Obesity Research & Clinical Practice xxx (xxxx) xxx

Contents lists available at ScienceDirect

Obesity Research & Clinical Practice

journal homepage: www.elsevier.com/locate/orcp

The Australian Obesity Management Algorithm: A simple tool to guide the management of obesity in primary care☆

Tania P. Markovic [a,b,*], Joseph Proietto [c], John B. Dixon [d], Georgia Rigas [e], Gary Deed [f,g], Jeffrey M. Hamdorf [h], Erica Bessell [b], Nathalie Kizirian [b], Sofianos Andrikopoulos [i], Stephen Colagiuri [b]

[a] Metabolism & Obesity Service, Royal Prince Alfred Hospital, Camperdown, NSW 2050, Australia
[b] Boden Initiative, Charles Perkins Centre, Faculty of Medicine and Health, University of Sydney, NSW 2006, Australia
[c] University of Melbourne, Parkville, VIC 3010, Australia
[d] Iverson Health Innovation Research Institute, Swinburne University of Technology, Hawthorn, VIC 3122, Australia
[e] St George Private Hospital, Kogarah, NSW 2217, Australia
[f] HealthCarePlus Medical Centre, Carindale, QLD 4152, Australia
[g] Monash University, Clayton, VIC 3800, Australia
[h] Medical School, University of Western Australia, Crawley, WA 6009, Australia
[i] Australian Diabetes Society, 145 Macquarie St, Sydney, NSW 2000, Australia

ARTICLE INFO

Keywords:
Obesity
Very low energy diet
Reduced energy diet
Low energy diet
Physical activity
Anti-obesity pharmacotherapy
Bariatric surgery

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/m^2 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 diet (VLED) or pharmacotherapy. For people with BMI 30–40 kg/m^2 or abdominal obesity and complications, or those with BMI > 40 kg/m^2 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/m^2 and complications and they should be referred to specialist care. Their treatment should include a VLED with or without pharmacotherapy and bariatric surgery.

☆ Guidelines prepared by representatives of Australian & New Zealand Obesity Society (ANZOS), Australian Diabetes Society (ADS), Australian & New Zealand Metabolic and Obesity Surgery Society (ANZMOSS) and Royal Australian College of General Practitioners (RACGP).

* Corresponding author at: Metabolism & Obesity Service, Royal Prince Alfred Hospital, Camperdown, NSW 2050, Australia.
E-mail addresses: tania.markovic@sydney.edu.au (T.P. Markovic), j.proietto@unimelb.edu.au (J. Proietto), profjohndixon@gmail.com (J.B. Dixon), drgeorgiarigas@gmail.com (G. Rigas), drgarydeed@outlook.com (G. Deed), jeffrey.hamdorf@uwa.edu.au (J.M. Hamdorf), erica.bessell@sydney.edu.au (E. Bessell), nathalie.kizirian@sydney.edu.au (N. Kizirian), ceo@diabetessociety.com.au (S. Andrikopoulos), stephen.colagiuri@sydney.edu.au (S. Colagiuri).

https://doi.org/10.1016/j.orcp.2022.08.003
Received 11 October 2021; Received in revised form 2 April 2022; Accepted 5 August 2022

1. Guiding principles

1.1. 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.

1.2. 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.

1.3. 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.

1.4. 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.

2. Treatment options for obesity

2.1. 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 programmes may be appropriate [19].

2.2. Reducing energy intake

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

2.2.1. 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.

2.2.2. 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.

2.2.3. Very low energy diet (VLED)

For individuals who have not responded to a RED or 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/m^2). VLEDs are low in carbohydrate, inducing mild ketosis, which has an anorexic effect, after 2–3 days.

2.2.4. 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 10–14.5% initial body weight after an eight-to-20 week VLED. At 12 months metabolic parameters improved in all the studies. In

DiRECT [23], which enroled only subjects with type 2 diabetes (of up to six years' duration), 46% were in remission at 12 months and 36% were still in remission at two years [24].

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 one 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 three meal replacements per day plus vegetables. The choice of the programme (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 [25].

2.2.4.1. 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)

2.2.4.2. Special groups.

- ***Diabetes treated with insulin or sulphonylurea:*** 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.
- ***Chronic kidney disease:*** Individuals with an estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m^2 need closer supervision, especially if eGFR < 30 mL/min/1.73 m^2.
- ***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.

2.3. 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 [26,27]. 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 need a gentler regimen. These people may benefit from seeing an exercise physiologist or engaging in community-based programmes.

2.4. 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 four medications have been approved by the Australian Therapeutic Goods Administration (TGA) for the treatment of obesity: phentermine, orlistat, liraglutide and naltrexone/bupropion.

2.5. TGA approved pharmacotherapy

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

Phentermine is a centrally acting adrenergic agonist that suppresses appetite [28,29]. 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 [28]. 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 [30].

2.5.2. Orlistat (Xenical®)

Orlistat inhibits pancreatic and gastric lipase, reducing fat absorption by 30%. Side effects include steatorrhoea, 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 [31]. The safety of orlistat has been established over the 4 years of the XENDOS study.

2.5.3. 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. Early studies suggested a possible increased risk of pancreatitis but this has not been borne out in large meta-analyses [32]. 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].

2.5.4. 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, 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/m^2 or BMI 27–45 kg/m^2 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 [37]. 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 ($p < 0.001$) [38].

2.6. 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.

2.6.1. 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 [39]. 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.

2.6.2. 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 [40].

2.6.3. Semaglutide (Ozempic®)

Semaglutide is a once weekly GLP1-RA used in type 2 diabetes, at a maximum dose of 1 mg weekly, which results in substantial weight loss. The recommended starting dose is 0.25 mg once weekly with monthly increments to 1 mg weekly. A phase 2 trial suggests 1 mg once weekly is as effective for weight loss as liraglutide 3 mg daily [41]. A number of phase 3 trials have recently been published using doses of 2.4 mg once weekly. In a study in adults with obesity or BMI $\geq$ 27 kg/m^2 with at least one weight-related condition, 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 [42]. The 2.4 mg once weekly dose has received US Federal Drug Administration and European Medicines Agency approval for weight loss. 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. The mechanism of this is unknown. Patients with a history of diabetic retinopathy need close monitoring for progression of retinopathy.

2.7. 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 multiprofessional 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 for multidisciplinary at a tertiary institution 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/m^2;
- BMI > 35.0–39.9 kg/m^2 and comorbidities that may improve with weight loss; or
- BMI > 30.0 – 34.9 kg/m^2 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 [43] on bariatric surgery as a treatment option for individuals with type 2 diabetes, which state that metabolic bariatric surgery is recommended for individuals with:

- BMI $\geq$ 40 kg/m^2 regardless of the level of glycaemic control or complexity of glucose lowering regimens; or
- BMI 35.0–39.9 kg/m^2 with inadequate glycaemic control despite lifestyle and optimal medical therapy.

Bariatric surgery should be considered in individuals with BMI 30.0–34.9 kg/m^2 with inadequately controlled hyperglycaemia despite optimal medical treatment by either oral or injectable medications (including insulin).

3. Algorithm for the management of obesity

3.1. Baseline assessment

3.1.1. Categorise the obesity

The algorithm considers two categories of BMI: BMI 30–40 kg/m^2 and BMI > 40 kg/m^2 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 [44,45], and for these populations the equivalent categories are BMI 27.5–37.5 and BMI > 37.5 kg/m^2 (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

Table 1
Pharmacotherapy for the treatment of obesity.

Drug	Phentermine Duromine® Metermine® Phentermine Juno®	Orlistat Xenical®	Liraglutide Saxenda®	Naltrexone/ bupropion Contrave®	Topiramate	Phentermine-Topiramate	Semaglutide Ozempic®
TGA status re weight	Approved	Approved*	Approved	Approved	Not Approved	Not Approved	Not Approved
TGA status					Approved for migraine/epilepsy		Approved for T2DM (1 mg)
Available doses	15 – 30 – 40 mg	120 mg	0.6 – 3 mg	Nal 8 mg/ Bup 90 mg	25 – 50 – 100 mg	Phen 15 mg Top 12.5 – 25 – 50 – 100 mg	0.25 – 0.5 – 1.0 mg
Starting dose	15–30 mg mane	120 mg tds	0.6 mg daily	Nal 8 mg/ Bup 90 mg mane	12.5 mg mane	Phen 15 mg mane Top 12.5 mg mane	0.25 mg once weekly
Dosage form	Tablet	Tablet	Injection	Tablet	Tablet	Tablet	Injection
Maximal dose	40 mg mane 12-week treatment only	120 mg tds	3.0 mg daily	Nal 16 mg/ Bup 180 mg bd	50 mg bd	Phen 15 mg mane Top 50 mg bd	2.4 mg once weekly
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	Hypersensitivity 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	Glaucoma Renal stones Pregnancy (if used for weight loss)	Uncontrolled hypertension Cardiac disease Glaucoma History of drug abuse MAO inhibitor or SSRI use Depression Renal stones	Hypersensitivity to semaglutide or any of its excipients Pregnancy
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	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	Nausea Vomiting Diarrhoea Constipation Pancreatitis Cholecystitis May exacerbate diabetic retinopathy
Cost of medication	15–40 mg[1]	120 mg[1]	3 mg	Nal Bup	Top	Phen Top	
Dose No./day	1	3	1	32 mg 360 mg	25 mg	15 mg[1] 25 mg[1]	0.25 – 1 mg
Cost/month	$108.50 - $144.90	$107.20	$387	4 Tablets of 8/90 mg $240	up to 4 (100 mg) $15	1 up to 4 $120 + $15	once wkly PBS $40 Private script $140

Mane, in the morning; tds, 3 times per day; bd, twice a day; MAO, monoamine oxidase; SSRI, selective serotonin reuptake inhibitor. TGA, Therapeutic Goods Administration. *Available through the veteran system. [1]Estimated 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 Aug 2021.

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 [46].

3.1.2. 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/m^2 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 [47]. 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.

3.2. Set weight loss targets

The algorithm provides a weight loss target of 10–15% in individuals

Table 2
The classification of weight by BMI and WC.

Classification	General population BMI (kg/m^2)	Population specific BMI (kg/m^2)*	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.

with BMI 30–40 kg/m^2 and > 15% in those with BMI > 40 kg/m^2. 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 the prevention of diabetes.

3.3. 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/m^2 without obesity-related complications. Shared care arrangements between primary care physicians and specialist services should be considered for people with BMI 30–40 kg/m^2 with complications or BMI > 40 kg/m^2 without complications. Individuals with BMI > 40 kg/m^2 with complications should be considered for referral to specialist care. (Fig. 1).

3.4. Weight loss strategies (Fig. 1)

3.4.1. Management of individuals with BMI 30–40 kg/m^2 without complications

Supervised lifestyle intervention is the mainstay of management for individuals with BMI 30–40 kg/m^2 without established complications. Initially this includes a reduced energy diet or a low energy diet, combined with a programme 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 programme 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.

3.4.2. Management of individuals with BMI 30–40 kg/m^2 with obesity-related complications or BMI > 40 kg/m^2 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.

3.4.3. Management of individuals with BMI > 40 kg/m^2 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.

4. Special clinical situations

4.1. 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/m^2 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 [48]. 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.

4.2. 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.

4.3. 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/m^2 and BMI ≥ 40 kg/m^2 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/m^2 with obesity-related complications, more intensive therapies are indicated, but maintaining physical function, favourable body composition and quality nutrition may require specific lifestyle programmes. When engaging older adults in intensive therapies, cardiovascular fitness should be considered.

4.4. Pregnancy

Obesity during pregnancy is associated with an increased risk of obstetric complications, hypertension, gestational diabetes, foetal macrosomia, birth defects and increased risk of obesity in the offspring. In women with BMI > 30 kg/m^2, a total weight gain not exceeding 9 kg is recommended [49,50]. Gestational weight gain (GWG) should be closely monitored in women with obesity, as well as in women with a

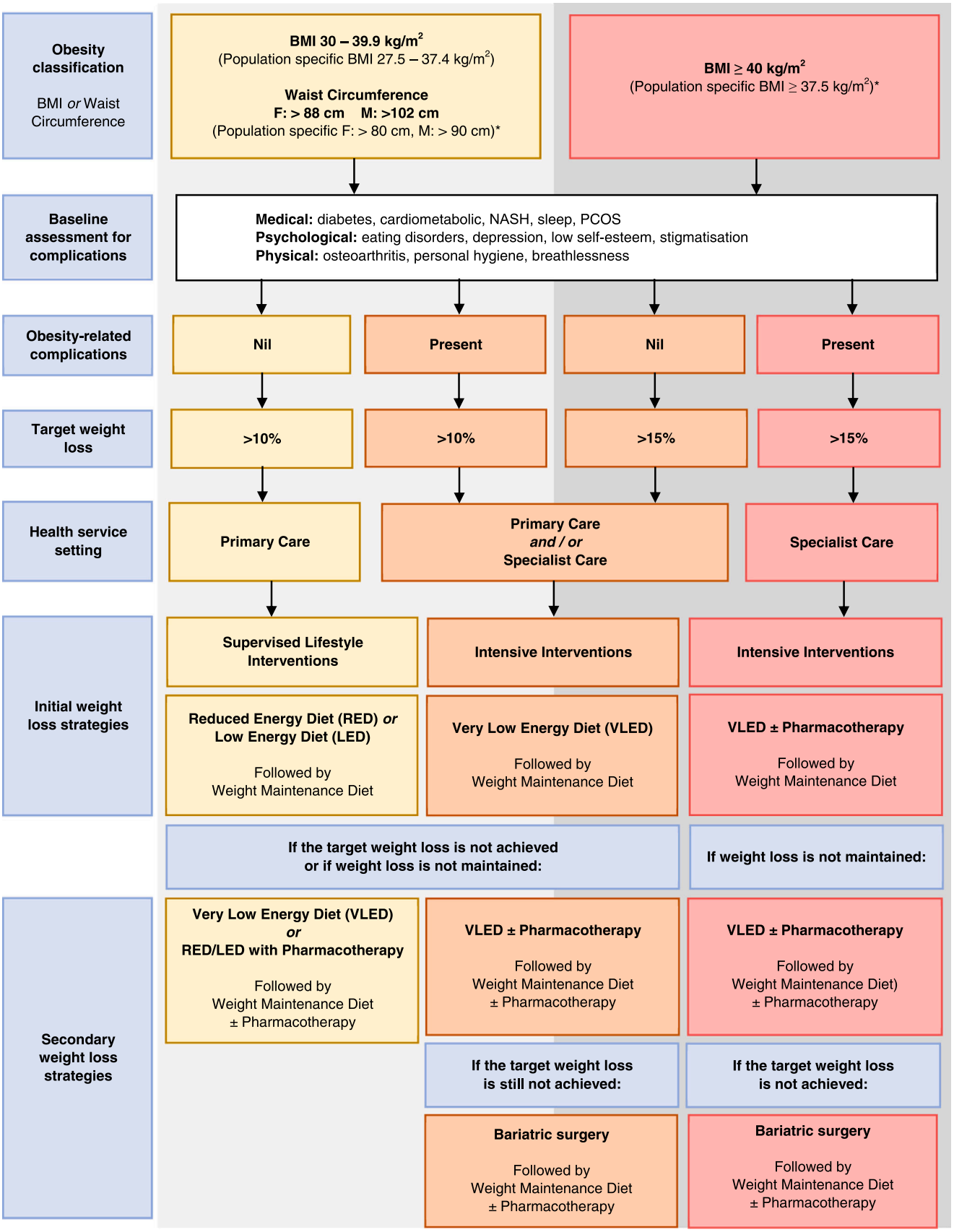

Fig. 1. Australian algorithm for the management of obesity. BMI, body mass index. LED, low energy diet. MASH, metabolic associated steatohepatitis. GORD, gastro-oesophageal reflux disease. OSA, obstructive sleep apnoea. PCOS, polycystic ovary syndrome. ED, erectile dysfunction. LUTS, lower urinary tract symptoms. RED, reduced energy diet. VLED, very low energy diet. *Cut-offs apply to Asian population and recommended for Australian indigenous population.

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 12–18 months prior to pregnancy. All medications available for weight loss in Australia are category B for use in pregnancy.

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 [27, 51–54].

CRediT authorship contribution statement

All authors contributed to the writing of this document. Nathalie Kirzirian collated the first draft based on input from all authors. Erica Bessell prepared the table entitled “Summary of current guidelines for management of overweight and obesity” based on literature review and re-configured the algorithm by amalgamating several pathways into one pictorial. All authors reviewed and approved this version (October, 2021) of the guidelines.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Competing interest statement

TPM has received support from Novo Nordisk for seminar presentations at conferences and Eli Lilly for participation in an advisory board. She serves on the Nestle Health Science VLCD Advisory Board. JP is on Advisory Boards for Novo Nordisk and iNOVA and has given lectures on obesity management for Novo Nordisk and iNova. JBD has received support from I-Nova as a consultant, advisory board and speaker fees; Nestle Health Science Consultant, advisory board and speaker fees; Novo Nordisk: Advisory board and speaker fees. GR has received consulting and speaking honoraria from NovoNordisk, iNova, Ethicon (Johnson & Johnson), Reshape HelathSciences and Nestle HealthScience. GD has received payments from Novo Nordisk, Inova pharmaceuticals for participation in advisory boards and seminar presentations at conferences. SC has received payments from Novo Nordisk and iNOVA pharmaceuticals for participation in advisory boards.

Acknowledgements

All persons who have made substantial contributions to the work reported in the manuscript (e.g., technical help, writing and editing assistance, general support), but who do not meet the criteria for authorship, are named in the Acknowledgements and have given us their written permission to be named. If we have not included an Acknowledgements, then that indicates that we have not received substantial contributions from non-authors.

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]	• 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 $\geq$ 27 kg/m^2 • If at least one severe complication: lifestyle/behavioural therapy with pharmacotherapy (BMI $\geq$27 kg/m^2) and consider bariatric surgery if BMI $\geq$ 35 kg/m^2
American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society[2]	• High-intensity comprehensive lifestyle intervention (moderate energy reduction, physical activity, behavioural strategies) • Consider pharmacotherapy as an adjunct if BMI $\geq$ 30 kg/m^2 (or $\geq$27 kg/m^2 with comorbidity) • Consider referral to bariatric surgery as an adjunct if BMI $\geq$ 40 kg/m^2 (or $\geq$35 kg/m^2 with comorbidity)
Obesity Canada Guidelines[3]	• Medical nutrition therapy delivered by a dietitian and physical activity recommendations • Psychological and behavioural interventions to affect change • Adjunctive pharmacotherapy if BMI $\geq$ 30 kg/m^2 (or $\geq$27 kg/m^2 with complications) • Consider bariatric surgery if BMI $\geq$ 40 kg/m^2 (or $\geq$35 kg/m^2 with at least one complication)
Saudi Clinical Practice Guideline[4]	• 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 $\geq$ 40 kg/m^2 (or $\geq$35 kg/m^2 with comorbidities)
European Guidelines[5]	• Nutrition and physical activity intervention delivered using cognitive behavioural therapy • Pharmacotherapy as an adjunct if BMI $\geq$ 30 kg/m^2 (or $\geq$27 kg/m^2 with comorbidities) • Consider bariatric surgery if other attempts unsuccessful and BMI $\geq$ 40 kg/m^2 (or $\geq$35 kg/m^2 with comorbidities, or $\geq$30 kg/m^2 with type 2 diabetes)
NICE: National Institute for Health and Care Excellence Guideline[6]	• 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 energy intake • Consider pharmacotherapy if lifestyle intervention unsuccessful • Consider bariatric surgery if BMI $\geq$ 40 kg/m^2 (or $\geq$35 kg/m^2 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	Celery	Radishes	Corn Green
Asparagus	Cucumber	Shallots	peas
Bean Sprouts	Eggplant	Silverbeet	Legumes
Bok Choy	Endive	Snow peas	Lentils
Broccoli	Green	Spinach	Potatoes
Brussels sprouts	beans	Squash	Sweet potato
Cabbage	Konjac noodles	Tomatoes	Parsnip
Capsicum	Lettuce (all types)	Watercress	Pumpkin
Carrots	Leeks Mushrooms	Zucchini	Turnip
Cauliflower	Onions		
Soups			
Stock cubes	Vegetable soups made from allowed vegetables		All other soups
Bonox® (in moderation)	Miso soup		
Sauces and condiments			
Lemon & lime juice	Soy sauce	Mustard	Cream
Vinegar	Chili	Tomato paste	High calorie simmer sauces and dressings
Worcestershire sauce	Diet, oil free or fat		
Tabasco sauce	free salad dressings		
Herbs and spices			
All spice	Curry powder	Oregano	
Basil	Dill	Paprika	
Celery flakes	Fennel	Parsley	
Chili Chives	Garlic	Pepper	
Cinnamon	Ginger	Rosemary	
Cloves	Lite salt	Sage	
Coriander	Mint		
Cumin	Mustard seed	Tarragon	
	Nutmeg	Thyme	
		Turmeric	
Others			

(*continued on next page*)

(*continued*)

Allowed	Avoid
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 interventions[1]

Intervention	Summary of effect
Lifestyle change	> 10% weight loss in few studies; weight loss difficult to maintain for many individuals. Study results: • 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
Combined lifestyle change and pharmacotherapy	> 10% weight loss in some but not all studies; weight loss maintained > 2 years in some participants. Study results: • Orlistat and dietary change: average weight loss 6–10 kg at 12 months; 2–3 kg at 5 years [31] • Phentermine and dietary change: average weight loss 6.4 kg 12 weeks [28] • Liraglutide and lifestyle change: average weight loss 8% at 56 weeks [32] and 6% at 3 yr [55] • Naltrexone/bupropion and lifestyle change: average loss 6.1% at 56 weeks [37] • Semaglutide and lifestyle: average weight loss 15% at 68 weeks [42]
Bariatric surgery with maintained lifestyle changes	> 15% weight loss consistently across studies; weight loss likely to be maintained > 5 years • 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 [54] • *Roux-en-Y* gastric bypass: average weight loss 33% at 12 months; 30% at 10 years [54]

[1]Adapted from the 2013 NHMRC table 6.4 [4].

References

[1] Australian Bureau of Statistics. National Health Survey: First Results, 2017–2018; 2018.
[2] Price Waterhouse Cooper. Weighing the cost of obesity: a case for action, 2015.
[3] Diabetes Prevention Program Research, 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] NHMRC. 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. N Engl J Med 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, et al. An adoption study of human obesity. N Engl J Med 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] Obesity Australia. 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. Med J Aust 2009;190(3):149–51.
[15] National Health and Medical Research Council. Australian Guide to Healthy Eating, 2013 ⟨http://www.eatforhealth.gov.au/guidelines/australian-guide-healthy-eating⟩.
[16] National Health and Medical Research Council. Australian Dietary Guidelines. Canberra, 2013.
[17] Hu T, Mills KT, Yao L, Demanelis K, Eloustaz M, Yancy Jr WS, 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. N Engl J Med 2009;360(9):859–73.
[19] Gudzune 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, et al. 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 Obes Metab 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] Proietto J. Obesity and weight management at menopause. Aust Fam Physician 2017;46(6):368–70.
[26] Australian Government. Physical activity and exercise guidelines for all Australians, 2021 (https://www.health.gov.au/health-topics/physical-activity-and-exercise-guidelines-for-all-australians).
[27] The Royal College of General Practitioners. Smoking, nutrition, alcohol, physical activity (SNAP): A population health guide to behavioural risk factors in general practice, 2nd edition, Melbourne, 2015.
[28] 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.
[29] 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.
[30] 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. Am J Clin Nutr 2012;95(2):297–308.
[31] 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.
[32] Monami M, Nreu B, Scatena A, Cresci B, Andreozzi F, Sesti G, Mannucci E. Safety issues with glucagon-like peptide-1 receptor agonists (pancreatitis, pancreatic cancer and cholelithiasis): Data from randomized controlled trials. Diabetes Obes Metab. 2017;19(9):1233–41.

[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. N Engl J Med 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. N Engl J Med 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 Obes Metab 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] 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.
[38] 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.
[39] 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. Obes Rev J Int Assoc Study Obes 2011;12(5):e338–47.
[40] Neoh SL, Sumithran P, Haywood CJ, Houlihan CA, Lee FT, Proietto J. Combination phentermine and topiramate for weight maintenance: the first Australian experience. Med J Aust 2014;201(4):224–6.
[41] 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.
[42] Wilding JPH, Batterham RL, Calanna S, Davies M, Van Gaal LF, Lingvay I, et al. Once-weekly semaglutide in adults with overweight or obesity. N Engl J Med 2021; 384(11):989.
[43] 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.
[44] 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.
[45] 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.
[46] 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. Nat Rev Endocrinol 2020;16(3): 177–89.
[47] 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.
[48] 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 2015;39(1):82–4.
[49] Rasmussen KM, Yaktine AL (Eds.). Weight gain during pregnancy: reexamining the guidelines. The National Academies Collection: Reports funded by National Institutes of Health. Washington (DC) 2009.
[50] Get Healthy New South Wales. Weight gain during pregnancy, 2021 ⟨http://www.gethealthynsw.com.au/assets/pdf/resources/GHS_Fact_Sheet_Weight_Gain_During_Pregnancy_online_Final.pdf⟩.
[51] 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.
[52] Grima M, Dixon JB. Obesity-recommendations for management in general practice and beyond. Aust Fam Physician 2013;42(8):532–41.
[53] 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.
[54] 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.
[55] 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.