve at least 60 minutes of moderate-to vigorous activity per day. All types of activities and outdoor play should be encouraged to promote sports participation and achievement of health benefits. • Engage in a variety of light activity throughout the day. These include taking the stairs or walking to destinations rather than taking transportation. • Build in regular breaks throughout the day. Prolonged sedentary behaviour is also harmful to health; building in breaks is important to improve concentration.
Sleep
• Regular, continuous sleep of at least nine hours (for 7–13 years old), at least eight hours (for 14–17 years old), and at least 7 hours (for 18 years old). Insufficient sleep duration is associated with obesity, hypertension, diabetes, poorer mental health, and cognitive functioning.
Screen time
• Limit recreational screen time as much as possible. Recreational screen time such as phone or device use of more than two hours per day is associated with the most adverse health outcomes such as increased adiposity and poorer psychosocial health. Screen time during meals is also discouraged to enable child to self-regulate during mealtimes.

Bariatric Surgery

Current international guidelines suggest that bariatric surgery should be considered in post-pubertal adolescents with BMI >40 kg/m² or a BMI >35 kg/m² with significant comorbidities and have failed a formal, intensive lifestyle modification programme.[28] Short- and mid-term results have been encouraging. BMI reduction at 1-year follow-up was -13.5 kg/m² and weight loss being maintained at a 5-year follow-up.[29,30] In addition, the reduction or even resolution of comorbidities after surgery is also significant. This procedure should be undertaken by an experienced multidisciplinary specialist team with expertise in the care of adolescents with obesity.

INDICATIONS FOR REFERRAL TO TERTIARY CARE

Primary care providers play an important role in the early identification of overweight and to provide brief intervention. However, primary care providers should provide follow-up for these patients and refer to a tertiary centre for further investigation or specialist management in the following situations:

- Increase in weight with slowing height growth
- Suspected secondary cause of obesity
- Severe obesity, especially if associated with obesity-related co-morbidities
- Rapid weight loss of more than 2 kg per month or concerns about eating disorder
- Failure of primary care management after a 6-month trial

Table IV: Summary of medications approved for the treatment of childhood obesity

Medication	Mechanism of action	FDA- and EMA-approved indications and age	Dose	Adverse effect(s)
Orlistat	Lipase inhibitor, blocks intestinal far absorption	Obesity >12years	PO 60–120 mg TDS	GI
Liraglutide	GLP-1 agonist	Obesity >12 years T2DM >10 years	SC 0.6–3 mg OD SC 0.6–1.8 mg OD	GI, hypoglycaemia, pancreatitis, renal failure
Semaglutide	GLP-1 agonist	Obesity ≥12 years	SC 0.25–2.4 mg once weekly	GI, hypoglycaemia, pancreatitis
Setmelanotide	MC4R agonist	POMC, PCSK1, or LEPR deficiency in >2 years	SC 0.5–2 mg OD	GI, hyperpigmentation
Metreleptin	Recombinant analogue of leptin	congenital leptin deficiency and generalised lipodystrophy	For weight <40 kg: SC 0.06–0.13 mg/kg OD For weight >40 kg: 2.5–10 mg/day	Fatigue, hypoglycaemia
Phentermine	TAAR1 agonist	FDA approved >16 years Not approved by EMA	PO 15–30 mg OD	Tachycardia, GI, dizziness, insomnia
Topiramate	Carbonic anhydrase inhibitor, appetite suppressant	Phentermine/topiramate FDA approved >12 years	PO P3.75 mg/T23 mg–P15 mg/T92 mg OD	Mood changes, memory issues, paraesthesia
Metformin	Inhibits gluconeogenesis, improves insulin sensitivity	T2DM >10 years *not approved for weight loss	PO 500–2,000 mg a day in divided doses	GI

Abbreviations:
FDA = Food and Drug Administration
EMA = European Medicines Agency
GI = gastrointestinal
POMC = proopiomelanocortin
PCSK1 = proprotein convertase subtilisin/kexin type 1
LEPR = leptin receptor
T2DM = type 2 diabetes mellitus
OD = once daily
TDS = three times a day
PO = oral
SC = subcutaneous

Note: *Drugs in Italics are available in Singapore.*

CONCLUSION

Paediatric obesity remains an ongoing serious health concern, with an increasing local prevalence that echoes the global trend. It results in both physical and psychosocial complications, and if allowed to continue into adulthood, endangers their adult health and longevity. Hence the prevention of obesity would be more relevant and cost-effective than to treat its complications. Primary care physicians are pivotal in this combat as they monitor growth and development of the child from birth. Each visit should provide an opportunity to provide anticipatory guidance throughout childhood and accord early preventive measures for at-risk children. Pharmacotherapy and bariatric surgery serve only to fill in the treatment gap for patients who are refractory to lifestyle modifications or have morbid obesity. Whilst they do show promising short-term effects, research is still ongoing to evaluate the long-term effects of these treatment modalities.

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LEARNING POINTS

- **Accurate weight and height should be taken at each visit to opportunistically screen for obesity. BMI-for-age should be monitored over time for early identification of young children at risk of being overweight or obese.**
- **Primary care providers can provide anticipatory guidance for caregivers of young children with up-trending or persistently high BMI-for-age (i.e., >90th percentile). This begins with early identification of at-risk children and education of caregivers on the potential long-term implications of childhood obesity.**
- **Primary care providers can provide preliminary assessment and management to address modifiable risk factors, such as adopting healthy nutrition and physical activity habits, to prevent childhood obesity.**
- **Management strategies to prevent obesity should be family-based (rather than focused on the child) and centred around long-term healthy behavioural and lifestyle changes.**
- **Pharmacotherapy and, to a smaller extent, bariatric surgery may be considered for children and adolescents with obesity only after a formal intensive lifestyle modification programme has failed to limit weight gain or ameliorate comorbidities. This should be done by clinicians experienced with the use of such medications under close supervision and done in conjunction with concomitant lifestyle modification programmes.**

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Unit No. 8

UNDERSTANDING MATTERS OF THE MIND IN OBESITY

Mr Adrian Toh

ABSTRACT

Obesity is a growing global concern and Singapore is not spared from this global epidemic. Apart from the increased risk of numerous serious diseases and health conditions, obese individuals are vulnerable to myriad psychological comorbidities. Obesity management through lifestyle changes can be limited by various barriers, increasing the challenge of implementation and leaving both clinicians and patients feeling frustrated and stressed. The paper examines the barriers identified in the literature, discusses the use of cognitive behavioural concepts and techniques to facilitate the lifestyle change process, and explores the use of motivation and readiness to change to guide the clinician's strategies.

Keywords; Weight Management Barriers; Cognitive Behavioural Therapy (CBT); Motivation; Adherence; Lifestyle Changes

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INTRODUCTION

Obesity is a growing global concern, having nearly tripled since 1975. In 2016, more than 1.9 billion adults (aged 18 and above)—39 percent of the world's population—were overweight (BMI ≥25 kg/m²) and 650 million were obese (BMI ≥30 kg/m²; 13 percent of the world's population).[1] Singapore is not spared from this global epidemic. The National Health Survey in 2010 reported that 29.3 percent of adult residents in Singapore were overweight while 10.8 percent were obese, suggesting that at least two in five adults in Singapore were either overweight or obese. The obesity prevalence in Singapore demonstrated a quadratic increasing trend within the survey years (1992–2010).[2] Apart from the increased risk for many serious diseases and health conditions (e.g., type 2 diabetes, high blood pressure, coronary heart disease, etc), obese individuals are vulnerable to a myriad psychological comorbidities such as mood, anxiety, and eating disorders.[3]

Despite the increasing prevalence rate and the threatening impact of obesity, it is believed that obesity is preventable through lifestyle changes such as dietary and physical activity modifications.[4] However, obesity management through lifestyle changes can be limited by various barriers, which increase the challenges of implementation and leave both clinicians and patients feeling frustrated and stressed.[5] The purposes of this scientific paper are: to examine the barriers identified in the literature; to discuss the use of cognitive behavioural concepts and techniques to facilitate the lifestyle change process; and to explore the use of motivation and readiness to change to guide the clinician's strategies.

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BARRIERS TO LIFESTYLE CHANGES

For lifestyle changes—such as dietary and physical activity modification—to be successful, it is necessary that the individuals adhere closely to the prescribed behaviours (e.g., having daily intake of 1.2 kcal for the next five days, or brisk-walking 2 km on three days in the next one week). Failing which, treatment effectiveness is hindered and results in poor treatment outcomes. Therefore, it is important to understand the barriers to individuals adhering to the prescribed behaviours.

Burgess, Hassmén, and Pumpa[6] identified barriers to lifestyle intervention in adults with obesity through systemic review, which included:

1. Poor Motivation

Patients and participants in weight loss programmes often anecdotally claimed that they are aware of what they need to do to manage their health and weight, but fail to motivate themselves to carry through with the behaviours. Motivation is key; in fact, patients themselves are aware of the importance of motivation.[7] It was suggested that one of the reasons for poorly sustained motivation might be the misinformation that significant weight loss is required for health improvements to be achieved, and subsequently lead to patients getting disheartened.[6]

2. Lack of Time

Another key barrier identified in the review was the lack of time.[6] In Singapore, the engagement of physical activity was perceived as time-consuming, when considered alongside family and work commitments.[8] Consequently, the lack of time and priority management, devoting time to health and physical activity, is challenging for individuals with obesity.

3. Environmental, Societal, and Social Pressures

The obesogenic environment, a term coined to describe "influences that the surroundings, opportunities, or conditions of life have on promoting obesity in individuals or populations", functions as a barrier to lifestyle changes.[9] These include the easy accessibility to less healthy food (especially more convenient with the trend of food delivery),

the "pro-sedentary" environment such as the convenient use of cars (and chauffeured rides), escalators and elevators, and passive leisure activities such as television and computer and mobile gaming.[10] Furthermore, when considering social norms, it is not unusual to *not* exercise. A local study found that at least one in three Singaporeans does not exercise.[11] Consequently, it will require a good amount of self-efficacy and motivation to live in the obesogenic environment and to be able to change and carry out the prescribed weight management behaviours.

4. Health and Physical Limitations

Individuals with obesity experience various health and physical limitations.[12] These include illness, injury, or complications associated with obesity or other chronic diseases, and these limitations present themselves as barriers to lifestyle changes.[6] The various health and physical limitations may result in the development of a vicious cycle that originates from the individuals' fear of pain and discomfort, leading to the limitation and avoidance of physical exercises, which consequently leads to further weight gain and/or poor health. At the same time, the presence of avoidance behaviours may further exacerbate the anxiety surrounding physical activity.

5. Negative Thoughts/Moods

Challenges in emotion regulation is a common barrier to lifestyle change.[6] Emotion regulation refers to an individual's ability to identify, understand, and accept one's emotions, and respond in adaptive ways. Studies demonstrated that poor mood and negative emotion precede unhealthy eating behaviours, maintaining obesity.[13] In addition, there is an interactive relationship between the individual's emotional distress and carbohydrate cravings.[14] Emotional distress was found to stimulate a craving for carbohydrate snack foods as consumption of these has the positive reinforcing effect of mood enhancement. The temporary positive benefits derived from the consumption will be sought after whenever emotional distress is experienced, creating a cycle of carbohydrate cravings and carbohydrate consumption.[14]

6. Socioeconomic Constraints

Socioeconomic constraints were also highlighted as barriers to lifestyle change. Locally, lower socioeconomic status (SES)—defined by education, income, and housing type—among Chinese women were associated with overweight and obesity.[15] Education is believed to enable individuals to integrate healthy behaviours into their lifestyle, such as dietary choices and exercises. Lowered income limits access to medical care, good housing and working conditions, and opportunities for healthy lifestyles. The lack of these might contribute to the association with overweight and obesity. On the flip side, studies showed that higher SES was positively associated with weight control behaviours such as physical activity, access to healthy foods, and less time spent watching television.[16–18]

7. Gaps in Knowledge/Lack of Awareness

As highlighted earlier, education enables individuals to integrate healthy behaviours into their lifestyle. The gaps in knowledge and/or lack of awareness of what healthy and detrimental lifestyle behaviours are make lifestyle change challenging. Burgess, Hassmén, and Pumpa highlighted that the lack of understanding regarding dietary and physical exercise recommendations are common among adults with obesity.[6]

8. Lack of Enjoyment of Exercise

The lack of enjoyment of exercise also presents as a barrier to lifestyle changes. Studies showed that enjoyment of exercise was positively correlated with exercise level, thus when working with individuals with obesity, the enjoyment of exercise is important for the long-term effectiveness of healthcare-based interventions.[19]

COGNITIVE BEHAVIOURAL CONCEPTS AND TECHNIQUES

Cognitive behavioural therapy (CBT) is one of the most extensively researched forms of psychotherapy. CBT has demonstrated effectiveness in addressing unhealthy eating habits, the lack of physical activity, and obesity.[20–23] CBT is particularly useful in managing unhelpful thoughts and behaviours, which prevent individuals with obesity from adhering to prescribed weight loss behaviours. Unsurprisingly, when compared with traditional dietary treatment, weight loss programmes that incorporate CBT strategies to promote lifestyle change were able to achieve better weight losses (of between 5 and 20 percent of weight vs 3 percent) and lower dropout rates (average dropout rates of 20 percent vs rates as high as 58 percent).[24] The following concepts and techniques can be useful in promoting lifestyle changes that will facilitate weight loss:

1. Self-Monitoring

Individuals with obesity can use self-monitoring to keep careful record of their own experiences, such as what, when, how much, and where they eat. Through it, they can learn about the factors (e.g., in social situations, in stressful periods) that put them at risk. With the increased awareness of triggers, they can be empowered to apply various strategies to reduce the risk of reacting to the triggers.[25] The use of self-monitoring on online platforms has also been found to be effective.[26]

2. Attentional Retraining

People who are battling obesity will often show an attentional bias in favour of food cues. For example, an individual with obesity might orient towards food cues, such as appealing high-calorie foods or a store window with rich foods. Attentional retraining demonstrates effectiveness in altering attentional biases for rewarding food cues.[27,28] It involves disrupting or at least reducing the automatic attentional bias by use of distractions, such as focusing on other aspects of the environment or engaging in physical activity.

3. Stimulus Control

The immediate food environment has powerful effects on eating. Thus, individuals with obesity can be trained to modify the stimuli in the environment that triggers their eating behaviours. Modifications include purchasing low-calorie foods and limiting high-calorie foods kept in the house. Confining eating to a specific place and time of the day can also be helpful, for example, to eat dinner at 7pm at the kitchen table, with a special placemat and to eat only when those stimuli are present. Additionally, in order to battle stress or emotional eating, creating and increasing non-food-related enjoyable activities in one's life can help in managing obesity.

4. Controlling Eating

Some strategies to increase the control over the process of eating include counting each mouthful of food and putting down eating cutlery after every few mouthfuls until the food in the mouth is chewed and swallowed. The lengthened duration between mouthfuls encourages slow eating, which promotes reduced food intake.[29] The process of mindful eating can also help reduce impulsive food choices that might impede weight gain.[30]

5. Self-Reinforcement

Positive reinforcement can further strengthen the carrying out of planned behaviours. For example, self-reinforcement following keeping to a specific diet or physical activity can include going to a movie or playing a video game. Through this process of self-reinforcement, a sense of self-control over eating can be developed. The sense of self-control can help people overcome temptations. Furthermore, being successful in weight loss is tied to greater vitality and psychological well-being, which function as further sources of self-reinforcement.[31]

6. Cognitive Restructuring

A key part of CBT in weight management is the application of cognitive restructuring. Unhealthy behaviours (e.g., poor eating habits and the lack of exercise) can be maintained by unhelpful thoughts or monologues such as "I will never lose weight" or "This weight loss attempt is going to end up like the last 28 attempts: failure". Thus, it is necessary to identify the unhelpful thinking and to consider an alternative or more balanced perspectives. An example of such would be "I have not lost as much weight as I had wished for in my previous attempts; however, I have learnt from the experience to know what behaviours led to the loss of 2 kg. Also, my past attempts do not determine the outcome of my current attempt." The process of cognitive restructuring improves the individual's self-efficacy, the belief that one will be able to lose weight.[32] Individuals with higher self-efficacy were found to have better outcomes with weight, and those with lower self-efficacy were more likely to drop out of treatments.[33,34]

7. Contingency Contracting

Contingency contracting has been found in many studies to be effective in increasing individuals' compliance with weight loss behaviours and significant weight loss was observed.[35] Contingency contracting involves a cost (e.g., forfeit of deposit money) for failure to attain a goal (e.g., abstaining from sweetened beverages) and/or provision of reward (e.g., praises or money) for attainment of a goal. The use of contingency contracting was found to reduce dropout rate as well.[36]

MOTIVATION AND READINESS TO CHANGE

The Transtheoretical Model (TTM) offers a promising framework for weight management intervention.[37,38] TTM uses stages of change to integrate processes and principles of change across major theories of intervention. The stages describe behaviour changes in an individual from less healthy behaviours to healthier ones. The five stages of change are pre-contemplation, contemplation, preparation, action, and maintenance. They are briefly described below:

1. Pre-contemplation

The individual has no intention of change and many at this stage are not aware of the problem (e.g., being within an unhealthy weight range). There are also some individuals at this stage who seek treatments because they have been pressurised by others, and consequently, they often revert to their old behaviours.

2. Contemplation

The individual in this stage gains increased awareness of the problem and the benefits of changing. However, the individual is also considering the costs involved in this change, weighing the pros and cons of changes.

3. Preparation

The individual intends to take steps to change but may not yet begin to do so. Some reasons for not starting might include being unsuccessful in the past or delaying until they can get over a stressful period of time. It is suggested that individuals in this stage should be recruited for action-oriented programmes.

4. Action

In this stage, the individual overtly modifies lifestyle behaviours to overcome the problem. Being in the action stage requires the commitment of time and energy to make the behavioural changes.

5. Maintenance

In the maintenance stage, the individual works to stabilise behaviour changes and to remain free of the old behaviours, or they might relapse.

To illustrate these with the situation of someone who is overweight: At the start, the person might not consider that he has a problem or that he is overweight. In this pre-contemplation stage, it is unlikely that he will change his behaviours. Later, he might acknowledge that he is overweight, and he considers the benefits and costs of changing his eating and exercise behaviours, or joining a weight loss programme. He is in the stage known as contemplation. After some time, he might arrive in the preparation stage, where he decides to lose weight and starts planning for the changes to be made. In the action stage, he takes actions and makes changes to address the weight issue. Over time, when the changes and new behaviours have become habits, he transits into the stage known as maintenance.

While these might sound sequential, and the eventual stage is maintenance, changes sometimes do not last due to the fact that humans make mistakes. The person who had worked hard to make healthier changes might slide back into old behaviours, where he is less active, eats less healthily, and regains weight. This stage is known as relapse. He has a number of options at this point: he can remain in relapse, move into contemplation or preparation, or straight back into action. Often, people move around between stages, going forwards, then backwards, and entering and leaving the cycle many times before they settle on a stable set of behaviours.

It can be useful to utilise tools to assess the individual's motivation and readiness to change for weight management and control. The S-weight is a tool that is easy to administer and was considered (among the assessment tools) to be more efficient in assessing the individual's readiness to change.[39] Through the assessment, it promptly identifies the stage of change the individual is in and the psychological obstacles towards weight management.[39] For example, educating, increasing importance of the cognitive dissonance, and using gamification and extrinsic rewards are useful intervention strategies for individuals in the precontemplation, contemplation, and preparation stages. Intervention strategies such as increasing the individuals' awareness of their current behavioural patterns are likely more useful for individuals in all the other stages of change, than individuals within the precontemplation stage. Thus, knowing the stage that the individual is in helps the clinician to understand the individual's challenges and to provide relevant intervention strategies accordingly.

Understanding the matters of the mind in obesity is the first step to effective lifestyle changes as an intervention for obesity. These include understanding the various barriers to lifestyle changes, the use of cognitive behavioural concepts and techniques to facilitate the process of change, and harnessing the knowledge of one's motivation and readiness to change to guide the intervention strategies.

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LEARNING POINTS

- **Barriers to lifestyle intervention in adults with obesity include: poor motivation; lack of time; environmental, societal, and social pressures; health and physical limitations; negative thoughts/moods; socioeconomic constraints; gaps in knowledge/lack of awareness; and lack of enjoyment of exercise.**
- **Cognitive behavioural therapy (CBT) is effective at addressing unhealthy eating habits, the lack of physical activity, and obesity. Some of the concepts and techniques that can be useful in promoting lifestyle changes include self-monitoring, attentional retraining, stimulus control, controlling eating, self-reinforcement, cognitive restructuring, and contingency contracting. Understanding the five stages of change and being able to identify the individual's stage of change can guide the intervention strategies. The five stages include pre-contemplation, contemplation, preparation, action, and maintenance.**

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†The patients in the cited studies were adults with type 2 diabetes

References: 1. Rybelsus® Singapore Prescribing Information. 2. Rodbard HW, et al. Diabetes Care. 2019;42(12):2272-2281. 3. Rosenstock J, et al. JAMA. 2019;321(15):1466-1480. 4. Rivera FB, et al. Curr Med Res Opin. 2024;40(9):1495-1514. 5. Singh AK, et al. Diabetes Metab Syndr. 2024;18(5):103024. 6. Gibbons C, et al. Diabetes Obes Metab. 2021 Feb;23(2):581-588.

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ASSESSMENT OF 30 MCQS

FPSC NO : 135
MCQS ON BASIC OBESITY MANAGEMENT ACCREDITATION 6
SUBMISSION DEADLINE: 8 December 2026, 12 NOON

INSTRUCTIONS
- **To submit answers to the following multiple choice questions, you are required to log on to the College Online Portal (https://lms.wizlearn.com/cfps/)**
- **Please contact sfp@cfps.org.sg if you have not received an email on the new LMS account.**
- **Attempt ALL the following multiple-choice questions.**
- **There is only ONE correct answer for each question.**
- **The answers should be submitted to the College of Family Physicians Singapore via the College Online Portal before the submission deadline stated above.**
- **There will be NO further extension of the submission deadline**

1. Which of the following statements best reflects the current understanding of obesity as a chronic disease?

A. The primary cause of obesity is usually a lack of discipline regarding diet and exercise

B. The assessment of obesity should focus mainly on BMI as the causes of obesity are usually self-evident

C. The assessment of adiposity-based chronic disease should include reviewing contributing factors, evaluating for obesity-related complications, and reviewing limitations to function and activities of daily living and barriers to treatment

D. Psychosocial factors are only relevant if the patient has a diagnosed mental health condition

E. Patients who regain weight after treatment generally require stronger motivation rather than reassessment

2. A patient says, "I regained weight because I lack discipline." Which of the following responses is most accurate?

A. Weight regain is uncommon when patients follow a strict diet

B. Weight regain occurs because patients eventually stop trying hard enough

C. Psychological and social factors have little influence on obesity

D. Weight regain can occur because weight loss triggers biological adaptations that increase hunger and reduce energy expenditure

E. Long-term obesity management rarely requires ongoing treatment

3. A 27-year-old female is seeing you for newly diagnosed prediabetes. She participated in a research study, during which a 75 g oral glucose tolerance test showed that her fasting glucose was 5.9 mmol/L and her 2-hr post glucose was 10.3 mmol/L. She was told that she has prediabetes and instructed to see her GP.

She has been having irregular menses for the past three years, which coincided with gradual weight gain. She was evaluated by a gynaecologist two years ago and was found to have polyendocrine metabolic ovarian syndrome (PMOS), but she has since not returned for the follow-up. She is currently not sexually active but is planning to get married soon.

On examination, her weight is 85.5 kg, BMI 34.4 kg/m², and BP 148/92 mmHg.

In the assessment and management of the patient, which of the following statements is FALSE?

A. Further assessment should include identification of obesity-related complications beyond pre-diabetes, including hypertension, MASLD, OSA, and reproductive dysfunction

B. Evaluation for secondary contributors to weight gain should include review of weight chart, targeted history, and examination for features of hypothyroidism, hypogonadism, and glucocorticoid excess

C. Obesity severity and treatment decisions should be based primarily on BMI classification

D. Future pregnancy plans are important to discuss because they influence the choice and timing of obesity treatment

E. Screening for common endocrine disorders that may contribute to weight gain, such as hypothyroidism, should be considered if not previously performed

4. A 24-year-old woman presents with progressive weight gain. Two years ago, her weight was 68 kg, BMI 25kg/m². Which of the following clinical features would most strongly prompt investigation for an underlying endocrine cause of weight gain?

A. She has gained 4 kg over two years while receiving intermittent courses of prednisolone for rheumatoid arthritis flares

B. She has gained 7 kg gradually over the past five years since starting full-time employment

C. She has gained 3 kg over three months after a knee injury that significantly reduced her physical activity

D. She has recurrent episodes of fasting and nocturnal symptomatic hypoglycaemia, with documented capillary glucose values as low as 1.8 mmol/L (45 mg/dL), occurring over the past four months

E. She retained 8 kg of gestational weight six months after delivery despite attempts at lifestyle modification

5. A 42-year-old woman with a history of controlled hypertension and major depressive disorder comes to the clinic to discuss obesity pharmacotherapy. She is no longer on anti-depressants. Her BMI is 34 kg/m². Which statement is MOST accurate?

A. Contrave (Naltaxone/Bupropion) may be considered, but patients should be monitored for neuropsychiatric symptoms and worsening mood
B. Contrave (Naltaxone/Bupropion) is contraindicated because all patients with depression are at high risk of psychiatric deterioration
C. GLP-1 receptor agonists are contraindicated in patients with a history of depression
D. Orlistat commonly causes central nervous system adverse effects requiring psychiatric monitoring
E. Phentermine is contraindicated solely because the patient has depression

6. A 45-year-old Chinese woman attends your clinic for follow-up of hypertension. Her blood pressure is well controlled. You notice that her weight has increased by 6 kg over the past year, and her BMI is now more than 30 kg/m². She has not previously discussed weight management with you.

According to a patient-centred approach to obesity care, what is the most appropriate first step?

A. Explain the health risks of obesity and benefits of sustained weight loss
B. Identify barriers to weight management and arrange appropriate support
C. Ask whether she would be comfortable discussing her weight and its impact on her health
D. Establish mutually acceptable behavioural and weight-management goals
E. Assess potential drivers of weight gain and obesity-related complications

7. 7. A 45-year-old patient with obesity asks which diet is the most effective for long-term weight loss. Which of the following is the best response based on current evidence?

A. There is no single dietary approach proven to be superior in the long term; treatment should be individualised
B. A low-carbohydrate diet is superior to all other dietary approaches
C. A very-low-calorie diet is the preferred approach for all patients with obesity
D. Intermittent fasting is consistently superior to continuous energy restriction
E. Meal replacement should be recommended for all patients with obesity

8. A patient with obesity asks how much weight loss would provide meaningful health benefits. Which of the following is most accurate?

A. Health benefits occur only after sufficient weight loss is achieved to reach a healthy BMI
B. Weight loss below 10% has no metabolic benefit
C. Modest weight loss mainly improves cosmetic outcomes without improving metabolic health
D. Weight loss of 5–10% is associated only with improved lipid profile
E. Modest weight loss of 5–10% can improve glycaemia, blood pressure, lipid profile, mobility, and quality of life

9. A patient with obesity has not achieved the desired weight or metabolic targets with a conventional energy-restricted diet. Under appropriate clinical supervision, which statement about a very low energy diet (VLED) is most accurate?

A. VLEDs should routinely be continued indefinitely to maintain weight loss
B. VLEDs provide approximately 1,200–1,500 kcal/day
C. VLEDs based on total meal replacements provide approximately 800 kcal/day or less and require supervision by trained healthcare professionals
D. VLEDs are associated with a higher risk of eating disorders than moderate energy-restricted diets
E. VLEDs do not require a structured transition back to normal eating

10. Which of the following best describes the importance of dietary counselling in achieving sustainable weight loss in patients with obesity?

A. It allows dietary recommendations to be individualised according to the patient's health status, preferences, and ability to sustain the plan, with ongoing support to manage weight regain and weight plateaus throughout the process
B. It ensures that patients follow the same standardised diet plan based on evidence-based practice
C. It helps patients achieve the greatest possible amount of weight loss
D. It focuses primarily on providing a meal plan for patients to follow strictly
E. It is only required during the initial phase of weight loss to provide necessary knowledge and is less important during weight maintenance

11. Which is the most accurate statement about aerobic exercise for weight loss in adults with overweight or obesity?

A. Keeping to just 150 minutes of aerobic exercise per week is sufficient for weight maintenance after weight loss
B. Increasing exercise volume has no effect on waist circumference or body fat
C. Aerobic exercise is effective only when performed at vigorous intensity

D. Aerobic exercise generally produces modest weight loss, with greater benefits when combined with dietary energy restriction

E. All individuals undertaking the same exercise programme can achieve similar weight loss

12. Why is resistance exercise particularly valuable during dietary weight reduction in older adults with obesity?

A. It helps preserve lean mass and improve muscular strength

B. It guarantees that no muscle mass will be lost

C. It produces greater weight loss than all other exercise modalities

D. It eliminates the need for aerobic exercise

E. It improves strength only when body weight increases

13. A person with obesity has exercised regularly but has not lost weight. Which statement is most appropriate?

A. The exercise programme has been ineffective

B. Cardiometabolic benefits occur only after substantial weight loss

C. Exercise should be stopped until dietary weight loss is achieved

D. Stable body weight indicates that visceral fat has not changed

E. Exercise may still improve insulin sensitivity, blood pressure, and fat distribution

14. Which is the correct recommended guideline for aerobic and resistance exercise?

A. Aerobic: 30 minutes per day, low intensity, most days of the week. Resistance: moderately heavy load, at least twice a week, covering major muscle groups

B. Aerobic: at least 150 minutes per week, moderate intensity, most days of the week. Resistance: once a week, covering major muscle groups

C. Aerobic: twice per week, moderate intensity. Resistance: most days of the week, covering major muscle groups

D. Aerobic: at least 150 minutes per week, moderate intensity, most days of the week. Resistance: moderately heavy load, at least twice a week, covering major muscle groups

E. Aerobic: achieve 10,000 steps daily. Resistance: moderately heavy load, at least twice a week

15. In which of the following patients would obesity pharmacotherapy generally NOT be initiated at the present time, either because it is not indicated or because there are major contraindications/precautions that must first be addressed?

i. A 30-year-old man with BMI 40 kg/m², no obesity-related complications, and multiple previous unsuccessful weight loss attempts

ii. A 28-year-old woman with BMI 24 kg/m², no central obesity or obesity-related complications, requesting medication for cosmetic weight loss

iii. A 35-year-old woman with BMI 34 kg/m² and prediabetes who is actively trying to conceive within the next few months

iv. A 28-year-old woman with BMI 27.5 kg/m², MASLD, and PMOS, who is not planning pregnancy

v. A 68-year-old man with BMI 29 kg/m², hypertension, and type 2 diabetes mellitus, currently receiving dialysis for end-stage kidney disease

A. II only

B. II and III only

C. III and V only

D. II, III, V

E. All of the above

16. Which of the following statements about GLP-1 receptor agonists are TRUE?

i. The lack of gastrointestinal side effects experienced by the patient implies that the medication has no effect on the patient

ii. Subcutaneous weekly semaglutide and tirzepatide should be stopped for at least two weeks in women planning for pregnancy

iii. In patients with established cardiovascular disease and BMI of ≥30 kg/m², Semaglutide 2.4 mg/week can reduce the risk of major adverse cardiovascular events

iv. Tirzepatide has been shown to reduce death from cardiovascular causes or worsening of heart failure in patients with heart failure with preserved ejection fraction (HFpEF) and obesity

v. Caution should be practised in patients with symptomatic gallstones when initiating GLP-1 receptor agonists

A. II, III, V

B. III, IV, V

C. I, III, IV

D. I, II, III, V

E. All of the above

17. Ms Tan, a 28-year-old Chinese female, comes to you with oligomenorrhoea and hirsutism. She is currently on treatment via her psychiatrist for anxiety disorder and is being investigated for a thyroid nodule. You diagnose her with polyendocrine metabolic ovarian syndrome (PMOS) after a work-up, during which time she was found to have an elevated fasting plasma glucose of 6.4 mmol/dL (confirmed on repeat sample on a separate day, with a HbA1c of 6%).

Her BMI is 28.5 kg/m², waist circumference 92 cm, BP 130/77 mmHg. You counsel her that weight loss is part of the treatment of her current medical conditions. She has had obesity since adolescence. Since starting to swim three times a week six months ago, she has kept her weight stable. She is open to starting obesity medications.

Which of the following on the use of obesity medications in her is FALSE?

A. Treatment with orlistat and semaglutide in the long term can reduce her risk of developing diabetes mellitus
B. Orlistat can help her lose about 3–5% of her weight in addition to lifestyle
C. The presence of a thyroid nodule alone is not a contraindication to GLP-1 receptor agonist therapy, although a history of medullary thyroid carcinoma or MEN2 should be excluded
D. Phentermine is a good option for her as it is low cost
E. Regardless of choice of medication, she must practise active contraception and discontinue the medication immediately should she get pregnant

18. The use of a glucagon-like peptide 1 (GLP-1) receptor agonist is not recommended in the following patients with BMI >30 kg/m²:

i. A patient with type 2 diabetes mellitus and renal impairment

ii. A patient with long-standing type 2 diabetes mellitus and frequent vomiting after food

iii. A patient with a thyroid nodule of undetermined origin

iv. A patient with history of severe pancreatitis of unknown aetiology

v. A patient with known heart failure (HFpEF) and a history of myocardial infarction

A. I, III, IV
B. II, III, IV
C. I, II, III
D. I, II, III, IV
E. All of the above

19. A 55-year-old man with a BMI of 33 kg/m² has hypertension, OSA, and stable coronary artery disease. He starts on subcutaneous semaglutide in addition to lifestyle intervention. The dose is currently titrated up to 1 mg after two months. At his review one month later, he has lost 1.5% of his initial body weight despite good adherence.

Which of the following is the most appropriate next step?

A. Continue the medication unchanged as any amount of weight loss predicts long-term success
B. Reassure him that efficacy cannot be assessed until least one year later
C. Review adherence and contributing factors, and continue dose escalation toward the maintenance dose if treatment is well tolerated
D. Double the dose above to the approved maximum dose
E. Discontinue semaglutide as the early weight loss response is inadequate and hence it is ineffective

20. Which statement best describes laparoscopic sleeve gastrectomy?

A. About 70–80% of the stomach is resected, with no foreign body placed and no diversion of food passage
B. A silicone ring is placed around the upper stomach and adjusted every 4–6 weeks
C. A small gastric pouch is created and connected to the mid-jejunum
D. The stomach is sutured from within during gastroscopy to reduce its volume
E. The duodenum is attached to the jejunum

21. In the multi-centre RCT cited in the article, endoscopic sleeve gastroplasty (ESG) achieved what total body weight loss at one year in class 1 and 2 obesity?

A. 5.2%
B. 13.6%
C. 25%
D. 30%
E. 40%

22. Several months after bariatric surgery, a patient has persistent pain and vomiting. The most appropriate consideration is:

A. Normal adaptation, so reassure and review in a year
B. Simply advise eating faster to clear the food
C. Underlying stenosis or ulcer, which may require investigation and treatment
D. Regret about the procedure, so refer for reversal counselling
E. Inadequate rehabilitation. Referral for Physiotherapy

23. A 10-year-old boy has a BMI at the 95th percentile on the Singapore BMI-for-age chart. According to Health Promotion Board definitions, he should be classified as:

A. Underweight
B. Normal weight
C. At risk of overweight
D. Overweight
E. Severely overweight (obesity equivalent)

24. A 15-year-old adolescent with obesity is reviewed in primary care. Which set of investigations is most appropriate for initial screening of obesity-related complications?

A. Full blood count, ESR, renal panel
B. Thyroid function tests, cortisol, insulin level
C. Fasting lipids, HbA1c, alanine aminotransferase (ALT)
D. Echocardiography, chest radiograph, pulmonary function tests
E. Growth hormone level, ACTH, prolactin

25. A 14-year-old adolescent with severe obesity has not achieved adequate improvement despite intensive lifestyle intervention. Which anti-obesity medication has demonstrated the greatest mean reduction in BMI in adolescent clinical trials?

A. Orlistat
B. Metformin
C. Liraglutide
D. Topiramate
E. Semaglutide

26. According to current recommendations for children and adolescents, which statement is CORRECT?

A. Recreational screen time should be encouraged if educational
B. Children should perform muscle-strengthening activities only once weekly
C. Fruit juice can be consumed freely because it is natural
D. Children should accumulate at least 60 minutes of moderate-to-vigorous physical activity daily
E. Sugary beverages are preferred over artificially sweetened beverages

27. Which approach is most consistent with recommended management of childhood obesity?

A. Focus exclusively on the child's eating habits
B. Use scare tactics to improve adherence
C. Wait for obesity-related complications to develop before intervening
D. Emphasise rapid weight loss as the principal goal
E. Adopt a family-based approach and use motivational interviewing techniques

28. A 42-year-old man with obesity (BMI 34 kg/m²), hypertension, and prediabetes is found to have hepatic steatosis on ultrasonography performed during a health screening. Viral hepatitis screening is negative, and his alcohol intake is minimal. The diagnosis of metabolic dysfunction-associated steatotic liver disease (MASLD) is made.

Which of the following statements is TRUE?

A. MASLD is a benign condition and does not increase the risk of cardiovascular disease
B. Lifestyle intervention is a key component of MASLD treatment
C. A weight loss of 3–5% is usually required to improve hepatic fibrosis
D. Patients with MASLD should avoid physical activity until liver enzymes normalise
E. Pharmacotherapy for obesity has no role in the management of MASLD

29. A 58-year-old woman presents for evaluation of progressive bilateral knee pain due to osteoarthritis (OA), which severely limits her mobility. She is scheduled for total knee replacement and has been asked to lose weight before her surgery. She has a history of type 2 diabetes and hypertension. Her current weight is 92 kg, height is 158 cm (BMI 36 kg/m²). She has no features of Cushing's syndrome. However, she has noticeable muscle weakness in her lower extremities, difficulty rising from a chair without using her arms, and a slow gait speed.

Beyond managing her glycaemic control and joint pain, which of the following is the most appropriate comprehensive management strategy for addressing her metabolic health and functional limitations?

A. Initiate a GLP-1 receptor agonist for weight loss and glycaemic control, paired with a very-low-calorie diet to accelerate weight loss and offloading of the osteoarthritic knees
B. Initiate a GLP-1 receptor agonist combined with dietitian guidance to optimise protein intake, and a structured progressive resistance training programme
C. Initiate very-low-calorie diet and avoid GLP-1 receptor agonists due to the risk of exacerbating muscle atrophy
D. Rely solely on a GLP-1 receptor agonist without lifestyle modification, as drug-induced weight loss will automatically restore her lower extremity muscle mass and function
E. Avoid obesity medications entirely, focusing strictly on high-intensity aerobic exercise to offset the muscle-wasting effects of ageing

30. A 41-year-old Chinese man comes to your clinic. He has a history of obesity and impaired fasting glucose. He currently takes no medications. He has a BMI of 42kg/m². His BP is 134/72 mmHg. His physical exam is unremarkable. Laboratory testing is notable for 2+ protein on urinalysis and a urine protein/creatinine ratio of 2.1 g/day. His renal panel showed eGFR 60 ml/min. HbA1c is 6.2%. Liver panel and inflammatory markers are normal.

Which statement best reflects an additional potential benefit of GLP-1 receptor agonist therapy beyond weight loss?

A. GLP-1 receptor agonists are contraindicated in patients with chronic kidney disease because they accelerate decline in kidney function

B. GLP-1 receptor agonists improve renal outcomes only through appetite suppression

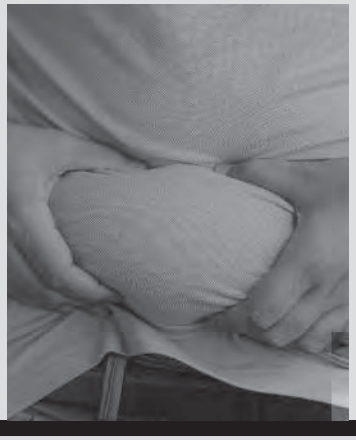
C. Renal benefits of GLP-1 receptor agonists have been demonstrated only in patients without diabetes

D. GLP-1 receptor agonists should be discontinued when eGFR falls below 45 mL/min/1.73 m²

E. GLP-1 receptor agonists may reduce albuminuria and slow progression of chronic kidney disease regardless of diabetes status

FPSC 133 "Chronic Disease Management 2026" Answers to 30 MCQs					
1.	D	11.	B	21.	A
2.	B	12.	C	22.	D
3.	A	13.	A	23.	C
4.	E	14.	B	24.	B
5.	C	15.	D	25.	C
6.	E	16.	C	26.	D
7.	D	17.	C	27.	B
8.	A	18.	B	28.	C
9.	C	19.	E	29.	D
10.	E	20.	A	30.	E

FPSC 134 "Multiple Myeloma" Answers to 15 MCQs					
1.	E	6.	C	11.	D
2.	D	7.	A	12.	C
3.	B	8.	A	13.	B
4.	D	9.	B	14.	D
5.	C	10.	D	15.	A



READINGS

A SELECTION OF TEN READINGS ON TOPICS RELATED TO
2026 FAMILY PRACTICE SKILLS COURSE:
BASIC OBESITY MANAGEMENT ACCREDITATION 6

https://doi.org/10.33591/sfp.52.5.ur

A SELECTION OF TEN READINGS ON TOPICS RELATED TO 2026 FAMILY PRACTICE SKILLS COURSE: BASIC OBESITY MANAGEMENT ACCREDITATION 6

FPSC135 – SATURDAY, 03 OCT 2026 & SUNDAY 04 OCT 2026: 2.00pm–5.30pm
All are available as free full text

Selection of readings made by A/Prof Goh Lee Gan

READING 1. WHY DO WE LOSE BONE DURING WEIGHT LOSS?

Shapses SA,[1,2] McGuire BD.[3] Why Do We Lose Bone During Weight Loss: Can It Be Prevented? Curr Osteoporos Rep. 2026 Sep 9;24(1):38. PMID: 42714789.

doi: 10.1007/s11914-026-00984-z. Free full text.

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ABSTRACT
PURPOSE OF REVIEW: Weight loss is recommended for obesity management, but it also increases bone turnover and reduces bone mineral density (BMD) and bone quality (microarchitecture, geometry, and strength). This review examines mechanisms and the multiple factors that influence skeletal responses to caloric restriction.

RECENT FINDINGS: Bone loss during weight loss likely reflects multiple interacting mechanisms, including alterations in endocrine signalling, impaired calcium absorption, reduced mechanical loading, and alterations in the gut microbiota. The extent of bone loss is further influenced by age, sex, initial body weight, nutrient intake, and method of weight loss, with greater loss in older and lean adults, and after severe caloric restriction, such as with incretin mimetics or bariatric surgery. Current evidence emphasises the importance of adequate nutrient intake (e.g., calcium, protein, etc) and exercise as countermeasures to attenuate bone loss. Emerging evidence also suggests that additional countermeasures, including osteoporosis medications and other lifestyle interventions, might affect weight-loss-induced bone loss, and randomised trials are needed.

Weight-loss-induced bone loss is multifactorial, involving endocrine, metabolic, nutritional, mechanical, and gut microbiota-mediated mechanisms that remain incompletely understood. These mechanisms influencing skeletal responses to weight loss can inform targeted strategies to preserve bone health and reduce fracture risk during obesity management.

READING 2. RESEARCH ADVANCES IN DIAGNOSIS AND TREATMENT OF MASLD/MASH

Wang D,[1] Miao J,[1] Zhang L,[1] Zhang L.[1] Research advances in the diagnosis and treatment of MASLD/MASH. Ann Med. 2025 Dec;57(1):2445780. PMID: 41421798.

doi: 10.1080/07853890.2024.2445780. Free full text.

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ABSTRACT
Background: The global increase in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) and its progression to Metabolic Dysfunction-Associated Steatohepatitis (MASH) pose significant public health challenges.

Objective: To review key research advancements in the diagnosis and treatment of MASLD/MASH over the past two decades.

Methods: This article synthesises recent findings on diagnostic and therapeutic strategies for MASLD/MASH, focusing on non-invasive diagnostic tools and emerging pharmacological treatments.

Results: Non-invasive diagnostic techniques, including high-resolution CT, MRI, FIB-4 index, and FAST score, have enhanced early detection and diagnostic accuracy.

Treatment strategies encompass pharmaceuticals such as THR-β agonists, FXR agonists, PPAR agonists, GLP-1 receptor agonists, and SGLT2 inhibitors, alongside lifestyle interventions like dietary changes, weight management, and exercise. Surgical options, including gastric bypass and liver transplantation, are effective, particularly for obese and advanced MASH patients. However, challenges remain in standardising treatment protocols, validating long-term efficacy, and addressing patient-specific factors.

Conclusion: MASLD/MASH treatment has advanced significantly with new diagnostic tools and therapies. Future research should focus on personalised approaches, long-term treatment outcomes, and multidisciplinary collaboration to improve patient care and treatment efficacy.

READING 3. CARDIORENAL OUTCOMES OF WEIGHT LOSS INTERVENTIONS IN PEOPLE WITH CKD AND T2DM

Wilkinson TJ,[1,2] Goldney J,[1,2] Yates T,[1,2] Henson J,[1,2] Zaccardi F,[1,2] Khunti K,[1–3] Webb D,[2,4] Papamargaritis D,[1–3] Davies MJ,[1–3] Cardiorenal outcomes of weight loss interventions in people with CKD and type 2 diabetes. Nephrol Dial Transplant. 2026 Jun 30;41(7):1262–1273.PMID: 41344888.

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ABSTRACT

BACKGROUND AND HYPOTHESIS: In patients with type 2 diabetes mellitus (T2DM) and chronic kidney disease (CKD), there remains a paucity of data from large, high-quality, real-world studies on the efficacy of weight-lowering interventions, including the glucagon-like peptide-1-based therapies semaglutide and tirzepatide, as well as bariatric surgery (BS). This retrospective cohort study evaluates these interventions on cardiorenal outcomes and mortality using health records from TriNetX US Collaborative Network.

METHODS: Three cohorts with T2DM and CKD living with overweight or obesity, prescribed semaglutide or tirzepatide, or with a BS procedure were propensity-score matched with a cohort receiving dipeptidyl peptidase 4 inhibitors (DPP4i, weight-neutral control). We investigated the hazard ratio (HR) of: (i) end-stage renal disease (ESRD); (ii) myocardial infarction (MI); (iii) stroke; and (iv) all-cause mortality.

RESULTS: Cohorts included 17,749 (semaglutide, age 64.1±10.9 years, 57.4% female), 4,211 (tirzepatide, 63.7±10.8, 58.1%), and 2,603 (BS, 56.1±11.2, 74.1%) patients. Compared with DPP4i, semaglutide reduced the risk of ESRD [HR 0.78 (95% CI 0.71 to 0.85)], while tirzepatide showed a reduction of 42 percent (0.58, 0.45–0.75). BS lowered ESRD risk (0.79, 0.67 to 0.92). Semaglutide reduced MI (0.80, 0.72–0.88) and stroke (0.85, 0.77–0.95) risk, while tirzepatide lowered MI and stroke by 24 percent. BS was associated with reduced MI (0.45, 0.35–0.58) and stroke (0.57, 0.44–0.74) risk. All-cause mortality risk was reduced by semaglutide (0.64, 0.59–0.70), tirzepatide (0.47, 0.35–0.63), and BS (0.68, 0.57–0.81).

CONCLUSIONS: Using a real-world dataset, semaglutide, tirzepatide, and BS-interventions are shown to elicit weight loss and improve glycaemic control. They have a myriad of benefits on cardio-renal outcomes and mortality, supporting their use as disease-modifying therapy options in T2DM and CKD.

READING 4. TEN-YEAR WEIGHT REGAIN AFTER BARIATRIC SURGERY

Ribeiro S,[1,2] Ferreira P,[3,4] Lima B,[5] Moreno T,[1,2] Gonçalves J,[1,2] Urbano Ferreira H,[1,2] Meira I,[1,2] Menino J,[1,2] Leite AR,[1,2] Borges-Canha M,[1,2] Silva MM,[1,2] Guerreiro V,[1,2] Pedro J,[1,2,6] Varela A,[1,2,6] Festas Silva D,[1,2,6] Freitas P,[2,6] Lima da Costa E,[6] Queirós J.[1] Ten-Year Weight Regain after Bariatric Surgery: Prevalence, Predictors, and Metabolic Impact. Obes Facts. 2026;19(3):249–264.PMID: 41217996.

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ABSTRACT

INTRODUCTION: Bariatric surgery (BS) is an effective intervention for severe obesity, but long-term weight regain (WR) can occur and may impact comorbidity outcomes.

METHODS: In this retrospective study, we analysed patients who underwent BS with a 10-year follow-up. WR was calculated as the percentage of maximum weight loss (%MWL) that had been regained from the nadir weight. Patients were categorised as "Maintainers" (WR ≤20% of %MWL) or "Regainers" (WR >20%).

RESULTS: Among the 353 included patients 317 underwent Roux-en-Y gastric bypass (RYGB) and 36 underwent sleeve gastrectomy (SG), 90.4 percent were female, with a mean age of 42±11 years and a mean body mass index of 44.6 kg/m^2. Mean WR at 10 years was 28 percent (±25), higher in SG vs RYGB (41.37% vs 26.17%, p=0.03). Overall, 56.7 percent were Regainers. Baseline type 2 diabetes mellitus (T2D) was associated with an approximately 40 percent reduced risk of WR >20% (OR=0.59; 95% CI: 0.38–0.93; p=0.023). WR was not significantly associated with the recurrence or remission of T2D, hypertension, or dyslipidaemia.

CONCLUSION: WR is common a decade after BS, particularly following SG. While WR does not appear to significantly impact comorbidity recurrence, its clinical relevance warrants further study. Standardised definitions are urgently needed to guide long-term management.

READING 5. "OBESITY FIRST" APPROACH IN HEALTHCARE

Mulder CJJ,[1,2] Bayoumy AB,[3,4] Ansari AR.[5] The "Obesity First" approach: Redefining the future of healthcare. Indian J Gastroenterol. 2026 Feb;45(1):15–19. PMID: 41152558.

doi: 10.1007/s12664-025-01882-5. Free full text.

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ABSTRACT

Obesity is a highly prevalent chronic disease driven by food addiction and associated with increased premature mortality. Obesogenic environments promote unhealthy behaviour, making weight management challenging. Until recently, effective pharmacological treatments were lacking.

The introduction of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) represents a major breakthrough in obesity care, with the potential to transform treatment strategies. Despite their efficacy, high costs (as of 2025) limit accessibility, particularly in low and middle-income regions, where parallel, unregulated use is emerging. Obesity remains under-recognised as a primary medical condition, especially in populations prone to metabolic complications, including metabolic dysfunction-associated steatotic liver disease (MASLD). Gastroenterology has historically underestimated the role of GLP-1 RAs in the past.

Moving forward, the choice between GLP-1 therapy and bariatric endoscopy/surgery will become a central research focus, with treatment failures in one modality already leading to crossover. GLP-1 RAs are expected to significantly impact obesity-related comorbidities, including hypertension, dyslipidaemia, type-2 diabetes, sleep apnoea, MASLD, and inflammatory bowel disease (IBD).

An "Obesity First" approach may reshape healthcare by addressing obesity as the primary topic cause for chronic disease. By 2035, the role of GLP-1 RAs as potential lifelong treatment will become clearer, with generic market expansion anticipated following patent expirations (China 2026; Europe 2031).

READING 6. PHARMACOTHERAPY FOR OBESITY MANAGEMENT IN ADULTS

Pedersen SD,[1] Manjoo P,[1] Dash S,[1] Jain A,[1] Pearce N,[2] Poddar M.[1] Pharmacotherapy for obesity management in adults: 2025 clinical practice guideline update. CMAJ. 2025 Aug 10;197(27):E797–E809. PMID: 40789597.

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ABSTRACT

BACKGROUND: Pharmacotherapy is a key component of comprehensive obesity management, alongside behavioural therapy and metabolic and bariatric surgery. In this guideline, we update the pharmacotherapy recommendations in the 2020 Canadian clinical practice guideline on obesity in adults and in the 2022 pharmacotherapy for obesity management revision to provide current recommendations for clinicians on the efficacy, safety, and appropriate use of pharmacotherapy in the management of obesity in adults.

METHODS: This guideline update follows the same methodology as the 2020 Canadian guideline on obesity in adults, adhering to the Appraisal of Guidelines for Research and Evaluation instrument and using the Shekelle framework to assess and grade evidence and to formulate recommendations. Building on the search conducted for the 2022 pharmacotherapy revision, we conducted a systematic literature review (search dates January 2022 to July 2024), supplemented by relevant trials published through May 2025, to identify studies assessing the efficacy of pharmacotherapy for weight management. We also conducted 13 targeted searches on the management of weight-related complications in 13 subpopulations with important adiposity-related health issues. We engaged primary care physicians, obesity medicine specialists, and people with lived experience of obesity to provide feedback on the recommendations.

RECOMMENDATIONS: This update includes six new and seven revised recommendations since the 2022 pharmacotherapy guideline revision (all 2020 pharmacotherapy recommendations are updated). Measures of central adiposity, in addition to ethnicity-specific body mass index and adiposity-related complications, should be used to guide the decision to initiate pharmacotherapy. Obesity pharmacotherapy should be used in conjunction with health behaviour changes and individualised based on a person's specific health needs and in keeping with their values and preferences.

Recommendations support long-term use of obesity pharmacotherapy for sustained weight loss and maintenance of weight loss. We provide recommendations for use of specific obesity pharmacotherapies with proven benefit in specific subpopulations—atherosclerotic cardiovascular disease, heart failure with preserved ejection fraction, metabolic dysfunction-associated steatohepatitis, prediabetes, type 2 diabetes, obstructive sleep apnoea, osteoarthritis—and for those with certain specific monogenic causes of obesity. We recommend against the use of compounded medications or medications other than those approved for weight loss in people with excess adiposity.

INTERPRETATION: Pharmacotherapy in obesity facilitates clinically meaningful weight loss and important improvements in obesity-related health complications. Clinicians who treat people with obesity with or without obesity-related health complications should appropriately use pharmacotherapy as an integral part of their treatment paradigm.

READING 7. PREVALENCE AND CORRELATES OF ANAEMIA FOLLOWING ROUX-EN-Y GASTRIC BYPASS

Gao Z,[1,2] Liang Y,[3] Zheng C,[4] Wu Y,[5] Xiang S,[5] Zhang X,[6] Li M,[2] Zhang Z,[7] Yang J.[1] Prevalence and correlates of anaemia following Roux-en-Y gastric bypass: a systematic review and meta-analysis. Int J Surg. 2025 Nov 1;111(11):8462–8479. PMID: 40788038.

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ABSTRACT

OBJECTIVE: To estimate the prevalence and associated factors of anaemia after Roux-en-Y gastric bypass (RYGB).

METHODS: A comprehensive literature search was conducted across PubMed, EMBASE, and CENTRAL from inception through 18 January 2025, utilising search terms including anaemia, haemoglobin, Hb, gastric bypass, and RYGB. Eligible studies reported anaemia prevalence post-RYGB with data on either follow-up duration or Roux limb length.

RESULTS: From 2,787 screened records, 74 studies involving 12,262 patients met inclusion criteria. Pooled analysis revealed a 26 percent overall postoperative anaemia prevalence. Temporal stratification revealed a progressive increase in prevalence: 15 percent at ≤1 year, 27 percent at >1–5 years, and 35 percent at >5 years post-surgery. Meta-regression confirmed a significant positive correlation between anaemia prevalence and follow-up duration. Studies with inadequate supplementation of iron, vitamin B12, and folic acid reported higher anaemia rates compared to those with adequate supplementation. Aetiological analyses identified strong associations of anaemia with vitamin B12 and iron deficiencies, but not with folic acid deficiency. Demographic risk factors included preoperative anaemia, female sex, and baseline BMI >45 kg/m^2. RYGB with longer Roux limbs showed a trend toward higher anaemia risk.

CONCLUSION: This study quantifies a substantial burden of anaemia following RYGB, demonstrating its temporal progression. The findings underscore the necessity for protocolised long-term haematologic surveillance, optimised nutrient repletion strategies targeting iron and vitamin B12, and personalised risk mitigation approaches for high-risk cohorts, particularly women and those with long Roux limbs, preoperative anaemia, or severe obesity.

READING 8. OBESITY ASSESSMENT AND TREATMENT ACROSS THE CARE CONTINUUM

Kushner RF,[1] Shapiro M.[2] Obesity: assessment and treatment across the care continuum. Ann Med. 2025 Dec;57(1):2521433. PMID: 40568728.

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ABSTRACT

BACKGROUND: Obesity is a chronic condition of dysregulated energy balance that is caused by a confluence of nutritional, neurological, hormonal, and metabolic factors. Clinically, obesity is associated with myriad consequences to overall health. Treatment should be based on a shared decision-making process between the patient and their professional team that considers the stage of the disease, wellness goals, and desired lifestyle and can include a combination of behavioural and lifestyle interventions, pharmacological therapies, and surgery. The chronic nature of obesity necessitates adjustments in treatment plans to match the evolving needs and goals of the patient over time, establishing a "care continuum".

METHODS: We conducted a broad, narrative literature search using PubMed for articles published on the assessment, diagnosis, and treatment of adults with obesity.

RESULTS: In this narrative literature review, we outline evidence-based best practices for the diagnosis and assessment of obesity along with the various available treatment modalities. We also present considerations for treating patients with obesity with a focus on the selection of obesity medications based on severity, concurrent conditions, mechanisms of action, and treatment goals. Finally, we discuss lifestyle management, the shift from weight loss to weight maintenance, and the implications of these changes in the care continuum.

CONCLUSION: Continued, individualised treatment of patients with obesity from diagnosis to weight maintenance is imperative for sustained weight reductions, and strategies should be tailored to the changing needs of patients over time.

READING 9. BARIATRIC SURGERY AND RELEVANT COMORBIDITIES

Wunker C,[#,1] Kumar S,[#,2] Hallowell P,[3] Collings A,[4] Loss L,[5] Bansal V,[6] Kushner B,[7] Zoumpou T,[8] Kindel TL,[9] Overby DW,[10] Chang J,[11] Ayloo S,[12] Sabour AF,[13] Ghanem OM,[14] Aleassa E,[15] Reid A,[16] Rodriguez N,[15] Haskins IN,[17] Hilton LR,[18] Slater BJ,[19] Palazzo F.[20] Bariatric surgery and relevant comorbidities: a systematic review and meta-analysis. Surg Endosc. 2025 Mar;39(3):1419–1448. PMID: 39920373.

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ABSTRACT
BACKGROUND: Obesity is a growing epidemic in the United States, and with this has come an increasing volume of metabolic surgery operations. The ideal management of obesity-associated medical conditions surrounding these operations is yet to be determined. This review sought to investigate the routine use of intraoperative cholangiogram (IOC) with cholecystectomy during or after a bypass-type operation, the ideal management of post-sleeve gastrectomy gastroesophageal reflux disease (GERD), and the optimal bariatric operation in patients with known inflammatory bowel disease (IBD).

METHODS: Using medical literature databases, searches were performed for randomised controlled trials (RCTs) and non-randomised comparative studies from 1990 to 2022. Each study was screened by two independent reviewers from the SAGES Guidelines Committee for eligibility. Data were extracted while assessing the risk of bias using the Cochrane Risk of Bias 2.0 Tool and the Newcastle-Ottawa Scale for RCTs and cohort studies, respectively. A meta-analysis was performed using random effects.

RESULTS: Routine use of IOC was associated with a significantly decreased rate of common bile duct injury and a trend towards decreased intraoperative complications, perioperative complications, and mortality. The rates of reoperation, postoperative pancreatitis, cholangitis, and choledocholithiasis were low in the routine use of the IOC group, but no non-routine use studies evaluated these outcomes. After sleeve gastrectomy, GERD-specific quality of life was significantly higher in the surgically treated group compared to the medically treated group. Bypass-type operations had worse outcomes of IBD sequelae than sleeve gastrectomy, including pain, patient perception, and fistula formation. Sleeve patients had lower mortality and fewer short- and long-term complications.

CONCLUSIONS: Low-quality data limited the conclusions that were drawn; however, trends were observed favouring the routine use of IOC during cholecystectomy for patients with bypass-type anatomy, surgical treatment of GERD post-sleeve gastrectomy, and sleeve gastrectomy in IBD patients. Future research proposals are suggested to further answer the questions posed.

READING 10. BARIATRIC SURGERY FOR MORBID OBESITY IN ADULTS

Park KB,[1,2] Jun KH.[1,2] Bariatric surgery for treatment of morbid obesity in adults. Korean J Intern Med. 2025 Jan;40(1):24–39. PMID: 39778524.

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ABSTRACT
Morbid obesity requires active intervention, with treatment options including lifestyle modification, pharmacotherapy, and surgery. As the prevalence of obesity continues to rise in Korea, it is crucial for specialists and general practitioners to have a comprehensive understanding of obesity and its management. Bariatric surgery is the most effective treatment modality for obesity, leading to significant weight loss and metabolic benefits. It involves surgical alterations of normal anatomical structures to improve overall health. Therefore, selecting the appropriate procedure based on the individual characteristics of patients is crucial.

This review highlights the two most commonly performed bariatric procedures worldwide, including in Korea: sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB). Furthermore, it provides a comprehensive overview of the surgical techniques involved in SG and RYGB, addresses potential complications, and presents findings from key studies on the weight loss and metabolic outcomes of these surgeries. Additionally, to support clinical application, the review provides outcome data for these procedures based on studies conducted in Korean populations.

In addition to SG and RYGB, this review briefly introduces other surgical and endoscopic options, as well as pharmacological treatments that are currently available or might become viable options in the near future.

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  Where there are more than six authors, the first three should be named and then followed by "et al".

  Example:
  Moussavi S, Chatterji S, Verdes E, Tandon A, Patel V, Ustun B. Depression, chronic diseases, and decrements in health: results from the World Health Surveys. Lancet. 2007 Sep;370(9590):851–8. https://doi.org/10.1016/S0140-6736(07)61415-9.

Tables
Tables should be submitted on a separate page. Label them in Roman-numeric sequence [I, II, III, etc] and ensure they are clear and with explanatory legends as required. Give each column a short or abbreviated heading. Place Table explanations in the footnotes, not in the heading. Explain in footnotes all non-standard abbreviations that are used in each Table.

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Concluding Paragraph
Summarise your main findings and its clinical implication, preferably in a single paragraph and no more than 3-4 sentences. Link the conclusions with the goals of the study but avoid unqualified statements and conclusions not adequately supported by the data. In particular, distinguish between clinical and statistical significance, and avoid making statements on economic benefits and costs unless the manuscript includes the appropriate economic data and analyses. Avoid claiming priority or alluding to work that has not been completed. State new hypotheses when warranted, but label them clearly.

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Include a minimum of three (3) Learning Points as a take-home message for readers.

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Title
The title should define the key focus of the case study.

Case Presentation
The author(s) will provide a pertinent summary of the medical and/or psychosocial issue pertaining to the health or disease management of the case. It should cover the situation and relevant background of the case. Author(s) should conceal the identity of the subject and/or related or accompanying personnel; abbreviation should be used instead, if necessary.

Diagnoses/Problems identified
The assessment of the diagnoses/problems identified will constitute a problem list and will serve as a focus for the management of the case. If the case was a diagnostic dilemma, the author(s) should showcase the diagnostic challenges and their work in narrowing to the correct diagnosis and/or differential diagnoses.

Management of the case
This section covers the approach to the management of the case by the author(s).

Literature review on latest evidence/guidelines (related to diagnosis and/or management)

The author(s) should provide a literature review of current evidence/guidelines, if any, of the basis of the case's diagnosis/management, or to highlight the gaps of knowledge if such evidence is lacking.

The author(s) will provide a concise summary of the lessons learnt from this case study.

Clinical Practice pointers (up to three (3))
The author(s) will suggest ways to apply the new knowledge in clinical practice or to highlight the limitations of its applications, if any.

RECOMMENDED FORMAT FOR PRISM (Patients' Revelations as Insightful Studies of their Management) SECTION

Authors planning to submit their case studies to the PRISM section should structure their article according to these headings:

Title
The title should be framed into a question to define the key focus of the case study.

Patient's revelation: What happened?
The author(s) will provide a concise description of the setting in which the subject raised his/her medical or psychosocial issue pertaining to their health or disease management. It should cover the background, encounter, and interaction of patient with the healthcare professional (doctor, nurse, or allied healthcare professional).

Author(s) should conceal the identity of the subject and/or related or accompanying personnel: abbreviation should be used instead, if necessary.

Gaining insight: What are the issues?
The issue(s) raised by the patient should be framed into question(s). The question(s) will constitute a problem list and will serve as a focus for the management of this subject.

Study the management: How do we apply in our clinical practice?
This section covers the approach to the management of the subject by the author(s). The author(s) should provide a literature review of current evidence, if any, of the basis of the subject's management, or to highlight the gaps of knowledge if such evidence is lacking. The author(s) will suggest ways to apply the new knowledge in clinical practice or to highlight the limitations of its applications, if any.

Conclusion
The author(s) will provide a concise summary of the lessons learnt from this case study.

The article submitted to the PRISM section should be written by no more than three authors. Each article should not exceed 2,000 words. Photographs or charts may be included but should conform to the specific instructions for any other articles submitted to *The Singapore Family Physician*.

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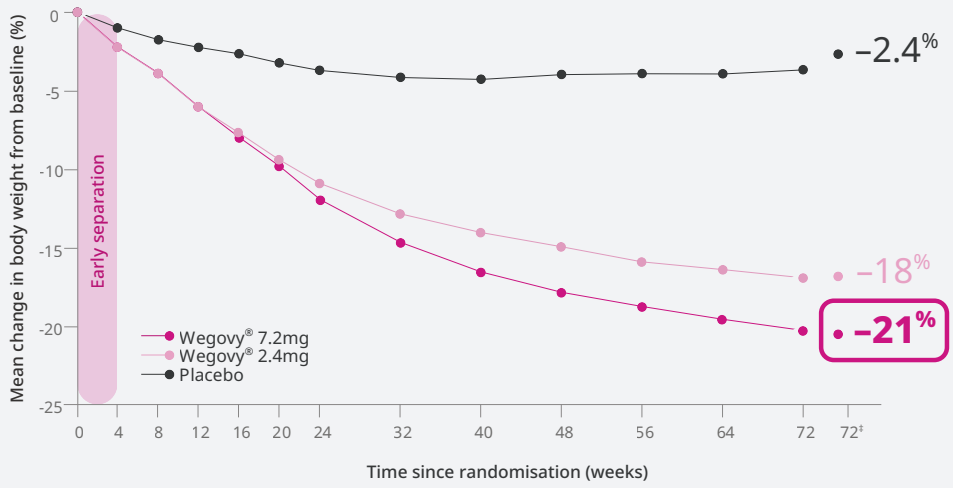
The **STEP-UP** trial: In adults with obesity, along with diet and exercise,

Wegovy® delivered weight loss as early as 4 weeks[1]

Body weight reduction during the on-treatment observation period[1*†]

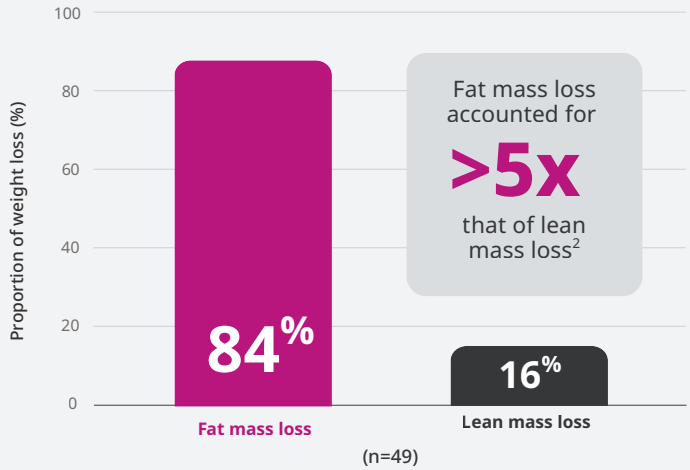


≥25%
WEIGHT LOSS
achieved by 1 in 3 patients[1]
~21% mean weight loss[1*]

Wegovy® delivered quality weight loss[2]

The STEP-UP trial investigated the effect of Wegovy® on body composition and proximal muscle strength[2]

Fraction of weight loss from adipose and lean tissue from Th9 to the knee[2#^]



84%
FAT MASS LOSS
from total weight loss[2]

Th9, the ninth thoracic vertebra

* Estimated treatment difference, -18.2 percentage points; (95% CI: -19.9 to -16.6), p<0.0001.

† From baseline to week 72. Data presented here from the STEP UP trial are based on the trial product estimand, which describes the assessed treatment effect as though all participants had remained on treatment for the duration of the trial and had not initiated other obesity therapies, whereas treatment policy estimand describes the assessed treatment effect among all randomised participants, regardless of trial product discontinuation, dose reduction, or initiation of any other obesity therapies.

The change in adipose tissue mass as a proportion of mass reduction was calculated as: {[(absolute volume change [L] in adipose tissue x 0.92)/[(absolute volume change [L] in adipose tissue x 0.92) + (absolute volume change [L] in lean tissue x 1.1)]} x 100 = {(11.0 x 0.92)/[(11.0 x 0.92) + (1.7 x 1.1)]} x 100 = 84.4%.

^ Results shown are from a pooled analysis including Wegovy® 2.4 mg and 7.2 mg doses.

References: 1. Wharton S, et al. Lancet Diabetes Endocrinol. 2025;13(11):949-963. 2. Hjelmesæth J, et al. Presented at the 61st EASD Annual Meeting; September 15-19, 2025; Vienna, Austria. Abstract 147, Diabetologia (2025) 68 (Suppl 1):S81.

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≥25%
WEIGHT LOSS
achieved by 1 in 3 patients[1]

~21% mean weight loss[1*]

84%
FAT MASS LOSS
from total weight loss[2]

74%
REDUCTION
in the constant thoughts about food[3]

4 WEEKS ONSET OF WEIGHT LOSS[1]

Now Approved for MASH (with F2-F3 fibrosis)[4,5^]


AI generated patient portrayal

MASH, metabolic dysfunction-associated steatohepatitis.

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*From baseline to week 72. Data presented here from the STEP UP trial are based on the trial product estimand, which describes the assessed treatment effect as though all participants had remained on treatment for the duration of the trial and had not initiated other obesity therapies, whereas treatment policy estimand describes the assessed treatment effect among all randomised participants, regardless of trial product discontinuation, dose reduction, or initiation of any other obesity therapies.

References: 1. Wharton S, et al. Lancet Diabetes Endocrinol. 2025;13(11):949-963. **2.** Hjelmesæth J, et al. Presented at the 61st EASD Annual Meeting; September 15-19, 2025; Vienna, Austria. Abstract 147, Diabetologia (2025) 68 (Suppl 1):S81. **3.** Arnaut T, et al. Impact of food noise after initiating semaglutide treatment: results from a US survey (INFORM). Presented at: The 61st European Association for the Study of Diabetes (EASD) Annual Meeting; September 15-19, 2025; Vienna, Austria. **4.** Wegovy® Singapore Prescribing Information. **5.** Sanyal AJ, et al. Phase 3 trial of semaglutide in MASH. N Engl J Med. 2025;392(17)

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