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*Participants in the STEP study had a mean body weight of 105.4 kg and a BMI of 37.8.

+Participants in SELECT study had a mean body weight of 96.5 kg and a BMI of 33.3.

Abbreviations: MACE, Major Adverse Cardiovascular Events; **CV,** Cardiovascular; **CVD,** Cardiovascular Disease

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Basic Obesity Management

Dr Chiang Shu Hui Grace

SFP2025; 51(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 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 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, Drs Lee Phong Ching, Sarah Tan, and Tham Kwang Wei provide an overview of sarcopenic obesity and its management.

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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Distance Learning Course on "Basic Obesity Management 5"

- Unit 1: Understanding Obesity: How and Why?
- Unit 2: A Practical Approach to the Patient with Obesity
- Unit 3: Dietary Interventions for Weight Loss
- Unit 4: Understanding Matters of the Mind in Obesity
- Unit 5: Use of Pharmacotherapy in Obesity Management
- Unit 6: Intensifying Treatment: Bariatric Surgery
- Unit 7: Approach to Childhood and Adolescent Obesity
- Unit 8: Sarcopenic Obesity: What, When, and How

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 last 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]

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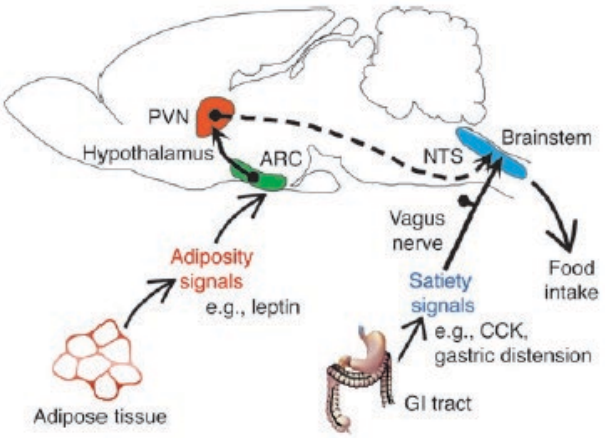
Obesity has often been viewed as a result of a simple lack of self-discipline and willpower to "eat less and move more" or plain laziness on the part of the person with obesity.[4] This oversimplified perception results from the lack of understanding that beyond the passive accumulation of excess energy 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 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]

Figure 1. Model for regulation of the hindbrain response



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]

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 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]

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

OBESITY AS A DISEASE: HEALTH CONSEQUENCES

Obesity is not benign. The failure of adipose tissues to continually expand leads 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 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

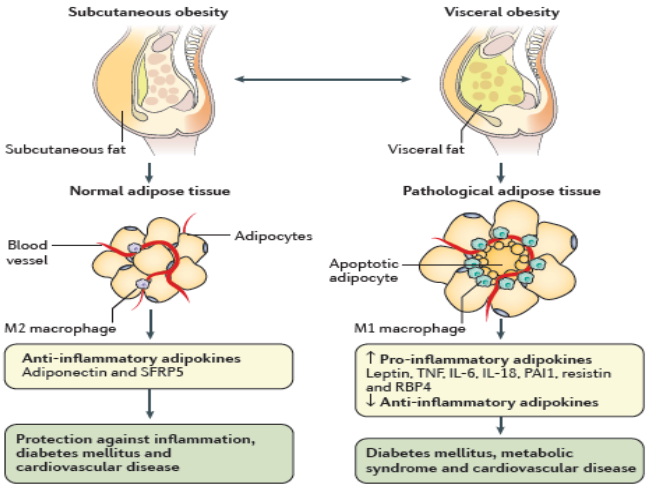


Figure 4. Organisations that have declared obesity as a disease

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 percent), 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 percent 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 may 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 now recognised as a disease and has been described as a complex, chronic medical condition with a major negative impact on human health.[27] Many associations and organisations, including the World Health Organisation (WHO), have now declared obesity as a disease, and this is an important first step to 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 now 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, 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.**

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

Over the past four decades, the global prevalence of obesity has risen markedly from 7 percent in 1980 to 12.5 percent in 2015,[1] affecting all regions including Singapore.[1,2] Obesity is not benign and it has been linked to many other health conditions, such as type 2 diabetes, cardiovascular disease, mood disorders, and certain cancers, resulting in reduced quality of life, higher healthcare costs, and increased mortality.[3-6] Obesity has clearly become a major global and regional health problem.

However, according to international surveys and interviews, people with obesity might not perceive their weight to be a significant problem.[7] Even if they do, it might take several years of struggling with excess weight before they finally consult a healthcare professional.[8] 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.[9] 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.[9] However, observational and self-report evidence suggests that less than half of patients with obesity are advised by their physicians to lose weight.[8,10] Primary care physicians often cite lack of motivation by the patient as the main reason for not discussing weight during consultations.[7,8] Other barriers described include limited understanding of obesity care, concern about negative consequences of raising a sensitive topic, and limited time and resources.[10]

On the other hand, studies on patients with obesity suggest that patients 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.[8,11] Only 3 percent were offended by such a conversation, according to an online survey conducted in 11 countries involving 14,502 participants.[8] 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.[8] 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 behaviour change, which was initially developed to support smoking cessation (refer to **Figure 1**).[12] 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.[13] 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.[14] 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.[15] 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 behaviour change at a pace that is suited to the patient's situation.[13]

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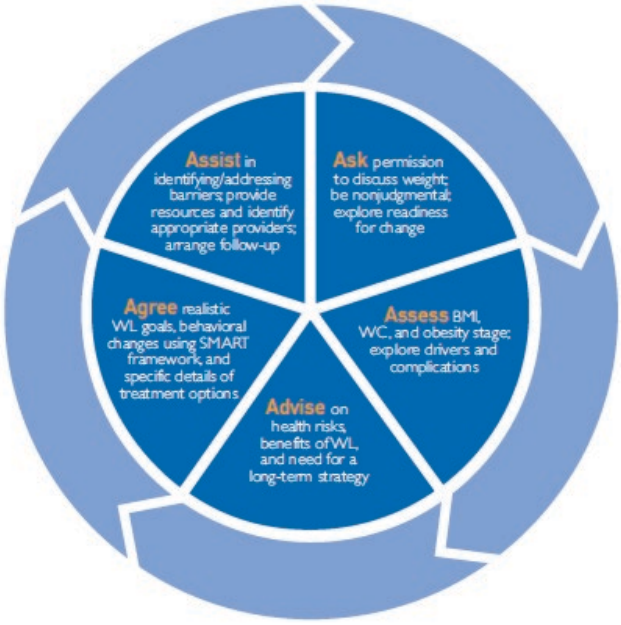
Figure 1: The 5 As framework for obesity counselling



BMI = body mass index
SMART = specific, measurable, achievable, relevant, time-based
WC = waist circumference
WL = weight loss[12]

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.[16] When a patient presents with a problem that could be attributed to weight, primary care physicians usually have no issues initiating the conversation.[7] 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.[17,18]

Regardless, there are two main points to highlight regarding a successful conversation about obesity.[17] 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.[17,18] 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.[17] 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.[12,17]

Second, in order to have a motivating conversation so that behaviour change can be elicited, the physician should employ motivational interviewing (MI).[19] MI is a patient-centred, goal-directed counselling technique aimed at increasing intrinsic motivation for change by exploring and resolving ambivalence to change.[19] 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).[19] In a meta-analysis of 11 Randomised Control Trials involving 1,448 participants, MI significantly enhanced weight loss in overweight and obese patients.[20] 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**).[21,22] 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[21,22]

	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 may 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 to support 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.[23] The causes of obesity are summarised in **Table 2**.[24]

Table 2: Causes of obesity[24]

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 Psychological Depression[b] Eating disorders Drug-induced Anti-depressants Anti-psychotics Anticonvulsants Sulphonylureas Insulin Thiazolidinediones Steroids Oral contraceptives
[a]Controversial whether hypothyroidism causes obesity or exacerbates obesity [b]Depression associated with overeating or binging

Table 3: STOP-BANG Questionnaire[29]

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 they can start making changes to improve their 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.[25,26] 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.

Disordered sleep patterns are associated with weight gain. Shift workers who have disrupted circadian rhythms have an increased risk of weight gain.[27] Disrupted sleep or poor sleep quality may also be due to obstructive sleep apnoea (OSA), which has a high prevalence in patients with obesity.[28] A useful tool in assessing for OSA is the STOP-BANG questionnaire to screen for OSA (refer to **Table 3**).[29]

Past Medical History

Many medical conditions are caused or contributed to by obesity. These include type 2 diabetes mellitus (T2DM), hypertension, dyslipidaemia, non-alcoholic fatty liver disease (NAFLD), polycystic 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
Non-alcoholic fatty liver disease
Polycystic ovarian syndrome
Coronary artery disease
Cancer—breast, endometrial, 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.[30]

Another useful clinical measurement of obesity is waist circumference, widely recognised as a measure of central adiposity. An increased waist circumference is associated with increased visceral adiposity and increased risk of cardiometabolic disease.[31,32] 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. 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.[33] However, waist circumference is preferred because it is a better marker of abdominal fat content and is easier to measure and interpret.[34] Neck circumference has also been associated with visceral adiposity and is used in the STOP-BANG score for OSA.[29,35]

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, especially cardiovascular- and metabolic-related complications. Acanthosis nigricans, a dark velvety discolouration of the skin on the neck and armpits, may be seen 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 as obesity management is now more "complications-centric" instead of being based on BMI classification alone. **Table 6** suggests investigations to consider in patients with obesity.

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)[30]**
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)[33]**	**WC threshold in Asians (cm)[40]**
Female	>88	>80
Male	>108	>90

Table 6. Investigations in a patient with obesity

<u>To investigate underlying causes</u> Thyroid function test 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 hypothyroidism Exclude Cushing's syndrome
<u>Screening for complications of obesity</u> *Clinical* Blood pressure STOP-BANG score *Laboratory investigations* Fasting glucose and HbA1C Lipid panel Renal panel Liver panel Others (e.g., Mammogram, pap smear, referrals as required)	 Screen for hypertension Screen for OSA Screen for DM, IFG, and IGT Screen for dyslipidaemia Screen for abnormal renal function Screen for NAFLD Screen for cancers
OSA: Obstructive sleep apnoea DM: Diabetes mellitus IFG: Impaired fasting glucose IGT: Impaired glucose tolerance NAFLD: Non-alcoholic fatty liver disease	

In the assessment of obesity, there are new 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).[36] 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.[36,37] The presence of obesity-related comorbidities and a higher EOSS stage may suggest more urgent action required to address obesity. For example, a patient with obesity with T2DM, NAFLD, 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 7. Edmonton obesity staging scale (EOSS)[36]

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, PCOS) 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.[38] Patients who share their perspectives achieve better outcomes.[22,39] 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.[32] 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 more 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 psychosocial behavioural goal. Examples include intake of fruits and vegetables, avoidance of 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 goal 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 prescription of medications for weight management and/or adjustment of medications that contribute to weight gain by adjusting doses or using weight-friendly alternatives, if possible. It is also important to assist the patient in further management by providing educational resources, arranging follow-ups, or referral to other community resources or healthcare providers.

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 and respectful manner with empathy.**
- **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.**

Unit No. 3

DIETARY INTERVENTIONS FOR WEIGHT LOSS

Ms Izabela Kerner

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 to compare 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 might 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, fruits 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.**

Unit No. 4

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/m^2) and 650 million were obese (BMI ≥30 kg/m^2; 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 to 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 Pumpa6 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 the 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]

BARRIERS TO LIFESTYLE CHANGES

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 he made changes and the 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.**

Unit No. 5

USE OF PHARMACOTHERAPY IN OBESITY MANAGEMENT

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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

TThe 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 1 in 4 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 render 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 percent) 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 percent) 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 may 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 6.1–8.6 percent[16,18] at one year, which can be maintained at six percent over ten 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/m^2 or BMI ≥27 kg/m^2 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:

1. 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).
2. Sequential: When initial lifestyle interventions implemented result in inadequate weight loss to achieve improvement or resolution of ORC, or greater weight loss is desired to meet patient's goals.
3. Weight regain after lifestyle interventions.
4. 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 percent) and 3.6 kg (3.3 percent) 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 percent 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

AAn 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 percent 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 percent over placebo) in one year.[18,38] Such adjunctive treatments are feasible in the primary care setting (total weight loss of 7.5 percent 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 HbA1c.[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 diarrhea.[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

SSemaglutide 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 T2DM28 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 percent) 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 percent vs 34.1 percent) and improvement in fibrosis (37 percent vs 22.5 percent).[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 ≥27kg/m^2, 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 HbA1c (2.2 percent) compared to semaglutide 2.4 mg (1.9 percent) and cagrilintide (0.9 percent) alone. Mean weight loss at week 32 was also significantly greater with CagriSema (15.6 percent) compared to semaglutide (5.1 percent) and cagrilintide (8.1 percent),[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 HbA1c 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 may serve to be 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 percent) 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, many 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 (HCl) 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.**

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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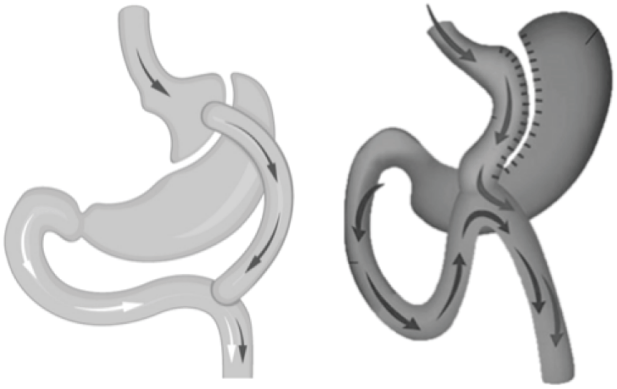
TYPES OF PROCEDURES

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 (refer to **Figure 1**).[3,4] 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 as 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)



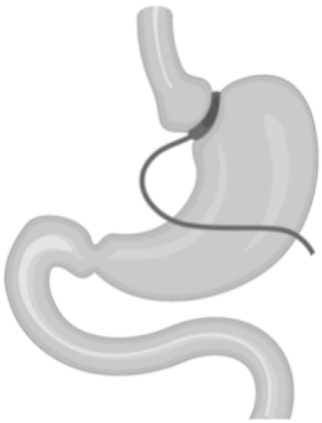
Gastric Band

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.

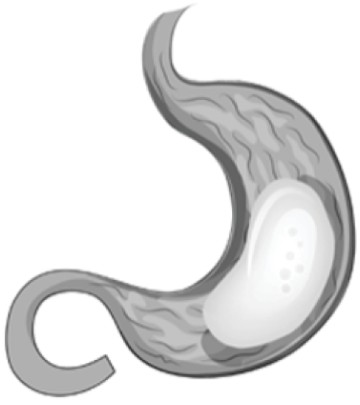
Gastric sleeve numbers have overtaken all other procedures as the most popular bariatric surgery worldwide.[9] The main reason for this lies with the simplicity and efficacy of the sleeve: reduction of meal portions without requiring any foreign body placement or diversion of food passage. The main problem 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

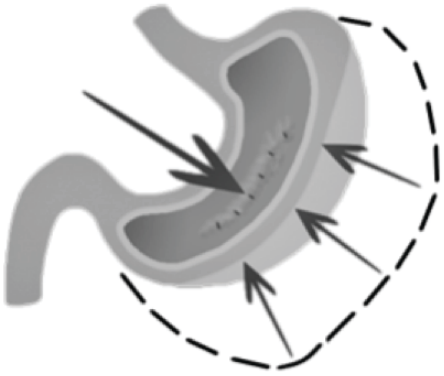
The first is the Elipse pill balloon. The balloon is tightly packed into a capsule that can be swallowed in the clinic. No anaesthesia or endoscopy is required. An x-ray is done to check that the capsule has entered the stomach prior to inflation. The entire placement can be completed in 20 minutes with 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 require 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 2025, 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.**

Unit No. 7

APPROACH TO CHILDHOOD AND ADOLESCENT OBESITY

Dr Elaine Chew Chu Shan, Dr Chin Xinyi

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]

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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 may 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**).

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
		Blount disease
	Bowing of tibia	Pes planus
	Foot pain	
Reproductive system	Tanner staging	Altered onset of puberty
		To evaluate height potential
	Breast development (in males)	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 more are 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.

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 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, active 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 and building in breaks is important to improve concentration.
Sleep
• Regular, continuous sleep of at least 9 hours (for 7–13 years old), at least 8 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 2 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.

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 licensed 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/m^2 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 had 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 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
Setmelanotide	MC4R agonist	POMC, PCSK1, or LEPR deficiency in >6 years	SC 1–2 mg OD	GI, hyperpigmentation
Metreleptin	Recombinant analogue of leptin	congenital leptin deficiency and generalised lipodystrophy	SC Max 0.13 mg/kg OD	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

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

Bariatric Surgery

Current international guidelines suggest that bariatric surgery should be considered in post-pubertal adolescents with BMI >40kg/m^2 or a BMI >35 kg/m^2 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/m^2 [29] and weight loss being maintained at a 5-year follow-up.[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

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.**

Unit No. 8

SARCOPENIC OBESITY: WHAT, WHEN, AND HOW

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ABSTRACT

While obesity has garnered significant attention as one of the most common chronic diseases, the awareness and scientific understanding around sarcopenic obesity (SO) remains lacking. SO is characterised by the co-existence of sarcopenia (loss of skeletal muscle mass and function/strength) and obesity (excess or abnormally distributed body adiposity, which impairs health), which can impact people of all ages. SO warrants attention as it serves as an independent risk factor for poorer health and outcomes including increased frailty and mortality, and more adverse metabolic outcomes. With the increasing use of obesity management interventions that result in significant weight loss, SO can either develop in susceptible individuals or worsen in those with pre-existing SO. Management of SO poses unique challenges as it mandates a balance of adequate weight loss with the preservation of skeletal muscle mass and strength. This article provides an overview of SO and its management.

Keywords: Sarcopenia, obesity, prevention, diagnosis, management

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INTRODUCTION

Sarcopenic obesity (SO) is the co-existence of sarcopenia (loss of skeletal muscle mass (SMM) and physical function or strength) and obesity (excess or abnormally distributed body adiposity, which impairs health).

With the advent of a rapidly ageing population, increasing life expectancy, and the rising prevalence of obesity in Singapore, SO warrants urgent attention as it is associated with poorer health outcomes for frailty and geriatric syndromes, for comorbidities of various chronic conditions and increased mortality, compared to either sarcopenia or obesity alone.[1-3] In Singapore, the prevalence of sarcopenia ranged from 13.6 percent to 25 percent among community-dwelling older adults.[4] The National Population Health Survey 2022 (Singapore) revealed that abdominal (central) obesity increased with age, particularly in women, with the highest prevalence (57.4 percent) among women aged 60–74 years old compared to women 18–29 years old (23.1 percent).[5] In the Geri-LABS study, which studied Singapore adults 50 years and older, the prevalence of SO was 6.2 percent in men and 12.4 percent in women.[6]

SO can develop under various clinical scenarios. Sarcopenia develops typically as a consequence of ageing, which sees a paralleled change in body composition favouring relative or absolute body fat accrual, setting the metabolic milieu for the development of SO. Furthermore, adipose tissue-associated inflammation of muscle can exacerbate the process of sarcopenia. On the other hand, sarcopenia can develop in people with obesity due to changes in skeletal muscle metabolism, a very sedentary lifestyle, inadequate protein intake and malnourishment (e.g., from chronic dieting or after metabolic bariatric surgery), or after an acute illness that induces skeletal muscle loss. While SO is more common in people of older age, it is also highly prevalent in people with obesity across their lifespan.

In addition, with the availability and use of obesity treatments that can result in significant and rapid weight loss as seen with the newer incretin-based therapies and metabolic bariatric surgery, SO can worsen in those with undiagnosed and untreated SO or develop in at-risk individuals.

Primary care physicians play a pivotal role in the early detection and management of SO to reduce the risk of health sequelae from untreated SO. This article aims to provide an overview of the screening, diagnosis, mechanisms, and management of SO.

PREVALENCE OF SARCOPENIC OBESITY

The prevalence of SO is variable due to the inconsistencies in definitions used and populations studied, highlighting the challenges faced in the diagnostic criteria used for SO. In a study of 535 community-dwelling Singaporean adults aged 21–90 years (the Yishun study), the overall prevalence of SO ranged from 0.4 percent to 7.6 percent, when BMI and waist circumference (WC) were respectively used to define obesity.[7] Among Singaporean adults aged 50 years and above (Geri-LABS study), the prevalence of SO was lowest for BMI (0.5 percent) compared to fat mass percentage (10.0 percent) and WC (10.5 percent), with WC correlating with worse functional outcomes, underscoring the need to prioritise assessment of central obesity over BMI for the diagnosis of obesity in Singaporeans.[6]

SO is highly prevalent among people with obesity and type 2 diabetes (T2D). Among Singaporean adults with a

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mean age of 61 years and T2D, the prevalence of SO was nearly 20 percent.[8] In a cohort of 599 people with obesity (mean age 51 years, mean BMI 43.1 kg/m^2) presenting for obesity treatment, SO was present in nearly 50 percent. The prevalence increased with age, with 31 percent of those aged 18–40 years old having SO compared to ~60 percent in those 60 years and above, with SO being twice as prevalent among women (~51 percent) than in men, and in those with lower BMI.[9] These variable prevalence rates inform us that we need to be vigilant with case-finding not just among people of older age but also among people with co-existing chronic diseases that can predispose to sarcopenia (e.g., T2D) and obesity.

DIAGNOSING SARCOPENIC OBESITY

The diagnosis of SO begins with high vigilance and an approach of case-finding (rather than universal screening).[4,10,11] Locally, the Singapore Clinical Practice Guidelines for Sarcopenia 2022 recommends case-finding for sarcopenia in adults aged 65 years and above.[4] In addition, screening for SO is recommended for all individuals with risk factors or with signs or symptoms of SO regardless of age (see **Table 1**).[4,11]

Commonly encountered clinical scenarios include someone with a very high BMI who loses a disproportionately higher amount of SMM during weight loss (e.g., from physical inactivity and inadequate dietary protein intake) or someone with a pre-disposing chronic disease or frailty who gains weight mainly in ectopic sites and develops central obesity.

There is a lack of a universally established SO definition and diagnostic criteria especially among Asians. Regardless, the diagnosis of SO remains as the establishment of a loss of SMM coupled with reduction in muscle strength or function in people with obesity (including those with central obesity), based on ethnic/population-specific established criteria.[12]

For obesity, screening and diagnosis involve establishing the presence of excess adiposity using ethnic-specific BMI cutoffs (e.g., Asians in Singapore, BMI ≥27.5 kg/m^2) and/or abnormally distributed adiposity using ethnic and gender-specific anthropometric measurements such as waist circumference (e.g., Asians in Singapore: males ≥90cm; females ≥80cm) or waist-hip ratio (e.g., Asians in Singapore: males ≥1.0; females ≥0.85). The use of BMI alone can often underdiagnose SO particularly in older adults and in Asians. Hence, adding a surrogate assessment of excess or abnormal distribution of adiposity is highly recommended.[6,7] In addition to anthropometrics, measurement of percentage body fat has been advocated by some experts for diagnosis of obesity in SO although there is no standardisation at present.[13] The WHO uses body fat cutoffs of >25 percent in males and >35 percent in females while recent studies use >30 percent and >40 percent in males and females respectively to define excess total body fat.[6,14]

The presence of any of the following clinical conditions that predispose to sarcopenia (e.g., functional decline or limitation; recurrent falls; malnutrition or chronic conditions such as chronic heart, lung, liver, and kidney disease) serves as a positive screening and will warrant confirmation of sarcopenia with further assessments.

In the absence of clinical signs or symptoms of sarcopenia, the Singapore Clinical Practice Guidelines for Sarcopenia (2022) and the Asian Workgroup for Sarcopenia (AWGS) 2019 Consensus call for the adoption of a case-finding approach in adults ≥60 years old with the measurement of the calf circumference (in the standing position) or the use of questionnaires (e.g., SARC-F or SARC-CaIF).10 If any of the following parameters are met—calf circumference (M: <34 cm, F: <33 cm) or SARC-F ≥4 or SARC-CalF ≥11—further assessments must be carried out to confirm the diagnosis of sarcopenia. The SARC-CaIF adds 10 points to the SARC-F scoring for calf circumference below cutoffs.

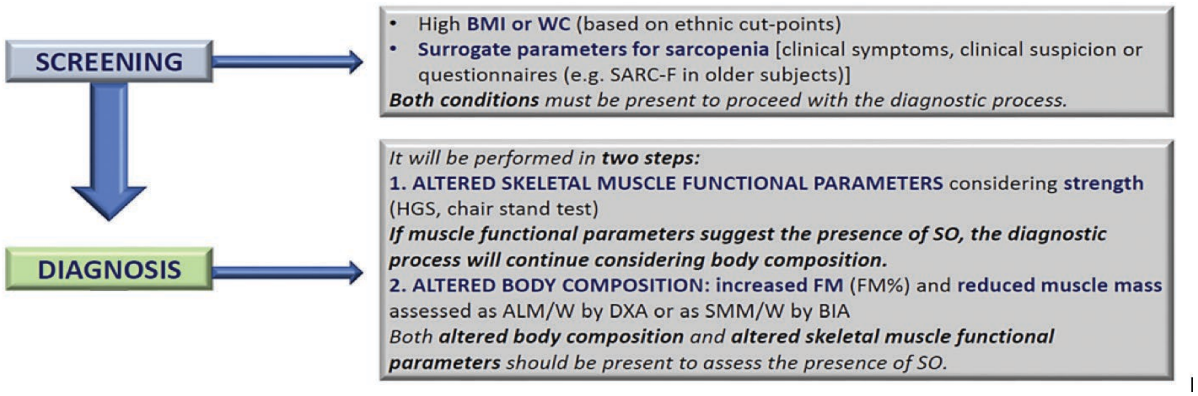
Table 1. Clinical Symptoms or Risk Factors for Sarcopenic Obesity

Age >70 years
Chronic disease diagnosis (e.g., inflammatory diseases and organ failure or chronic disease) including but not limited to:
Chronic heart failure
Chronic kidney disease (particularly renal replacement therapy)
Chronic bowel failure or dysfunction
Chronic liver disease (particularly NASH and liver cirrhosis)
Chronic respiratory disease
Chronic neurologic and neurodegenerative diseases
Chronic cognitive impairment
Depression
Organ transplantation
Endocrine diseases (e.g., metabolic syndrome, diabetes mellitus, hypercortisolism, hypogonadism and corticoid treatment)
Osteoarthritis
Cancer (especially but not limited to chemotherapy of breast or prostate cancer)
Recent acute disease/nutritional events:
Recent hospitalization (particularly but not limited to COVID-19, ICU stay, surgery)
Recent major surgery or trauma with/without complications
Recent sustained immobilization or reduced mobility (e.g., trauma, fracture, orthopaedic disease)
Recent history of reduced food intake (e.g., <50% for >2 weeks)
Recent weight loss (including diet-induced voluntary weight loss and weight cycling syndrome)
Recent rapid increase in weight
Long-standing restrictive diets and bariatric surgery
History – complaint of:
Repeated falls
Weakness, exhaustion
Fatigability
Perceived progressive movement limitations

Adapted from: Donini LM, Busetto L, Bischoff SC, et al. Definition and Diagnostic Criteria for Sarcopenic Obesity: ESPEN and EASO Consensus Statement. Obes Facts. 2022;15(3):321-335. doi: 10.1159/000521241. Epub 2022 Feb 23. PMID: 35196654; PMCID: PMC9210010.[11]

Following a positive screening for sarcopenia, the diagnosis of sarcopenia requires the confirmation of both (1) reduced skeletal muscle function or strength and (2) reduced SMM, in a 2-stage approach (refer to **Figure 1**).

Figure 1. Diagnostic procedure for the assessment of sarcopenic obesity



Legend:
ALM/W: appendicular lean mass adjusted to body weight; bioelectrical impedance analysis
BMI: body mass index
DXA: dual X-ray absorptiometry
FM: fat mass
HGS: handgrip strength
SMM/W: total skeletal muscle mass adjusted by weight
SO: sarcopenic obesity
WC: waist circumference
SARC-F: strength, assistance with walking, rising from a chair, climbing stairs and falls.

Adapted from: Donini LM, Busetto L, Bischoff SC, et al. Definition and Diagnostic Criteria for Sarcopenic Obesity: ESPEN and EASO Consensus Statement. Obes Facts. 2022;15(3):321-335. doi: 10.1159/000521241. Epub 2022 Feb 23. PMID: 35196654; PMCID: PMC9210010.[11]

For Asians, sarcopenia should be diagnosed using the AWGS 2019 criteria.[10] Poor physical function (performance) is defined as 6-metre walk (gait speed) <1.0 m/s or 5-time chair stand test ≥12 seconds or Short Physical Performance Battery (SPPB) ≤9 while reduced muscle strength is defined as handgrip strength (HGS) of <28 kg and <18 kg for men and women respectively (refer to **Table 2**). However, the validation of these cutoffs is not available for younger people with obesity.

The assessment of muscle mass can be performed using dual energy X-ray absorptiometry (DEXA) for the analysis of appendicular skeletal mass (ASM) corrected for height (ASM/height^2). A low ASM is defined as <7.0 and <5.4 kg/m^2 in men and women respectively, as measured by DXA (refer to **Table 2**).[10] While DEXA is the recommended modality, it is not as accessible in the primary care setting and not every diagnostic facility has the capability to perform a body composition analysis with a DEXA scan. DEXA incurs higher cost (compared to bioelectrical impedance analysis (BIA)) and X-ray exposure, which may impose a barrier for the uptake of assessment. Recognising these challenges, some experts recommend the use of BIA, which is more readily available in most clinical settings. When performed under controlled conditions (e.g., fasted state with light clothing) and in subjects without fluid overload (or issues with lower extremity swelling), BIA can be a practical and reproducible assessment for monitoring of treatment.

Table 2. Cutoffs for the Screening and Diagnosis of Sarcopenia in Asians[10]

Screening		
Clinical Situation	**Measurements/Assessment**	**Positive Cutoff(s)**
Presence of any clinical condition or, signs or symptoms of sarcopenia	Functional decline or limitation; unintentional weight loss; depressive mood; cognitive impairment; repeated falls; malnutrition OR Chronic conditions (as listed in **Table 1**)	Presence of any
Without clinical conditions, signs or symptoms of sarcopenia	Calf circumference **or**	M <34 cm, F<33 cm
	SARC-F **or**	≥4
	SARC-CalF	≥11
Diagnosis		
Muscle strength*	Handgrip strength	M <28 kg, F <18 kg
Physical performance (function)*	6-metre walk **or**	<1.0 m/s
	5-time chair stand test **or**	≥12 s
	Short Physical Performance Battery	≤9
Appendicular skeletal muscle mass (ASM)*#	Dual-energy X-ray absorptiometry **or**	M <7.0 kg/m^2 F <5.4 kg/m^2
	Bioelectrical impedance analysis	M <7.0 kg/m^2 F <5.7 kg/m^2

M: males
F: females
*Assessments must be performed in sequence and all three must be present/positive for the diagnosis of sarcopenia
#Use of appendicular lean mass (ALM) or SMM corrected for weight (ALM/weight for DEXA or SMM/weight by BIA) has been alternatively recommended (cutoffs of 35.7% (M) and 30.7% (F) by BIA has been used for a predominantly East Asian population)[15]

In people with obesity in whom total body mass can be high, the use of ASM for SMM assessment can under-diagnose sarcopenia. Relatively reduced SMM in the presence of high fat and total body mass can have clinical implications including functional impact, even in the absence of absolute SMM loss. Hence, the use of appendicular lean mass (ALM) or SMM corrected for weight (ALM/weight for DEXA or SMM/weight by BIA) is recommended although this cutoff has not been determined in Asians.[2,11] Cutoffs of 1–2 standard deviations corresponding to 31.5–37.0 percent (males) and 22.1–27.6 percent (females) have been recommended,[16] while in the Korean SO Study, cutoffs of 35.7 percent (males) and 30.7 percent (females) by BIA have been used for a predominantly East Asian population.[15]

In the primary care setting, screening can be done using a case-finding approach with a BMI and waist circumference measurement followed by sarcopenia screening in those with signs or symptoms (refer to **Table 1**), a calf circumference measurement, or a SARC-F questionnaire in those with very high BMI or history of lower extremity oedema (in whom a calf circumference will not be accurate). Confirmation of sarcopenia can be easily carried out using a handheld dynamometer to measure handgrip strength repeated at least twice (for assessment of muscle strength) or the 5-time chair stand test or 6-metre walk test (for assessment of muscle function) followed by confirmation of reduced SMM (body composition) using a BIA scan, provided the patient does not have fluctuating body fluid issues.not have fluctuating body fluid issues.

PATHOGENESIS AND AETIOLOGY OF SARCOPENIC OBESITY

The aetiology and pathophysiology of SO is complex. Different factors that can trigger the development of SO include ageing (age-related changes in body composition), hormonal imbalances, dietary and lifestyle factors (e.g., malnutrition, physical activity and mental stress), presence of co-existing acute or chronic conditions, and systemic inflammation to myocellular mechanisms (e.g., oxidative stress).[2,11] Regardless of the metabolic scenario, the main mechanism of pathogenesis is the cross-talk between adipose tissue and skeletal muscle inflammation, and both weight gain and weight loss can contribute to the development of SO.

In people with obesity, the constellation of systemic inflammation, insulin resistance, and oxidative stress, systemically and within the muscles, can lead to a muscle-catabolic state, potentially promoting a "resistance to anabolism" in the skeletal muscle (i.e., reduced muscle protein synthesis in response to nutrients is blunted).[2,17] In response to weight gain, this leads to a preferential increase in fat mass with proportionately reduced SMM accrual, favouring the development of SO.

Ectopic fat deposition in the muscle (myosteatosis), commonly seen in people with central obesity and insulin resistance, can lead to muscle oxidative stress with reduction in SMM and strength.[1] Reduced physical activity, a pertinent risk factor of weight gain and aetiology of obesity, can have further direct negative impact on muscle protein synthesis and muscle function. Obesity is associated with a myriad of other acute and chronic conditions/diseases (e.g., T2D, heart failure, chronic kidney disease, obstructive sleep apnoea, asthma, musculoskeletal conditions, prolonged hospital stays), which can perpetuate the process of SO either through further reduction in physical activity and/or through systemic inflammation and oxidative stress.[2,11,13,14]

Weight loss (interventions) inevitably results in SMM loss, especially with rapid or large amounts of weight loss such as seen with metabolic bariatric surgery, very-low calorie diets, and when protein intake and physical activity are lacking during weight loss.[11] Body weight cycling and inadequate nutrition, as often seen in chronic dieting in the process of management of obesity, can lead to development of or perpetuate pre-existing SO.[11,12] Hence, SO is rather prevalent even in younger individuals with obesity. Regardless, as people with obesity increase in age, the physiological changes in body composition favouring a loss in SMM and function with preferential fat mass gain and aggravate pre-existing SO, especially in the presence of risk factors (refer to **Table 1**).

Ageing is associated with physiologic body composition changes involving SMM loss (beginning in middle age) along with the development of myosteatosis and oxidative stress within the muscles, an increase in fat mass, and propensity for visceral adiposity, which favour the development of SO. These changes result from hormonal changes (e.g., reduced growth hormone, testosterone, and estrogen), low levels of physical activity, changes in energy metabolism, and diet (e.g., low protein, calcium, vitamin D intake). When older adults are inflicted with acute or chronic conditions, the above factors are augmented through increased inflammation, immobility, and changes in nutrition, which can lead to either weight gain or weight loss, triggering the onset or accelerating the development of SO.[2,16]

CLINICAL IMPLICATIONS OF SARCOPENIC OBESITY

The co-existence of obesity and sarcopenia synergistically creates a vicious twin-cycling of SMM loss and adipose tissue gain with further aggravation of SO if untreated. Multiple studies demonstrate an increased risk of metabolic disorders, geriatric syndromes, morbidity, and mortality related to SO.

The clinical consequences of SO include worsening metabolic dysfunction with development of metabolic syndrome, T2D, increased risk of geriatric syndromes (further reduction in physical activity with functional limitation and disability, increased frailty, cognitive impairment and dementia, risk of falls and fractures, depression), prolonged hospital length of stay, increased risk of all-cause mortality and mortality from various conditions such as cardiovascular disease, heart failure, post-hospitalisation, worse morbidity outcomes of various chronic diseases (e.g., stroke, lung diseases) and cancer outcomes (e.g., reduced overall disease-free survival), and development of various clinical conditions (e.g., hospitalisation, reduced quality of life, poor nutritional status).[2,3,8,15,16]

MANAGEMENT OF SARCOPENIC OBESITY

Regardless of the phenotypic presentation or primary aetiology of SO, the overall management of SO requires an approach of vigilance and case-finding in susceptible individuals with early detection and halting of the process, along with management strategies which address fat loss without further SMM loss (preservation) and, where possible, increase in SMM.

However, the treatment of SO poses unique challenges. By reducing fat mass, intramyocellular inflammation can be reduced. Yet, with weight loss, there is inevitable concurrent muscle loss. Hence, effective management strategies of SO will need to focus on combining exercise and nutritional interventions targeted at reducing fat (weight) while preserving or increasing muscle mass and function. This approach aims to create a negative energy balance, leading to adipose tissue reduction, improved adipose markers, and decreased inflammation, while also enhancing muscle mass.

Exercise Interventions

Exercise is essential for the prevention and management of SO.[18] Regular physical activity, particularly resistance and aerobic exercises, has been shown to improve muscle mass, strength, and overall physical function. Resistance training, in particular, is highly effective in enhancing muscle quality, function, strength, and flexibility in older adults, and it is recommended for at least two non-consecutive days per week, gradually increasing intensity and volume over time.[19,20]

Aerobic exercise, such as walking, cycling, or swimming, is also important as it improves muscle aerobic capacity and cardiovascular function, increases insulin sensitivity, promotes mitochondrial adaptation, increases capillary density of muscle tissue, and reduces oxidative stress and adipose tissue.[21,22] A combination of resistance and aerobic exercises is more effective in improving muscle mass and function and reducing fat than either form of exercise alone.[2] High-intensity interval training (HIIT), which alternates between short bursts of intense exercise and recovery periods, has been particularly noted for its ability to stimulate muscle protein synthesis and improve muscle function and insulin sensitivity.[23]

The Singapore Physical Activity Guidelines (SPAG), developed by the Health Promotion Board and SportSG advises adults to engage in 150 to 300 minutes of moderate-intensity aerobic activity weekly, which can be spread across any duration throughout the week.[24] SPAG encourages

incorporating various activities, such as strength training and light-intensity exercises, to break up sedentary periods and enhance overall physical and mental well-being. For older adults, staying active while enhancing muscular strength and functional balance is crucial, as it aids in managing frailty and common chronic conditions like diabetes and hypertension.

Nutritional Interventions

Nutritional strategies play a vital role in preventing and treating SO. While hypocaloric diets can effectively reduce fat mass, they may also lead to muscle loss by downregulating muscle protein synthesis and increasing proteolysis. There is also a risk of micronutrient deficiencies and bone loss.[25] Currently, there is insufficient evidence regarding the impact of macronutrient manipulation (such as fat versus carbohydrate restriction) compared to conventional calorie restriction (total calorie intake) on weight loss and body composition, particularly in relation to the SO phenotype.[26]

A modest reduction in calorie intake, typically between 200–700 kcal per day, is recommended to achieve gradual weight loss of 0.5–1 kg per week, while minimising muscle loss.[27] Protein intake is particularly important in preserving muscle mass, especially in older adults who are at higher risk of anabolic resistance. A daily protein intake of 1.0–1.2 g/kg body weight is generally recommended to maintain and recover muscle mass and function, with higher amounts (1.2–1.5 g/kg) suggested for individuals with multiple co-morbidities.[28,29] Additionally, protein intake should be spread evenly across meals to optimise muscle protein synthesis, as no significant anabolic benefits have been observed with protein intake exceeding 30 grams per meal.[30]

Apart from the quantity of protein, the source of protein may also be vital for preserving muscle mass. Animal-based proteins, particularly whey, are more effective at promoting muscle protein synthesis due to their higher leucine content and faster digestion and absorption rates.[31] Supplementation with leucine, an essential amino acid, of 2.0–2.5 g per day has been associated with increased muscle protein synthesis in older adults, independent of other amino acid intake.[32]

Time-restricted eating (TRE) involves limiting food intake to specific periods of the day (e.g., an 8-hour feeding window over a 24-hour period). Some studies have suggested that TRE can promote weight loss while preserving muscle mass and improving cardiovascular health and other physiological markers.[33] Hence, TRE could be an appealing, safe, and practical dietary approach for patients with SO, but more research is needed.

The combination of exercise and nutrition interventions offers the best outcomes for individuals with SO. Existing studies suggest that combining exercise with adequate protein intake can lead to significant reductions in fat mass, increases in muscle mass, and improvements in physical function, such as handgrip strength and gait speed.[2]

Role of Nutritional Supplements

Several nutrients and dietary components have been proposed to support muscle health due to their anabolic, anti-inflammatory, anticatabolic, and antioxidant properties. These include omega-3 fatty acids, β-hydroxy β-methylbutyrate (HMB), carotenoids, selenium, and vitamins D, E, and C. However, the evidence supporting these interventions is inconclusive and sometimes conflicting due to limited studies and small sample sizes.[2] Further research is needed to clarify their roles in the treatment of SO.

Impact of Novel GLP-1 Based Anti-Obesity Medications on Muscle Mass and Function

Newer glucagon-like peptide 1 (GLP-1) receptor agonist-based anti-obesity medications, such as semaglutide and tirzepatide, have garnered significant attention in recent years due to impressive weight loss of 17 percent with semaglutide and 21 percent with tirzepatide,[34,35] along with substantial improvements in both subjective and objective measures of physical function. However, these AOMs have not been specifically studied in populations with SO, and existing clinical trials have often limited the enrolment of older adults and individuals with chronic diseases. While semaglutide and tirzepatide can lead to significant adipose tissue loss, they may also result in concomitant muscle loss. In the STEP 1 trial, semaglutide treatment led to lean mass loss of 5.26 kg, representing ~38 percent of total weight loss[34]—exceeding the usual "quarter fat-free mass" rule.[36]

Nevertheless, the extent of muscle mass loss relative to fat loss and the improvement in muscle composition, including reduction in myosteatosis and ectopic fat, remain unclear. These factors are crucial as they play a central role in the pro-inflammatory cycle driving SO.[2] Animal studies also suggest that GLP-1 may improve muscle cell function by enhancing blood flow and nutrient delivery, and even stimulating muscle growth through specific signalling pathways.[37]

Bariatric Surgery

While bariatric surgery is highly effective in treating severe obesity, resulting in significant fat loss and relative improvements in physical function, it also causes substantial loss of muscle mass and strength.[38] Research specifically targeting individuals with SO is limited. In a study of 71 patients, sleeve gastrectomy was shown to better preserve bone and muscle compared to Roux-en-Y bypass.[39] However, remission rates for comorbidities were similar after surgery, regardless of sarcopenia.[40] Given the marked calorie deficit and weight loss in the first year after bariatric surgery, these patients should be managed at specialised multidisciplinary centres during this period, with a focus on nutrition, exercise, and additional medical support.

EMERGING MEDICAL THERAPIES

Myostatin Inhibitors

Myostatin inhibits skeletal muscle growth and development and elevated myostatin levels are observed in individuals with sarcopenia, making it a key target in SO treatment. Its inhibition leads to muscle cell hyperplasia and hypertrophy, suppresses irisin, and downregulates pro-inflammatory cytokines, with potential benefits on metabolism, adiposity, and insulin sensitivity.[41] Interestingly, myostatin levels can be decreased following a DASH diet, with improved body composition and cardiometabolic biomarkers in older adults.[42]

In a trial of the activin receptor type 2B inhibitor bimagrumab among 180 older adults with sarcopenia, it improved functional parameters and lean mass, although no specific subanalysis for those with obesity was conducted.[43] In another trial involving adults with type 2 diabetes and BMI 28–40 kg/m², bimagrumab, combined with diet and exercise, led to significant adipose tissue loss, increased lean mass, and metabolic improvements.[43] However, there was no distinct analysis for those with SO.

Case Study

Mdm H is a 61-year-old woman who was referred to Dr N for treatment of obesity. She describes a gradual increase in weight since her early thirties, with difficulty sustaining weight loss via lifestyle measures. Her current weight is 92 kg (BMI 33.8 kg/m^2) and she has a waist circumference of 92 cm. She also has hypertension, type 2 diabetes (diagnosed four years ago; latest HbA1c 7.3%), and obstructive sleep apnoea. Her medications include metformin 1 g BD, empagliflozin 25 mg OM, and losartan 50 mg OM.

On further history, she describes that she has tried to control her weight by limiting caloric intake. As she is a vegetarian, her current dietary intake consists mainly of vegetables and rice or noodles. She shares that it has been difficult for her to exercise as she gets tired easily. She also describes muscle fatigue even when playing with or carrying her baby grandson. She works as a part-time administrator (sedentary) and spends the rest of her day caring for her grandson at home. She does not smoke or drink alcohol.

Suspecting sarcopenic obesity, Dr N conducted the SARC-F screening questionnaire:

Question	Response	Points
How much difficulty do you have in lifting or carrying 4.5 kg?	Some	1
How much difficulty do you have walking across a room?	None	0
Rising from a chair: How much difficulty do you have transferring from a chair or bed?	Some	1
How much difficulty do you have climbing a flight of 10 stairs?	A lot	2
How many times have you fallen in the past year?	None	0
Total		4

Individual Item Scoring:
None – 0 Some – 1 A lot – 2

A Total Score of ≥4 for SARC-F or ≥11 for SARC-CalF is a positive screening, which warrants confirmation of diagnosis of sarcopenia.

To confirm the diagnosis, further tests were performed:

Test	Result	Cutoff for Sarcopenic Obesity
Muscle Strength		
5 times chair stand test	16 seconds	≥12 seconds
Handgrip strength	15 kg	<18 kg (F)
Body Composition via Bioimpedance Analysis (BIA)		
Muscle mass (SMM/W)	26%	<30.7%(F)*
Fat mass 45%		>35% (F)

* via BIA based on an East Asian population

This confirms the diagnosis of sarcopenic obesity. Dr N explains to Mdm H that the treatment goals will focus on reducing fat mass, while improving her muscle mass and strength. She is started on obesity pharmacotherapy and referred to the dietitian and physiotherapist to guide her on increasing protein intake and resistance exercises.

CONCLUSION

Sarcopenic Obesity poses a real threat to the health of rapidly ageing populations especially with concurrent high prevalence of central obesity. Early detection through screening with a case-finding approach is key to breaking the vicious cycle of SO, which can occur throughout the entire life cycle. Despite challenges in diagnostic criteria, diagnosing SO involves the confirmation of excess or abnormal distribution of adiposity and loss of SMM and function/strength using the relevant population-specific cutoffs. The management of SO requires a multifaceted approach that includes exercise, nutrition, digital technology, and personalised care. Combining resistance and aerobic exercises with adequate protein intake and other nutritional strategies can effectively reduce fat, preserve or increase muscle mass, and improve overall physical function in individuals with SO. More research is needed to refine these interventions and explore the potential benefits of novel pharmacological agents and additional nutritional supplements to treat SO. In the meantime, it is essential to increase the awareness of SO and use simple screening tools in primary care to identify this important condition so

that at-risk individuals can receive appropriate treatments to improve their clinical outcomes.

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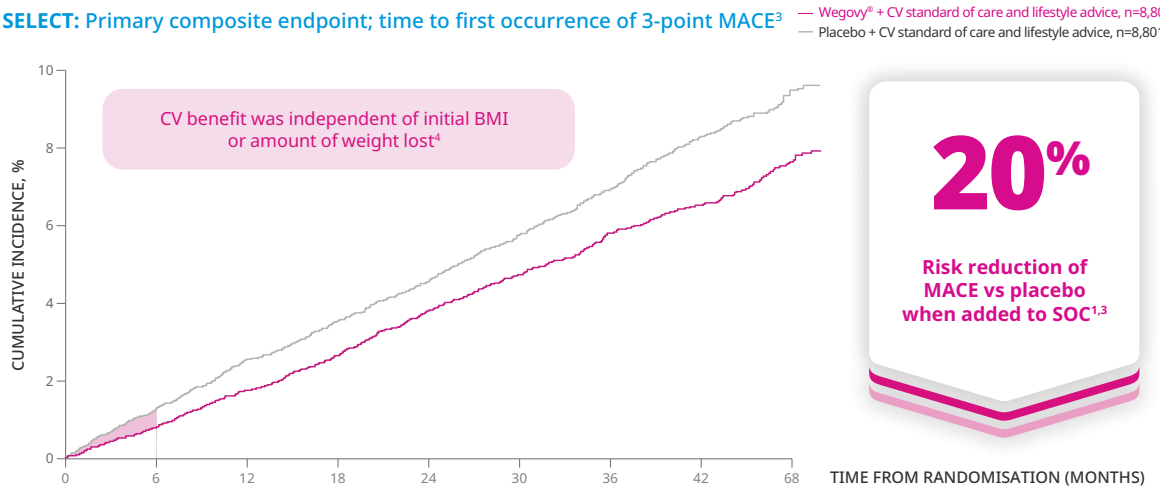

Adapted from Lincoff AM, *et al.* 2023.[3]

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 , respectively.[3]

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.[3]

For healthcare professionals only.

BMI, body mass index; **CV,** cardiovascular disease; **MACE,** major adverse cardiovascular events; **MI,** myocardial infarction; **SOC,** standard of care.

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ASSESSMENT OF 30 MCQS

FPSC NO : 129
MCQS ON BASIC OBESITY MANAGEMENT ACCREDITATION 5
SUBMISSION DEADLINE: 16 December 2025, 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. Select the statement that is true.

A. Weight regain after weight loss is uncommon
B. Assessing for other lifestyle factors like stress levels, psychosocial support in the patient's life will not help to elicit contributory factors of obesity
C. During and after weight loss, there is an adaptive increase in energy expenditure, which stimulates appetite and can persist for many years
D. The release of gastro-intestinal hormones such as Amylin, CCK, PYY is reduced after weight loss, resulting in a loss of appetite suppression and satiety
E. Patients with obesity are to be blamed for their lack of discipline in lifestyle habits as a cause of their obesity

2. Which of the following is true?

A. Healthcare professionals should use condition-first language when conversing with people with obesity
B. People with obesity need more help because they are usually weak-willed
C. Multiple studies on people with obesity suggests that >80% of the people are not willing to be helped with their weight
D. Motivational Interviewing is a doctor-centred approach designed to help doctors during their consultation
E. People with obesity might not perceive their weight to be a significant problem

3. Regarding obesity, which of the following statements is true?

A. Obesity is not a chronic disease as it is the result of a person's choice of lifestyle
B. Socio-economic factors and food insecurity decrease one's likelihood of consuming highly processed, energy-dense foods
C. As a chronic disease, obesity has its distinct disruption to physiology, associated with multiple aetiologies, aggravating factors, and complications
D. As with other chronic disease management, there is no need to assess the patient's expectations and readiness to change before initiating therapy
E. Weight discrimination and obesity stigma has a positive health impact on people living with obesity by motivating them to lose weight

4. A 26-year-old woman presents with progressive weight gain. Which of the following clinical scenarios, if present, would most strongly warrant investigation for a secondary cause of obesity?

A. She gained 12 kg during pregnancy and has been unable to lose it despite lifestyle changes
B. She started antipsychotic medication one year ago and subsequently gained 18 kg
C. She transitioned to a sedentary office job three years ago and has gradually increased her BMI
D. She reports frequent emotional eating during periods of stress at work
E. Over six months, she gained 15 kg and developed new purple abdominal striae and proximal muscle weakness

5. A 44-year-old man with obesity snores loudly, feels fatigued during the day, and has a neck circumference of 42 cm. What is the most appropriate next step to assess his risk of obstructive sleep apnoea (OSA)?

A. Mallampati airway score
B. STOP-BANG questionnaire
C. Epworth Sleepiness Scale
D. Edmonton Obesity Staging System
E. Pittsburgh Sleep Quality Index

6. In the *Agree* step of the 5As framework, which of the following best represents the recommended approach to goal-setting?

A. Physician sets a target of 15% weight loss over six months to optimise metabolic risk
B. Patient and physician agree on a SMART behavioural goal, such as reducing sugar-sweetened beverages
C. Weight discussion is avoided to maintain rapport until the patient initiates it
D. Pharmacotherapy is initiated first-line in all patients with BMI ≥27.5 with comorbidities
E. Patient is instructed to self-monitor calories and weight daily, regardless of readiness

7. A 27-year-old female is seeing you for newly diagnosed prediabetes. She participated in a research study, during which she underwent a 75 g oral glucose tolerance test. The 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 was informed 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 polycystic ovarian syndrome, 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/m^2, and BP 148/92 mmHg.

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

A. It is important to ask the patient about her plans for fertility as it will affect the management of her weight and other medical conditions
B. Further history and clinic examination for evidence of glucocorticoid excess is required
C. Weight loss with intensive lifestyle intervention will reduce the risk of progression of prediabetes to diabetes mellitus
D. The high blood pressure measured in clinic can be assumed to be white coat hypertension, and thus is not a concern
E. A blood test for thyroid function should be performed, if not already done

8. A 40-year-old woman has a history of type 2 diabetes mellitus, hypertension, and schizophrenia. She noticed progressive weight gain over the past year. Which of the following medication most likely contributes to weight gain?

A. Metformin
B. Canagliflozin
C. Valsartan
D. Fluoxetine
E. Risperidone

9. What is the 3Hs concept introduced by the Dietitian to reduce overall calorie intake?

A. My Healthy plate, Healthy Calorie intake, Healthy cooking methods & eating out
B. My Healthy plate, Healthy living, and Healthy physical movement
C. My Healthy plate, Healthy home cooking, Healthy eating out
D. Healthy Calorie intake, Healthy living, Healthy cooking methods & eating out
E. Healthy calorie intake, Healthy food swaps, Healthy living

10. Which of the following statements about intermittent fasting (IF) is most accurate?

A. IF guarantees sustainable long-term weight loss result
B. Skipping breakfast on IF improves basal metabolic rate
C. IF can be restrictive, may disrupt hunger/ fullness cues, and long-term safety is unknown
D. IF eliminates the risk of hyperglycaemia in people with diabetes
E. IF accelerates weight loss from skeletal muscle, adipose tissue, and water

11. When patients are on anti-obesity medications (AOMs) with calorie restriction, what is the key nutrition strategy to help preserve muscle mass and support weight maintenance?

A. Focus only on reducing fat intake
B. Ensure adequate protein intake, especially high-quality sources
C. Recommend complete fasting to accelerate fat loss
D. Avoid all carbohydrates to prevent energy storage
E. Reduce calories coming from fat, sugar, and alcohol

12. The Institute of Medicine recommends 0.8 g/kg/day minimally of protein for healthy adults. In patients on anti-obesity medications with calorie restriction, which statement best reflects current practice?

A. 0.8 g/kg/day is sufficient for all patients, regardless of weight loss or activity
B. Protein needs may be higher, especially with physical activity, to preserve muscle mass
C. Protein intake should always be restricted to avoid kidney strain
D. Protein is only relevant for athletes, not patients with obesity
E. Only total calories matter; protein quality and quantity are less important

13. Individuals with obesity are vulnerable to many psychological comorbidities such as:

A. Depressive disorders
B. Anxiety disorders
C. Somatic symptom disorder
D. Eating disorders
E. All except C

14. When considering socioeconomic constraints as a barrier to lifestyle change, which of these is/are least associated with overweight and obesity in Singapore?

A. Education
B. Income
C. Housing type
D. Employment
E. All except D

15. Which of the following was/were identified as a factor contributing to the lack of time to pursue health and physical activity activities in Singapore?

A. Transportation concerns
B. Social commitment
C. Family and work commitment
D. Medical appointments
E. All of the above

16. An example of contingency contracting includes:

A. A forfeit of deposit amount $300 if the individual failed to attain a goal
B. Referral to specialist if the individual failed to attain a goal
C. Reward the individual with a cheat day if he attains a goal
D. Refund the programme fee if the individual fail to attain a goal
E. None of the above

17. Increasing the individuals' awareness of their current behavioural pattern is less useful for individuals in which stage?

A. Pre-Contemplation
B. Contemplation
C. Preparation stage
D. Action
E. Maintenance

18. In which of the following patients should pharmacotherapy NOT be recommended as an adjunctive treatment with lifestyle therapy?

I. A 30-year-old man, BMI 35 kg/m² with no known medical problems and at least five past attempts of weight loss
II. A 28-year-old female, BMI 24 kg/m² with no central obesity or known medical problems, requesting medications for quick weight loss
III. A 35-year-old female, BMI 34 kg/m² with pre-diabetes, actively planning for a family
IV. A 28-year-old female, BMI 27.5 kg/m², with MASLD and PCOS with no active family plans
V. A 68-year-old man, BMI 29 kg/m² with hypertension, well-controlled type 2 diabetes mellitus and near-end stage renal failure (CKD stage 5)

A. I and III
B. I, II, III, V
C. II, III, V
D. All of the above
E. None of the above

19. 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 gall-stones when initiating GLP-1 receptor agonists

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

20. 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 polycystic ovarian syndrome 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 liraglutide in the long term can reduce her risk of developing diabetes mellitus
B. Orlistat can help her lose about 4-5% of her weight in addition to lifestyle
C. Phentermine is a good option for her as it will not worsen her anxiety
D. GLP-1 receptor agonists should not be started until the thyroid nodule is fully investigated
E. Regardless of choice of medication, she must practise active contraception and discontinue the medication immediately should she get pregnant

21. Which of the following treatment of obesity can lead to significant muscle loss?

I. Lifestyle modification with high-protein diet and exercise
II. GLP1-RA
III. Bariatric surgery

A. III only
B. II and III
C. I and II
D. All of the above
E. None of the above

22. A 47-year-old male patient comes to see you five years after Roux-en-Y gastric bypass surgery for metabolic syndrome. His BMI is currently 32.4 kg/m² from 48 kg/m². He has frequent abdominal pain that worsens after eating. A recent oesophagogastroduodenoscopy done shows the presence of marginal ulcers. What are some treatment options available in the clinic setting?

A. Start anti-obesity medications
B. Smoking cessation
C. Acid suppression (PPIs)
D. B and C
E. All of the above

23. The recent international guidelines states that bariatric surgery can be considered in patients with:

A. BMI ≥27.5 kg/m² or BMI ≥23 kg/m² in patients with obesity-related co-morbidities (minus 2.5 BMI points for Asians)
B. BMI ≥25 kg/m² for all patients regardless of obesity-related co-morbidities (minus 2.5 BMI points for Asians)
C. BMI ≥35 kg/m² or BMI ≥30 kg/m² in patients with medically uncontrolled Diabetes (minus 2.5 BMI points for Asians)
D. BMI ≥45 kg/m² or BMI ≥40 kg/m² in patients with obesity-related co-morbidities (minus 2.5 BMI points for Asians)
E. BMI ≥40 kg/m² or BMI ≥35 kg/m² in patients with obesity-related co-morbidities (minus 2.5 BMI points for Asians)

24. Which of the following procedures has the highest risk of gastroesophageal reflux?

A. Sleeve gastrectomy
B. Roux-en-Y gastric bypass
C. One-anastomosis gastric bypass
D. Bilo-pancreatic diversion
E. Endoscopic sleeve gastroplasty

25. After metabolic bariatric surgery, the following patients should be reviewed by a multi-disciplinary team except for:

A. Mr H is now six years after a sleeve gastrectomy with an average weight loss 25% and his type 2 diabetes has been in remission since surgery
B. Mdm T had a gastric bypass three years ago with remission of her type 2 diabetes and has just found out she is pregnant
C. After maintaining a weight loss of 15% ~18 months after his sleeve gastrectomy, Mr Y now returns with a weight gain of 10% over the last six months
D. Ms A has been able to maintain her 25% weight loss three years after her sleeve gastrectomy and has been having gastric reflux symptoms intermittently
E. Ms W is 24 months after her gastric bypass. She has been having frequent episodes of tremulousness and sweating after food and numbness of both her legs for the last one month

26. According to Singapore's Health Promotion Board guidelines, a child aged 10 years with a BMI at the 95th percentile would be classified as:

A. Normal weight
B. Overweight
C. Severely overweight (obesity equivalent)
D. Requiring immediate bariatric surgery referral
E. Requiring immediate pharmacological intervention

27. Which of the following is the most appropriate initial screening test combination for a 14-year-old with obesity presenting to primary care?

A. Full blood count, urea and electrolytes, thyroid function tests
B. Fasting lipids, glycated haemoglobin, alanine aminotransferase
C. Oral glucose tolerance test, insulin levels, cortisol
D. Echocardiogram, chest X-ray, pulmonary function tests
E. Complete metabolic panel, growth hormone levels, bone age

28. A 13-year-old patient with obesity has been following lifestyle modifications for eight months without significant improvement and has developed MASLD. Which medication would be most appropriate to consider as an adjunct to lifestyle interventions?

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

29. According to the 24-hour activity guidelines mentioned in the article, which recommendation is INCORRECT for children and adolescents?

A. At least 60 minutes of moderate-to-vigorous activity per day

B. At least nine hours of sleep for 7–13 year olds

C. Limit recreational screen time to maximum four hours per day

D. Avoid sugar-sweetened beverages including fruit juices

E. Encourage regular family mealtimes

30. A primary care doctor should refer a child with obesity to tertiary care in all of the following situations EXCEPT:

A. A 12-year-old with BMI at 92nd percentile whose parents are concerned about weight

B. An 8-year-old showing increase in weight with slowing height growth

C. A 15-year-old with severe obesity and newly diagnosed type 2 diabetes

D. A 10-year-old who has lost 3 kg per month after starting dietary changes

E. A 14-year-old with suspected Prader-Willi syndrome

FPSC 128 "Ageing with Vitality" Answers to 15 MCQs					
1.	B	6.	E	11.	C
2.	C	7.	C	12.	D
3.	A	8.	D	13.	A
4.	B	9.	E	14.	C
5.	C	10.	C	15.	D

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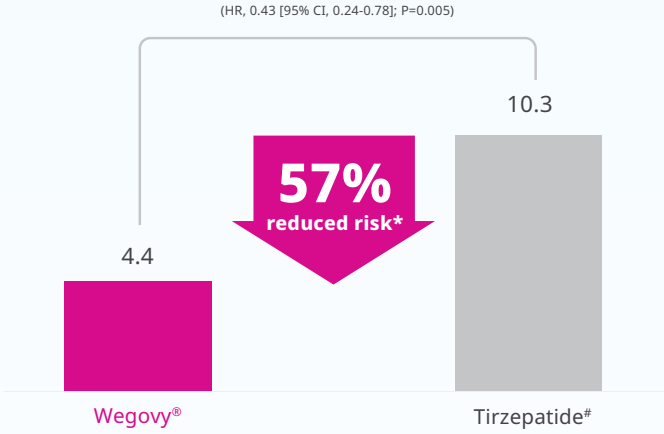

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CI, confidence interval; **CV,** cardiovascular disease; **HR,** hazard ratio; **MACE,** major adverse cardiovascular events.

References: 1. Wilson L, *et al.* Semaglutide is associated with lower risk of cardiovascular events compared with tirzepatide in patients with overweight or obesity and ASCVD and without diabetes in routine clinical practice. Presentation. ESC Congress. 31 August 2025.

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READINGS

A SELECTION OF TEN READINGS ON TOPICS RELATED TO
2025 FAMILY PRACTICE SKILLS COURSE:
BASIC OBESITY MANAGEMENT ACCREDITATION 5

A SELECTION OF TEN READINGS ON TOPICS RELATED TO 2025 FAMILY PRACTICE SKILLS COURSE: BASIC OBESITY MANAGEMENT ACCREDITATION 5

FPSC129 – SATURDAY, 11 OCT 2025 & SUNDAY 12 OCT 2024: 2.00pm–5.30pm
All are available as PMC free full text

Selection of readings made by A/Prof Goh Lee Gan

READING 1 – LIFESTYLE INTERVENTIONS FOR TREATMENT AND REMISSION OF TYPE 2 DIABETES AND PREDIABETES IN ADULTS

Rosenfeld RM,[1] Grega ML,[2] Karlsen MC,[3] Staffier KL,[3] Abu Dabrh AM,[4] Aurora RN,[5] Bonnet JP,[6] Donnell L,[7] Fitzpatrick SL,[8] Frates B,[9] Joy EA,[10] Kapustin JF,[11] Noe DR,[12] Panigrahi G,[13,14] Ram A,[15] Levine Reisner LS,[16] Valencia WM,[17] Weatherspoon LJ,[18] Weber JM,[19] Gulati M.[20] Lifestyle Interventions for Treatment and Remission of Type 2 Diabetes and Prediabetes in Adults: A Clinical Practice Guideline From the American College of Lifestyle Medicine. Am J Lifestyle Med. 2025 Jun 10;19(2 Suppl):10S-131S. PMID: 40546761.

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ABSTRACT

OBJECTIVE: Diabetes is a defining disease of the 21st century because of its rising prevalence, association with obesity, and enormous health impact. Abundant evidence shows that lifestyle interventions can delay or prevent type 2 diabetes (T2D) in adults, offer relief, and sometimes achieve complete remission. Despite this empowering message, there are no clinical practice guidelines that focus primarily on lifestyle interventions as first-line management of prediabetes and T2D. Our objective, therefore, is to offer pragmatic, trustworthy, and evidence-based guidance for clinicians in using the six pillars of lifestyle medicine-nutrition; physical activity; stress management; sleep; social connectedness; and avoidance of risky substances-for managing adults with T2D and in preventing T2D in adults with prediabetes or a history of gestational diabetes mellitus.

METHODS: We used well-established, peer-reviewed guideline methodology to develop evidence-based key action statements (recommendations) that facilitate quality improvement in clinical practice. The guideline development group included 20 members representing consumers, advanced practice nursing, cardiology, clinical pharmacology, behavioural medicine, endocrinology, family medicine, lifestyle medicine, nutrition and dietetics, health education, health and wellness coaching, sleep medicine, sports medicine, and obesity medicine. Recommendation strength was based on the aggregate evidence supporting a key action statement plus a comparison of associated benefits vs harms/costs. Multiple literature searches, conducted by an information specialist, identified eight relevant guidelines, 118 relevant systematic reviews, and 112 randomised clinical trials. The guideline underwent extensive internal, external, and public review and comment prior to publication.

RESULTS: We developed 14 key action statements and associated evidence profiles, each with a distinct quality improvement goal in the context of lifestyle interventions for T2D. Strong recommendations were made regarding advocacy for lifestyle interventions; assessing baseline lifestyle habits; establishing priorities for lifestyle change; prescribing aerobic and muscle strength physical activity; reducing sedentary time; identifying sleep disorders; prescribing nutrition plans for prevention and treatment; promoting peer/familial support and social connections; counselling regarding tobacco, alcohol, and recreational drugs; and establishing a plan for continuity of care. Recommendations were made regarding identifying the need for psychological interventions and for adjusting (deprescribing) pharmacologic therapy. We include numerous tables and figures to facilitate implementation, a plain-language summary for consumers, and an executive summary for clinicians as separate publications.

CONCLUSIONS: There is robust research evidence supporting the efficacy of lifestyle interventions in preventing, treating, and achieving remission of T2D in adults. Our multidisciplinary guideline development group successfully synthesised this evidence into 14 key action statements that can be used by clinicians and other healthcare professionals to improve quality of care for adults with, or at-risk for, T2D. Despite the research gaps and implementation challenges we highlight in the guideline, we believe strongly that our recommendations have immediate relevance and can help raise awareness and shift the paradigm of T2D management towards optimal use of lifestyle interventions.

READING 2 – INTERNATIONAL CONSENSUS ON SURGERY FOR TYPE 2 DIABETES MELLITUS

Kermansaravi M,[1,#] Omar [1,2,3,#] Finer N,[4] Le Roux C,[5] Carbajo MA,[6] Sarwer D,[7] Busetto L,[8] Ponce J,[9] Logue J,[10] Parretti HM,[11] O'Kane M,[12] Shahabi S,[13] Khunti K,[14] Blakemore AI,[15] Stenberg E,[16] Abbott S,[17] Alqahtani A,[18] Aminian A,[19] Amr B,[20] Balibrea JM,[21] Batterham RL,[22] Behrens E,[23] Bhatt DL,[24] Chesworth P,[25] Chowbey P,[26] Clare K,[27] Neto MG,[28] Graham Y,[29] Goel R,[30] Hanif W,[31] Herrera MF,[32] Kasama K,[33] Kassir R,[34] Knop FK,[35] Kothari SN,[36] Kristinsson JA,[37] McGowan B,[38] McKechnie A,[39] Miller K,[40] Miras AD,[41] Morton J,[42] Ogden J,[43] Peterli R,[44,45] Pinkney JH,[46] Pournaras D,[47] Pouwels S,[48] Prager G,[49] Salminen P,[50,51] Serlie MJ,[52,53] Shabbir A,[54] Singhal R,[55] Taheri S,[56] Tahrani AA,[57] Weiner R,[58] Shikora SA,[59] Mahawar K.[60] International expert consensus on surgery for type 2 diabetes mellitus. BMC Endocr Disord. 2025 Jul 1;25(1):151. PMID: 40598146.

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ABSTRACT

INTRODUCTION: Metabolic and bariatric surgery (MBS) has been an established treatment option for patients with Type 2 diabetes mellitus (T2DM), but there is a relative paucity of evidence-based guidelines on preoperative, operative, and postoperative considerations concerning metabolic surgery for T2DM patients. To address this gap, we initiated a Delphi consensus process with a diverse group of international multidisciplinary experts.

METHOD: We embarked on a Delphi consensus-building exercise to propose an evidence-based expert consensus covering various aspects of MBS in patients with T2DM. We defined the scope of the exercise and proposed statements and surveyed the literature through electronic databases. The literature summary and voting process were conducted by 52 experts, who evaluated 44 statements. The quality of evidence was assessed using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) criteria.

RESULTS: Consensus, defined as >80% agreement, was reached for 43 out of 44 statements. The experts reached an agreement on the nature, terminology, and mechanisms of action of MBS. The currently available scores for predicting remission of T2DM after surgery are not robust enough for routine clinical use, and there is a need for further research to enable more personalised treatment. Additionally, they agreed that metabolic surgery for T2DM is cost-effective, and MBS procedures for treating T2DM vary in their safety and efficacy.

CONCLUSION: This Delphi expert consensus statement guides clinicians on various aspects of metabolic surgery for T2DM and also grades the quality of the available evidence for each of the proposed statements.

READING 3 – PORTFOLIO DIET AND LDL-C IN A YOUNG MULTIETHNIC COHORT

Chen V,[1,2] Zeitoun T,[1] El-Sohemy A,[1] Kavanagh ME,[1,2] Chiavaroli L,[1-3] Kendall CWC,[1,2,6] Jenkins DJA,[1-3,7,8] Mahdavi S,[1,4] Glenn AJ,[4,5] Sievenpiper JL.[9-13] Portfolio diet and LDL-C in a young, multiethnic cohort: cross-sectional analyses with cumulative exposure modelling. BMC Public Health. 2025 May 13;25(1):1761. PMID: 40361017.

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ABSTRACT

BACKGROUND: The Portfolio Diet is a plant-based dietary pattern of cholesterol-lowering foods that has demonstrated clinically meaningful reductions in low-density lipoprotein cholesterol (LDL-C) and other cardiovascular risk factors. However, the Portfolio Diet has not been assessed in an ethnoculturally diverse population of young adults.

OBJECTIVE: To examine the association of the Portfolio Diet Score (PDS) with LDL-C and other established cardiovascular risk factors in a young adult population.

METHODS: This cross-sectional analysis included 1,507 men and women (mean age, 23±3 years) of diverse ethnocultural backgrounds from the Toronto Nutrigenomics and Health Study. Diet was assessed by a validated Toronto-modified Harvard 196-item food frequency questionnaire with adherence to the Portfolio Diet measured using the Portfolio Diet Score. Data were analysed using multiple linear regressions with adjustment for potential confounders. Modelling analyses related LDL-C levels according to absolute adherence to the Portfolio Diet with cumulative LDL-C and onset of rising cardiovascular risk by age.

RESULTS: Participants were Caucasian (49%), East Asian (34%), South Asian (11%), or other (7%) with a mean LDL-C of 2.3±0.7mmol/L. A 1-point higher PDS and higher PDS tertiles were associated with lower LDL-C (ß [95% CI] per 1-point: -0.009 mmol/L [-0.016, -0.002], P=0.013; Ptrend across tertiles=0.040), non-HDL-C (-0.010 mmol/L [-0.018, -0.002], P=0.014; Ptrend=0.028), total cholesterol (-0.011 mmol/L [-0.019, -0.003], P=0.011; Ptrend=0.038), systolic blood pressure (-0.150 mmHg [-0.250, -0.050], P=0.003; Ptrend<0.001) and diastolic blood pressure (-0.133 mmHg [-0.219, -0.046], P=0.003; Ptrend<0.001). Higher PDS tertiles were associated with lower triglycerides (Ptrend=0.039). A 1-point higher PDS was also associated with lower BMI (-0.038 kg/m^2 [-0.071, -0.004], P=0.026), waist circumference (-0.092 cm [-0.171, -0.013], P=0.022), body weight (-0.124 kg [-0.229, -0.019], P=0.021) and FMI (-0.019 kg/m^2 [-0.037, -0.001], P=0.039). There was no association with HDL-C, CRP, or fasting glucose. Modelling analyses suggest that, compared to low adherence, 50% and 100% adherence to the Portfolio Diet may delay the onset of rising cardiovascular risk by an estimated six and 13 years, respectively.

CONCLUSIONS: Among young adults, the PDS was inversely associated with LDL-C and several other established cardiovascular risk factors. Early adherence to the Portfolio Diet may limit lifetime exposure to LDL-C and could delay the age at which cardiovascular events begin.

READING 4 – GLOBAL PATTERNS OF NONINVASIVE TESTS FOR THE CLINICAL MANAGEMENT OF METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE

Allen AM,[1,2] Mark HE,[1] Lazarus JV,[1,3,4] Alkhouri N,[1,5] Noureddin M,[1,6] Wong VW,[1,7] Tsochatzis EA,[1,8] de Avila L,[1,9] Racila A,[1,9] Nader F,[1,9] Henry L,[1,9] Stepanova M,[1,9] Younossi ZM,[1,9,11] Castera L.[1,10] Global patterns of utilisation of noninvasive tests for the clinical management of metabolic dysfunction-associated steatotic liver disease. Hepatol Commun. 2025 Apr 30;9(5):e0678. PMID: 40304566.

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ABSTRACT
BACKGROUND: Noninvasive tests (NITs) are used to risk-stratify metabolic dysfunction-associated steatotic liver disease. The aim was to survey global patterns of real-world use of NITs.

METHODS: A 38-item survey was designed by the Global NASH Council. Providers were asked about risks for advanced fibrosis, which NITs (cutoff values) they use to risk-stratify liver disease, monitor progression, and which professional guidelines they follow.

RESULTS: A total of 321 participants from 43 countries completed the survey (54% hepatologists, 28% gastroenterologists, and 18% other). Of the respondents, 85% would risk-stratify patients with type 2 diabetes, obesity (82%), or abnormal liver enzymes (73%). Among NITs to rule out significant or advanced fibrosis, transient elastography (TE) and fibrosis-4 (FIB-4) were most used, followed by NAFLD Fibrosis Score, Enhanced Liver Fibrosis, and magnetic resonance elastography. The cutoffs for ruling out significant fibrosis varied considerably between practices and from guidelines, with only 50% using TE <8 kPa, 65% using FIB-4 <1.30 for age <65, and 41% using FIB-4 <2.00 for age ≥65. Similar variability was found for ruling in advanced fibrosis, where thresholds of FIB-4 ≥2.67 and TE ≥10 kPa were used by 20% and 17%, respectively. To establish advanced fibrosis, 48% would use two NITs while 23% would consider 1 NIT, and 17% would confirm with liver biopsy. TE was used by >75% to monitor, and 66% would monitor (intermediate or high risk) annually. Finally, 65% follow professional guideline recommendations regarding NITs.

CONCLUSIONS: In clinical practice, there is variability in NIT use and their thresholds. Additionally, there is suboptimal adherence to professional societies' guidelines.

READING 5 – LIFESTYLE AND SURGICAL INTERVENTIONS TO ACHIEVE WEIGHT LOSS IN PEOPLE WITH OVERWEIGHT OR OBESITY

Idris I,[1,2] Anyiam O.[1,2] The latest evidence and guidance in lifestyle and surgical interventions to achieve weight loss in people with overweight or obesity. Diabetes Obes Metab. 2025 Apr;27 Suppl 2(Suppl 2):20-34. PMID: 40026042.

doi: 10.1111/dom.16296. PMID: 40026042. Free full text.

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ABSTRACT
BACKGROUND: The prevalence of obesity and related co-morbidities has reached epidemic proportions. Effective evidence-based treatment approaches are therefore important. Lifestyle intervention remains the mainstay of the treatment strategy to manage obesity. Increased evidence has also emerged regarding the efficacy of metabolic bariatric surgery (MBS) to induce significant and sustained weight loss while also reducing the progression of obesity-related co-morbidities for people living with obesity.

AIMS & METHODS: This article aims to bring together current evidence, guidance, and best practice for the prevention and management of people living with overweight or obesity by means of lifestyle and behavioural intervention, as well as by MBS.

RESULT: Lifestyle intervention encompasses dietary strategies, physical activity, and behavioural intervention. Discussion on MBS will focus on current indications, comparison between different MBS procedures, novel endoscopic techniques, potential complications, and pre-operative management.

PLAIN LANGUAGE SUMMARY: The number of people living with excess weight and complications associated with being overweight is alarmingly high. Effective treatment approaches that are supported by clinical studies are therefore important. Lifestyle changes remain important in managing excess weight. Increased evidence has also shown the benefits of weight loss surgery to produce significant weight loss that can be sustained, while also reducing the risk of developing medical conditions associated with excess weight. This article aims to bring together current evidence, guidance, and best practice for the prevention and management of people living with excess weight by means of lifestyle and behavioural changes, as well as by weight loss surgery. Lifestyle intervention encompasses dietary strategies, physical activity, and behavioural intervention. Discussion on weight loss surgery will focus on current criteria for suitability, comparison between different weight loss surgery procedures, new techniques, possible complications, and appropriate management prior to weight loss surgery.

READING 6 – POSITION STATEMENT AND GUIDELINES ABOUT ENDOSCOPIC SLEEVE GASTROSCOPY

Baratte C,[1] Poghosyan T,[1] Sebbag H,[2] Arnalsteen L,[3] Auguste T,[4] Blanchet MC,[5] Benchetrit S,[6] Abou-Mrad A,[7] Reche F,[8] Genser L,[9] Caiazzo R,[10] Lazzati A,[11] Catheline JM,[12] Pourcher G,[13] Leyre P,[14] Kamoun-Zana S,[15] Stenard F,[16] Coste T,[17] Sterkers A,[18] Blanchard C,[19] Pattou F,[20] Perretta S,[21] Robert M.[22] Position statement and guidelines about Endoscopic Sleeve Gastroplasty (ESG) also known as "Endo-sleeve". J Visc Surg. 2025 Feb;162(1):71-78. PMID: 39794164.

doi: 10.1016/j.jviscsurg.2024.12.003. PMID: 39794164. Free full text.

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ABSTRACT
IS ESG EFFECTIVE IN THE TREATMENT OF OBESITY AND ASSOCIATED COMORBIDITIES?: Endoscopic Sleeve Gastroplasty (ESG) is more effective than lifestyle modifications alone for weight loss and improving obesity-related comorbidities. While it has less effect on weight loss compared to Laparoscopic Sleeve Gastrectomy (LSG) in the short to medium term, it offers similar comorbidities resolution to LSG.

ABSTRACT

IS ESG EFFECTIVE IN THE TREATMENT OF OBESITY AND ASSOCIATED COMORBIDITIES?: Endoscopic Sleeve Gastroplasty (ESG) is more effective than lifestyle modifications alone for weight loss and improving obesity-related comorbidities. While it has less effect on weight loss compared to Laparoscopic Sleeve Gastrectomy (LSG) in the short to medium term, it offers similar comorbidities resolution to LSG.

IS ESG A SAFE PROCEDURE, AND WHAT ARE ITS RISKS?: The safety profile of ESG is consistently supported in the literature. Surgical complications after ESG, ranging from 1.5–2.3%, such as bleeding, perforation, fistula, or upper bowel obstruction, are rare and typically managed endoscopically. The incidence of new-onset gastro-oesophageal reflux disease (GERD) is deemed negligible and occurs less frequently after ESG compared to SG.

WHAT ARE THE INDICATIONS AND MANAGEMENT METHODS?: Multidisciplinary care for patients undergoing ESG should be provided in an accredited centre authorised to perform bariatric and metabolic surgery, with validation through a multidisciplinary consultation meeting (RCP). Perioperative management should be personalised and ideally modelled after the protocols already in place for bariatric and metabolic surgery to ensure satisfactory and lasting weight and metabolic outcomes. Adherence to follow-up visits is a significant predictor of successful weight loss outcomes after ESG. Additionally, all endoscopic surgical procedures should be documented in a registry affiliated with a recognised scientific society, as is standard for other bariatric surgical procedures.

WHICH HEALTHCARE PROFESSIONALS CAN PERFORM ESG?: ESG must be performed by a practitioner trained in endoscopy and obesity management, capable of ensuring thorough preoperative care and comprehensive postoperative follow-up, supported by an experienced multidisciplinary team. In France, Notice No. 2021.0040/AC/SEAP of 10 June 2021, issued by the Haute Autorité de santé (HAS) college, specifies that "the technology of ESG via the trans-oral approach, involving wide plication of the greater gastric curvature […] with an endoscopic suture placement device, enables a gastroenterologist or a visceral and digestive surgeon to perform gastric plication through digestive endoscopy by placing sutures in the stomach". Ideally, this should take place in an accredited centre authorised to perform bariatric and metabolic surgery, such as those approved by the Agence régionale de santé (ARS), in accordance with Article R6123-212 of December 2022 of the French Public Health Code.

WHAT ARE THE RECOMMENDATIONS AND VIEWS OF OTHER INTERNATIONAL SCIENTIFIC SOCIETIES?: ESG is an integral part of the therapeutic arsenal available to bariatric and metabolic surgeons, offering an effective and valuable treatment option for obesity in specific patient populations. The International Federation for the Surgery of Obesity (IFSO) Bariatric Endoscopy Committee, following a comprehensive systematic review and meta-analysis, endorsed ESG as an effective and valuable treatment for obesity. ESG is particularly beneficial for patients with class I and II obesity, as well as for those with class III obesity who are not suitable candidates for metabolic bariatric surgery. Additionally, it can be proposed as an addition to lifestyle interventions in adolescent patients with class II obesity. The SOFFCOMM endorses endoscopic sleeve gastroplasty (ESG) as an effective and valuable treatment for obesity and highlights the importance of appropriate patient selection, coupled with rigorous evaluation of long-term outcomes, to refine its indications further.

READING 7 – SAGES GUIDELINES FOR MANAGEMENT OF CO-MORBIDITIES RELEVANT TO METABOLIC AND BARIATRIC SURGERY

Kumar SS,[1] Wunker C,[2] Collings A,[3] Bansal V,[4] Zoumpou T,[5] Chang J,[6] Rodriguez N,[7] Aleassa EM,[7] Sabour A,[8] Hilton LR,[9] Ghanem OM,[10] Kushner BS,[11] Loss LJ,[12] Haskins IN,[13] Ayloo S,14 Reid A,[15] Overby DW,[16] Hallowell P,[17] Kindel TL,[18] Slater BJ,[19] Palazzo F.[20] SAGES guidelines for the management of comorbidities relevant to metabolic and bariatric surgery. Surg Endosc. 2025 Jan;39(1):1-10. PMID: 39663246.

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Erratum in Surg Endosc. 2025 Feb;39(2):1407. doi: 10.1007/s00464-025-11537-3.

ABSTRACT
BACKGROUND: Patients who are under consideration for or have undergone metabolic and bariatric surgery frequently have comorbid medical conditions that might make their perioperative care more complex. These recommendations address routine intraoperative cholangiography in patients with bypass-type anatomy, the management of reflux disease after sleeve gastrectomy, and the optimal bariatric procedure for patients with comorbid inflammatory bowel disease.

METHODS: A systematic review was conducted including studies published from 1990 to 2022 to address these questions. These results were then presented to a panel of bariatric surgeons who formulated recommendations based on the best available evidence or utilised expert opinion when the evidence base was lacking.

RESULTS: Conditional recommendations were made in favour of routine intraoperative cholangiography in patients with bypass-type anatomy undergoing laparoscopic cholecystectomy, trialling medical management prior to surgical management in patients with reflux after sleeve gastrectomy, and sleeve gastrectomy rather than Roux-en-Y gastric bypass in patients with inflammatory bowel disease. The strength of these recommendations was limited by the quality of evidence available. Recommendations for future research were made for all questions.

CONCLUSIONS: These recommendations should provide guidance regarding management of these comorbidities in patients who are under consideration for or have undergone metabolic and bariatric surgery. These recommendations also identify important areas where future research should focus on to strengthen the evidence base.

READING 8 – PHARMACOTHERAPY FOR OBESITY MANAGEMENT IN ADULTS

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

doi: 10.1503/cmaj.250502. PMID: 40789597. Free full text.

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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 9 – NUMBER NEEDED TO TREAT FOR SEMAGLUTIDE IN POPULATIONS WITH OVERWEIGHT OR OBESITY

Lübker C,[1] Bhavsar J,[2] Duque do Vale R,[3] Nørtoft E,[3] Tarp JM,[3] Emerson SS,[4] Plutzky J,[5] Roberts G,[6] Lincoff AM.[7] The Composite Number Needed to Treat for Semaglutide in Populations with Overweight or Obesity and Established Cardiovascular Disease Without Diabetes. Adv Ther. 2025 May;42(5):2513-2525. PMID: 40156748.

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ABSTRACT

INTRODUCTION: Number needed to treat (NNT), an outcome measure derived from the estimated risk results of clinical trials, is widely used to demonstrate value to stakeholders by identifying how many patients require treatment to avoid one event of interest. However, NNTs calculated for primary trial endpoints might underestimate a treatment's value by not considering other outcomes. In this secondary analysis of data from the SELECT cardiovascular (CV) outcomes trial, we aimed to determine the NNT for semaglutide for major adverse cardiovascular events (MACE), in addition to NNTs when other clinically and payer-relevant outcomes are included.

METHODS: This study is a secondary analysis of data from the randomised, double-blind SELECT trial (ClinicalTrials.gov NCT03574597) of once-weekly subcutaneous administration of semaglutide compared with placebo in 17,604 patients with overweight or obesity and with established cardiovascular disease (CVD) (39.8 months mean follow-up). The outcomes were NNT3P-MACE (based upon the trial's composite primary endpoint of death from cardiovascular causes, non-fatal myocardial infarction, non-fatal stroke), NNTEXTENDED (inclusive of NNT3P-MACE, hospitalisation for any cause, coronary revascularisation, and non-CV death), and NNTCKM (inclusive of NNTEXTENDED, glycated haemoglobin level [HbA1c] ≥6.5%, and a 5-point nephropathy composite).

RESULTS: The relative risk reductions observed for the events comprising the NNTs were 20% (NNT3P-MACE), 20% (NNTEXTENDED), and 41% (NNTCKM). At 1- and 4-years post-initiation of semaglutide, NNT3P-MACE was 125 and 58, NNTEXTENDED was 49 and 25, and NNTCKM was 20 and 11, respectively.

CONCLUSION: When clinically and payer-relevant outcomes from the SELECT trial are included in calculations of NNT, semaglutide was associated with greater risk reductions and lower estimates of NNT than for the primary endpoint alone. Our findings suggest that including the broader effects of semaglutide beyond the primary trial endpoint recognises additional value to stakeholders.

READING 10 – STRATEGIES FOR MINIMISING MUSCLE LOSS DURING USE OF INCRETIN-MIMETIC DRUGS FOR TREATMENT OF OBESITY

Mechanick JI,[1] Butsch WS,[2] Christensen SM,[3] Hamdy O,[4] Li Z,[5] Prado CM,[6] Heymsfield SB.[7] Strategies for minimising muscle loss during use of incretin-mimetic drugs for treatment of obesity. Obes Rev. 2025 Jan;26(1): e13841.PMID: 39295512.

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ABSTRACT

The rapid and widespread clinical adoption of highly effective incretin-mimetic drugs (IMDs), particularly semaglutide and tirzepatide, for the treatment of obesity has outpaced the updating of clinical practice guidelines. Consequently, many patients might be at risk for adverse effects and uncertain long-term outcomes related to the use of these drugs. Of emerging concern is the loss of skeletal muscle mass and function that can accompany rapid substantial weight reduction; such losses can lead to reduced functional and metabolic health, weight cycling, compromised quality of life, and other adverse outcomes.

Available evidence suggests that clinical trial participants receiving IMDs for the treatment of obesity lost 10% or more of their muscle mass during the 68- to 72-week interventions, approximately equivalent to 20 years of age-related muscle loss. The ability to maintain muscle mass during caloric restriction-induced weight reduction is influenced by two key factors: nutrition and physical exercise. Nutrition therapy should ensure adequate intake and absorption of high-quality protein and micronutrients, which might require the use of oral nutritional supplements. Additionally, concurrent physical activity, especially resistance training, has been shown to effectively minimise loss of muscle mass and function during weight reduction therapy.

All patients receiving IMDs for obesity should participate in comprehensive treatment programmes emphasising adequate protein and micronutrient intakes, as well as resistance training, to preserve muscle mass and function, maximise the benefit of IMD therapy, and minimise potential risks.

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References: 1. Wegovy® Singapore Prescribing Information, August 2025. **2.** Garvey WT, *et al. Nat Med*. 2022;28(10):2083-2091 (plus supplementary appendix). **3.** Rubino D, *et al. JAMA*. 2021; 325:1414–25 (plus supplementary appendix). **4.** Lincoff AM, *et al. N Engl J Med*. 2023;389:2221–32. **5.** Wilson L, *et al.* Semaglutide is associated with lower risk of cardiovascular events compared with tirzepatide in patients with overweight or obesity and ASCVD and without diabetes in routine clinical practice. Presentation. ESC Congress. 31 August 2025. **6.** Deanfield J, *et al. Obes Facts*. 2024;17(suppl 1):7-515. **7.** Butler J, *et al. Lancet*. 2024; 403: 1635–48. **8.** Zhao Z, *et al. N Engl J Med*. 2023;389(24)2221-2232.

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The title should be concise and highlight the key elements of the article.

Include on the title page the first and last names, designation, qualifications, present appointments, and type and place of practice of each contributor.

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The summary should describe why the article was written and present the main argument or findings.

Limit words as follows: 250 words for major articles; 200 words for case reports.

All Original articles (examples: randomised controlled trials, cohort studies, observational studies, and review articles) must be accompanied by a structured abstract while all other categories of manuscripts (examples: PRISM and Case Records of Family Medicine) should have unstructured abstracts.

Structured Abstract – Organise the abstract according to the following headings:

- **Introduction** – states the purposes/aims of the study/ investigation
- **Methods** – describes the selection of study subjects/ experimental animals, observational, and analytical methods
- **Results** – provides specific data and its statistical significance, if possible
- **Conclusion** – succinct emphasis of new and important aspects of the study or observations

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Add, at the end of summary in alphabetical listing, **key words** of up to five in number that will be used for article indexing and retrieval under Medical Subject Headings or MeSH. MeSH is the NLM controlled vocabulary thesaurus used for indexing articles for WPRIM and PubMed. Please refer to www.nlm.nih.gov/mesh/ for details.

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- **Technical Information (under methods)**
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  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.

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

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The Case Records of Family Medicine is a newly created series to encourage submissions from Family Medicine teaching programmes and for Family Medicine departments to submit cases of learning value to the *Singapore Family Physician*. Cases discussed during peer review learning and Family Medicine grand ward round teachings are just some examples of submissions that are suitable for this

series. Authors planning to submit their case studies to the Case Records of Family Medicine section should structure their article according to these headings:

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.

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

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

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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).

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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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References: 1. Wegovy® Singapore Prescribing Information, February 2025. **2.** Weghuber D, et al. *N Engl J Med*. 2022;387:2245–57 (Plus Supplementary Appendix).

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References: 1. Wegovy® Singapore Prescribing Information, August 2025. **2.** Lincoff AM, *et al. N Engl J Med.* 2023;389:2221–32. **3.** Wilson L, *et al.* Semaglutide is associated with lower risk of cardiovascular events compared with tirzepatide in patients with overweight or obesity and ASCVD and without diabetes in routine clinical practice. Presentation. ESC Congress. 31 August 2025.

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