

The Australian Obesity Management Algorithm - a simple tool to guide the management of obesity in primary care

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Abstract

Obesity is a complex and multifactorial chronic disease with genetic, environmental, physiological and behavioural determinants that requires long-term care. Obesity is associated with a broad range of complications including type 2 diabetes, cardiovascular disease, dyslipidaemia, metabolic associated fatty liver disease, reproductive hormonal abnormalities, sleep apnoea, depression, osteoarthritis and certain cancers. An algorithm has been developed (with PubMed and Medline searched for all relevant articles from 1 Jan 2000-1 Oct 2021) to (i) assist primary care physicians in treatment decisions for non-pregnant adults with obesity, and (ii) provide a practical clinical tool to guide the implementation of existing guidelines (summarised in Appendix 1) for the treatment of obesity in the Australian primary care setting.

Main recommendations and changes in management: Treatment pathways should be determined by a person's anthropometry (body mass index (BMI) and waist circumference (WC)) and the presence and severity of obesity-related complications. A target of 10-15% weight loss is recommended for people with BMI 30-40 kg/m² or abdominal obesity (WC > 88 cm in females, WC > 102 cm in males) without complications. The treatment focus should be supervised lifestyle interventions that may include a reduced or low energy diet, very low energy diets (VLED) or pharmacotherapy. For people with BMI 30-40 kg/m² or abdominal obesity and complications or those with BMI >40 kg/m² a weight loss target of 10-15% body weight is recommended, and management should include intensive interventions such as VLED, pharmacotherapy or bariatric surgery, which may be required in combination. A weight loss target of >15% is recommended for those with BMI >40 kg/m² and complications and they should be referred to specialist care. Their treatment should include a VLED with or without pharmacotherapy and bariatric surgery.

Keywords: obesity, very low energy diet, reduced energy diet, low energy diet, physical activity, anti-obesity pharmacotherapy, bariatric surgery

Section 1: Guiding principles

1. The benefits of weight loss

In 2017-18, 67% of Australians aged 18 years and over were above a healthy weight, with 31% having obesity (1). Obesity and its related complications place a considerable financial burden on Australia. In 2014-15, the direct and indirect costs of obesity were estimated at \$8.65 billion (2). Although the focus of this document is on weight loss interventions for the management of obesity, maintaining a healthy lifestyle and preventing weight gain in individuals whose weight is in the healthy or overweight range is an important and essential strategy to prevent a worsening of the current obesity epidemic.

Weight loss in people with obesity has proven medical benefits in reducing the risk of diabetes, other obesity-related complications and mortality (3). The weight loss required to achieve some of these benefits is relatively small, with weight loss in the order of 5% showing reduction in diabetes risk (4). However, some people with more severe obesity will require greater degrees of weight loss to improve their health, function and wellness.

2. The role of primary care

Primary care is critical to addressing Australia's obesity problem. It is essential that primary care practitioners identify and treat medical conditions that are largely driven by obesity as summarised in Appendix 2. Management of obesity in primary care requires a personalised approach, often in a shared care arrangement, with regular monitoring and the application of a variety of weight loss strategies, intensified over time if weight loss and health targets are not achieved.

Routine and regular consideration and assessment of weight are essential initial steps that allow identification of:

- individuals whose weight is affecting their health and who may benefit from weight management interventions; and,
- individuals who are gaining weight and require counselling and weight management interventions to prevent further weight gain.

3. Weight bias – stigma - discrimination

Weight bias in obesity care is a common and important obstacle that can interfere with effective obesity treatment. Bias arises in part from the erroneous belief that obesity is caused by lifestyle choices alone. There is strong evidence that obesity is predominantly genetic (5, 6) caused by classical genetic mutations (7-9) and epigenetic mechanisms (10, 11). Weight bias is common among healthcare providers with accumulating evidence that individuals with obesity are perceived as lacking self-control, unmotivated to improve health,

noncompliant with treatment, and personally to blame for their weight (12). Those who perceive bias from their healthcare providers have less trust in them, experience more difficulty losing weight and avoid preventive health services and medical appointments (13). Conversely, provider weight bias may result in less willingness to help individuals with obesity compared to those with healthier weight. Healthcare professionals should reflect on their attitude towards individuals with obesity and the potential for weight bias as they can be major barriers to appropriate care.

4. A person-centred approach

Weight and weight loss can be sensitive issues. Most individuals with obesity have attempted multiple weight loss interventions and may not be ready to make another attempt. Poor body image, low self-esteem, psychological problems and eating disorders, such as binge eating and food addiction, are common and will influence the effectiveness of treatment. Even weight measurement may be upsetting for some people. The objective of reducing weight and improving lifestyle behaviour should be discussed at the outset and differing expectations between medical and non-medical benefits reconciled. This discussion should be used to inform the setting of individual realistic and sustainable weight loss targets according to the treatment selected.

Section 2: Treatment options for obesity

1. Lifestyle interventions

Supervised lifestyle interventions are an essential component of all weight loss strategies. Treatment goals focus on reducing energy intake, optimising diet quality and increasing energy expenditure. The consumption of a high energy diet does not necessarily equate to a nutritionally sound diet and consideration of the nutritional adequacy of an individual's diet needs to be considered (14). On the other hand, improving diet quality and increasing physical activity improves health outcomes even without weight reduction. General advice on healthy eating is defined in the Australian Dietary Guidelines and the Australian Guide to Healthy Eating (15, 16). Similar weight loss can be achieved with diets of different macronutrient content (17, 18). Involving a multidisciplinary team, such as an accredited practicing dietitian, exercise physiologist, lifestyle coach or psychologist, should be considered. For some individuals, established commercial programs may be appropriate (19).

1.1 Reducing energy intake

The following are options for reducing energy intake and achieving an energy deficit:

1.1.1 Reduced Energy Diet (RED)

An RED aims to produce a modest energy deficit of 2000 - 4000 kJ/day (480 - 960 kcal/day). This can be achieved by encouraging the intake of vegetables, fruit, wholegrains, legumes, nuts, seeds, lean meat, poultry, fish, eggs and low-fat milk, cheese and yogurt and minimising the intake of discretionary foods.

1.1.2 Low Energy Diet (LED)

An LED aims to reduce total daily energy intake to 4200 - 5000 kJ (1000 - 1200 kcal) for which a more prescriptive diet is needed. Specific meal plans can be provided, or prepared low energy meals can be obtained from commercial providers. LEDs can also be achieved by substituting one or two meals with one or two specially formulated meal replacements.

1.1.3 Very Low Energy Diet (VLED)

For individuals who have not responded to a RED/LED, either a VLED or addition of weight reducing pharmacotherapy (see section 2) should be advised. A VLED aims to reduce energy intake to less than 3300 kJ/day (800 kcal/day) by substituting meals with formulated meal replacements. VLEDs can be an initial weight loss strategy when supervised lifestyle interventions have been unsuccessful in reducing weight or when rapid weight loss is required (eg prior to bariatric or general surgery), particularly in patients with super-obesity (BMI

> 50 kg/m²). VLEDs are low in carbohydrate, inducing mild ketosis, which has an anorexic effect, after 2-3 days.

VLED in practice

VLEDs are often recommended for up to 12 weeks but can be continued for 6-12 months under careful supervision (20), depending on baseline and target weight. Recent studies (21-23) have demonstrated that these diets can be effectively prescribed in primary care with excellent outcomes. Participants achieved a mean weight loss of approximately 10 to 14.5% (initial body weight) after an eight-to-20 week VLED. At 1 year metabolic parameters improved in all the studies. In DiRECT (23), which enrolled only subjects with type 2 diabetes (of up to six years' duration), 46% were in diabetes remission at 12 month and 36% were still in remission at 2 years (24). In DiRECT-Aus, an open label single arm intervention trial conducted in 25 primary care practices in NSW, at 12 months 56% of people with type 2 diabetes of up to 6 years' duration achieved remission with a VLED (25).

Regular clinical review is essential and should occur at least monthly. Individuals may follow a partial or a complete VLED regimen. The partial regimen is more palatable and is based on two meal replacements per day (typically breakfast and lunch) and 1 serve of lean protein (usually for dinner) with vegetables (Appendix 3). A teaspoon of olive oil should be added to induce contraction of the gall bladder and reduce the risk of gallstone formation. The complete VLED regimen is based on 3 meal replacements per day, plus vegetables. The choice of the program (partial *vs* complete) depends on the target weight and the individual's ability to tolerate the VLED. Detailed instructions on how to prescribe a VLED are available here (26).

1.1.3.1 Contraindications to VLED

- Pregnancy or lactation
- Severe psychological condition (e.g. unstable anxiety disorders, major depression), alcoholism or drug dependence
- Recent myocardial infarction, cerebrovascular event or unstable angina
- Porphyria
- Age > 65 years (use with caution as there are limited safety data)

1.1.3.2 Special Groups

- ***Diabetes on insulin or sulphonylureas:*** Doses of sulphonylurea or insulin should be reduced by 50% on commencement of the VLED. Subsequent dosage adjustments are based on frequent self-monitoring of blood glucose. Treatment of hypoglycaemia with carbohydrate takes precedence over the diet.
- ***Diabetes on SGLT2i:*** Monitor ketones or consider stopping SGLT2i while using a VLED to reduce risk of euglycaemic ketoacidosis.

- **Chronic kidney disease:** Individuals with an estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73m² need closer supervision, especially if eGFR < 30 mL/min/1.73m².
- **Taking Warfarin:** Vegetable intake is often increased during a VLED and this may alter the international normalised ratio (INR). Individuals on warfarin should be instructed to test INR one week after commencing the VLED in case the warfarin dose requires adjustment. The absolute quantity of green vegetables is not the issue, rather the level of intake must be kept constant throughout the VLED.

1.2 Increasing energy expenditure

Regular physical activity is essential for well-being and to address obesity-related complications. All adults are recommended to follow the guidelines for physical activity (27, 28). Physical activity (particularly anaerobic-resistance exercise) can protect and improve muscle mass and strength and thus may prevent sarcopenia. Aerobic exercise improves cardiovascular fitness. People with musculoskeletal problems may need alternative forms of exercise, such as water-based activities. Individuals with cardiovascular or respiratory disease may also need a gentler regimen. These people may benefit from seeing an exercise physiologist or engaging in community-based programs.

2. Pharmacotherapy (Table 1)

Anti-obesity pharmacotherapy may be useful in assisting with the initial weight loss and may be combined with a RED or LED if these approaches have not been successful. Pharmacotherapy can also help with weight loss maintenance after a VLED or the prevention of weight regain regardless of the approach for the initial weight loss. While weight loss pharmacotherapy will usually be required long-term, data on its long-term safety and effectiveness are limited. Only five medications have been approved by the Australian Therapeutic Goods Administration (TGA) for the treatment of obesity: phentermine, orlistat, liraglutide, naltrexone/bupropion and semaglutide (2.4 mg).

2.1 TGA approved pharmacotherapy

Phentermine (Duromine®, Metermine®, Phentermine Juno®)

Phentermine is a centrally acting adrenergic agonist that suppresses appetite (29, 30). Duromine and Metermine are delivered in a slow release resin complex, while Phentermine Juno is in a hydrophobic wax matrix that contains hydrophylic release modifiers that are digested in the gastro-intestinal tract. Side effects include tachycardia, hypertension, insomnia and dry mouth. Phentermine should not be used with anti-depressant drugs or in individuals with coronary artery disease, arrhythmias or uncontrolled hypertension because of its cardiac stimulant actions. Phentermine is registered for short-term use (3 months) as an adjunct to lifestyle management of obesity. In conjunction with a hypocaloric diet, weight reduction of 5-10% is achieved with 12 weeks of

treatment (29). The longer-term safety of phentermine has been evaluated in the SEQUEL trial in which combined phentermine (maximum dose 15 mg) and topiramate (Qysmia) was continued for 2 years (31).

Orlistat (Xenical®)

Orlistat inhibits pancreatic and gastric lipase, reducing fat absorption by 30%. Side effects include steatorrhea, oily spotting and flatulence if more than 30 g of fat is consumed daily. Potential complications of its long-term use are deficiencies of fat-soluble vitamins A, D, E and K and the development of oxalate kidney stones. In conjunction with lifestyle intervention, results from the XENDOS study reported a weight loss of 10.6 kg at 1 year and 5.8 kg at 4 years in the orlistat group compared to 6.2 kg and 3.0 kg weight loss in the placebo group, respectively. A 37% reduction in progression to type 2 diabetes was also reported in the orlistat group (32). The safety of orlistat has been established over the 4 years of the XENDOS study.

Liraglutide 3 mg (Saxenda®)

Liraglutide is a once-daily glucagon-like peptide-1 receptor agonist (GLP1-RA) that slows gastric emptying and suppresses appetite. The starting dose is 0.6 mg daily by subcutaneous injection, with a weekly increment of 0.6 mg to minimise gastrointestinal side effects. While the recommended and maximal daily dose is 3.0 mg, some individuals may achieve good weight loss with lower doses. Since liraglutide is a blood glucose-lowering medication, other glucose-lowering medications may need adjustment in people with diabetes. Common adverse effects include nausea, vomiting, constipation and diarrhoea, which can be reduced by slowing the dose escalation schedule. There is an increased risk of gallstones and cholecystitis requiring cholecystectomy independent of weight loss. While very rare, GLP1-RAs are associated with an increased risk of pancreatitis (29). Results from the SCALE trial, a 56-week placebo-controlled trial, demonstrated that treatment with 3 mg liraglutide in combination with lifestyle intervention, resulted in a mean weight loss of 8% compared to 2.6% in the placebo group (33). While the LEADER Study showed that liraglutide 1.8 mg daily in people with type 2 diabetes improved cardiovascular outcomes compared with placebo (34), no differences in cardiovascular risk were shown in a post hoc analysis of data from 5 trials (n = 5908 participants) using the 3 mg daily dose compared to placebo (35).

Naltrexone and bupropion (Contrave®)

Naltrexone 8 mg and bupropion 90 mg are available in an extended release (ER) tablet formulation. Although the precise mode of action of naltrexone and bupropion as anorectic agents is unknown, they are thought to act in both the hypothalamic hunger system and the mesolimbic reward centres of the brain (36). The main side effects of this combination therapy are nausea and vomiting (37), hence the recommendation to gradually escalate the dose starting with one tablet daily and increasing the daily dose by one tablet per week to two tablets twice daily. The COR-I phase 3 clinical trial, in 1742 participants with BMI of 30 - 45 kg/m² or BMI 27 - 45 kg/m² with dyslipidaemia or hypertension, showed naltrexone 32 mg/bupropion 360 mg

ER, when added to a hypocaloric diet and exercise, resulted in an average weight loss of 6.1% versus 1.3% with placebo ($p < 0.001$) at 56 weeks (38). A study in people with type 2 diabetes and obesity found that naltrexone 32 mg/bupropion 360 mg ER treatment resulted in a 5% decrease in body weight from baseline (vs 1.8% placebo, $p < 0.001$) and a 0.6% reduction in HbA1c compared to 0.1% on placebo ($p < 0.001$) (39).

Semaglutide (Ozempic®, Wegovy®)

Semaglutide is a once weekly GLP-1 approved for the management of type 2 diabetes, at a maximum dose of 1 mg weekly (Ozempic®), and for obesity at a maximal dose of 2.4 mg once weekly (Wegovy®). The recommended starting dose is 0.25 mg once weekly with monthly increments to 2.4 mg weekly. In a study of adults with obesity or BMI ≥ 27 kg/m² with at least one weight-related condition treated with semaglutide 2.4 mg once weekly, participants achieved a mean weight loss of 14.9% body weight. Eighty-six percent of participants on active treatment had $\geq 5\%$, 69% $\geq 10\%$ and 50% had $\geq 15\%$ body weight loss (40). As liraglutide (33), semaglutide lowers blood pressure, improves the lipid profile, reduces glucose levels, reduces C-reactive protein and improves physical functioning (40). In people without diabetes but with obesity and pre-existing cardiovascular disease, 2.4 mg semaglutide once weekly has been shown to reduce the risk of death from cardiovascular causes, nonfatal myocardial infarction or nonfatal stroke by 20% over a median of 3.25 years (41). In people with type 2 diabetes and chronic kidney disease, semaglutide 1 mg once weekly reduced the risk of clinically important renal outcomes and death from cardiovascular disease (42). In a 72-week phase 2 study of patients (n=320) with metabolic dysfunction associated steatohepatitis (MASH) treated with semaglutide 0.1, 0.2 or 0.4 mg daily there was a reduction in steatosis but not fibrosis (43). However, in a 48-week smaller trial (n=71) of semaglutide 2.4 mg once weekly in people with biopsy confirmed MASH and compensated cirrhosis there was no significant improvement in fibrosis or resolution of MASH compared to placebo (44). The side effects are similar to those of liraglutide apart from an additional adverse effect of worsening diabetic retinopathy in people with a pre-existing history of this condition. However, there are no GLP-1 receptors in the eye so it is most likely that any worsening of retinopathy is related to the rapid fall in glucose levels with these agents (45). Patients with a history of diabetic retinopathy need close monitoring for progression of retinopathy.

2.2 Off-label pharmacotherapy

Other medications not approved by TGA for weight loss therapy are being used off-label in Australia for the management of obesity by practitioners experienced in obesity care.

Topiramate

Topiramate is an anticonvulsant medication for difficult to control epilepsy and migraine. This medication has a powerful appetite suppressant effect, resulting in weight loss. A meta-analysis of 10 randomised

controlled trials of at least 16 weeks duration concluded that the topiramate-treated group had additional weight loss of 5.3 kg compared with the placebo-treated group (46). Effective doses are between 25 and 100 mg per day. Side effects include depression, difficulty concentrating, paraesthesia (common) and closed angle glaucoma (rare). Topiramate can be considered for use “off label” in people with obesity who have not responded to other weight loss medications or in whom other therapies are contraindicated.

Combined low dose phentermine and topiramate

The combination of these two monotherapies is useful for weight management. An Australian study investigating the safety, tolerability and efficacy of the combination showed that in about 40% of people the combination was not well tolerated, predominantly due to topiramate (25 mg mane) side effects. In those who tolerated the combination, the 10% weight loss achieved with a VLED was maintained over a mean duration of pharmacotherapy of 10 months. A group of people who continued phentermine-topiramate for 22 months had a further mean weight loss of 6.7 kg (47).

Tirzepatide (Mounjaro®)

Tirzepatide, a single molecule dual GLP-1 and glucose-dependent insulinotropic peptide (GIP) co-agonist has been TGA-approved for type 2 diabetes, but not obesity. It has been approved for obesity management in the USA, Europe and the UK. In a 72 week trial in people with obesity tirzepatide 5 mg, 10 mg and 15 mg weekly resulted in 15.0%, 19.5% and 20.9% weight loss respectively (49). Tirzepatide has also been shown to reduce blood pressure, improve lipids and reduce glucose and CRP levels (49). In a 52-week phase 2 trial of people with MASH and moderate to severe fibrosis, tirzepatide resulted in greater resolution of MASH without worsening of fibrosis (50). In patients with moderate to severe obstructive sleep apnoea, tirzepatide 10-15 mg once weekly reduced the apnoea-hypopnoea index, hypoxia, systolic blood pressure, weight and hsCRP and improved sleep-related patient-reported outcomes (51). The side effect profile of tirzepatide is similar to other GLP-1 agonist based therapies – these include gastrointestinal effects (nausea, vomiting, constipation and diarrhoea) and increased risk of acute pancreatitis.

3. Bariatric surgery

Bariatric surgery remains the most efficacious weight loss intervention for the treatment of obesity. Bariatric surgery should be considered as part of a comprehensive treatment delivered by a multidisciplinary team including primary care practitioners, physicians, surgeons, dietitians and psychologists. The potential benefits of surgery need to be assessed for each individual by suitably trained and experienced practitioners and balanced against the individual risk profile. Components of successful bariatric surgery care include an informed patient, tailored operation, optimisation of health prior to surgery, committed multi-professional team care and long-term follow up. All individuals considered for bariatric surgery need a careful risk to benefit assessment and optimisation of health prior to surgery. Not all individuals in whom surgery is a potential treatment option will

be suitable for surgery, especially if they have multiple and advanced complications. These people should be referred to multidisciplinary tertiary institutions for ongoing care. A detailed description of available bariatric surgical procedures is discussed in the 2013 National Health and Medical Research Council (NHMRC) Guidelines (4).

The NHMRC clinical practice guidelines for the management of overweight and obesity (4) state that, taking into account the individual situation, bariatric surgery may be considered for adults with:

- BMI > 40 kg/m²;
- BMI > 35.0 - 39.9 kg/m² and comorbidities that may improve with weight loss; or
- BMI > 30.0 – 34.9 kg/m² who have poorly controlled type 2 diabetes and are at increased risk of cardiovascular disease.

The Australian Diabetes Society endorsed the 2nd Diabetes Surgery Summit meeting guidelines (52) on metabolic surgery as a treatment option for individuals with type 2 diabetes, which state that metabolic bariatric surgery is recommended for individuals with:

- BMI ≥ 40 kg/m² regardless of the level of glycaemic control or complexity of glucose lowering regimens; or
- BMI 35.0–39.9 kg/m² with inadequate glycaemic control despite lifestyle and optimal medical therapy.

Metabolic bariatric surgery should be considered in individuals with BMI 30.0–34.9 kg/m² with inadequately controlled hyperglycaemia despite optimal medical treatment by either oral or injectable medications (including insulin).

Section 3: Algorithm for the management of obesity

1. Baseline assessment

1.1 Categorise the obesity

The algorithm considers two categories of BMI: BMI 30-40 kg/m² and BMI >40 kg/m² and the presence of abdominal obesity. While BMI has some limitations, it remains a useful measure for guiding management decisions. The association between BMI and fat distribution varies according to a number of factors including race such that South Asian, South Eastern Asian, Eastern Asian and Australian Aboriginal & Torres Strait Islander populations are characterised by higher adiposity for a given BMI (53, 54), and for these populations the equivalent categories are BMI 27.5-37.5 and BMI >37.5 kg/m² (**Table 2**). Similarly, there are racial differences for the cut offs for abdominal obesity, but the categories also differ according to sex (**Table 2**). Although abdominal obesity tends to increase with BMI it is important to be aware that some people with a BMI in the normal or overweight range may have abdominal obesity. This occurs particularly in the elderly in association with sarcopenia. While the gold standard for measurement of abdominal fat is with computed tomography or magnetic resonance imaging, measurement of the waist circumference provides a very good estimate of metabolic disease risk. Currently there is no consensus on the optimal protocol for this measure with data supporting its measurement either at the midpoint between the lowest rib and the iliac crest; the level of the iliac crest; or, the narrowest point of the torso. Consistency of the method used is important as values differ with each technique (55).

1.2 Assess for obesity-related complications

Assessment of obesity-related complications (Appendix 2) provides additional guidance for the treatment pathway. Complications are grouped under medical, psychological and those resulting in physical limitations. The majority of individuals with a BMI > 40 kg/m² will have obesity-related complications. Some complications are more responsive to weight loss including type 2 diabetes, metabolic associated fatty liver disease (MAFLD), polycystic ovary syndrome (PCOS), hypogonadism and hypertension and will benefit most from weight loss treatments (56). Although there are various systems to stage obesity, this algorithm adopts a simplified approach to obesity staging. Given the chronic progressive nature of obesity, assessment of obesity-related complications needs to be ongoing and repeated at regular intervals.

2. Set weight loss targets

The algorithm provides a weight loss target of 10 - 15 % in individuals with BMI 30 - 40 kg/m² and > 15% in those with BMI >40 kg/m². These targets are indicative only and personalised weight loss targets should be set

between the clinician and the person with obesity. Lesser degrees of weight loss can still have medical benefits, especially in diabetes prevention.

3. Using Health Services

The management of obesity requires a multidisciplinary team and a long-term chronic disease approach. The algorithm suggests that primary care is ideally placed to manage the care of people with BMI 30- 40 kg/m² without obesity-related complications. Shared care arrangements between primary care physicians and specialist services should be considered for people with BMI 30-40 kg/m² with complications or BMI >40 kg/m² without complications. Individuals with BMI >40 kg/m² with complications should be considered for referral to specialist care.

4. Weight loss strategies (Figure 1)

4.1 Management of individuals with BMI 30-40 kg/m² without complications

Supervised lifestyle intervention is the mainstay of management for individuals with BMI 30-40 kg/m² without established complications. Initially this includes a reduced energy diet or a low energy diet, combined with a program to increase regular physical activity. Referral to multidisciplinary care such as an accredited practicing dietitian, exercise specialist (ie exercise physiologist, physical educator, sports physician or physiotherapist), lifestyle coach or an established commercial weight loss program can be considered. If weight loss is insufficient or weight regain is experienced, a VLED can be considered or a RED, LED or VLED can be combined with pharmacotherapy.

4.2 Management of individuals with BMI 30-40 kg/m² with obesity-related complications or BMI > 40 kg/m² without complications

This group of individuals requires more intensive interventions. Three main options are available and the choice of therapies should be guided by previous weight loss interventions and response.

1. VLED may be an initial option for individuals who have not tried this previously and are willing to use meal replacements. If effective in achieving adequate weight loss, the meal replacements can be reduced, and the diet can be replaced with a weight maintenance diet. If weight is regained the VLED can be reintroduced. A VLED can also be used in the short-term to reduce liver volume prior to abdominal surgery and to reduce surgical complications.

2. Pharmacotherapy can be used in combination with a RED or LED, or considered in individuals who do not have an adequate initial response to the VLED, are unwilling to follow a VLED or regain weight once the VLED is relaxed.

3. Bariatric surgery is an option for individuals who do not respond to the VLED plus pharmacotherapy or

have previously tried this approach without satisfactory weight loss, or who have type 2 diabetes.

4.3 Management of individuals with BMI > 40 kg/m² with obesity-related complication

These individuals should be considered for intensive medical interventions and are best managed in specialist care. The combination of a VLED and pharmacotherapy should be considered as initial treatment. Subsequent management is guided by response. Bariatric surgery should be recommended, especially in the presence of weight responsive complications or when previous interventions have not resulted in sustainable weight loss or health improvements.

Section 4: Special clinical situations

1. Age

The nadir for lowest mortality associated with weight is not constant and varies with age, ethnicity and the presence of other disease. Obesity defined by BMI ≥ 30 kg/m² does not appear to carry the same mortality risk in older adults (>65 years). With ageing, lowest mortality is associated with a BMI higher than the normal range (57). The mortality risk associated with weight loss (including intentional) increases with age, generating an altered risk to benefit ratio. Healthy ageing should therefore focus on lifestyle, quality nutrition and physical activity to improve cardiovascular fitness, optimise functional independence and quality of life.

1.1 Young adults (18-35 years)

Adolescence and early adulthood are often associated with a decrease in physical activity and rapid weight gain. Particular attention should be given to early detection and management of individuals on a positive weight trajectory and with high cardio-metabolic risk. Interventions should be initiated early to prevent weight gain, complications and end-organ damage.

1.2 Older adults (>65 years)

Research in the elderly is scant. There is no clear BMI target in this age group. With aging there is a reduction in muscle mass, height loss and an increase in abdominal obesity, none of which are accounted for in the BMI. The main goal in older adults with obesity is to improve physical function and minimise the impact of obesity-related complications. In individuals with BMI 30-40 kg/m² and BMI ≥ 40 kg/m² without complications, the aims of treatment are to maintain health and physical function, prevent weight gain and generate a more moderate intentional weight loss. In individuals with a BMI ≥ 40 kg/m² with obesity-related complications, more intensive therapies are indicated, but maintaining physical function, favourable body composition and quality nutrition may require specific lifestyle programs. When engaging older adults in intensive therapies, cardiovascular fitness should be considered.

2. Pregnancy

Obesity during pregnancy is associated with an increased risk of obstetric complications, hypertension, gestational diabetes, fetal macrosomia, birth defects and increased risk of obesity in the offspring. In women with BMI >30 kg/m², a total weight gain not exceeding 9 kg is recommended (58, 59). Gestational weight gain (GWG) should be closely monitored in women with obesity, as well as in women with a healthy pre-pregnancy weight, as excessive GWG is associated with poorer maternal and neonatal outcomes. In women with excessive weight gain, weight management strategies should be implemented. If indicated, bariatric surgery should be performed at least 12 to 18 months prior to pregnancy. All medications available for weight loss in Australia are category B for use in pregnancy. Semaglutide, tirzepatide and topiramate are category D for use in pregnancy in Australia.

Section 5: Implementation in primary care and linkage with specialist care

Obesity is a chronic disease that requires lifelong management. Primary care physicians play a key role in identifying individuals with obesity and implementing appropriate interventions to support weight loss and prevent weight regain. Primary care should assist individuals to access obesity support services that address the complexity surrounding obesity. General practices can strategically improve services for these individuals and carers through personal education, education of practice staff, development of obesity focused practice resources (e.g. weighing scales to monitor individuals across the obesity ranges as a matter of routine) and development of a referral network of specialist services or multidisciplinary teams able to manage obesity and weight-related complications. Practice systems that exist for chronic disease management can be used for weight management such as recall systems and reviews focusing on high risk individuals with complications and those who have had bariatric surgery (28, 60-63).

APPENDIX 1: Summary of current guidelines for management of overweight and obesity

Guideline	Main Recommendations
American Association of Clinical Endocrinologists and American College of Endocrinology ¹	<ul style="list-style-type: none"> • No complications: Lifestyle/behavioural therapy and consider pharmacotherapy if lifestyle alone is not effective • If one or more mild-to-moderate complications: lifestyle/behaviour therapy and consider pharmacotherapy if BMI ≥ 27 kg/m² • If at least one severe complication: lifestyle/behavioural therapy with pharmacotherapy (BMI ≥ 27 kg/m²) and consider bariatric surgery if BMI ≥ 35 kg/m²
American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society ²	<ul style="list-style-type: none"> • High-intensity comprehensive lifestyle intervention (moderate calorie reduction, physical activity, behavioural strategies) • Consider pharmacotherapy as an adjunct if BMI ≥ 30 kg/m² (or ≥ 27 kg/m² with comorbidity) • Consider referral to bariatric surgery as an adjunct if BMI ≥ 40 kg/m² (or ≥ 35 kg/m² with comorbidity)
Obesity Canada Guidelines ³	<ul style="list-style-type: none"> • Medical nutrition therapy delivered by a dietitian and physical activity recommendations • Psychological and behavioural interventions to affect change • Adjunctive pharmacotherapy if BMI ≥ 30 kg/m² (or ≥ 27 kg/m² with complications) • Consider bariatric surgery if BMI ≥ 40 kg/m² (or ≥ 35 kg/m² with at least one complication)
Saudi Clinical Practice Guideline ⁴	<ul style="list-style-type: none"> • Lifestyle intervention including diet and physical activity, delivered through individualised counselling • Intensive lifestyle modification in those at higher risk of comorbidities • Preferred pharmacotherapy is metformin and orlistat • Bariatric surgery should be considered in those with BMI ≥ 40 kg/m² (or ≥ 35 kg/m² with comorbidities)
European Guidelines ⁵	<ul style="list-style-type: none"> • Nutrition and physical activity intervention delivered using cognitive behavioural therapy • Pharmacotherapy as an adjunct if BMI ≥ 30 kg/m² (or ≥ 27 kg/m² with comorbidities) • Consider bariatric surgery if other attempts unsuccessful and BMI ≥ 40 kg/m² (or ≥ 35 kg/m² with comorbidities, or ≥ 30 kg/m² with type 2 diabetes)
NICE: National Institute for Health and Care Excellence Guideline ⁶	<ul style="list-style-type: none"> • Referral to weight management programs which include behaviour change strategies delivered by trained professionals • Provide advice on increasing physical activity, improving diet quality, and reducing calorie intake • Consider pharmacotherapy if lifestyle intervention unsuccessful • Consider bariatric surgery if BMI ≥ 40 kg/m² (or ≥ 35 kg/m² with comorbidities) and other interventions unsuccessful

1. Garvey WT, et al. American Association of Clinical Endocrinologists and American College of Endocrinology Comprehensive Clinical Practice Guidelines for Medical Care of Patients with Obesity. *Endocr Pract.* 2016;22 Suppl 3:1-203. 2. Jensen MD, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *J Am Coll Cardiol.* 2014;63(25 Pt B):2985-3023. 3. Wharton S, et al. Obesity in adults: a clinical practice guideline. *CMAJ.* 2020;192(31):E875-E91. 4. Alfadda AA, et al. The Saudi clinical practice guideline for the management of overweight and obesity in adults. *Saudi Med J.* 2016;37(10):1151-62. 5. Yumuk V, et al. European Guidelines for Obesity Management in Adults. *Obes Facts.* 2015;8(6):402-24. 6. (NICE) National Institute for Health and Care Excellence. Obesity: identification, assessment and management – clinical guideline. 2014; <https://www.nice.org.uk/guidance/cg189>

APPENDIX 2: Screening and assessment of obesity-related complications

Type 2 diabetes mellitus	Glycated haemoglobin (HbA1c) and fasting glucose to screen for diabetes
Cardiovascular disease	Electrocardiogram (ECG), cardiac ultrasound and cardiac risk assessment Referral to cardiology if high cardiovascular risk, presence of cardiac symptoms or abnormal ECG or cardiac ultrasound
Metabolic associated fatty liver disease	Liver function tests Consider abdominal ultrasound fibro-scan if liver enzymes elevated, particularly if associated with hyperglycaemia, specifically to detect fibrotic liver disease
Gastro-oesophageal reflux disease	If presence of severe heartburn or acid reflux consider referral for endoscopy
Obstructive sleep apnoea (OSA)	Screening questionnaire (eg STOP-BANG) to identify those at risk for OSA Referral to sleep specialist if STOP-BANG score ≥ 3
Asthma	Underdiagnosed – wheezing or short of breath – refer to respiratory physician Undertreated - asthma plan review
Idiopathic Intracranial Hypertension	Headaches, women aged 20-50 at greatest risk. Assess for papilloedema and visual disturbance. Refer to neurologist for further investigation
Arthralgia	Usually from degenerative joint disease but consider rheumatoid arthritis as risk increased with obesity If neurological signs present imaging is necessary
Lymphoedema	Optimal treatment is compression after ensuring peripheral circulation is normal; Often misdiagnosed as cardiac failure, will worsen with diuretics Consider referral to occupational therapist for compression bandaging if very severe
Reproductive hormonal dysfunction	Central hypogonadism (low testosterone and low gonadotrophins) common in males, can lead to reduced libido, depression and may exacerbate cardiovascular risk Central hypogonadism can occur in females, polycystic ovary syndrome (PCOS) much more common; both can result in amenorrhoea and reduced fertility, but PCOS also has features of androgen excess

Disordered eating	Enquire about binge eating, purging or night eating Referral to dietitian or clinical psychologist with expertise in this area if suggestive symptoms
Depression	Screening questionnaire (eg K10 screening tool for anxiety and depression or Patient Health Questionnaire (PHQ)-9 Referral to clinical psychologist or psychiatrist if high risk identified

APPENDIX 3: Examples of foods allowed and to avoid while on a VLED

Allowed			Avoid
Low starch vegetables			
Alfalfa sprouts Asparagus Bean Sprouts Bok Choy Broccoli Brussels sprouts Cabbage Capsicum Carrots Cauliflower	Celery Cucumber Eggplant Endive Green beans Konjac noodles Lettuce (all types) Leeks Mushrooms Onions	Radishes Shallots Silverbeet Snow peas Spinach Squash Tomatoes Watercress Zucchini	Corn Green peas Legumes Lentils Potatoes Sweet potato Parsnip Pumpkin Turnip
Soups			
Stock cubes Bonox® (in moderation)	Vegetable soups made from allowed vegetables Miso soup		All other soups
Sauces and condiments			
Lemon & lime juice Vinegar Worcestershire sauce Tabasco sauce	Soy sauce Chili Diet, oil free or fat free salad dressings	Mustard Tomato paste	Cream High calorie simmer sauces and dressings
Herbs and spices			
All spice Basil Celery flakes Chili Chives Cinnamon Cloves Coriander Cumin	Curry powder Dill Fennel Garlic Ginger Lite salt Mint Mustard seed Nutmeg	Oregano Paprika Parsley Pepper Rosemary Sage Tarragon Thyme Turmeric	
Others			
Low joule jellies Artificial sweeteners Tea, coffee, diet drinks			Fruit and fruit juice Alcohol Milk Sugary drinks Flavoured mineral water Discretionary foods

APPENDIX 4: Summary of weight loss interventions¹

Intervention	Summary of effect
<p>Lifestyle change</p>	<p>>10% weight loss in few studies; weight loss difficult to maintain for many individuals.</p> <p>Study results:</p> <ul style="list-style-type: none"> • Dietary change: average weight loss 3-5 kg at 12 months; 0 kg at 5 years • Dietary change and exercise: average weight loss 5-10 kg at 12 months; 0-3 kg at 5 years • Lifestyle change and psychological intervention: average weight loss 3-4 kg at 5 years
<p>Combined lifestyle change and pharmacotherapy</p>	<p>>10% weight loss in some but not all studies; weight loss maintained > 2 years in some participants.</p> <p>Study results:</p> <ul style="list-style-type: none"> • Orlistat and dietary change: average weight loss 6-10 kg at 12 months; 2-3 kg at 5 years • Phentermine and dietary change: average weight loss 6.4 kg 12 weeks (64) • Liraglutide and lifestyle change: average weight loss 8% at 56 weeks (29) and 6% at 3 yr (65) • Naltrexone/bupropion and lifestyle change: average weight loss 6.1% at 56 weeks (32) • Semaglutide (2.4 mg) and lifestyle change: average weight loss 14.9% at 68 weeks (40) • Tirzepatide and lifestyle change: average loss 20.9% with 15 mg dose at 72 weeks (49)
<p>Bariatric surgery with maintained lifestyle changes</p>	<p>>15% weight loss consistently across studies; weight loss likely to be maintained >5 years</p> <ul style="list-style-type: none"> • Laparoscopic adjustable gastric banding: average weight loss 20% at 12 months; 12% at 10 years • Sleeve gastrectomy: average weight loss 25% at 12 months; 16% at 10 years (63) • <i>Roux-en-Y</i> gastric bypass: average weight loss 33% at 12 months; 30% at 10 years

¹ Adapted from the 2013 NHMRC table 6.4 (4).

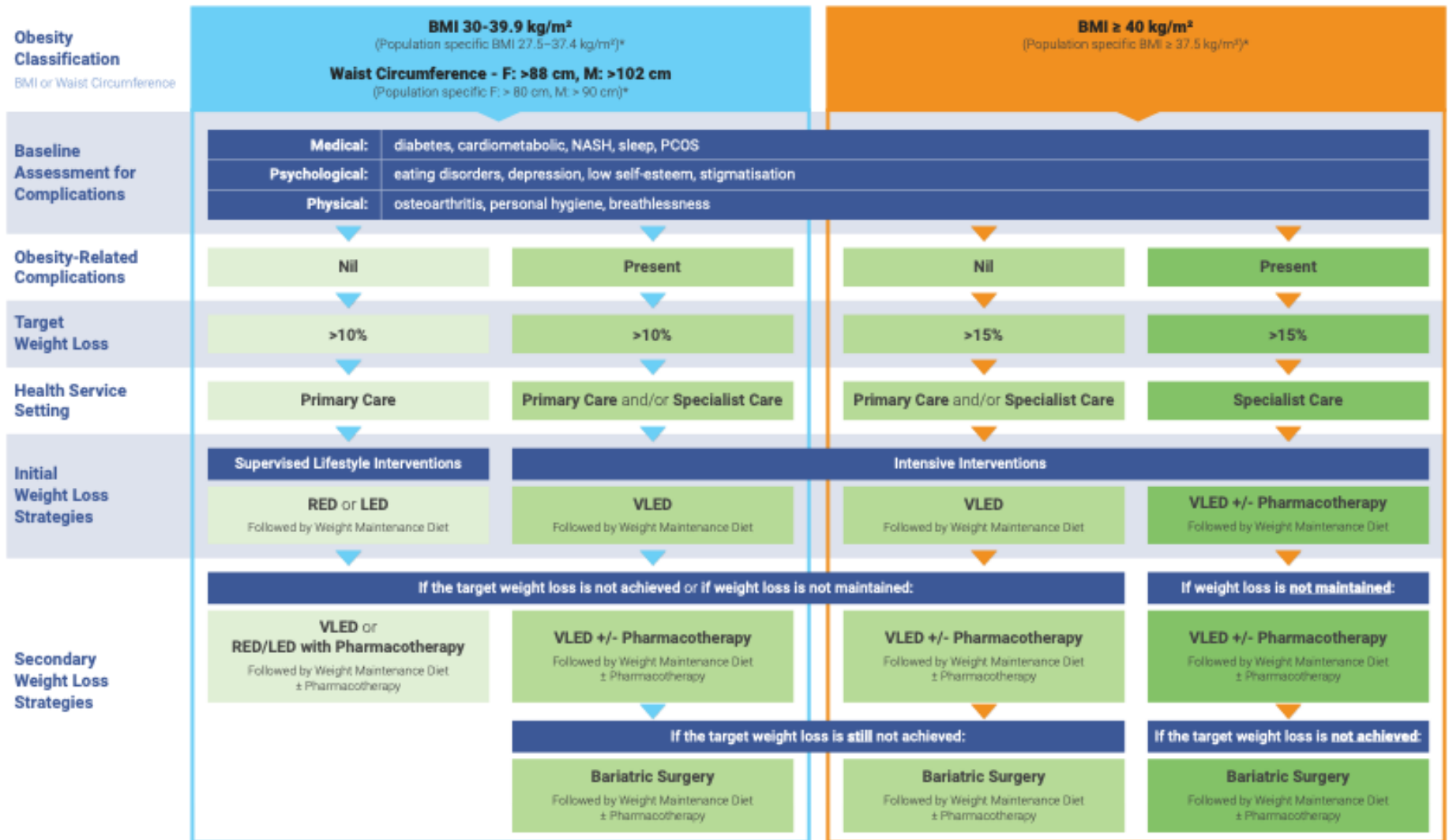
References

1. Statistics ABo. National Health Survey: First Results, 2017-2018. 2018.
2. PriceWaterhouseCooper. Weighing the cost of obesity: A case for action. 2015.
3. Diabetes Prevention Program Research G, Knowler WC, Fowler SE, Hamman RF, Christophi CA, Hoffman HJ, et al. 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet*. 2009;374(9702):1677-86.
4. Council NHaMR. Clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children in Australia. 2013.
5. Bouchard C, Tremblay A, Despres JP, Nadeau A, Lupien PJ, Theriault G, et al. The response to long-term overfeeding in identical twins. *The New England journal of medicine*. 1990;322(21):1477-82.
6. Stunkard AJ, Harris JR, Pedersen NL, McClearn GE. The body-mass index of twins who have been reared apart. *N Engl J Med*. 1990;322(21):1483-7.
7. Montague CT, Farooqi IS, Whitehead JP, Soos MA, Rau H, Wareham NJ, et al. Congenital leptin deficiency is associated with severe early-onset obesity in humans. *Nature*. 1997;387(6636):903-8.
8. Stunkard AJ, Sorensen TI, Hanis C, Teasdale TW, Chakraborty R, Schull WJ, Schulsinger F. An adoption study of human obesity. *The New England journal of medicine*. 1986;314(4):193-8.
9. Vaisse C, Clement K, Guy-Grand B, Froguel P. A frameshift mutation in human MC4R is associated with a dominant form of obesity. *Nat Genet*. 1998;20(2):113-4.
10. Huypens P, Sass S, Wu M, Dyckhoff D, Tschop M, Theis F, et al. Epigenetic germline inheritance of diet-induced obesity and insulin resistance. *Nat Genet*. 2016;48(5):497-9.
11. Ravelli GP, Stein ZA, Susser MW. Obesity in young men after famine exposure in utero and early infancy. *N Engl J Med*. 1976;295(7):349-53.
12. Australia O. Rethink Obesity: A media guide on how to report on obesity. 2015.
13. Fruh SM, Nadglowski J, Hall HR, Davis SL, Crook ED, Zlomke K. Obesity Stigma and Bias. *J Nurse Pract*. 2016;12(7):425-32.
14. Markovic TP, Natoli SJ. Paradoxical nutritional deficiency in overweight and obesity: the importance of nutrient density. *The Medical journal of Australia*. 2009;190(3):149-51.
15. Council NHaMR. Australian Guide to Healthy Eating
<http://www.eatforhealth.gov.au/guidelines/australian-guide-healthy-eating> [
16. Council NHaMR. Australian Dietary Guidelines. 2013.
17. Hu T, Mills KT, Yao L, Demanelis K, Eloustaz M, Yancy WS, Jr., et al. Effects of low-carbohydrate diets versus low-fat diets on metabolic risk factors: a meta-analysis of randomized controlled clinical trials. *Am J Epidemiol*. 2012;176 Suppl 7:S44-54.
18. Sacks FM, Bray GA, Carey VJ, Smith SR, Ryan DH, Anton SD, et al. Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. *The New England journal of medicine*. 2009;360(9):859-73.
19. Gudzone KA, Doshi RS, Mehta AK, Chaudhry ZW, Jacobs DK, Vakil RM, et al. Efficacy of commercial weight-loss programs: an updated systematic review. *Ann Intern Med*. 2015;162(7):501-12.
20. Sumarithran P, Proietto J. Safe year-long use of a very-low-calorie diet for the treatment of severe obesity. *Med J Aust*. 2008;188(6):366-8.
21. Astbury NM, Aveyard P, Nickless A, Hood K, Corfield K, Lowe R, Jebb SA. Doctor Referral of Overweight People to Low Energy total diet replacement Treatment (DROPLET): pragmatic randomised controlled trial. *BMJ*. 2018;362:k3760.
22. Christensen P, Meinert Larsen T, Westerterp-Plantenga M, Macdonald I, Martinez JA, Handjiev S, et al. Men and women respond differently to rapid weight loss: Metabolic outcomes of a multi-centre intervention study after a low-energy diet in 2500 overweight, individuals with pre-diabetes (PREVIEW). *Diabetes, obesity & metabolism*. 2018;20(12):2840-51.
23. Lean ME, Leslie WS, Barnes AC, Brosnahan N, Thom G, McCombie L, et al. Primary care-led weight management for remission of type 2 diabetes (DiRECT): an open-label, cluster-randomised trial. *Lancet*. 2018;391(10120):541-51.
24. Lean MEJ, Leslie WS, Barnes AC, Brosnahan N, Thom G, McCombie L, et al. Durability of a primary care-led weight-management intervention for remission of type 2 diabetes: 2-year results of the DiRECT open-label, cluster-randomised trial. *Lancet Diabetes Endocrinol*. 2019;7(5):344-55.
25. Hocking SL, Markovic TP, Lee CMY, Picone TJ, Gudorf KE, Colagiuri S. Intensive Lifestyle Intervention for Remission of Early Type 2 Diabetes in Primary Care in Australia: DiRECT-Aus. *Diabetes care*. 2023.
26. Proietto J. Obesity and weight management at menopause. *Australian Family Physician*. 2017;46(6):368-70.

27. Government A. More than half of all Australian adults are not active enough. 2014.
28. Practitioners RACoG. Smoking, nutrition, alcohol, physical activity (SNAP): A population health guide to behavioural risk factors in general practice. 2015.
29. Kang JG, Park CY, Kang JH, Park YW, Park SW. Randomized controlled trial to investigate the effects of a newly developed formulation of phentermine diffuse-controlled release for obesity. *Diabetes Obes Metab.* 2010;12(10):876-82.
30. Munro JF, MacCuish AC, Wilson EM, Duncan LJ. Comparison of continuous and intermittent anorectic therapy in obesity. *Br Med J.* 1968;1(5588):352-4.
31. Garvey WT, Ryan DH, Look M, Gadde KM, Allison DB, Peterson CA, et al. Two-year sustained weight loss and metabolic benefits with controlled-release phentermine/topiramate in obese and overweight adults (SEQUEL): a randomized, placebo-controlled, phase 3 extension study. *The American journal of clinical nutrition.* 2012;95(2):297-308.
32. Torgerson JS, Hauptman J, Boldrin MN, Sjostrom L. XENical in the prevention of diabetes in obese subjects (XENDOS) study: a randomized study of orlistat as an adjunct to lifestyle changes for the prevention of type 2 diabetes in obese patients. *Diabetes Care.* 2004;27(1):155-61.
33. Pi-Sunyer X, Astrup A, Fujioka K, Greenway F, Halpern A, Krempf M, et al. A Randomized, Controlled Trial of 3.0 mg of Liraglutide in Weight Management. *The New England journal of medicine.* 2015;373(1):11-22.
34. Marso SP, Daniels GH, Brown-Frandsen K, Kristensen P, Mann JF, Nauck MA, et al. Liraglutide and Cardiovascular Outcomes in Type 2 Diabetes. *The New England journal of medicine.* 2016;375(4):311-22.
35. Davies MJ, Aronne LJ, Caterson ID, Thomsen AB, Jacobsen PB, Marso SP, et al. Liraglutide and cardiovascular outcomes in adults with overweight or obesity: A post hoc analysis from SCALE randomized controlled trials. *Diabetes, obesity & metabolism.* 2018;20(3):734-9.
36. Billes SK, Sinnayah P, Cowley MA. Naltrexone/bupropion for obesity: an investigational combination pharmacotherapy for weight loss. *Pharmacol Res.* 2014;84:1-11.
37. Hong K, Herrmann K, Dybala C, Halseth AE, Lam H, Foreyt JP. Naltrexone/Bupropion extended release-induced weight loss is independent of nausea in subjects without diabetes. *Clin Obes.* 2016;6(5):305-12.
38. Greenway FL, Fujioka K, Plodkowski RA, Mudaliar S, Guttadauria M, Erickson J, et al. Effect of naltrexone plus bupropion on weight loss in overweight and obese adults (COR-I): a multicentre, randomised, double-blind, placebo-controlled, phase 3 trial. *Lancet.* 2010;376(9741):595-605.
39. Hollander P, Gupta AK, Plodkowski R, Greenway F, Bays H, Burns C, et al. Effects of naltrexone sustained-release/bupropion sustained-release combination therapy on body weight and glycemic parameters in overweight and obese patients with type 2 diabetes. *Diabetes Care.* 2013;36(12):4022-9.
40. Wilding JPH, Batterham RL, Calanna S, Davies M, Van Gaal LF, Lingvay I, et al. Once-Weekly Semaglutide in Adults with Overweight or Obesity. *The New England journal of medicine.* 2021;384(11):989.
41. Lincoff AM, Brown-Frandsen K, Colhoun HM, Deanfield J, Emerson SS, Esbjerg S, et al. Semaglutide and Cardiovascular Outcomes in Obesity without Diabetes. *The New England journal of medicine.* 2023.
42. Perkovic V, Tuttle KR, Rossing P, Mahaffey KW, Mann JFE, Bakris G, et al. Effects of Semaglutide on Chronic Kidney Disease in Patients with Type 2 Diabetes. *The New England journal of medicine.* 2024.
43. Newsome PN, Buchholtz K, Cusi K, Linder M, Okanoue T, Ratziu V, et al. A Placebo-Controlled Trial of Subcutaneous Semaglutide in Nonalcoholic Steatohepatitis. *The New England journal of medicine.* 2020.
44. Loomba R, Abdelmalek MF, Armstrong MJ, Jara M, Kjaer MS, Krarup N, et al. Semaglutide 2.4 mg once weekly in patients with non-alcoholic steatohepatitis-related cirrhosis: a randomised, placebo-controlled phase 2 trial. *Lancet Gastroenterol Hepatol.* 2023.
45. Nauck MA, Quast DR, Wefers J, Pfeiffer AFH. The evolving story of incretins (GIP and GLP-1) in metabolic and cardiovascular disease: A pathophysiological update. *Diabetes, obesity & metabolism.* 2021;23 Suppl 3:5-29.
46. Kramer CK, Leitao CB, Pinto LC, Canani LH, Azevedo MJ, Gross JL. Efficacy and safety of topiramate on weight loss: a meta-analysis of randomized controlled trials. *Obesity reviews : an official journal of the International Association for the Study of Obesity.* 2011;12(5):e338-47.
47. Neoh SL, Sumithran P, Haywood CJ, Houlihan CA, Lee FT, Proietto J. Combination phentermine and topiramate for weight maintenance: the first Australian experience. *The Medical journal of Australia.* 2014;201(4):224-6.

48. O'Neil PM, Birkenfeld AL, McGowan B, Mosenzon O, Pedersen SD, Wharton S, et al. Efficacy and safety of semaglutide compared with liraglutide and placebo for weight loss in patients with obesity: a randomised, double-blind, placebo and active controlled, dose-ranging, phase 2 trial. *Lancet*. 2018;392(10148):637-49.
49. Jastreboff AM, Aronne LJ, Ahmad NN, Wharton S, Connery L, Alves B, et al. Tirzepatide Once Weekly for the Treatment of Obesity. *New England Journal of Medicine*. 2022.
50. Loomba R, Hartman ML, Lawitz EJ, Vuppalanchi R, Boursier J, Bugianesi E, et al. Tirzepatide for Metabolic Dysfunction-Associated Steatohepatitis with Liver Fibrosis. *The New England journal of medicine*. 2024.
51. Malhotra A, Grunstein RR, Fietze I, Weaver TE, Redline S, Azarbarzin A, et al. Tirzepatide for the Treatment of Obstructive Sleep Apnea and Obesity. *New England Journal of Medicine*. 2024.
52. Rubino F, Nathan DM, Eckel RH, Schauer PR, Alberti KG, Zimmet PZ, et al. Metabolic Surgery in the Treatment Algorithm for Type 2 Diabetes: A Joint Statement by International Diabetes Organizations. *Diabetes Care*. 2016;39(6):861-77.
53. W. H. O. Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet*. 2004;363(9403):157-63.
54. Piers LS, Rowley KG, Soares MJ, O'Dea K. Relation of adiposity and body fat distribution to body mass index in Australians of Aboriginal and European ancestry. *Eur J Clin Nutr*. 2003;57(8):956-63.
55. Ross R, Neeland IJ, Yamashita S, Shai I, Seidell J, Magni P, et al. Waist circumference as a vital sign in clinical practice: a Consensus Statement from the IAS and ICCR Working Group on Visceral Obesity. *Nature reviews Endocrinology*. 2020;16(3):177-89.
56. Ryan DH, Yockey SR. Weight Loss and Improvement in Comorbidity: Differences at 5%, 10%, 15%, and Over. *Curr Obes Rep*. 2017;6(2):187-94.
57. Dixon JB, Egger GJ, Finkelstein EA, Kral JG, Lambert GW. 'Obesity paradox' misunderstands the biology of optimal weight throughout the life cycle. *Int J Obes (Lond)*. 2015;39(1):82-4.
58. In: Rasmussen KM, Yaktine AL, editors. *Weight Gain During Pregnancy: Reexamining the Guidelines*. The National Academies Collection: Reports funded by National Institutes of Health. Washington (DC)2009.
59. NSW GH. Weight gain during pregnancy
http://www.gethealthynsw.com.au/assets/pdf/resources/GHS_Fact_Sheet_Weight_Gain_During_Pregnancy_online_Final.pdf [
60. Allied Health Sciences Section Ad Hoc Nutrition C, Aills L, Blankenship J, Buffington C, Furtado M, Parrott J. ASMBs Allied Health Nutritional Guidelines for the Surgical Weight Loss Patient. *Surg Obes Relat Dis*. 2008;4(5 Suppl):S73-108.
61. Grima M, Dixon JB. Obesity--recommendations for management in general practice and beyond. *Aust Fam Physician*. 2013;42(8):532-41.
62. Mechanick JI, Youdim A, Jones DB, Garvey WT, Hurley DL, McMahon MM, et al. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient--2013 update: cosponsored by American Association of Clinical Endocrinologists, the Obesity Society, and American Society for Metabolic & Bariatric Surgery. *Endocr Pract*. 2013;19(2):337-72.
63. Welbourn R, Dixon J, Barth JH, Finer N, Hughes CA, le Roux CW, et al. NICE-Accredited Commissioning Guidance for Weight Assessment and Management Clinics: a Model for a Specialist Multidisciplinary Team Approach for People with Severe Obesity. *Obes Surg*. 2016;26(3):649-59.
64. Fothergill E, Guo J, Howard L, Kerns JC, Knuth ND, Brychta R, et al. Persistent metabolic adaptation 6 years after "The Biggest Loser" competition. *Obesity (Silver Spring)*. 2016;24(8):1612-9.
65. le Roux CW, Astrup A, Fujioka K, Greenway F, Lau DCW, Van Gaal L, et al. 3 years of liraglutide versus placebo for type 2 diabetes risk reduction and weight management in individuals with prediabetes: a randomised, double-blind trial. *Lancet*. 2017;389(10077):1399-409.

AUSTRALIAN OBESITY MANAGEMENT ALGORITHM



BMI = body mass index, LED = low energy diet, NASH = non-alcoholic steatohepatitis, PCOS = polycystic ovary syndrome, RED = reduced energy diet, VLED = very low energy diet

*Out-of-ffs apply to Asian population and recommended for Australian indigenous population.

Table 1 Pharmacotherapy for the treatment of obesity

Drug	Phentermine Duromine® Metermine® Phentermine Juno®	Orlistat Xenical®	Liraglutide Saxenda®	Naltrexone/ Bupropion Contrave®	Semaglutide Wegovy®	Tirzepatide Mounjaro®	Topiramate	Phentermine/ Topiramate
TGA status re weight	Approved	Approved*	Approved	Approved	Approved	Not Approved	Not Approved	Not Approved
TGA status							Approved for migraine/epilepsy	
Available doses	15 – 30 – 40 mg	120 mg	0.6 – 3 mg	Nal 8mg/ Bup 90mg	0.25 – 0.5 – 1.0- 1.7 – 2.4 mg	2.5 – 5 – 7.5 – 10 – 12.5 – 15 mg	25 – 50 – 100 mg	Phen 15 mg Top 12.5 – 25 – 50 – 100 mg
Starting dose	15-30 mg mane	120 mg tds	0.6 mg daily	Nal 8mg/ Bup 90mg mane	0.25 mg once weekly	2.5 mg once weekly	12.5 mg mane	Phen 15 mg mane Top 12.5 mg mane
Dosage form	Tablet	Tablet	Injection	Tablet	Injection	Injection	Tablet	Tablet
Maximal dose	40 mg mane 12-week treatment only	120 mg tds	3.0 mg daily	Nal 16mg/ Bup 180mg bd	2.4 mg once weekly	15 mg once weekly	50 mg bd	Phen 15 mg mane Top 50 mg bd
Contraindications	Uncontrolled hypertension Cardiac disease Glaucoma Pregnancy History of drug abuse MAO inhibitors SSRI use	Anorexia Pregnancy Fat soluble vitamin deficiency Chronic malabsorption syndrome Cholestasis	Hyper-sensitivity to liraglutide or any of its excipients	Hypersensitivity to naltrexone, bupropion or any of the excipients Uncontrolled hypertension Seizure disorder or history of seizures Known CNS tumour Acute alcohol or benzodiazepine withdrawal Anorexia nervosa or bulimia Pregnancy Severe hepatic impairment End stage renal failure MAO inhibitor use	Hypersensitivity to semaglutide or any of its excipients Pregnancy	Hypersensitivity to tirzepatide or any of its excipients Pregnancy	Glaucoma Renal Stones Pregnancy (if used for weight loss)	Uncontrolled hypertension Cardiac disease Glaucoma History of drug abuse MAO inhibitors or SSRI use Depression Renal calculi

Side effects	Hypertension Tachycardia Insomnia Anxiety/ depression Restlessness Dry mouth Diarrhoea Constipation	Steatorrhoea Excessive flatus Fat soluble vitamin deficiency	Nausea Vomiting Diarrhoea Constipation Pancreatitis Cholecystitis	Nausea Vomiting Constipation Dizziness Headache Insomnia Dry mouth Word finding difficulty	Nausea Vomiting Diarrhoea Constipation Pancreatitis Cholecystitis May exacerbate diabetic retinopathy	Nausea Vomiting Diarrhoea Constipation Pancreatitis Cholecystitis	Paraesthesia Confusion Memory loss Glaucoma Renal stones Nausea Vomiting Pancreatitis	Hypertension Tachycardia Insomnia Restlessness Dry mouth Diarrhoea Constipation Paraesthesia Confusion Memory loss Glaucoma Renal stones
Cost of medication				Nal Bup				Phen Top
Dose	15 - 40 mg	120 mg	3 mg	32 mg 360 mg	0.25 – 2.4 wkly	2.5 – 15 wkly	25 mg	15 mg 25 mg
No./day	1	3	1	4 tablets 8/90 mg			1 – 4/day	1 1
Cost/month ¹	\$90 - \$140	\$65-110	\$387	\$250	0.25-1.0 mg \$260 1.7 mg \$380 2.4 mg \$460	2.5/5 mg \$395 7.5/10 mg \$545 12.5/15 mg \$695	\$15	\$90 + \$15

Mane, in the morning; tds, 3 times per day; bd, twice a day; MAO, monoamine oxidase; SSRI, selective serotonin reuptake inhibitors. TGA, Therapeutic Goods Administration.

*Available through the veteran system. ¹Estimated price from pharmacy websites. The estimates of price per month are calculated by dividing the cost by the number of tablets per script, multiplied by the dose/day. Prices listed as July 2024.

Table 2 The classification of weight by BMI and WC

Classification	General population BMI (kg/m²)	Population specific BMI (kg/m²)*	General population WC (cm)	Population specific WC (cm)*
Normal range	18.5 – 24.9	18.5 – 22.9	F < 80 M < 94	
Overweight	25.0 – 29.9	23.0 – 27.49	F 80 – 88 M 94 - 102	
Class I obesity	30.0 – 34.9	27.5 – 32.4	F > 88 M > 102	F > 80 M > 90
Class II obesity	35 – 39.9	32.5 – 37.4		
Class III obesity	≥ 40	≥ 37.5		

F = female, M = male. * Cut-offs apply to Asian population and recommended for Australian indigenous population.