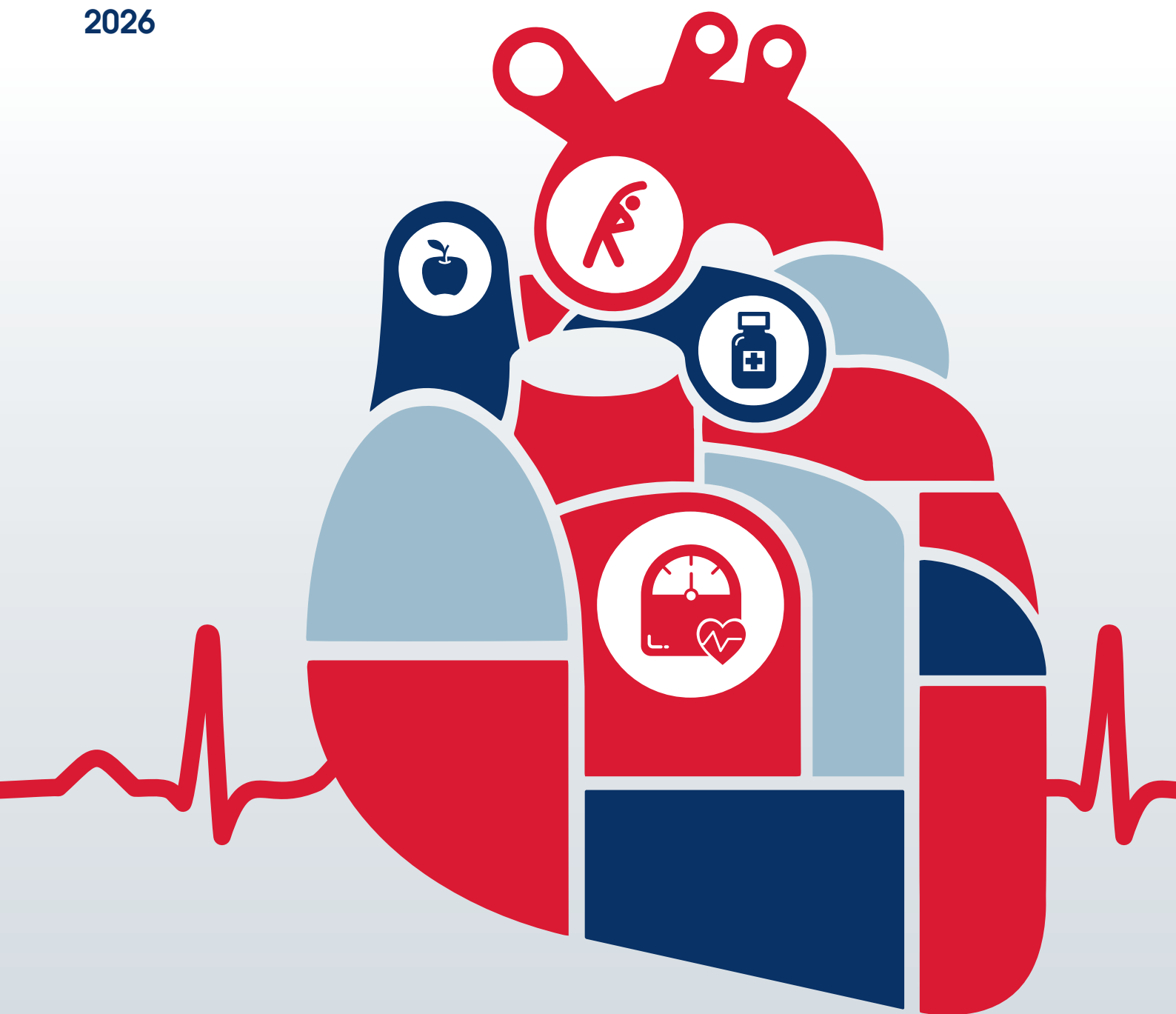


Obesity and cardiovascular disease



A clinical consensus statement
from the National Heart Foundation
of Australia

2026





Acknowledgement of Country

We acknowledge the Traditional Owners and Custodians of country throughout Australia and their continuing connection to land, waters and community. We pay our respects to them and their cultures, and Elders past, present and future.



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Summary of recommendations

Recommendations	Strength of advice
 Defining overweight and obesity	
<p>Recognise clinical obesity as a chronic, systemic condition driven by excess adiposity.</p>	<p>Moderate advice ● ● ○</p>
<p>Use body mass index (BMI) as a primary screening tool for obesity, complemented by additional anthropometric assessments, such as waist circumference, waist-to-height ratio, or waist-to-hip ratio.</p>	<p>Strong advice ● ● ●</p>
 Behaviour modifications Nutrition	
<p>In adults living with overweight or obesity and cardiovascular disease (CVD) or at high risk of CVD, recommend a heart-healthy eating pattern, which includes a wide variety of foods from all food groups, is naturally low in unhealthy fats, salt and added sugars, and limits discretionary food/beverages to reduce cardiovascular risk.</p>	<p>Moderate advice ● ● ○</p>
<p>For weight loss in adults living with overweight or obesity and CVD or at high risk of CVD, recommend a heart-healthy eating pattern with reduced energy intake (at least 2000 kJ/day deficit from estimated daily energy requirements) with a goal of achieving at least 5% weight loss over six months.</p>	<p>Strong advice ● ● ●</p>
<p>In adults living with overweight or obesity and CVD or at high risk of CVD who require more rapid weight loss to help manage complications, recommend either a low energy diet (LED) or very low energy diet (VLED) under clinical supervision.</p>	<p>Strong advice ● ● ●</p>
 Behaviour modifications Physical activity	
<p>In adults living with overweight or obesity and CVD or at high risk of CVD, recommend regular physical activity across the course of the day while reducing sedentary behaviour, irrespective of impact on weight, to support cardiovascular and overall health.</p>	<p>Strong advice ● ● ●</p>
<p>For adults living with overweight or obesity and CVD or at high risk of CVD, recommend an exercise routine combining moderate-to-vigorous aerobic activity and resistance training, tailored to a person's goals, to support cardiovascular and overall health.</p>	<p>Strong advice ● ● ●</p>

Recommendations	Strength of advice
 Pharmacological and surgical interventions Pharmacotherapy	
<p>Obesity management medications are indicated, in conjunction with behaviour modifications, when adequate weight-related health improvements cannot be attained through behaviour modifications alone.</p>	<p>Strong advice</p> <p>● ● ●</p>
<p>In adults living with established atherosclerotic CVD and BMI ≥ 27 kg/m², consider prescribing a GLP-1 receptor agonist with proven CVD benefit (semaglutide) to reduce the risk of major adverse cardiovascular events.</p>	<p>Moderate advice</p> <p>● ● ○</p>
<p>In adults living with heart failure with preserved ejection fraction (HFpEF) and obesity^a, consider semaglutide or tirzepatide to improve symptoms and functional capacity.</p>	<p>Moderate advice</p> <p>● ● ○</p>
<p>In adults living with type 2 diabetes and overweight or obesity, with or without CVD, consider a GLP-1 or GIP/GLP-1 receptor agonist with proven CVD benefit (semaglutide, liraglutide or tirzepatide) to reduce the risk of major adverse cardiovascular events.</p>	<p>Strong advice</p> <p>● ● ●</p>
<p>^a Defined in trials as BMI ≥ 30 kg/m² and ejection fraction $\geq 50\%$ (SUMMIT, tirzepatide) or ejection fraction $\geq 45\%$ (STEP-HFpEF, semaglutide). While these medications have demonstrated benefit for heart failure symptoms and quality of life, they are not yet approved by the Therapeutic Goods Administration for management of this condition.</p>	
 Pharmacological and surgical interventions Surgical interventions	
<p>In adults living with overweight or obesity and CVD or at high risk of CVD who have not attained adequate weight-related health improvements through behaviour modifications and pharmacological interventions, consider referral to multidisciplinary specialised obesity services.</p>	<p>Moderate advice</p> <p>● ● ○</p>
<p>In adults with CVD or at high risk of CVD with BMI ≥ 40 kg/m², or BMI ≥ 35 kg/m² with at least one weight-related comorbid condition[‡], refer for consideration of metabolic bariatric surgery to reduce the risk of major adverse cardiovascular events.</p>	<p>Moderate advice</p> <p>● ● ○</p>
<p>In adults with CVD or at high risk of CVD who have undergone metabolic bariatric surgery, offer lifelong multidisciplinary follow-up care to support long term health.</p>	<p>May be appropriate</p> <p>● ○ ○</p>
<p>[‡] Weight-related comorbid conditions include hypertension, dyslipidaemia, obstructive sleep apnoea, cardiovascular disease, prediabetes or type 2 diabetes.</p>	

Abbreviations: GIP, glucose-dependent insulintropic polypeptide; GLP-1, glucagon-like peptide-1.

Overview

Introduction

Overweight and obesity represent a critical and growing public health challenge in Australia and globally. Approximately two thirds of adults in Australia are currently living with these conditions.^{1,2} The prevalence of obesity alone has increased from 19% in 1995 to 32% in 2022,¹ with nearly half of the adults in Australia expected to be living with obesity by 2035.³ Overweight and obesity are associated with a range of chronic conditions including cardiovascular disease (CVD), cancer, type 2 diabetes, chronic kidney disease, metabolic dysfunction-associated steatotic liver disease, musculoskeletal disease, dementia, asthma and obstructive sleep apnoea.^{2,4,5} In 2024, overweight and obesity overtook tobacco use as the leading modifiable contributor to Australia's disease burden, accounting for 8.3% of total burden.⁶

In the context of CVD, which remains a leading cause of morbidity and mortality in Australia,¹ overweight and obesity contribute to hypertension, dyslipidaemia, insulin resistance and systemic inflammation.⁷ These mechanisms accelerate the development of coronary heart disease, stroke, heart failure and atrial fibrillation.⁸

Obesity is now widely recognised as a complex, chronic, relapsing condition, rather than solely a risk factor, arising from interacting biological, behavioural and environmental drivers.⁹ This contemporary disease framework underscores the need for long term, person-centred management approaches that address prevention, treatment and maintenance across the life course.¹⁰

Effective overweight and obesity management requires a multimodal, staged approach. Foundational to this approach is sustained behaviour modification, including nutrition and physical activity, supported in a non-stigmatising, culturally appropriate manner. For people living with severe obesity (obesity with complications or very elevated body mass index (BMI)), or for those for whom behaviour modifications alone cannot achieve the required outcomes, obesity management medications and metabolic bariatric surgery play an important adjunctive role.

Newer incretin-based therapies, such as glucagon-like peptide-1 (GLP-1) receptor agonists and dual glucose-

dependent insulinotropic polypeptide (GIP)/GLP-1 receptor agonists, have changed the obesity treatment landscape. These agents offer highly effective options for weight management, with demonstrated positive cardiovascular outcomes, including for people with established CVD.^{11,12}

Despite the strong causal link between obesity and several cardiovascular conditions, gaps persist in prevention and management across the Australian healthcare system. Obesity remains overlooked as a modifiable risk factor for CVD, while stigma and inequities in access to effective treatment further compound the challenges.¹³ Current clinical guidelines offer limited integration of obesity management within cardiovascular care pathways. This underscores the need for a coordinated, multidisciplinary approach that primary healthcare professionals, other specialists and allied healthcare professionals can collectively use to support people living with obesity.¹⁴

This consensus statement has been developed to help address these knowledge gaps and the growing need for practical guidance in this evolving area of medicine. It intends to assist healthcare professionals to deliver equitable, person-centred, evidence-based strategies to assess and manage overweight and obesity and improve long-term health outcomes for adults in Australia living with, or at high risk of, CVD. The statement is intended to complement the upcoming National Health and Medical Research Council (NHMRC)'s *Clinical practice guidelines for the management of overweight and obesity for adults, adolescents and children in Australia* ('NHMRC Clinical Practice Guidelines').¹⁴

Obesity is a multifaceted condition underpinned by a broad and complex range of drivers that extend beyond the scope of clinical intervention.⁹ System-level and environmental factors play a profound role, and while not addressed in detail in this consensus statement, are also critical to tackling the growing problem of obesity in Australia.¹⁵ Similarly, while out of scope for this consensus statement, primary prevention of overweight and obesity remains critical to reducing the incidence of CVD. Longer duration of obesity is

associated with greater cumulative cardiometabolic risk, earlier onset of hypertension, dyslipidaemia and type 2 diabetes, and higher lifetime risk of CVD events.¹⁶ This emphasises the importance of early intervention strategies that continue across the life course.

Purpose

The purpose of this consensus statement is to provide:

- a discussion of the link between overweight/obesity and CVD
- evidence-based recommendations for the definition, assessment and diagnosis of overweight and obesity, and discussion of the practical implications for cardiovascular health
- evidence-based recommendations for the management of overweight and obesity in the context of CVD, encompassing behaviour modifications, obesity management medications and metabolic bariatric surgery
- specific practice points and considerations for high-priority populations
- considerations for cardiovascular diagnostic imaging, cardiovascular-kidney-metabolic syndrome, and other conditions relevant to CVD and overweight/obesity
- practical advice, tools and resources to guide healthcare professionals on applying the recommendations in their clinical practice.

Intended audience

This consensus statement is intended for all healthcare professionals caring for people living with overweight or obesity with established CVD or at high risk of CVD. These include (but are not limited to) general practitioners and other primary healthcare professionals, cardiologists, endocrinologists, surgeons, gastroenterologists, nurses and nurse practitioners, First Nations health workers and practitioners, pharmacists, dietitians, exercise physiologists, physiotherapists and other allied healthcare professionals.

Scope

This consensus statement addresses the clinical assessment, diagnosis and management of adults (people aged 18 years or older) living with overweight or obesity with established CVD or at high risk of CVD.

- 'Established CVD' is a broad term that includes both atherosclerotic CVD (e.g. coronary heart disease) and non-atherosclerotic cardiovascular conditions (e.g. heart failure, atrial fibrillation).
- 'High risk' refers to high risk of atherosclerotic CVD and has been defined as per the below groups (Figure 1):
 - People with high calculated risk according to the *Australian CVD Risk calculator* ('Aus CVD Risk calculator') (estimated five-year CVD risk \geq 10%)
 - People with clinically determined high risk according to the *2023 Australian Guideline for assessing and managing cardiovascular disease risk*
 - People with very elevated individual risk factors (e.g. very high blood pressure, very elevated cholesterol, very high lipoprotein(a))
 - People with subclinical or 'pre-clinical' disease (e.g. very high coronary artery calcium score)

This consensus statement does not comprehensively address:

- i. overweight and obesity in children, adolescents, older adults or pregnant women,
- ii. extensive guidance on behaviour modifications,
- iii. eating disorders,
- iv. complementary medicines,
- v. detailed advice on metabolic bariatric surgical procedures, or
- vi. cost effectiveness of interventions.

A discussion of the system-level drivers of obesity and primary prevention is also out of scope of this document.

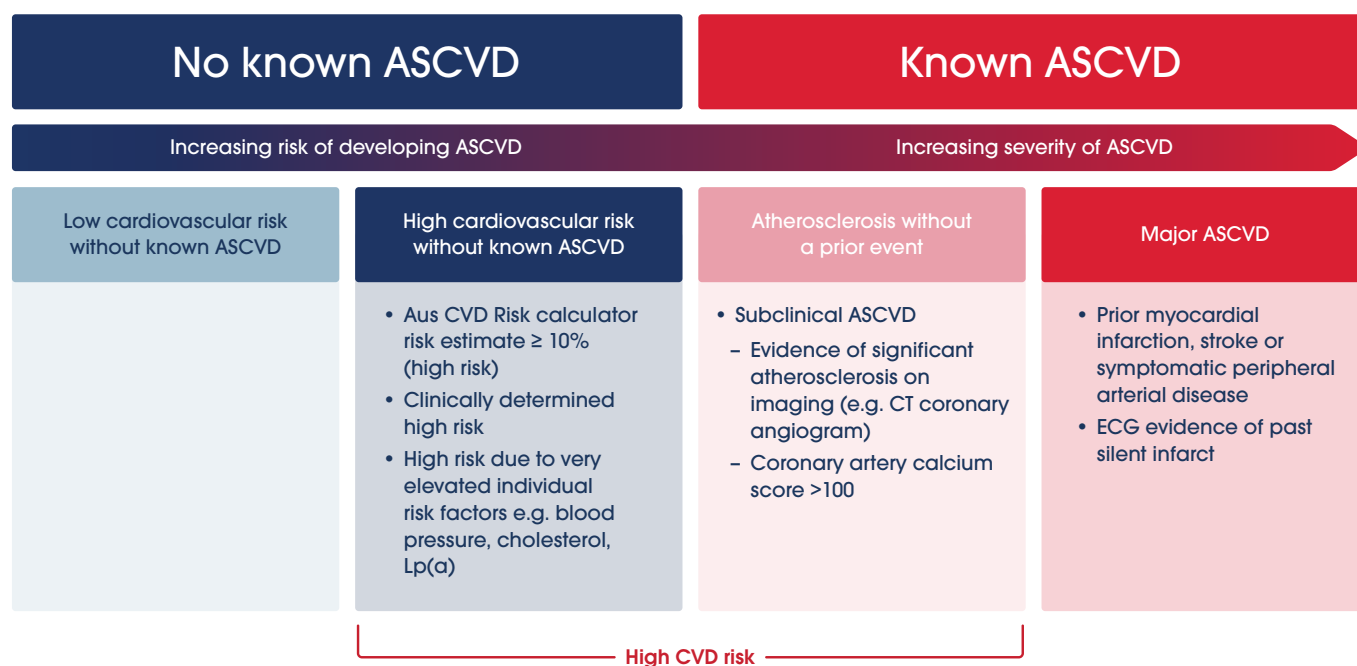


Figure 1: Continuum of atherosclerotic cardiovascular disease risk.

Abbreviations: ASCVD, atherosclerotic cardiovascular disease; CT, computed tomography; CVD, cardiovascular disease; ECG, electrocardiogram; Lp(a), lipoprotein (a).

Adapted with permission from the TIMI Study Group from VESALIUS-CV Late Breaking Clinical Trial presentation slides, presented at the American Heart Association Scientific Sessions 2025.

How the consensus statement was developed

This consensus statement was developed by the National Heart Foundation of Australia (Heart Foundation) under the guidance of a multidisciplinary taskforce ('the Taskforce') with expertise in cardiology, endocrinology, obesity, bariatric surgery, general practice, First Nations health, epidemiology, nutrition, dietetics, exercise physiology, pharmacy, public health, and consumers with lived experience. See *Appendix 1: Contributors to the consensus statement* for the full list of contributors.

In addition to expert guidance provided by the Taskforce, this consensus statement has been informed by the following:

- An independent evidence review completed by the University of Queensland involving a systematic synthesis of peer-reviewed literature conducted via targeted searches.
- An environmental scan assessing:
 - prominent international guidelines and position statements on obesity and CVD
 - existing local guidelines, policies, strategies, services and reports, including the draft NHMRC Clinical Practice Guidelines.
- A national stakeholder forum on obesity and CVD held in July 2025.
- Targeted consultation with a diverse group of individual experts, consumers, peak bodies and government health department representatives.

The consensus statement recommendations were developed by Taskforce members and informed by the evidence review, environmental scan, and clinical expertise.

To support the consistency and integrity of the recommendations, a structured framework for strength of advice was specifically developed to guide the evaluation of evidence and formulation of advice (Table 1). This framework was adapted from the European Society of Cardiology's Scientific Documents Policy.¹⁷

Based on evidence quality and consensus among Taskforce members, the strength of advice is both evidence-based and practical. Generally, the more high-quality studies and robust research designs included in an analysis, the stronger its evidence rating. Systematic reviews, meta-analyses and randomised controlled trials typically represent the highest level of evidence due to their rigorous methodology and strong indications of effectiveness.¹⁸

Table 1. Strength of advice hierarchy

<p>A. Strong advice, based on robust published evidence* and supported by expert consensus.** High confidence that benefits clearly outweigh risks. Supported by consistent robust high-quality evidence and expert consensus.</p>	<p>Strong advice ● ● ●</p>
<p>B. Moderate advice, based on some published evidence* and supported by expert consensus.** Benefits likely outweigh risks, but with some uncertainty. Evidence may not be as strong or expert opinion may not reach unanimous consensus.</p>	<p>Moderate advice ● ● ○</p>
<p>C. May be appropriate, based on published evidence* and expert consensus.** Insufficient evidence in this area. Recommendation based primarily on expert consensus.</p>	<p>May be appropriate ● ○ ○</p>
<p>D. Area of uncertainty. Insufficient evidence or consensus to support a clear position. Clinical practice may vary in this area.</p>	<p>Area of uncertainty ○ ○ ○</p>

* Systematic reviews, meta-analyses and randomised controlled trials constitute the highest level of evidence. Observational studies constitute a lower level of evidence and are considered less robust. Case studies and grey literature constitute the lowest level of evidence.

** While formal voting thresholds were not specified, recommendations were retained only where there was strong majority support and no substantive unresolved objections.

For more information on how this consensus statement was developed, please refer to *Appendix 2: Developing the clinical consensus statement*.

Conflicts of interest

Conflicts of interest were recorded and managed throughout the entire development of the consensus statement.

Conflicting interests were considered within a framework of both:

- i. the relationship (direct or indirect) of the participating individual to any third party with interest in the topic under consideration during the development process of the consensus statement
- ii. the nature (financial and non-financial) of the potential conflict.

Conflicting interests among the subgroups required appropriate management to ensure clinical recommendations were not compromised. Processes employed by the Heart Foundation project team aimed to ensure the integrity of the Taskforce and to strike an appropriate balance between the existence of interests

in a topic under review and the expertise required to make sound and meaningful recommendations.

More information on conflicts of interest management and a summary of all disclosures can be found in *Appendix 3: Conflicts of interest*.

Impact of overweight and obesity on cardiovascular risk and CVD outcomes

The underlying causes of overweight and obesity are complex and not fully understood, arising from a multifactorial interplay of genetic, epigenetic, neurobiological, environmental, psychological, physiological, behavioural and sociocultural influences (Figure 2).¹⁹

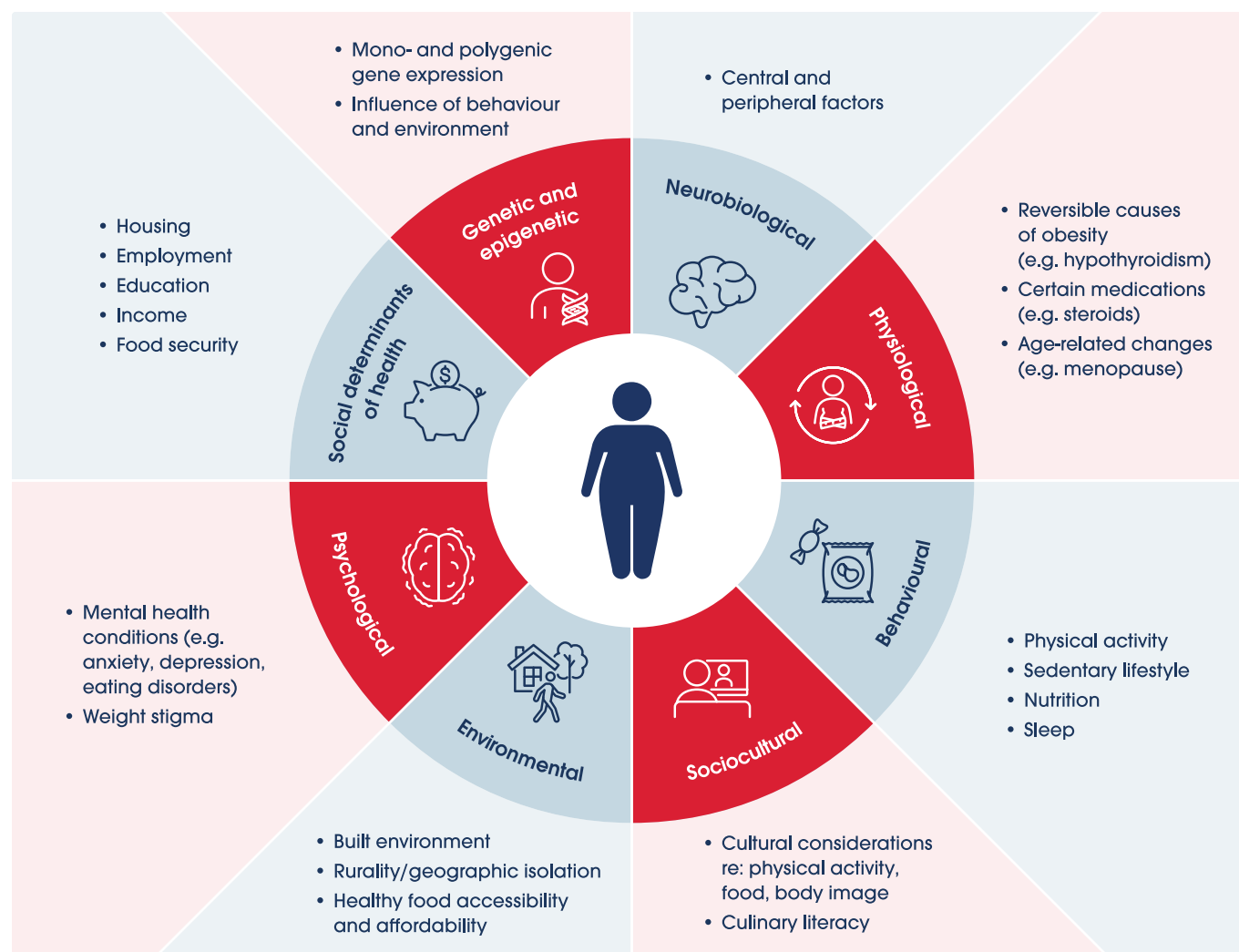


Figure 2: Multifactorial aetiologies of obesity.²⁰

Overweight and obesity are significant independent risk factors for CVD via both direct and indirect mechanisms (Figure 3).

Directly, excess adipose tissue causes endothelial dysfunction, systemic inflammation, oxidative stress and insulin resistance.²¹ Excess adiposity also promotes atherosclerosis and causes a range of functional and structural changes in the heart, resulting in poor cardiac function.¹⁹

Indirectly, excess adiposity contributes to the development of several well-established risk factors for CVD, including hypertension,^{22,23} type 2 diabetes²⁴ and dyslipidaemia.²⁵ The Aus CVD Risk calculator does not include BMI as a specific risk variable for the general population (it is included only for people with type 2 diabetes). This is because much, but not all, of the predicted risk associated with overweight/obesity is captured via the inclusion of downstream metabolic risk factors such as hypertension, type 2 diabetes and dyslipidaemia.²⁶



Figure 3: Direct and indirect impacts of excess adiposity on CVD and CVD subtypes.

Abbreviations: CVD, cardiovascular disease.

Within the ‘umbrella’ term of CVD, overweight/obesity is also a risk factor for several CVD subtypes including atrial fibrillation,²⁷⁻³¹ coronary heart disease,^{32,33} heart failure,^{34,35} peripheral arterial disease³⁶ and stroke.^{37,38} Similarly, people living with overweight and obesity have an increased risk of major adverse cardiovascular events including all-cause and CVD mortality.³⁹ Overweight and obesity confer a 46% and 64% higher

risk of myocardial infarction respectively,⁴⁰ and an increased risk of sudden cardiac death (21% and 52%, respectively).^{41,42}

Several modifying factors play a role in the relationship between overweight/obesity and CVD. These include adipose tissue distribution, cardiorespiratory fitness, comorbidities and metabolic health status (Figure 4).

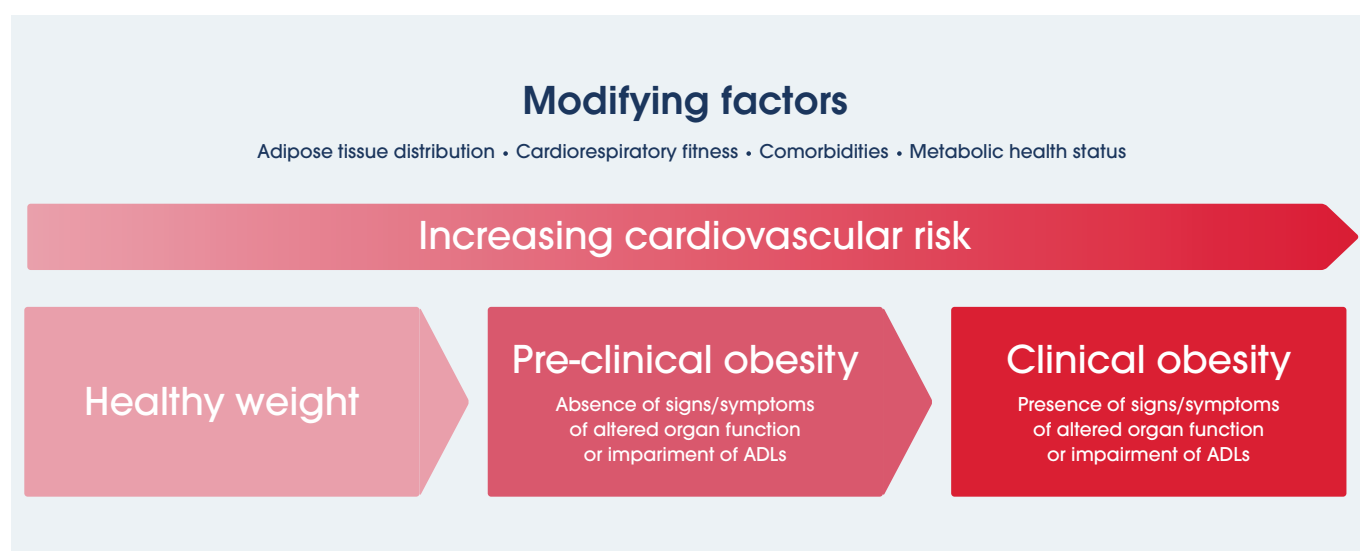


Figure 4: Modifying factors for the relationship between excess adiposity and cardiovascular risk.

Abbreviations: ADLs, activities of daily living.

While both general and central adiposity contribute to increased risk of CVD, central adiposity, particularly elevated visceral, perivascular and hepatic fat, is more strongly associated with adverse outcomes.⁴³ For example, incremental increases in waist circumference, a marker of elevated visceral and hepatic fat, are associated with increased risk of CVD in both women and men.⁴⁴ Similarly, people with ‘normal weight obesity’, defined as BMI within the healthy range but with an elevated body fat percentage, have a significantly increased risk of CVD and adverse cardiometabolic risk factors, including high triglycerides and hypertension.

Metabolic health status (generally defined by the presence or absence of hypertension, impaired fasting glucose and/or dyslipidaemia) and cardiorespiratory fitness are also important modifiers of the relationship between CVD and obesity.⁴⁵⁻⁴⁸

When elevated BMI is accompanied by poor metabolic health or low fitness, the risk of cardiovascular morbidity and mortality significantly increases.^{46,47} People with obesity and optimised metabolic risk factors have a lower risk of CVD than people with obesity with poor metabolic health.⁴⁹ However, having good metabolic health does not entirely negate the adverse impacts of excess adiposity. People with obesity without metabolic risk factors are still at increased risk of cardiovascular events.⁵⁰

To note, in people with *established* CVD, the association between BMI and CVD-related mortality or major adverse cardiovascular events is non-linear, with some studies suggesting a lower BMI may confer a higher risk of mortality, with excess adiposity being somewhat protective.⁵¹⁻⁵⁵ The latter is known as the 'obesity paradox'. However, this is regarded by many as an anomaly of epidemiological and observational studies.⁵⁶⁻⁶⁰

Intentional weight loss in people with overweight or obesity and CVD improves heart function and cardiovascular risk factors and reduces risk of mortality.^{61,62} Maintaining weight loss is essential for sustaining these benefits,⁶³ primarily through ongoing behaviour modifications and pharmacotherapy, if clinically indicated.

Cardiovascular-kidney-metabolic syndrome

Cardiovascular-kidney-metabolic (CKM) syndrome is a term used to describe the cluster of metabolic, kidney and cardiovascular disorders that share common risk pathways and mutually contribute to the development and accelerated progression of one another.⁶⁴ Evidence suggests that each additional CKM component (e.g. hypertension, type 2 diabetes, chronic kidney disease) increases all-cause mortality risk by ~ 22% and CVD mortality by ~ 37%.⁶⁵

The relationship between obesity and CKM syndrome is bidirectional. Excess adiposity accelerates the development and progression of cardiometabolic and renal disease, while established cardiovascular and kidney disease can further limit physical activity, alter metabolism and complicate obesity management.⁶⁴

Identification of CKM syndrome in people with obesity provides an opportunity for comprehensive, integrated care. Management should extend beyond weight reduction alone to include proactive assessment and optimisation of blood pressure, glycaemic control, lipid levels, renal function, sleep apnoea, smoking status, physical activity and nutrition. Addressing additional comorbid conditions can substantially reduce cumulative cardiovascular risk and improve long-term outcomes for people living with obesity.⁶⁴

Detailed clinical guidance for the diagnosis and management of CKM syndrome is beyond the scope of this document. However, practical considerations for healthcare professionals include the following:

- screening all people with overweight/obesity and CVD or at high risk of CVD for other metabolic risk factors including blood pressure, blood glucose, HbA1c, lipids and measures of kidney function (e.g. urine albumin-to-creatinine ratio, estimated glomerular filtration rate)
- utilising pharmacological therapies that confer cardiovascular, kidney and metabolic benefits (e.g. GLP-1 receptor agonists, GIP/GLP-1 receptor agonists and sodium-glucose co-transporter 2 (SGLT-2) inhibitors) where clinically relevant
- adopting a person-centred multidisciplinary approach, which ideally includes general practitioners, cardiologists, endocrinologists and renal specialists. Nurses and nurse practitioners, pharmacists and other allied healthcare professionals are also vital in supporting people to maintain adherence to medications and behaviour changes.

Recent research also highlights the overlapping role of liver dysfunction in CKM syndrome, coining a new term – cardiovascular-renal-hepatic-metabolic syndrome. This term recognises the bidirectional association of metabolic dysfunction-associated steatotic liver disease and CKM syndrome and re-emphasises the critical role of holistic management beyond organ-specific approaches.^{66,67}

Weight stigma

Weight stigma can be implicit or explicit, and refers to negative attitudes, stereotypes and discrimination directed at individuals living with overweight or obesity.⁶⁸ It is a recognised social and structural determinant of health and is associated with adverse mental and physical health outcomes,⁶⁹⁻⁷² social inequities,⁷³ increased cardiovascular risk,⁷⁴ and premature mortality independent of elevated BMI.⁷⁵

Weight stigma is embedded in healthcare systems, from clinical environments to models of care and unconscious biases held by healthcare professionals.^{76,77} This stigma may result in inadequate assessment and inequitable access to treatment and support for individuals living with overweight or obesity.⁷⁸

Weight stigma may cause treatment bias, where provision of, or referral to, evidence-based interventions, such as obesity management medications and metabolic bariatric surgery, is delayed or withheld due to stigmatising beliefs. Treatment bias further reinforces inequitable access to treatment.⁷⁹

People who experience stigma are more likely to delay or avoid healthcare, report poorer relationships with healthcare providers, and experience psychological distress. Fear of judgement or bias may prevent people from discussing sensitive issues like nutrition, physical activity or mental health, and those who feel judged are less likely to follow medical advice.^{80,81}

Creating stigma-free healthcare environments is a foundational equity-based requirement to ensure safe, respectable and inclusive access to care for people living with overweight and obesity. Stigma-free care is associated with improved engagement, healthcare use, and uptake of recommended care.⁸²

Healthcare professionals can take practical steps to minimise weight stigma and its impacts by:

- using person-first, non-stigmatising language (e.g. 'person living with obesity' rather than 'obese person') and avoiding terminology that implies blame or personal failure. Acknowledge obesity as a complex, chronic condition that is influenced by genetics, environment and social determinants, not just personal choice⁸³
- referring people with overweight or obesity to evidence-based treatments, including obesity management pharmacotherapy and metabolic bariatric surgery, and ensuring referral pathways are transparent, consistent and free from bias
- supporting psychological safety, recognising that previous experiences of stigma may affect engagement, trust and readiness for care
- focusing on the positive outcomes of healthy behaviours beyond anthropometric targets, such as psychosocial health, quality of life and functional status, and using shared decision-making to devise health-related goals⁸³
- ensuring clinical environments are inclusive, with features such as armless or wider chairs, high-capacity exam tables and scales, and appropriately sized gowns and medical equipment such as blood pressure cuffs.⁸³ Check imaging providers have adequate facilities to conduct the required investigations prior to referral
- building workforce capability, including healthcare professional training to recognise, reflect on and address implicit weight bias, and to communicate about weight and health in culturally safe, trauma-informed ways.

Resources and further reading

- The Obesity Collective: Quick guide: Considerations for discussing weight and health
- Australian Journal of General Practice: Supporting healthcare professionals to reduce weight stigma
- LiveLighter: Talking to patients about weight and health and other online learning
- WA Primary Health Alliance: Yarning about weight; information and videos about discussing weight with First Nations peoples
- World Obesity Strategic Centre for Obesity Professional Education (SCOPE): E-learning module Weight bias: A hidden harm

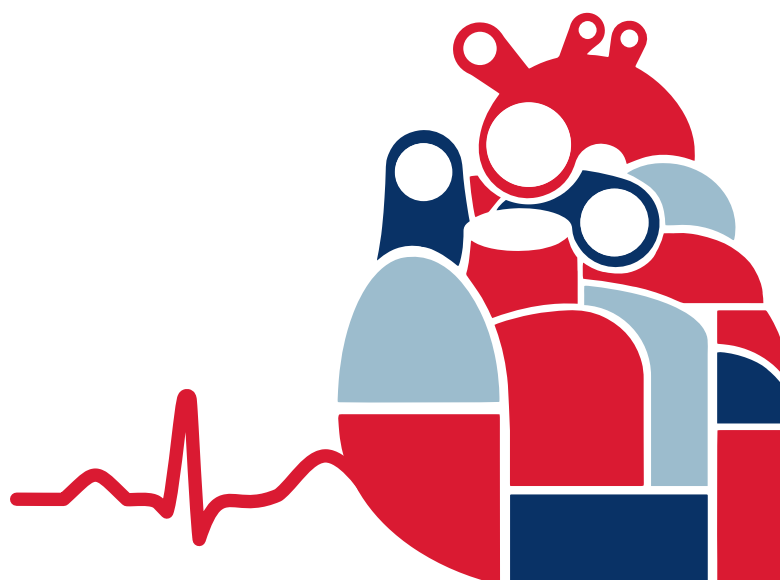
High-priority populations

Certain population groups in Australia experience a disproportionate burden of overweight and obesity, putting them at increased risk of CVD and other complications. These populations may also experience inequitable access to healthcare and services as a result of language, sociocultural, gender-based, geographical and financial barriers.

These populations include culturally and linguistically diverse (CALD) communities,⁸⁴ First Nations peoples,² people living in rural and remote areas,² people experiencing socioeconomic disadvantage,² people living with a mental health condition⁸⁵ and women (particularly post-menopausal women).^{86,87}

Equity-focused, integrated models of care should be person-centred, holistic and should identify unique barriers and challenges people may face in achieving a healthier weight and managing long-term CVD risk.^{88,89}

Specific practice points, tailored to meet the unique clinical needs of these populations, have been integrated throughout this consensus statement and are outlined further in Section 5: Equity considerations for high-priority populations.






Section 1: Defining overweight and obesity

Diagnosis and assessment

Clinical obesity is a chronic, systemic condition in which excess adiposity leads to changes in tissue, organ and overall physiological function.⁹ Pre-clinical obesity, also referred to as 'obesity without complications', is marked by excess adiposity with preserved metabolic, organ and functional integrity.⁹

Overweight and obesity are typically defined using BMI (Table 2).¹⁵ Despite its limitations, BMI remains the most widely used and validated screening tool in clinical and public health settings due to its simplicity, reproducibility and scalability. However, it does not

capture individual differences in adiposity, muscle mass or fat distribution, which independently influence cardiometabolic risk across BMI categories. There is growing recognition of the need to use anthropometric measures alongside BMI (Tables 3,4) as part of holistic and individualised metabolic health assessments, to more accurately assess cardiometabolic risk.^{83,90} Updated frameworks advocate for using both measures of adiposity and clinical staging, reflecting the presence or absence of obesity-related complications.^{9,91}

 Recommendations	Strength of advice
Recognise clinical obesity as a chronic, systemic condition driven by excess adiposity.	Moderate advice 
Use body mass index (BMI) as a primary screening tool for obesity, complemented by additional anthropometric assessments, such as waist circumference, waist-to-height ratio, or waist-to-hip ratio.	Strong advice 

Practice points

BMI and other measures of excess adiposity

- Routine opportunistic assessment of BMI and waist circumference in all adults is recommended.⁹² Unless BMI exceeds 40 kg/m² (whereby excess adiposity can be assumed), both measures should be used to assess general and central adiposity. Additional anthropometric measures may include waist-to-hip ratio, waist-to-height ratio or direct fat assessment, noting that appropriate practitioner training is required to ensure reliable and reproducible measurement.⁹³
- Healthcare professionals should be aware of the limitations of BMI in some groups (e.g. pregnant and menopausal women, athletes, people with atypical fat distribution).
- Interpret BMI using population-specific considerations where relevant, recognising limitations in measurement and applicability across different groups:
 - **First Nations peoples:** standard anthropometric measures such as BMI and waist circumference may not accurately reflect cardiometabolic risk,⁹⁴ and validated thresholds are lacking. Emerging evidence suggests that a lower BMI threshold of approximately 22 kg/m² and above (rather than 25 kg/m²) may be indicative of increased cardiometabolic risk, particularly in remote communities.¹⁴ CVD risk screening should begin earlier alongside proactive care planning and timely access to tailored interventions.

- **Older adults:** age-related sarcopenia, unintentional weight loss and changes in fat distribution diminish the reliability of BMI as a standalone predictor of cardiometabolic risk.^{95,96}
- **Pacific Islander and Māori populations:** typically have different body composition, including higher lean mass, which may limit the accuracy of BMI as a standalone measure.⁹⁷
- **Middle Eastern, Black African and African Caribbean populations:** may face elevated cardiometabolic risk at lower BMI thresholds,^{92,98,99} however not all population-specific cut-off points have been adequately defined.
- **Asian populations:** in many Asian populations, increased cardiometabolic risk occurs at lower BMI levels. Consider adopting lower BMI thresholds for risk assessment. For example, BMI 23–27.5 kg/m² may indicate increased risk (overweight) and BMI ≥ 27.5 kg/m² may indicate high risk (obesity).⁹⁹
- To mitigate the many limitations associated with BMI, particularly in the populations noted above, BMI should always be measured alongside another anthropometric measure for a more accurate assessment of cardiometabolic risk, beyond obesity. See Tables 3,4.
- Waist-to-height ratio has the advantage of not requiring sex or ethnicity-specific cut offs, with a threshold (≥ 0.5) which can be applied universally, offering a more equitable alternative to BMI.¹⁰⁰⁻¹⁰² The practical translation of this is 'make sure your waist circumference is less than half your height'. See Table 4.
- Healthcare professionals should be aware of the impact of menopause on fat distribution. Declining oestrogen levels contribute to increased adipose distribution around the abdomen, elevating cardiometabolic risk. Measuring waist circumference and waist-to-height ratio is particularly important in this group for more accurate risk assessment.^{103,104}
- Dual-energy X-ray absorptiometry (DEXA) scans, impedance scales and emerging body composition technologies can offer insights into body fat percentage, distribution and metabolic variability among individuals with similar BMI.¹⁰⁵ Emerging international literature supports the complementary use of functional and metabolic phenotyping alongside traditional anthropometric assessment to better reflect the biological complexity of obesity.^{106,107} However, these tools are currently limited by cost, access and reproducibility in the general practice setting. At the time of writing, DEXA scans are only covered by the Medicare Benefits Schedule for assessment of bone mineral density.¹⁰⁸

Table 2: BMI classification for adults* ^{99,109-111}

Classification	BMI cut off (kg/m ²)
Healthy weight	18.5–24.9
Overweight	25.0–29.9
Obesity	≥ 30

* To calculate, divide body weight (kg) by height squared (m²) or refer to online calculators.

Table 3: Waist circumference risk thresholds* ^{109,110,112,114}

Cultural background and sex	Increased risk of cardiometabolic disease	Substantially increased risk of cardiometabolic disease
European descent – men	≥ 94 cm	≥ 102 cm
European descent – women	≥ 80 cm	≥ 88 cm
South Asian, Chinese, Japanese – men	≥ 90 cm	N/A
South Asian, Chinese, Japanese – women	≥ 80 cm	N/A

* Note that waist circumference is not a validated assessment tool in children or pregnant women. Cut-off points have not been established in First Nations peoples, Pacific Islander and Māori populations and Middle Eastern and other Asian populations. Clinical judgement should guide assessment.

Table 4: Waist-to-height ratio (WHtR)[‡] and waist-to-hip ratio (WHR)[#] ^{93,113}

Measure	Increased risk of cardiometabolic disease
Waist circumference to height	≥ 0.5
Waist circumference to hip	≥ 0.90 (men) ≥ 0.85 (women)

[‡] To calculate, divide waist circumference (cm) by height (cm) or refer to online calculators.

[#] To calculate, divide waist circumference (cm) by hip circumference (cm) or refer to online calculators.

Practical considerations for cardiometabolic health assessment in people living with overweight/obesity

- In people living with overweight or obesity, prioritise cardiometabolic health assessment to guide timely intervention.^{114,115} This may include:
 - assessing and documenting weight history, including recent weight change and intentionality for weight loss
 - identifying underlying factors that may be contributing to weight gain (e.g. hormonal conditions and some medications)¹¹⁶
 - assessing behavioural risk factors (e.g. nutrition, physical activity, smoking and alcohol use)
- identifying metabolic changes (e.g. hypertension, low high-density lipoprotein cholesterol (HDL-C), elevated triglycerides or elevated low-density lipoprotein cholesterol (LDL-C), an increased total cholesterol-to-HDL-C ratio (TC:HDL-C), impaired fasting glucose/ elevated HbA1c (between 5.7–6.4%))
- assessing family history of obesity, CVD or type 2 diabetes
- identifying organ dysfunction (e.g. liver dysfunction, early nephropathy, left ventricular hypertrophy)
- detecting comorbidities/obesity complications such as obstructive sleep apnoea

- consideration of sex-specific cardiovascular risk factors (e.g. history of gestational diabetes, hypertensive disorders of pregnancy or premature menopause).
- Comprehensive CVD risk assessment should be conducted as per the *Australian Guideline for assessing and managing cardiovascular disease risk*.²⁶ Those eligible for formal CVD risk assessment include people without known CVD aged 45–79, people with diabetes without known CVD aged 35–79, and First Nations peoples without known CVD aged 30–79 years.
- GPs can use Medicare Benefits Schedule item number 715 for Aboriginal and Torres Strait Islander health assessments to facilitate effective, holistic conversations about overall health including weight and CVD, recognising that identity, health and wellbeing are grounded in kinship, Country and culture, not only individual factors.
- Pre-clinical obesity is associated with increased long-term CVD risk.¹¹⁷ A proactive approach is recommended to prevent transition to clinical obesity, including appropriate referral to allied healthcare professionals and connection to community resources.⁹
- Evaluate physical function and factors that may influence mobility, independence, and quality of life.¹¹⁸ This can include brief functional assessments such as sit-to-stand or walking speed,¹¹⁹ clinical observation of movement, strength, and balance, and discussion of symptoms such as fatigue, musculoskeletal pain, or difficulties with everyday tasks.¹¹⁸ In settings where resources allow, additional assessments such as handgrip strength or body composition measures may inform a more comprehensive evaluation, particularly within multidisciplinary or specialist care.

Summary of the evidence

- BMI has been the basis for nearly all major weight-related intervention trials, including those evaluating behaviour modifications, pharmacotherapy and metabolic bariatric surgery. Although it serves as a cost effective and practical screening tool, it does not directly quantify adiposity and therefore cannot be relied upon alone to diagnose overweight or obesity.¹²⁰⁻¹²²
- Contemporary international consensus defines obesity as a disease of excess or dysfunctional adipose tissue, rather than excess body weight alone. The 2025 Lancet Diabetes & Endocrinology Commission (see Resources and further reading) emphasises that BMI should be used primarily for population-level surveillance and screening, with diagnosis requiring confirmation of excess adiposity with other anthropometric measures, and assessment of adiposity-related impairment of organ or tissue function.⁹
- Diagnostic frameworks increasingly distinguish between preclinical and clinical obesity based on health impact. This approach recognises excess adiposity as a continuum and prioritises diagnosis and treatment according to the presence of adiposity-related disease, functional limitation or end-organ damage.^{9,87}

Resources and further reading

- Definition and diagnostic criteria of clinical obesity

BMI calculators and waist circumference tables

- Heart Foundation: BMI calculator
- Heart Foundation: How to correctly measure your waist
- Department of Health, Disability and Ageing: BMI and waist measurement tool

Calculating waist-to-hip ratio and waist-to-height ratio

- World Health Organization: Waist circumference and waist-to-hip ratio
- Queensland Health: Body measurements (adult); includes instructions for measuring waist circumference and calculating waist-to-height ratio

Implications of obesity for cardiovascular diagnostics

In people with overweight or obesity undergoing cardiovascular investigations, tailored diagnostic approaches are required. Excess adiposity can complicate accurate evaluation and diagnosis of CVD by introducing structural and functional

challenges which can impact the reliability of several diagnostic procedures (Table 5).

Healthcare professionals should be aware of these imaging limitations and procedural challenges.

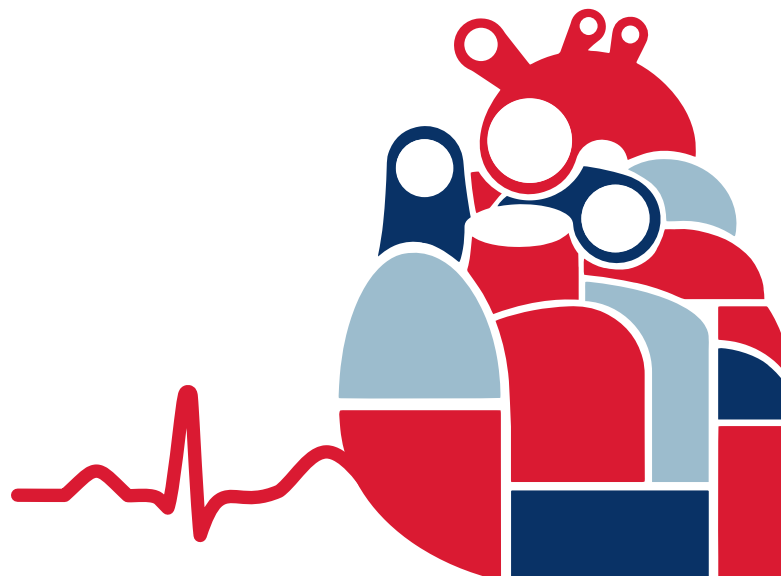
Where feasible and accessible, select modalities and techniques that accommodate body habitus. Prior to referral, consider confirming that the imaging or testing facility has the capacity to safely accommodate the person's body size and composition.

Table 5: Cardiovascular diagnostic tests that may be impacted by excess adiposity^{8,123-126}

Test	Impact of excess adiposity	Mitigation strategies
ECG+/- stress testing	<ul style="list-style-type: none"> • low QRS voltage • axis shifts • reduced ischaemia detection • limited ability to achieve target workload with standard exercise protocols 	<ul style="list-style-type: none"> • adjust lead placement • use modified exercise protocols or alternative exercise modalities (e.g. arm ergometry) where treadmill exercise is not feasible • may use dobutamine stress test as alternative to exercise stress testing
Transthoracic echo	<ul style="list-style-type: none"> • poor acoustic windows • limited wall motion visualisation 	<ul style="list-style-type: none"> • contrast-enhanced imaging improves endocardial definition • consider transoesophageal echo if feasible, especially if assessing valves
Stress echo	<ul style="list-style-type: none"> • image quality compromised during stress • limited ability to achieve target workload with standard exercise protocols 	<ul style="list-style-type: none"> • contrast-enhanced imaging improves endocardial definition • may use dobutamine stress test as alternative to exercise stress testing
Nuclear imaging (SPECT/PET)	<ul style="list-style-type: none"> • attenuation artefacts reduce perfusion accuracy • increased radiation dose required 	<ul style="list-style-type: none"> • apply attenuation correction • PET could be considered (if feasible/accessible)
Cardiac MRI	<ul style="list-style-type: none"> • generally unaffected • maintains high image quality 	<ul style="list-style-type: none"> • ensure tunnel diameter and weight limits are suitable

Test	Impact of excess adiposity	Mitigation strategies
Blood pressure measurement	<ul style="list-style-type: none"> • cuff sizing challenges may lead to inaccurate readings • discomfort may reduce adherence or cause incomplete readings 	<ul style="list-style-type: none"> • ensure appropriately sized cuffs are available • provide clear instructions and support to maximise wear time and data quality
Invasive coronary angiogram	<ul style="list-style-type: none"> • vascular access may be technically challenging • increased fluoroscopy time and radiation exposure • higher contrast volume may be required • procedural table weight limits and positioning challenges 	<ul style="list-style-type: none"> • consider radial access where feasible • optimise fluoroscopy settings and minimise exposure time • use contrast sparing techniques and monitor renal function • confirm table weight limits and procedural capability of the facility prior to referral
CT coronary angiogram	<ul style="list-style-type: none"> • image quality degrades with high BMI • increased radiation dose required 	<ul style="list-style-type: none"> • use dose modulation • optimise positioning • ensure tunnel diameter and weight limits are suitable

Abbreviations: BMI, body mass index; CT, computed tomography; ECG, electrocardiogram; MRI, magnetic resonance imaging; PET, positron emission tomography; SPECT, single photon emission computed tomography.



Section 2:

Behaviour modifications

Behaviour modifications are integral to the management of overweight/obesity and cardiovascular risk. Behaviour modifications to support long term risk reduction in this setting include addressing nutrition, physical activity, smoking and alcohol use, sleep and psychological wellbeing. Optimising nutrition and physical activity is an effective approach to reduce excess adiposity, improve cardiometabolic health and manage CVD risk.⁹¹ While nutrition and physical activity modifications confer modest weight loss compared with obesity management medications and metabolic bariatric surgery, they have consistently been shown to improve blood pressure, lipid profiles and cardiovascular outcomes, independent of weight loss.^{127,128}

Physical activity and nutrition vitally underpin other obesity management interventions, including pharmacotherapy and metabolic bariatric surgery, to maintain optimal nutritional status and musculoskeletal health, and to reduce CVD risk. These behaviour changes should continue across the life course, even when weight goals are achieved, to both maintain health benefits and manage CVD risk in the longer term (the latter should include management of all CVD risk factors including smoking and alcohol intake).

In clinical practice, co-prescribing nutrition and physical activity interventions is more effective in improving cardiovascular and metabolic outcomes than either intervention alone.¹²⁹ Social prescribing of physical activity interventions, in particular, has been shown to improve activity levels, quality of life and social isolation.^{130,131}

To promote adherence to behaviour modifications, advice and programs should be person-centred and tailored to each person's ability, physiology, comorbidities and preferences (including cultural), and focus on gradual, achievable goals co-developed with their healthcare team. Multidisciplinary support from a person's GP, nurses and nurse practitioners, other specialists, allied healthcare professionals and mental health professionals (where relevant) is also critical to achieving improved health outcomes. Medicare GP Chronic Condition Management Plans can facilitate this, enabling subsidised access to allied healthcare professionals.

Nutrition

Recommendations	Strength of advice
In adults living with overweight or obesity and cardiovascular disease (CVD) or at high risk of CVD, recommend a heart-healthy eating pattern, which includes a wide variety of foods from all food groups, is naturally low in unhealthy fats, salt and added sugars, and limits discretionary food/beverages to reduce cardiovascular risk.	Moderate advice ● ● ○
For weight loss in adults living with overweight or obesity and CVD or at high risk of CVD, recommend a heart-healthy eating pattern with reduced energy intake (at least 2000 kJ/day deficit from estimated daily energy requirements) with a goal of achieving at least 5% weight loss over six months.	Strong advice ● ● ●
In adults living with overweight or obesity and CVD or at high risk of CVD who require more rapid weight loss to help manage complications, recommend either a low energy diet (LED) or very low energy diet (VLED) under clinical supervision.	Strong advice ● ● ●

Practice points

Practical dietary advice

- The Heart Foundation's heart-healthy eating pattern incorporates principles of the Dietary approaches to stop hypertension (DASH) and Mediterranean diets, which are associated with improvements in cardiovascular risk factors.
- Encourage people to adopt a heart-healthy eating pattern which consists of:
 - plenty of vegetables and fruits
 - a variety of healthy protein-rich foods including legumes, nuts and seeds, fish and seafood. Include smaller amounts of eggs and lean poultry and limit red meat to 1–3 times a week
 - unsweetened milk, yoghurt and cheese (reduced fat varieties are the preferred choice for people with elevated LDL-C and those with coronary heart disease)
 - a variety of wholegrains rich in dietary fibre including grainy bread, wholemeal pasta, oats and brown rice
 - healthy fats and oils like avocados, olives, unsalted nuts and seeds and their oils for cooking
 - herbs and spices to flavour food instead of adding salt.
- Limit sodium intake to less than 2,000 mg per day, including sodium from processed foods. Excess dietary salt contributes to elevated blood pressure, a key modifiable risk factor for CVD. To help reduce sodium intake, particularly for those who add salt to their food, low-sodium, potassium-enriched salt substitutes may be considered unless contraindicated (e.g. chronic kidney disease, hyperkalaemia).¹³²
- Recommend reducing snacking frequency and choosing nutrient-dense snacks, while limiting discretionary and highly processed foods and sugar-sweetened beverages, to support appropriate energy intake and improve overall diet quality.
- Frozen or canned fruits, vegetables and legumes (with no added sugars or salts) can be appropriate alternatives to fresh fruits and vegetables when access and availability is limited.
- Access to fresh fish and seafood is variable across Australia. It is also expensive in some regions. Frozen and canned fish are acceptable alternatives. For canned varieties, encourage people to opt for products stored in spring water or olive oil rather than brine.
- Actively assess and limit intake of sugar-sweetened beverages (including soft drinks, flavoured milk, energy drinks and sweetened juices). Promote water as the drink of choice for hydration and health, encouraging it as a swap for sugar-sweetened beverages and alcohol, supported by gradual substitution strategies. Educate people that there is no safe limit of alcohol intake for heart health. Sugar-sweetened beverages and alcohol are nutrient-poor, calorie-dense fluids that can contribute to weight gain.

Reduced energy intake

- For weight loss, a person's daily energy intake should be at least 2,000 kJ (approximately 500 kcal) below their daily energy requirement. The target reduced energy intake should be further tailored to the individual's nutritional needs, medical conditions, cultural preferences and lifestyle to enhance adherence and effectiveness.
- Use a daily energy requirement calculator to calculate a person's daily energy requirements based on their target body weight (see Resources and further reading).
- To support kilojoule reduction, provide education on tracking intake using electronic or paper-based tools such as a diary or mobile apps. Several mobile apps are available in Australia. When recommending an app, ensure it is evidence-based, encourages behaviour change and aligns with reliable Australian nutrient databases and evidence-based resources on healthy eating such as the *Australian Dietary Guidelines*. People with limited health literacy or numerical literacy should be referred for more appropriate, tailored support.

- To minimise the risk of malnutrition and preserve nutritional status, energy restriction should not be recommended for individuals with conditions characterised by established catabolic dominance, such as cancer, unless under specialist supervision.
- Although caloric restriction may yield short-term improvements in weight and quality of life among people with heart failure, its use warrants caution due to observed signals of increased mortality and heart failure-related rehospitalisation.¹³³

Low energy diets (LEDs) and very low energy diets (VLEDs)

- An LED may be required to achieve steady weight loss, while a VLED may be considered where more rapid weight loss (up to 2 kg per week) is clinically indicated. The need for rapid weight loss should be guided by the presence and severity of obesity-related complications and comorbidities, such as advanced and/or poorly controlled cardiometabolic disease, the need for urgent pre-procedural or pre-operative risk reduction, or obesity-related conditions that directly exacerbate cardiovascular risk.
- LEDs set a daily energy intake target and encourage monitoring to achieve a specific deficit relative to energy expenditure. For an LED, the daily energy intake target is limited to 4,200–7000 kJ (approximately 1000–1700 kCal) per day.¹³⁴
- VLEDs most commonly use formulated meal replacement products to achieve a protein-sparing modified fast that facilitates rapid weight loss. For a VLED, the daily energy intake target is limited to 2700–3700 kJ (approximately 650–900 kCal) per day.¹³⁵
- For a VLED, limiting the kilojoule intake can be achieved by initially introducing formulated meal replacement products, plus some non-starchy vegetables and a small amount of oil. Individuals should receive appropriate education and support, including clear information that VLEDs alone may not be nutritionally complete and typically require clinical supervision, as they can be associated with adverse effects.¹³⁶
- VLEDs should be supported by coordinated care and ongoing clinical supervision. Where available, support from an Accredited Practising Dietitian is recommended to ensure safe and effective implementation, monitoring and maintenance once ceased. Where access is limited, appropriately trained healthcare professionals, including general practitioners or nurse practitioners, may provide dietary counselling and ongoing clinical monitoring.

Other dietary approaches

- The effectiveness of intermittent fasting for weight loss remains inconclusive, as current evidence demonstrates mixed results.¹³⁷⁻¹³⁹ While this approach can reduce weight compared with unrestricted eating, recent studies suggest there are no consistent benefits over continuous calorie restriction when energy intake is similar.^{140,141}
- Lower-carbohydrate eating patterns may be considered for selected individuals (e.g. those with insulin resistance, type 2 diabetes, metabolic syndrome or dyslipidaemia) to support short-term improvements in cardiometabolic risk markers. However, evidence for sustained weight loss or cardiovascular benefit is limited.¹⁴²
- Higher-protein eating patterns may be considered in selected individuals to support satiety, preservation of lean mass and short-term improvements in body composition and selected cardiometabolic risk markers. However, evidence for sustained weight loss or cardiovascular benefit is limited, and such approaches should prioritise healthy protein within nutritionally balanced, minimally-processed dietary patterns.¹⁴³

Considerations for providing nutritional advice

- Use easy to understand and culturally relevant visual aids (plates, bowls, culturally familiar foods) when explaining portion sizes, 'extras'/discretionary items and heart-healthy eating patterns (e.g. more vegetables/legumes, less deep-fried foods, sugary drinks).
- Ensure dietary advice is culturally appropriate and considers food availability, affordability, traditional

nutrition and cooking practices and individual preferences across diverse communities to achieve long term adherence.¹⁴⁴ Consider both health literacy and culinary literacy when providing dietary advice to ensure practical understanding and application.

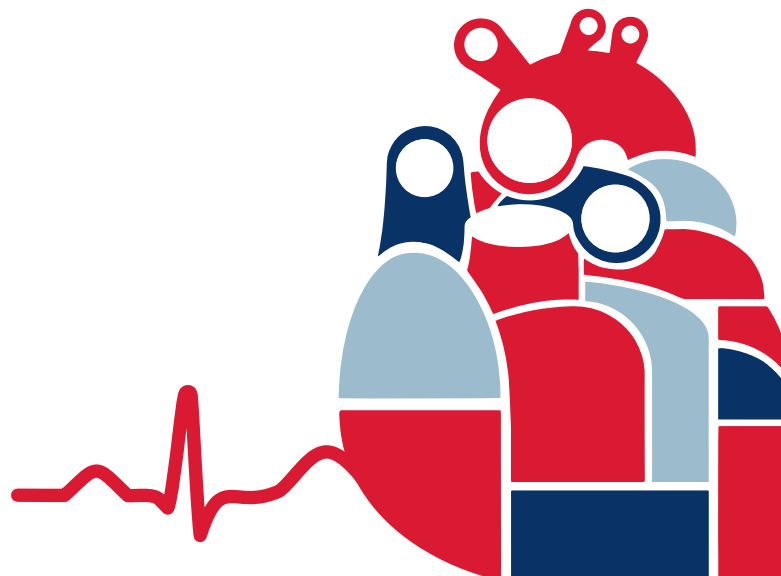
- Incorporate food label literacy into education by referring people to relevant online tools and resources (see Resources and further reading) or an Accredited Practising Dietitian or structured programs (e.g. community health clinics offering supermarket tours) for interpretation of nutrition panels and ingredient lists as part of comprehensive dietary counselling.
- Recognise that behaviour change is strongly influenced by psychological and emotional factors and that emotional eating is commonly reported in people living with overweight or obesity. Addressing the underlying drivers is essential to achieving sustained behaviour change. Consider referral to mental health professionals where appropriate.

For dietary advice for people taking incretin-based medications – refer to the Pharmacotherapy section for further detail.

For dietary advice for people undergoing metabolic bariatric surgery – refer to the Surgical interventions section for further detail.

Multidisciplinary care

- Consider referral to an Accredited Practising Dietitian for individualised nutrition care, including support for LED/VLED use where appropriate, and for people prescribed incretin-based therapies to minimise muscle loss, nutrient deficiencies and gastrointestinal side effects.
- Where access to an Accredited Practising Dietitian is limited, appropriately trained healthcare professionals may provide dietary support. Regular reviews should be scheduled based on individual clinical needs. For example, monthly reviews in primary care settings may be appropriate, which can be distributed across the multidisciplinary team.
- Leverage existing Medicare-subsidised primary care services (e.g. Mental Health Treatment Plans and Chronic Condition Management Plans) to enable multidisciplinary care, recognising that increased and regular touchpoints are associated with improved outcomes.¹²¹
- Where an eating disorder is suspected, refer for assessment under an Eating Disorder Treatment and Management Plan and to an appropriate mental health professional to support safe, coordinated care.



Summary of the evidence




- Observational and cohort studies demonstrate that healthy eating patterns are associated with a reduction in CVD risk^{145,146} and mortality.^{145,147}
- A network meta-analysis that compared 14 diets in adults with overweight or obesity demonstrated that most diets at six months led to a modest and comparable weight reduction of approximately 4–5 kgs and cardiovascular risk factor improvements (systolic blood pressure ~3–5 mmHg reduction; LDL-C ~0.03–0.18 mmol/L reduction; HDL-C ~0.05 mmol/L increase). The majority of effects diminished at 12 months, except for the reduction in LDL-C with Mediterranean diets (noting adherence was not generally reported).¹⁴⁸
- When dietary interventions are supported by dietitians, modest improvements in body weight, blood pressure and selected CVD risk factors have been observed.¹⁴⁹ Strong evidence demonstrates that reducing dietary salt intake results in decreased systolic and diastolic blood pressure.^{132,150,151}
- Reduced energy intake, typically creating a daily energy deficit of ~2000–3000 kJ (approximately 500–700 kCal), is widely recommended for weight management in adults with overweight or obesity.¹⁵² Reduced energy intake approaches often vary by macronutrient composition, including low carbohydrate and/or low fat, or may include partial meal replacement strategies to achieve a reduction in total energy intake. Evidence from randomised controlled trials and meta-analyses show that reduced energy intake diets lead to modest but clinically meaningful weight loss (~4–8 kg at six months)^{148,152,153} and modest improvements in cardiovascular risk factors, including reductions in systolic blood pressure.¹⁴⁸ Most benefits peak at six months without ongoing support, and by 12 months the effects on weight loss and improvements in cardiovascular risk factors are largely diminished.¹²⁸

- A primary care-randomised controlled trial found that a VLED delivered via total diet replacement resulted in greater weight loss at 12 months than usual care (mean difference 7.2 kg; 95% CI 4.9–9.4).¹⁵⁴

Resources and further reading

- Eatforhealth.gov.au: Daily energy requirements calculator
- Eatforhealth.gov.au: Eat for Health resources
- Heart Foundation:
 - Nutrition position statements and evidence summaries and reviews
 - Heart-healthy recipes
 - Reading food labels and other resources
- RACGP: Smoking, nutrition, alcohol, physical activity (SNAP)
- Refer to PHN HealthPathways for local referral pathways to nutrition supports
- LiveLighter: Recipes, practical tips and resources
- Healthy Eating With No Money & No Time: Recipes, practical tips and resources

Physical activity

 Recommendations	Strength of advice
In adults living with overweight or obesity and CVD or at high risk of CVD, recommend regular physical activity across the course of the day while reducing sedentary behaviour, irrespective of impact on weight, to support cardiovascular and overall health.	Strong advice 
For adults living with overweight or obesity and CVD or at high risk of CVD, recommend an exercise routine combining moderate-to-vigorous aerobic activity and resistance training, tailored to a person's goals, to support cardiovascular and overall health.	Strong advice 

Practice points

General considerations

- Educate people that whilst physical activity alone typically results in only modest weight loss, it can improve waist circumference, blood pressure, blood glucose, lipid profile and cardiorespiratory fitness and reduce cardiovascular risk.¹²⁷ Physical activity has other benefits for health including reduced risk of type 2 diabetes,¹⁵⁵ dementia (including Alzheimer's disease)¹⁵⁶ and other chronic diseases (including some cancers).¹⁵⁷
- Encourage and assist people to set small, measurable and realistic goals they can achieve early to build confidence and momentum. Support people to build volume of activity (total amount of activity), before focusing on increasing the intensity, especially in those who are just beginning or returning to physical activity, to reduce the risk of injury.
- In some cases, overweight/obesity is a barrier to being physically active. Adopt a person-centred approach in developing a physical activity and/or exercise program, with consideration given to current physical limitation, pain management, concurrent musculoskeletal injuries or disease, and stigma. Management of these conditions can improve physical activity and exercise tolerance. Advise people that any volume of physical activity is better than none.
- Where appropriate, refer to an appropriately qualified exercise specialist, such as an Accredited Exercise Physiologist or Physiotherapist, to offer tailored guidance, support safe progression and improve confidence with physical activity. A referral is particularly appropriate when overweight/obesity co-exists with cardiac limitations, type 2 diabetes, hypertension, metabolic syndrome, chronic kidney disease (early stages) and/or other co morbid conditions (e.g. cancer).
- Use GP Chronic Condition Management Plans, which can support subsidised access to individual or group-based exercise sessions.
- Link people from culturally and linguistically diverse (CALD) backgrounds to community and faith-based programs that feel safe and familiar (e.g. walking groups, women-only exercise sessions, culturally tailored health groups).

Types of physical activity

- Recommend 30 minutes or more of moderate-to-vigorous intensity aerobic physical activity on most days of the week (such that there is an accumulation of 2.5 hours or more per week) for cardiovascular, metabolic and health outcomes.^{158,159} The 'talk test' can be used to judge intensity of exercise. Moderate activity allows comfortable conversation, while vigorous activity makes talking difficult.¹⁶⁰ See Table 6 for specific physical activity recommendations for weight management goals, noting that these goals are general in nature and should be tailored to the individual.
- Recommend resistance training 2–3 times per week in combination with moderate-to-vigorous physical activity.¹⁵⁸ Resistance training is particularly beneficial for older adults and post-menopausal women to preserve bone density and muscle mass, improve functional capacity and reduce central adiposity.^{158,161-163} Where an individual is unable to perform both aerobic and resistance training, prescribing and engaging in either modality is substantially better than remaining inactive.
- Advise people to limit sedentary behaviour by minimising prolonged sitting and regularly breaking up sitting time throughout the day.¹⁵⁸
- Encourage activities that are enjoyable, accessible and easily maintained (e.g. walking, swimming, cycling or home-based programs) and personalise physical activity plans based on the person's preferences (e.g. individual or group activities), functional capacity, comorbidities, safety, access to resources and social/cultural context.
- Provide education that physical activity can be accumulated throughout the day rather than all at once. A practical strategy can be to advise people to adopt 'exercise snacking'.¹⁶⁴ This is brief bursts of moderate-to-vigorous movement incorporated within a person's daily routine. For example, walking to public transport or taking the stairs contributes meaningfully. This approach has been shown to improve cardiorespiratory fitness and can be more achievable and sustainable for people than longer sessions, especially those who identify as time poor.¹⁶⁵
- A daily target of ~7,000 steps (Table 7) can be a realistic and achievable goal for many people and is associated with measurable reductions in cardiovascular risk and overall mortality compared with lower step counts.¹⁶⁶ Wearable devices and tracking apps can monitor step count and keep people motivated.¹⁶⁷
- High intensity interval training may be particularly beneficial for improving cardiorespiratory fitness and reducing visceral fat, but should only be considered after comprehensive cardiovascular risk assessment and undertaken within a supervised setting.¹⁶⁸ Intensity should be tailored to the individual.¹⁶⁸

Table 6: Summary of Exercise & Sport Science Australia (ESSA) position statement - physical activity in the management of obesity in adults* ¹⁶⁹

Goal	Minutes per week	Type of physical activity
Prevention of weight gain and adiposity gain	>150 min but preferably 300	Moderate-vigorous intensity aerobic activity
Weight loss and total adiposity loss	Minimum of 300–420	Moderate-vigorous intensity aerobic activity
Prevention of weight regain	300	Moderate-vigorous intensity aerobic activity

* ESSA position statement is not specific to people living with CVD.

Table 7: Building steps into a sedentary workday: a practical example

Activity	Approx. steps	Cumulative daily total
Baseline (sedentary worker) – office day	~3,500 steps	3,500
Short morning walk (10 mins, ~1,000 steps)	+1,000	4,500
Walking during lunch break (10 mins, ~1,000)	+1,000	5,500
Evening walk after dinner (15 mins, ~1,500)	+1,500	7,000

Considerations in people with established CVD

- Before an individual with established CVD starts a moderate-to-vigorous physical activity program, complete a comprehensive pre-exercise clinical screening. If other relevant medical conditions are present (e.g. osteoporosis), ensure further assessment, screening and appropriate clinical support. This enables safe physical activity prescription, allows tailoring to underlying conditions, and reduces the risk of adverse outcomes.
- Refer to Sports Medicine Australia's *Pre-exercise Screening and Risk Factor Assessment guide* for more information (see Resources and further reading).

For people on incretin-based pharmacotherapy

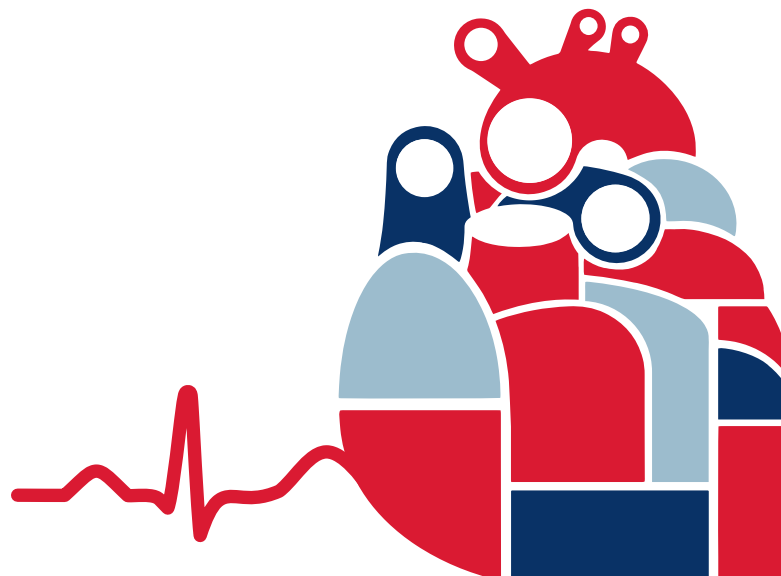
- Individuals on incretin-based pharmacotherapies are at increased risk of lean muscle loss and sarcopenic obesity.^{170,171} Regular structured resistance training two days per week or more is important to preserve lean muscle mass during weight loss.¹⁷²

Summary of the evidence

- Aerobic exercise appears to be most effective in reducing BMI and body weight, with improvements ranging from a 0.9 to 1.4 kg/m² reduction in BMI and 2–4 kg in weight.¹⁷³ In adults with metabolic syndrome, aerobic exercise has also been found to reduce waist circumference (-3.4 cm), fasting glucose (-0.15 mmol/L), triglycerides (-0.3 mmol/L), and improve HDL-C (0.05 mmol/L) and cardiorespiratory fitness (VO₂max) by approximately 4.2 mL/kg/min.¹⁷⁴
- In people with overweight or obesity specifically, resistance training has been shown to improve diastolic blood pressure (-2.96 mmHg; 95% CI -5.22–-0.70) vs control).¹⁷⁵
- Combined aerobic and resistance training is likely the most effective type of physical activity for improving cardiovascular risk factors in people with overweight or obesity. This type of training has been shown to reduce blood pressure (systolic -5.6 mmHg; diastolic -4.7 mmHg), fasting glucose (-0.3 mmol/L) and body fat (-2.8%), with reductions of ~2.6 kg in body weight and ~4 cm in waist circumference observed.¹⁷⁵
- Short bursts of intermittent physical activity have been shown to reduce the risk of major adverse cardiovascular events in people who are inactive. A 2023 prospective cohort study in people with a mean BMI in the overweight range (i.e. BMI 25.0–29.9 kg/m²) found that, compared with bouts of physical activity lasting less than a minute, the risk of major adverse cardiovascular events was lower for bouts lasting 1 to < 3 minutes (HR 0.71; 95% CI 0.54–0.93), 3 to < 5 minutes (HR 0.62; 95% CI 0.48–0.81) and 5 to < 10 minutes (HR 0.59; 95% CI 0.46–0.76).¹⁷⁶

Resources and further reading

- RACGP: Smoking, nutrition, alcohol, physical activity (SNAP)
- Department of Health, Disability and Ageing: movement guidelines for all Australians 2026
- AUSactive and Sports Medicine Australia: Pre-exercise screening and risk factor assessment
- Heart Foundation: Physical activity resources for health professionals
- Heart Foundation: Heart Foundation Walking
- LiveLighter: Recipes, practical tips and resources



Sleep

There is growing evidence to support the integration of sleep assessment and management into routine obesity and CVD management. Short sleep (six hours or less per night for adults) and poor sleep quality (insufficient, fragmented, non-restorative, or associated with daytime impairment, such as fatigue or reduced functioning) independently increase cardiometabolic risk and promote weight gain by impairing glucose tolerance and decreasing insulin sensitivity and leptin production, while increasing appetite.¹⁷⁷⁻¹⁷⁹ In addition, sleep deprivation contributes to daytime fatigue, reduced motivation and impaired recovery, which together can reduce engagement in physical activity and adherence to healthy behaviour modifications.¹⁸⁰

Weight loss is effective in improving sleep.¹⁸¹ While improved sleep duration and quality may have an effect on reducing cardiometabolic risk factors, independent of weight loss, further research is required.^{182,183}

Practice points

- Sleep should be routinely assessed in adults with obesity and increased cardiovascular risk.¹⁸⁴ Assessment should include sleep duration, perceived sleep quality, sleep regularity, daytime sleepiness or fatigue, and symptoms suggestive of common sleep disorders. Validated tools suitable for primary care include short questionnaires such as the Insomnia Severity Index or Epworth Sleepiness Scale, which can help identify clinically significant sleep disturbance and guide management or referral.
- Improving sleep duration and/or quality may enhance a person's capacity to engage in physical activity and adhere to dietary changes.¹⁸⁵ Educate about sleep hygiene, including maintaining regular sleep and wake times, limiting caffeine and alcohol intake (particularly in the evening), reducing screen exposure before bedtime, and promoting a sleep conducive environment.
- Referral to sleep specialists should be considered for people with suspected obstructive sleep apnoea (see Section 6. Other populations and conditions), treatment resistant insomnia or complex comorbidities.

Resources and further reading

- Australian Journal of General Practice: General practitioner assessment and management of insomnia in adults
- Sleep Health: Primary Care Resources
- Australian Prescriber: Management of insomnia in primary care

Section 3:

Pharmacological and surgical interventions

Pharmacological and surgical interventions play a critical role in the management of overweight and obesity, particularly among those at high cardiovascular risk or with established CVD.^{186,187}

Pharmacotherapy is indicated for obesity management, in conjunction with behaviour modifications, when adequate weight-related health improvements cannot be attained through behaviour modifications alone. Weight loss of approximately 5–10% of total body weight is generally required to attain these weight-related health improvements.^{188,189} Pharmacotherapy should complement, not replace, behaviour modifications.^{106,190,191}

Incretin-based medicines (including GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists) have changed the landscape of obesity management. High quality trials have demonstrated incretin-based medicines improve cardiometabolic risk factors and, in some cohorts, reduce the risk of major adverse cardiovascular events.^{11,12} Across trials, they have consistently led to substantial weight loss, achieving reductions exceeding 10% of baseline body weight.^{11,190,192,193} To note, real world observational data suggest that average weight loss is lower than that seen in randomised controlled trials due to a combination of lower achieved dosages, treatment interruptions and high discontinuation rates (up to 20–50% within the first year).^{194,195}

General practitioners are well-placed to support people in commencing obesity management medications and to support ongoing care. Weight regain is common after ceasing these agents, therefore they are likely to be needed long term.¹⁹⁶

For people who are unable to attain weight-related health improvements with behaviour modifications and pharmacotherapy, or unable to sustain weight loss after treatment discontinuation, referral to specialised care is generally required. This may include consideration of metabolic bariatric surgery to achieve weight goals and improve cardiovascular health.

Multiple systematic reviews and meta-analyses, of predominantly cohort studies, have shown that metabolic bariatric surgery is consistently associated with reduced cardiovascular morbidity and mortality among individuals with obesity (predominantly in people at high risk of CVD with some lower quality evidence demonstrating benefit in people with established disease).^{187,197-202}

Procedures such as sleeve gastrectomy and Roux-en-Y gastric bypass not only achieve sustained weight loss, but can also induce remission of type 2 diabetes, lower blood pressure, improve lipid profiles, and may reduce the risk of heart failure exacerbations.²⁰³⁻²⁰⁶ Metabolic bariatric surgery has also been associated with short term improvements in mental health, depressive symptoms and quality of life, but longer-term mental health outcomes are heterogeneous.²⁰⁷⁻²⁰⁹






Access and cost remain significant barriers for obesity management medications and metabolic bariatric surgery. There are currently no Therapeutic Goods Administration-approved incretin-based medicines available on the Pharmaceutical Benefits Scheme (PBS) for obesity management (Table 8)*. This means the cost remains prohibitive for many people.²¹⁰ At the time of writing, the Pharmaceutical Benefits Advisory Committee has made a positive recommendation for semaglutide in a subgroup of people with obesity and established CVD.²¹¹

Similarly, metabolic bariatric surgery has limited availability in the vast majority of Australian public hospitals, with more than 90% of procedures taking place in the private sector.²¹² Financial barriers are likely to compound inequities given the disproportionate burden of obesity and CVD in priority populations who may have limited access to private medical and surgical care.²¹⁰ With a growing prevalence of obesity and overweight in Australia, expediting mechanisms to improve access to multidisciplinary obesity care is critical.

For further detail on the role of pharmacological and surgical interventions, refer to *Management of overweight and obesity in adults with established CVD* (pg. 49-50).

* Pharmaceutical Benefits Scheme criteria noted throughout this consensus statement is correct at the time of writing. Refer to PBS website for current criteria and further information.

Pharmacotherapy

 Recommendations	Strength of advice
Obesity management medications are indicated, in conjunction with behaviour modifications, when adequate weight-related health improvements cannot be attained through behaviour modifications alone.	Strong advice 
In adults living with established atherosclerotic CVD and BMI ≥ 27 kg/m ² , consider prescribing a GLP-1 receptor agonist with proven CVD benefit (semaglutide) to reduce the risk of major adverse cardiovascular events.	Moderate advice 
In adults living with heart failure with preserved ejection fraction (HFpEF) and obesity ^a , consider semaglutide or tirzepatide to improve symptoms and functional capacity.	Moderate advice 
In adults living with type 2 diabetes and overweight or obesity, with or without CVD, consider a GLP-1 or GIP/GLP-1 receptor agonist with proven CVD benefit (semaglutide, liraglutide or tirzepatide) to reduce the risk of major adverse cardiovascular events.	Strong advice 

^a Defined in trials as BMI ≥ 30 kg/m² and ejection fraction $\geq 50\%$ (SUMMIT, tirzepatide) or ejection fraction $\geq 45\%$ (STEP-HFpEF, semaglutide). While these medications have demonstrated benefit for heart failure symptoms and quality of life, they are not yet approved by the Therapeutic Goods Administration for management of this condition.

General information

Obesity management medications approved for use in Australia by the Therapeutic Goods Administration (TGA) are listed in Table 8.

The injectable GLP-1 receptor agonists (e.g. semaglutide, liraglutide) and GIP/GLP-1 receptor agonists (tirzepatide) promote weight loss via several different mechanisms of action (Figure 5). Cardiovascular outcome trials of these agents have also demonstrated improvement in cardiometabolic markers including blood pressure and lipid profiles.^{190,213}

GLP-1 receptor agonists act by binding to and amplifying activation of the GLP-1 receptor, resulting in enhanced post-prandial insulin secretion, glucagon release inhibition, delayed gastric emptying and appetite suppression.²¹⁴

The cardioprotective effects of semaglutide may be independent of its impact on weight, suggesting additional underlying mechanisms, including anti-inflammatory effects such as reductions in systemic inflammatory markers, which may contribute to cardiovascular benefit.^{215,216}

The GIP/GLP-1 receptor agonists have an additional agonism effect at the GIP receptor, resulting in increased insulin secretion in a glucose dependent manner and combined effects on appetite regulation and weight loss.^{217,218}

Table 8: Therapeutic Goods Administration-approved obesity management medications, their indications and relevant PBS criteria

Medication name	TGA indications	PBS criteria (for obesity management)
Incretin-based agents		
Semaglutide	Management of weight in adults with a BMI ≥ 30 kg/m ² or ≥ 27 kg/m ² in the presence of one or more weight-related complications.	Not included on PBS*
Tirzepatide	Management of weight in adults with a BMI ≥ 30 kg/m ² or ≥ 27 kg/m ² in the presence of one or more weight-related complications.	Not included on PBS
Liraglutide	Management of weight in adults with a BMI ≥ 30 kg/m ² or ≥ 27 kg/m ² in the presence of one or more weight-related complications.	Not included on PBS
Oral agents		
Orlistat	Management of weight in adults with a BMI ≥ 30 kg/m ² or ≥ 27 kg/m ² in the presence of one or more weight-related complications.	Included on Repatriation Pharmaceutical Benefits Scheme, for individuals with BMI ≥ 35 kg/m ² with no known co-morbidities or BMI ≥ 30 kg/m ² with at least one co-morbidity
Naltrexone/bupropion	Management of weight in adults with a BMI ≥ 30 kg/m ² or ≥ 27 kg/m ² in the presence of one or more weight-related complications.	Not included on PBS
Phentermine	Management of weight in adults with a BMI ≥ 25 kg/m ² .	Not included on PBS

* At the time of writing, the Pharmaceutical Benefits Advisory Committee has made a positive recommendation for semaglutide in a subgroup of people with obesity and established CVD.²¹¹

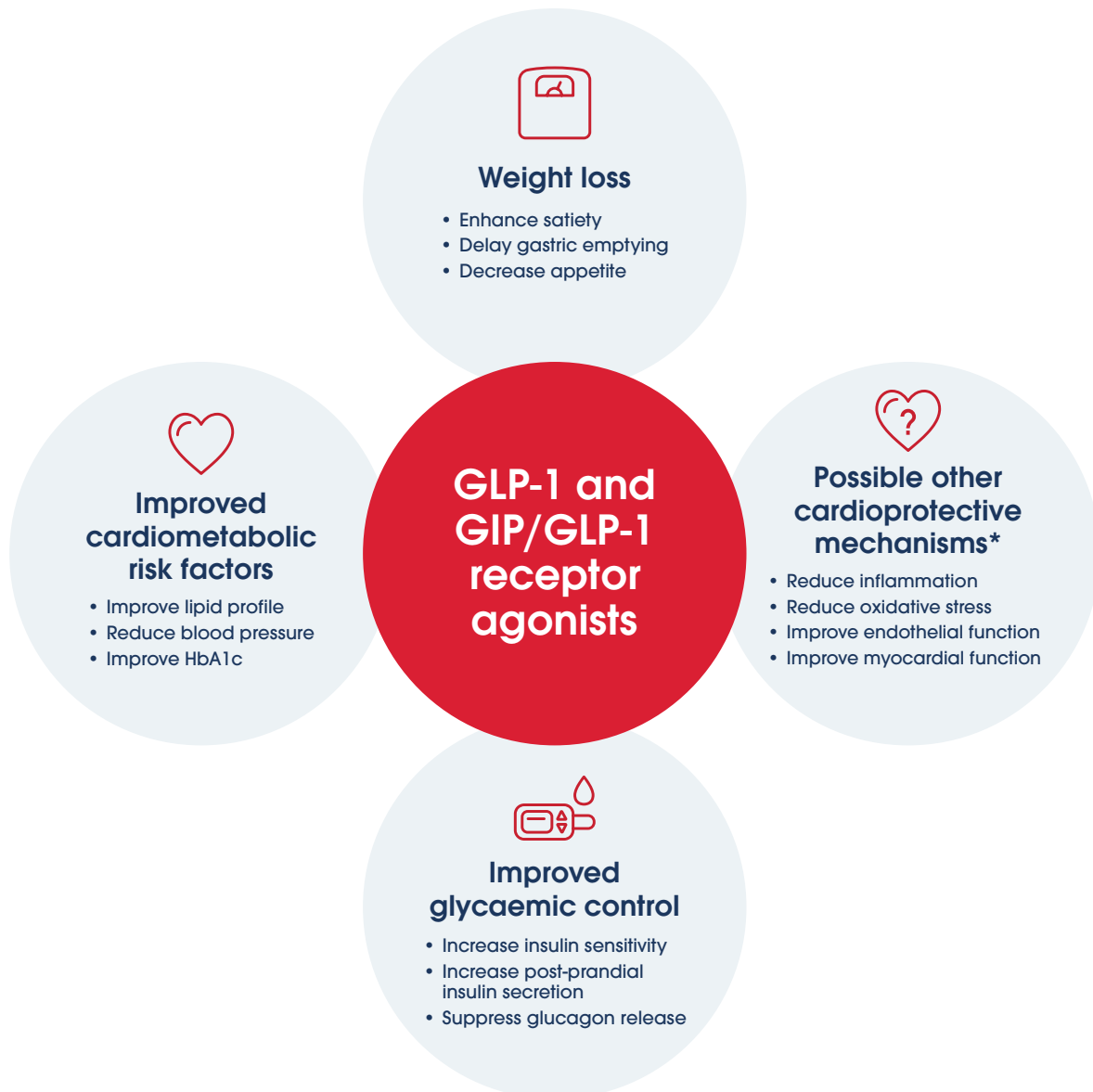


Figure 5: Mechanisms of action of GLP-1 receptor agonists and GIP/GLP-1 receptor agonists.

*Further research is required to fully understand the cardioprotective mechanisms of these agents.

Abbreviations: GIP, glucose-dependent insulinotropic polypeptide; GLP-1, glucagon-like peptide-1; HbA1c, haemoglobin A1c.

Compared with injectable pharmacotherapies, the oral agents orlistat, naltrexone/bupropion and phentermine are less effective for weight loss and lack CVD outcome data (Table 9).

For phentermine, there are several CVD-related contraindications (e.g. valvular disease, arrhythmias

and advanced arteriosclerosis) and precautions (e.g. mild hypertension). Similarly, naltrexone/bupropion is contraindicated in people with uncontrolled hypertension and is cautioned in people with controlled hypertension.²²³⁻²²⁵ Refer to individual agent product information for more detail.

Table 9: Pharmacotherapies for obesity management^{†219,223,224,226-243}

Medication name and medication class	Mechanism of action	TGA indications for obesity management in adults*	Dose	Common side effects	Efficacy for weight loss (approximate % of total body weight lost ^{‡§})	Impact on CV outcomes compared with placebo
Incretin-based agents						
Semaglutide (Wegovy) GLP-1 receptor agonist	<ul style="list-style-type: none"> • Enhances satiety • Delays gastric emptying • Decreases appetite 	BMI ≥ 30 kg/m ² OR ≥ 27 kg/m ² in the presence of one or more weight-related complications	<ul style="list-style-type: none"> • Start at 0.25 mg subcut. once weekly • Titrate: <ul style="list-style-type: none"> - Weeks 1–4: 0.25 mg - Weeks 5–8: 0.5 mg - Weeks 9–12: 1 mg - Weeks 13–16: 1.7 mg - Maintenance dose: 2.4 mg once weekly 	Nausea, vomiting, diarrhoea, constipation, dyspepsia, abdominal pain, injection site reactions	9–10% ²²⁸	<ul style="list-style-type: none"> • Reduced risk of CV death, non-fatal MI and non-fatal stroke in people with established atherosclerotic CVD (without type 2 diabetes)²²⁸ and in people with type 2 diabetes at high risk of CVD²²⁹ • Reduced risk of CV events in people with type 2 diabetes and chronic kidney disease²⁴⁴
Tirzepatide (Mounjaro) GIP/GLP-1 receptor agonist	<ul style="list-style-type: none"> • Enhances satiety • Delays gastric emptying • Decreases appetite 	BMI ≥ 30 kg/m ² OR ≥ 27 kg/m ² in the presence of one or more weight-related complications	<ul style="list-style-type: none"> • Start at 2.5 mg subcut. once weekly. After four weeks, increase dose to 5 mg subcut. once weekly • If needed, dose increases can be made in 2.5 mg increments • Maximum dose is 15 mg once weekly 	Nausea, vomiting, diarrhoea, constipation, dyspepsia, abdominal pain, injection site reactions	11–12% ²⁴⁵	Reduced risk of CV death, non-fatal MI and non-fatal stroke in people with type 2 diabetes and atherosclerotic CVD ^{245#}
Liraglutide (generics available) GLP-1 receptor agonist	<ul style="list-style-type: none"> • Enhances satiety • Delays gastric emptying • Decreases appetite 	BMI ≥ 30 kg/m ² OR ≥ 27 kg/m ² in the presence of one or more weight-related complications	<ul style="list-style-type: none"> • Start at 0.6 mg subcut. once daily • Increase weekly by 0.6 mg (as tolerated) to a maintenance dose of 3.0 mg once daily 	Nausea, vomiting, diarrhoea, constipation, dyspepsia, abdominal pain, injection site reactions	6–8% ^{246 ζ}	Reduced risk of CV death, non-fatal MI or non-fatal stroke in people with type 2 diabetes at high risk of CVD ²²⁷

Footnotes:
 * Consult TGA indications and Pharmaceutical Benefits Scheme criteria to guide pharmacotherapy choice, tailored to the individual context.
 ‡ Approximate percentage total body weight lost is based on trials including people with type 2 diabetes where pharmacotherapy dose may not represent the highest dose available for weight loss.
 § Weight loss efficacy for each agent varies by patient cohort; figures for injectable medications have been derived from published cardiovascular outcome trials.
 # Non-inferiority cardiovascular outcome trial with dulaglutide as comparator (not placebo controlled).
 ζ The SCALE trial was used as the reference for approximate percentage of total body weight lost, in preference to the LEADER cardiovascular outcome trial (used approximately half the maintenance dose of liraglutide that is indicated for obesity management).

Table 9: (Continued)

Medication name and medication class	Mechanism of action	TGA indications for obesity management in adults*	Dose	Common side effects	Efficacy for weight loss (approximate % of total body weight lost)	Impact on CV outcomes compared with placebo
Oral agents						
Naltrexone/bupropion Opioid antagonist/noradrenaline-dopamine reuptake inhibitor	<ul style="list-style-type: none"> Not fully known; may impact central appetite regulation and reward system 	BMI \geq 30 kg/m ² OR \geq 27 kg/m ² in the presence of one or more weight-related complications	<ul style="list-style-type: none"> Start at 8 mg/90 mg tablet once daily. Dose should be escalated over a period of four weeks: <ul style="list-style-type: none"> Week 1: one tablet AM Week 2: one tablet AM + one tablet PM Week 3: two tablets AM + one tablet PM Week 4 ongoing: two tablets twice daily 	Nausea, vomiting, constipation, dizziness, headache, insomnia, dry mouth, anxiety, agitation, difficulty concentrating	~5–6%	Unknown
Orlistat Lipase inhibitor	<ul style="list-style-type: none"> Inhibits gastrointestinal lipase, reducing absorption of consumed dietary fat 	BMI \geq 30 kg/m ² OR \geq 27 kg/m ² in the presence of one or more weight-related complications	<ul style="list-style-type: none"> 120 mg orally up to three times a day with main meals 	Flatulence, faecal urgency/incontinence, loose oily stools, headache, fatigue, decreased absorption of fat-soluble vitamins, oxalate kidney stones	~3–4%	Unknown
Phentermine Anorectic, sympathomimetic amine	<ul style="list-style-type: none"> Stimulates central nervous system to reduce appetite 	BMI \geq 25 kg/m ²	<ul style="list-style-type: none"> 15mg orally once daily at breakfast. Increase to maximum of 40 mg once daily if required Recommended for short term treatment only (up to 12 weeks) 	Tachycardia, arrhythmia, elevated blood pressure, precordial pain, restlessness, agitation, insomnia, tremor, dizziness, headache, diarrhoea, rash	~2–5%	Unknown

Footnotes:

* Consult TGA indications and Pharmaceutical Benefits Scheme criteria to guide pharmacotherapy choice, tailored to the individual context.

Abbreviations: BMI, body mass index; CV, cardiovascular; CVD, cardiovascular disease; MI, myocardial infarction; TGA, Therapeutic Goods Administration

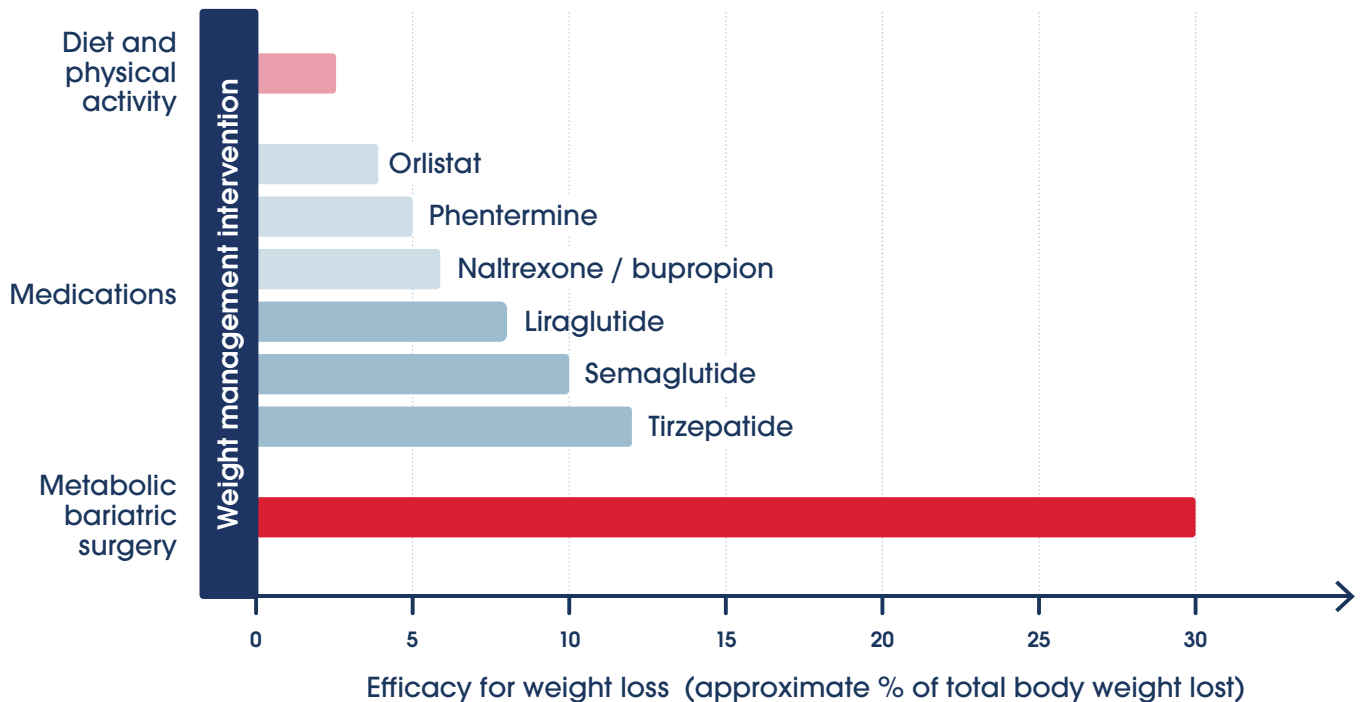


Figure 6: Efficacy of weight management interventions for weight loss. *83,228,236-238,245-249

*To note, the efficacy for weight loss percentages represent findings from several studies of varying cohorts and study quality. They do not represent head-to-head comparison studies and are intended to provide a general indication of comparative efficacy. Figures for semaglutide and tirzepatide have been derived from published cardiovascular outcome trials. In the case of liraglutide, the SCALE trial was used as the reference for percentage total body weight lost, in preference to the LEADER cardiovascular outcome trial. The latter used approximately half the maintenance dose of liraglutide that is indicated for weight management. For each intervention, the bar represents the upper end of the range for approximate percentage of total body weight lost.

Practice points

Initiation and titration

- Pharmacotherapy may be considered when weight-related health improvements, such as improvements in lipids, blood glucose levels, blood pressure and metabolic dysfunction-associated steatotic liver disease, have not been attained with behaviour modifications alone, or the weight loss required is higher than what can be achieved through behaviour modifications.
- Pharmacotherapy for obesity management should be initiated as part of a comprehensive management plan, which includes the continuation of behaviour modifications (nutrition and physical activity).
- In certain cases, especially among individuals with a higher BMI and existing complications, initiation of pharmacotherapy may be warranted as a first-line intervention along with behaviour modifications. Shared decision-making is critical at that stage, factoring in the person's preferences and values. The high cost of obesity management medications, particularly incretin-based agents, means they remain inaccessible for many people.

- The starting dose of semaglutide is 0.25 mg subcutaneous once weekly, which can be increased incrementally every four weeks to a maintenance dose of 2.4 mg:²⁵⁰
 - weeks 1–4: 0.25 mg
 - weeks 5–8: 0.5 mg
 - weeks 9–12: 1 mg
 - weeks 13–16: 1.7 mg
 - maintenance dose: 2.4 mg.

Slow initiation helps to minimise gastrointestinal side effects.

To note, Wegovy (semaglutide) is a higher dose semaglutide formulation which has been approved by the TGA for chronic weight management at a maximum dose of 2.4 mg weekly. Ozempic is only TGA-approved for type 2 diabetes. Oral formulations of semaglutide exist (one is TGA-approved for diabetes) but are not commercially available in Australia at the time of writing. A higher dose injectable semaglutide formulation (7.2 mg) is currently pending TGA approval.²⁵¹

- The starting dose of tirzepatide is 2.5 mg subcutaneous once weekly. After four weeks, the dose can be increased to 5 mg weekly. Dose can be further increased by 2.5 mg every four weeks to a maximum dose of 15 mg once weekly as required.²⁵²
- The starting dose of liraglutide is 0.6 mg subcutaneous once daily. Doses should be increased in 0.6 mg increments at intervals of at least one week to a maintenance dose of 3 mg daily.^{219,234}
- Oral agents (e.g. orlistat, naltrexone/bupropion and phentermine) have a limited role in the management of overweight/obesity in people with or at high risk of CVD due to their minimal impact on weight, unknown CVD benefits and poor tolerability.
- After commencement of pharmacotherapy, regular assessment of weight, cardiometabolic risk factors and treatment tolerability should be undertaken. The dose of pharmacotherapy should be adjusted if required to achieve health targets (increasing or decreasing the dose). Monitor for nutritional adequacy if clinical concerns such as poor diet

quality, disordered eating behaviour or very rapid weight loss occur (typically > 1.5–2 kg per week). See section on 'Dietary advice for people on incretin-based medications'. Engage multidisciplinary team, including pharmacists and allied healthcare professionals, to support adherence.

- Advise people that ongoing pharmacotherapy, in conjunction with behaviour modifications, is likely to be needed to maintain a healthy weight and effectively manage CVD risk in the long term. Weight regain is common after cessation of pharmacotherapy. Some studies show up to 60% of weight lost during treatment is regained one-year post cessation,¹⁹⁶ and cardiometabolic risk factor improvements may be reversed (e.g. HbA1c, cholesterol and blood pressure).²⁵³

Side effects

- Advise people that the most common side effects of incretin-based obesity management medications are gastrointestinal and can include nausea, diarrhoea, constipation and vomiting. These side effects have been reported in approximately 20–40% of people and are most common during dose initiation and escalation.^{250,252} In most people, these side effects can be mitigated by commencing at a lower dose and titrating the dose gradually over weeks.
- People should be advised that having smaller, more frequent meals and avoiding fatty foods can alleviate nausea and vomiting side effects during treatment initiation.²⁵⁴ Adequate fluid and fibre intake can also help alleviate the side effect of constipation.²⁵⁴
- The use of GLP-1 receptor agonists has been associated with rarer side effects including an increased risk of acute pancreatitis, gastroparesis and intestinal obstruction, although incidence is rare (<0.1%).²¹⁹ Inform people about the symptoms of pancreatitis and when to seek medical attention.²⁵⁰ If pancreatitis occurs, the GLP-1 receptor agonist should not be restarted. Similarly, exercise caution when commencing these medications in people with a history of acute pancreatitis.

- A newly identified safety signal, based on a small number of case reports, indicates a possible association between GLP-1 receptor agonists (including tirzepatide) and suicidal ideation.^{255,256} Whilst international regulators (e.g. US Food and Drug Administration) have now removed such warnings, the Therapeutic Goods Administration has updated product safety warnings and advised healthcare professionals to monitor people for changes in mood, behaviour or emerging suicidal thoughts or behaviours.
- Case reports have linked GLP-1 receptor agonists and GLP-1/GIP receptor agonists to an increased potential risk of regurgitation and pulmonary aspiration during anaesthesia due to delayed gastric emptying.²⁵⁷ Preprocedural clinical consensus advice should be followed (see Resources and further reading).
- Advise people that most over-the-counter and online weight loss products, including complementary and herbal supplements, have limited or no evidence for their safety and efficacy. Counterfeit medications disguised as weight loss products are not uncommon. These agents are illegal and can contain harmful, undisclosed ingredients, including sibutramine which has been shown to increase the risk of heart attack and stroke.²⁵⁸
- Strategies to mitigate lean muscle loss while on treatment include optimising dietary protein, recognising that GLP-1 and GIP/GLP-1 receptor agonists may suppress appetite and substantially reduce overall energy intake, increasing the risk of inadequate protein consumption.²⁶⁰⁻²⁶²
- Regular resistance exercise training can help preserve lean muscle mass during weight loss and should be performed on two or more days per week, where clinically appropriate.¹⁵⁹

Dietary advice for people on incretin-based medications

Lean muscle mass loss

- Sarcopenia (loss of muscle strength, mass and function) can be exacerbated by chronic comorbidities, including CVD, and is associated with faster disease progression and higher mortality in these groups.²⁵⁹
- Maintenance of lean mass is important during weight loss because muscle mass facilitates a higher metabolic rate and makes it easier to lose and maintain body weight after a weight loss intervention.
- While dietary weight loss strategies typically cause some loss of lean muscle, GLP-1 and GIP/GLP-1 receptor agonists are associated with significant loss of lean mass ranging from 15–45% of total weight loss,²⁶⁰ varying by agent.
- Prescribers of obesity management medications should consider concurrent referral to an Accredited Practising Dietitian to optimise outcomes.²⁶³ Incretin-based medications increase the risk of muscle loss, sarcopenic obesity and nutrient deficiencies.²⁶³
- People on incretin-based medications should prioritise adequate protein intake (1.2–1.60 g/kg of body weight per day, or an absolute target of 80–120 g/day where appropriate),²⁵⁴ combined with structured resistance exercise, to support preservation of lean mass during weight loss. Increased protein intake alone is unlikely to preserve muscle mass in the absence of resistance training.
- Supplementation may assist in meeting protein and micronutrient requirements when dietary intake is insufficient.²⁶³
- Reduced appetite with GLP-1 receptor agonists may result in unintentional intermittent fasting. Individuals should be encouraged to consume meals at regular intervals, as prolonged fasting without adequate protein intake or dietary variety may increase the risk of nutritional inadequacy.²⁵⁴
- More aggressive caloric restriction strategies including LEDs and VLEDs are generally not appropriate in combination with incretin-based medications unless under close specialist supervision with monitoring of body composition and nutritional status.

Considerations in people with type 2 diabetes

- In adults living with type 2 diabetes and overweight or obesity, consider prescription of glucose-lowering agents with dual weight management and cardiovascular benefits to reduce the risk of major adverse cardiovascular events.
- GLP-1 receptor agonists with demonstrated cardiovascular benefit in people with type 2 diabetes include liraglutide,²²⁷ semaglutide,²²⁹ and dulaglutide.²⁶⁴
- Tirzepatide, a GIP/GLP-1 receptor agonist, has also demonstrated cardiovascular benefit in people with type 2 diabetes.²⁴⁵
- At the time of writing, semaglutide and dulaglutide can only be prescribed on the PBS for type 2 diabetes specifically, not obesity management.
- SGLT-2 inhibitors (such as empagliflozin and dapagliflozin) are also indicated for the treatment of type 2 diabetes and have been shown to have modest weight-lowering effects in this population.²⁶⁵ These drugs offer significant cardiovascular and renal benefits.²⁶⁶
- Healthcare professionals should be aware of potential drug interactions with the use of GLP-1 receptor agonists and GIP/GLP-1 receptor agonists in combination with other glucose-lowering agents (e.g. sulfonylureas, insulin) due to an increased risk of hypoglycaemia.^{250,252} Monitor and consider titrating the dose of sulfonylurea or insulin when commencing a GLP-1 receptor agonist or GIP/GLP-1 receptor agonist.²⁶⁷
- In people with type 2 diabetes, assess for retinopathy during GLP-1 receptor agonist initiation and titration, due to the increased risk of retinopathy associated with a rapid reduction in glycosylated haemoglobin (HbA1c).²⁶⁸
- Emerging safety signals suggest a possible rare association between GLP-1 receptor agonists (particularly semaglutide) and GIP/GLP-1 receptor agonists, and non-arteritic anterior ischemic optic neuropathy in people with type 2 diabetes, however further research is warranted.²⁶⁹⁻²⁷¹

Considerations in older people

- Evidence for the safety and efficacy of obesity management medications in older people is limited. Healthcare professionals should consider the increased risk of sarcopenia and nutrient deficiencies when prescribing these medications and reinforce appropriate nutrition and resistance training.^{263,272}

Considerations in women

- Women of reproductive age taking a GLP-1 receptor agonist or tirzepatide should be advised to use contraception concurrently due to potential safety concerns associated with taking these pharmacotherapies in pregnancy (as shown in animal studies).^{273,274} Semaglutide and tirzepatide are both classified as pregnancy category D medications.^{250,252}
- Tirzepatide and GLP-1 receptor agonists may decrease absorption of oral contraceptives due to delayed gastric emptying, however the evidence is inconsistent.²⁷⁵ As a precaution to avoid unplanned pregnancy, current product information for tirzepatide includes advice that women should switch to a non-oral contraceptive or add a barrier method of contraception for four weeks after initiation, and for four weeks after each dose escalation.²⁷⁶ This advice has not been extended to GLP-1 receptor agonists (liraglutide and semaglutide) at the time of writing.

Considerations in people with heart failure

- When initiating and escalating semaglutide or tirzepatide in people with obesity and heart failure with preserved ejection fraction (HFpEF), monitor kidney function and electrolytes and adjust other medications (e.g. diuretics, antihypertensives, glucose-lowering agents) if required, particularly if the person is experiencing gastrointestinal side effects.²⁷⁷

- Semaglutide and tirzepatide have been shown to improve quality of life and functional capacity in people with heart failure with preserved ejection fraction (HFpEF) and BMI ≥ 30 kg/m², but insufficient evidence exists to conclude cardiovascular outcome benefits.^{278,279}
- There is a lack of high-quality trial data evaluating the cardiovascular impact of GLP-1 receptor agonists in people with heart failure with reduced ejection fraction.
- SGLT-2 inhibitors are also indicated for the management of both reduced and preserved ejection fraction heart failure and may have modest weight-lowering effects in these populations.²⁸⁰ These medications include empagliflozin and dapagliflozin, which are included on the PBS for the indication of chronic heart failure.

Considerations in people with other comorbidities

- Tirzepatide is also indicated for the treatment of moderate-to-severe obstructive sleep apnoea in adults with obesity.²⁵²
- GLP-1 receptor agonists have emerged as an important treatment option for diabetic kidney disease in the context of type 2 diabetes, with semaglutide showing reduced risk of clinically important kidney outcomes and death from cardiovascular causes in people with type 2 diabetes and chronic kidney disease.²⁸¹
- Incretin-based therapies are emerging as a treatment for metabolic dysfunction-associated liver disease (formerly non-alcoholic fatty liver disease), with evidence suggesting benefits in liver histology and a reduction in liver fat.^{282,283} The Therapeutic Goods Administration has provisionally approved semaglutide for the treatment of non-cirrhotic metabolic dysfunction-associated steatohepatitis (MASH) in adults with moderate to advanced liver fibrosis.^{231,284}

Summary of the evidence

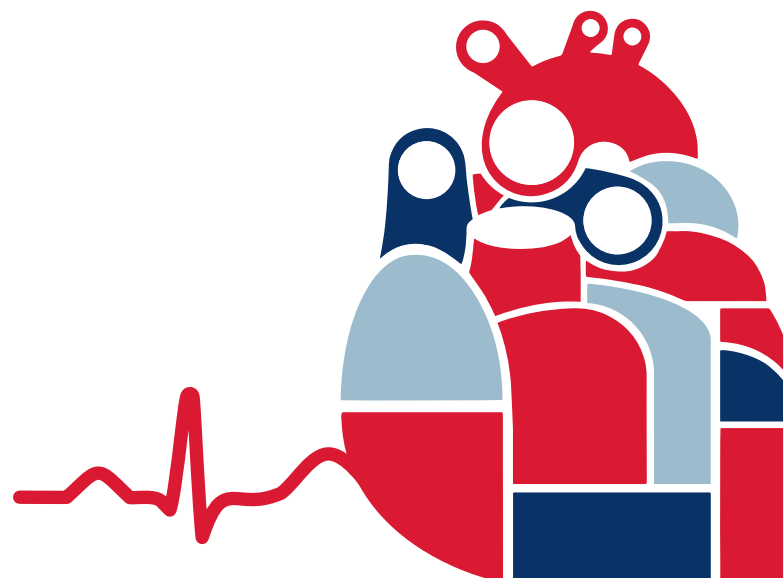
- Evidence for the use of incretin-based therapies in a CVD primary prevention cohort largely draws from trials involving people with type 2 diabetes. The LEADER, SUSTAIN-6 and REWIND trials collectively demonstrate the cardiovascular outcome benefits of GLP-1 receptor agonists in people with type 2 diabetes and at high CVD risk.
 - LEADER (9,340 participants, median 3.8 years) showed liraglutide reduced major adverse cardiovascular events compared with placebo (HR 0.87; 95% CI 0.78–0.97).²²⁷
 - SUSTAIN-6 (3,297 participants, median two years) found semaglutide provided an even greater relative risk reduction compared with placebo (HR 0.74; 95% CI 0.58–0.95).²²⁹
 - REWIND (around 9,900 participants, approximately one third with established CVD, median 5.4 years) demonstrated dulaglutide lowered major adverse cardiovascular event risk compared with placebo (HR 0.88; 95% CI 0.79–0.99).²⁶⁴
- The cardiovascular outcome trial for tirzepatide (SURPASS-CVOT) has demonstrated cardiovascular benefit in people with type 2 diabetes and atherosclerotic CVD.^{229,245,285} In SURPASS-CVOT, tirzepatide was found to be non-inferior to dulaglutide (HR 0.92; 95% CI 0.83–1.01).²⁴⁵ At the time of writing, the SURMOUNT-MMO trial is currently underway to investigate the cardiovascular outcomes of tirzepatide in people with overweight/obesity without type 2 diabetes.²⁸⁶
- The landmark SELECT trial has demonstrated the cardiovascular benefits of the GLP-1 receptor agonist semaglutide (2.4 mg subcutaneous once weekly), in people established atherosclerotic CVD and with a BMI ≥ 27 kg/m² (without type 2 diabetes). In this double-blind, placebo-controlled randomised

clinical trial of 17,604 adults, semaglutide reduced the incidence of cardiovascular death, myocardial infarction and stroke (HR 0.80; 95% CI 0.72–0.90).²²⁸

- There is growing evidence to support the role of GLP-1 receptor agonists and GIP/GLP-1 receptor agonists in improving heart failure-related symptoms and quality of life in people with HFpEF in the setting of obesity,^{278,279} and in concurrent obesity and type 2 diabetes.²⁸⁷ In the SUMMIT trial, tirzepatide showed a significant reduction in heart failure events in people with HFpEF but results should be interpreted with caution due to small event numbers.²⁷⁹
- Emerging unpublished observational data suggest that GLP-1 receptor agonists may also play a secondary prevention role in people with atrial fibrillation, obesity and type 2 diabetes, although further research is underway.²⁸⁸

Resources and further reading

- Australian Register of Therapeutic Goods Product Information
- Pharmaceutical Benefits Scheme
- Australian Diabetes Society. Clinical practice recommendations: Periprocedural use of GLP-1 RAs and GLP-1/GIP RAs




Emerging pharmacotherapies

Several novel obesity management medications and formulations are currently being investigated in clinical trials. Some selected new therapies on the horizon are discussed below.

- Retatrutide, a triple agonist against GLP-1, GIP and glucagon receptors, has demonstrated significant weight loss (up to a 24% reduction in body weight at the highest dose) and metabolic benefits in people with obesity, both with and without type 2 diabetes in phase 2 clinical trials.^{289,290}
- Survodutide is a novel dual glucagon receptor and GLP-1 receptor agonist.²⁹¹ In phase 2 trials, it conferred significant weight loss (up to 19% of total body weight) in people with obesity without type 2 diabetes.²⁹² Phase 3 clinical trials are currently underway in both people with and without type 2 diabetes, in addition to a cardiovascular outcomes trial.²⁹³
- Oral semaglutide (25 mg) daily achieved 11.4% reduction in body weight compared with placebo (95% CI -13.9 to -9.0) in people with overweight and obesity.²⁹⁴ While not a head-to-head study, this is comparable to observed body weight reduction achieved with subcutaneous semaglutide 2.4 mg weekly. Trials have demonstrated that oral semaglutide reduces the risk of major adverse cardiovascular events (HR 0.86; 95% CI 0.77 to 0.96) in people with type 2 diabetes and established CVD and/or chronic kidney disease, without an increase in risk of serious adverse events.²⁹⁵
- A novel oral GLP-1 receptor agonist – orforglipron – has shown significant glycaemic and weight loss benefits in people with and without type 2 diabetes.²⁹⁶⁻²⁹⁸
- CagriSema, a combination of cagrilintide and semaglutide, is a dual GLP-1 receptor agonist and long-acting amylin analogue. The REDEFINE trials have shown that CagriSema confers clinically significant weight loss (approximately 20% of total body weight) compared with placebo, in both people with and without type 2 diabetes.^{299,300}
- Phase 2 trials of once weekly administration of the amylin receptor agonist, eloralintide, in adults with overweight or obesity and without type 2 diabetes, have demonstrated significant weight loss, with a mean decrease in body weight ranging from 9–16%, compared with 0.4% in the placebo group.³⁰¹ Combination therapy of amylin and semaglutide has also shown promise in weight management, with over 40% of trial participants achieving a decrease in body weight of $\geq 25\%$.³⁰²
- Monthly injectable agents have shown promise in the treatment of obesity in adults with and without type 2 diabetes. Maridebart cafraglutide (MariTide) is a combined GLP-1 receptor agonist and GIP receptor antagonist. Results from a phase 2 clinical trial suggest this pharmacotherapy produces clinically significant weight loss compared with placebo.³⁰³

Several other pharmacotherapies are used off label, including lisdexamfetamine and a combination of phentermine and topiramate, but none are Therapeutic Goods Administration-approved for weight loss in Australia.

Surgical interventions

 Recommendations	Strength of advice
In adults living with overweight or obesity and CVD or at high risk of CVD and who have not attained adequate weight-related health improvements through behaviour modifications and pharmacological interventions, consider referral to multidisciplinary specialised obesity services.	Moderate advice ● ● ○
In adults with CVD or at high risk of CVD with BMI ≥ 40 kg/m ² or, BMI ≥ 35 kg/m ² with at least one weight-related comorbid condition [‡] , refer for consideration of metabolic bariatric surgery to reduce the risk of major adverse cardiovascular events.	Moderate advice ● ● ○
In adults with CVD or at high risk of CVD who have undergone metabolic bariatric surgery, offer lifelong multidisciplinary follow-up care to support long term health.	May be appropriate ● ○ ○

[‡] Weight-related comorbid conditions include hypertension, dyslipidaemia, obstructive sleep apnoea, cardiovascular disease, prediabetes or type 2 diabetes.

General information

Differences in eligibility criteria for metabolic bariatric surgery differ locally and internationally. The BMI criteria presented in this statement align with the current Medicare Benefits Schedule criteria for metabolic bariatric surgery.³⁰⁴ These criteria also reflect the absence of randomised controlled trial evidence of improved cardiovascular outcomes in people with a lower BMI (30–34.9 kg/m²) undergoing metabolic bariatric surgery. While surgery is highly effective for weight loss, evidence of cardiovascular benefit in this cohort is largely based on observational studies and small randomised controlled trials demonstrating improvement in surrogate cardiovascular measures only.³⁰⁵⁻³⁰⁷ The first randomised controlled trial investigating the impact of metabolic bariatric surgery in improving cardiovascular outcomes is currently underway.^{304,308}

Practice points

Eligibility and pre-surgical assessment

- Multidisciplinary specialised obesity services provide medically led, multidisciplinary care that incorporates nutrition and physical activity interventions, psychological therapies, pharmacological treatment, and access to metabolic bariatric surgery where clinically appropriate and feasible. Referral to multidisciplinary specialised obesity services from primary care should be considered:
 - when sustained weight-related health improvements (such as improvements in lipids, blood glucose, blood pressure or metabolic dysfunction-associated steatotic liver disease) have not been attained via behaviour modifications and pharmacotherapy, or
 - in complex clinical scenarios, or
 - in individuals who meet the Medicare Benefits Schedule criteria for metabolic bariatric surgery.

- The most common types of metabolic bariatric surgery performed in Australia are sleeve gastrectomy, Roux-en-Y gastric bypass and single anastomosis gastric bypass (Table 10).
- Discuss the potential benefits and risks of metabolic bariatric surgery with people, including the requirement for lifelong monitoring and multidisciplinary follow up. Referral to specialist care for consideration of surgery should be undertaken as part of a shared decision-making process.
- Comprehensive pre-surgical assessment by a multidisciplinary specialist team is important. This should include assessment of cardiometabolic risk, screening for mental health conditions, eating disorders and nutritional deficiencies and an assessment of psychological and overall fitness for surgery. Ensure input from surgeons, endocrinologists, cardiologists, psychologists and dietitians as appropriate.
- Monitor people for dumping syndrome following bariatric surgery, which can occur when a person consumes high sugar or high carbohydrate foods and drinks. These types of foods/drinks pass too quickly from the stomach into the small intestine, causing gastrointestinal symptoms (e.g. nausea, vomiting, bloating, diarrhoea) and hypoglycaemia.
- Some people may require obesity management medication post-surgery to support weight maintenance. Healthcare professionals should assess the need to commence pharmacotherapy (or continue pharmacotherapy if commenced prior to surgery), monitor for side effects, and titrate the dose accordingly. Involve pharmacists as appropriate.
- A small subset of people experience increased risk of alcohol or other substance use disorders post-operatively, particularly following gastric bypass procedures.³⁰⁹ Population data suggest new-onset substance use disorder occurs in a small minority.³¹⁰ Risk should be addressed through pre-operative screening and longitudinal monitoring, particularly in individuals with prior substance use and mental health disorders.

Post-surgical monitoring and follow up

- People who have undergone metabolic bariatric surgery should be monitored for:
 - micronutrient deficiencies
 - protein-energy malnutrition
 - mental health conditions (e.g. depression, addictive disorders, eating disorders)
 - weight regain or excessive weight loss
 - gastrointestinal side effects or complications, including gall stones
 - medication side effects due to altered pharmacokinetics
 - bone health.

Refer to Table 11 for further detail.

- Post-surgery care should include an integrated care plan such as support for nutrition, physical activity, behavioural strategies, and psychological wellbeing.

Dietary advice post-bariatric surgery

- Advise consumption of small, frequent meals (typically 4–6 per day) eaten at regular intervals to improve tolerance, optimise nutrient intake, and reduce the risk of dumping syndrome and post-prandial hypoglycaemia.³¹¹
- Protein-rich foods should be included with each meal and snack to support lean mass preservation, enhance satiety and to moderate post-prandial glycaemic responses.³¹²
- Foods and drinks high in added sugars or refined carbohydrates should be minimised, as they can precipitate early dumping symptoms and late post-prandial (reactive) hypoglycaemia following bariatric procedures.^{313,314}

- People are advised to avoid drinking with meals and to separate fluids from solid food intake (commonly by ~30 minutes) to slow gastric emptying, improve nutrient absorption, and reduce dumping related symptoms.³¹¹
- Encourage slow eating, thorough chewing, and mindful portion control to improve gastrointestinal tolerance and support recognition of early satiety.³¹⁵
- Soluble fibre and modest amounts of dietary fat may help slow nutrient absorption and reduce glycaemic variability, though tolerance should be individualised and guided by dietitian review.³¹³
- Long term dietary management following bariatric surgery should be supported by regular dietitian review to address nutritional adequacy, symptom management, and evolving risks such as dumping syndrome, hypoglycaemia or micronutrient deficiencies.

Accessibility

- Accessibility of metabolic bariatric surgery services remains a significant barrier in Australia, with only a very small minority of Australian public hospitals offering these services.³¹⁶ Around 90% of metabolic bariatric surgeries, which are typically associated with significant costs, take place in the private sector.³¹⁶
- Uptake of metabolic bariatric surgery is impacted by both financial and geographical barriers including access to private health insurance with appropriate surgical cover, limited service availability in rural areas, and low referral rates.³¹⁷

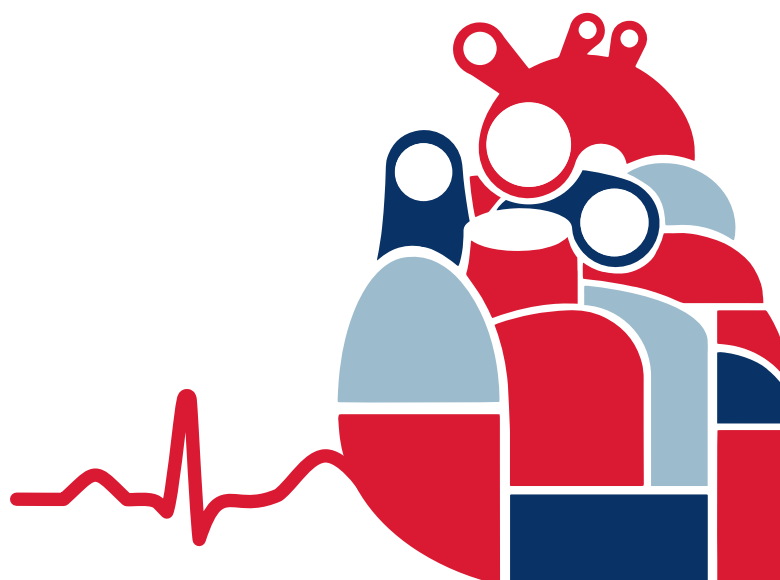


Table 10. Comparison of selected metabolic bariatric procedures in Australia.³¹⁸⁻³²⁷

	Sleeve gastrectomy	Roux-en-Y gastric bypass (RYGB)	Single anastomosis (one anastomosis) gastric bypass	Single anastomosis duodenal-ileal interposition
Procedure details	Removes ~75–80% of the stomach, leaving a narrow sleeve	Creates a small gastric pouch and reroutes small intestine	Similar to RYGB but with one intestinal anastomosis	Combines a sleeve gastrectomy with a single anastomosis connecting the duodenum to the distal ileum
Proportion of bariatric surgeries	~77%	~9%	~13%	<1%
Mean % weight loss	~25–30% at 1–2 years	~25–30% at 1–2 years	~30–35% at 1–2 years	~30–40% at 1–2 years
Nutritional concerns	<ul style="list-style-type: none"> • Nutritional deficiencies • Annual vitamin check required • Lifelong supplementation 	<ul style="list-style-type: none"> • Greater frequency of nutritional deficiencies • Six monthly vitamin check required • Strict lifelong supplementation 	<ul style="list-style-type: none"> • Even greater frequency of nutritional deficiencies • Six monthly vitamin check required • Very strict lifelong supplementation 	<ul style="list-style-type: none"> • Higher risk of protein-energy malnutrition • Six monthly vitamin check required
Advantages	<ul style="list-style-type: none"> • Shorter operative time • Fewer long-term complications 	<ul style="list-style-type: none"> • Excellent metabolic benefits • Less reflux 	<ul style="list-style-type: none"> • Greater weight loss • Strong metabolic effect 	<ul style="list-style-type: none"> • Even greater weight loss and more durable • Good revision option for sleeve
Disadvantages and risks	<ul style="list-style-type: none"> • Irreversible • Higher risk of GORD • Staple line leaks (difficult to manage) 	<ul style="list-style-type: none"> • Risk of dumping syndrome • Internal hernias • Marginal ulcers (difficult to manage) • Requires individual to maintain lifelong connection with surgeon for early detection of complications 	<ul style="list-style-type: none"> • Risk of bile reflux • Risk of dumping syndrome • Internal hernias (rare) • Marginal ulcers (difficult to manage) 	<ul style="list-style-type: none"> • Marginal ulcers (rare)

Abbreviations: GORD, gastroesophageal reflux disease.

Table 11: Common issues following metabolic bariatric surgery and actions primary healthcare professionals can consider.^{312,328-340}

Post-surgical issue	Risk	Primary healthcare professional actions to consider
Nutritional deficiencies	Common nutritional deficiencies include iron, folate, calcium (check PTH), vitamins A, B1, B6, B12, D	<ul style="list-style-type: none"> • Conduct regular blood tests (every six months initially post-surgery, then six monthly or annually) • Refer to an Accredited Practising Dietitian, or another appropriately qualified healthcare professional if not available • Prescribe lifelong multivitamin and mineral supplements (and procedure-specific additional supplements)
Weight regain	Weight regain is to be expected and should be managed early as required	<ul style="list-style-type: none"> • Monitor weight regularly • Support people to maintain ongoing nutrition and physical activity • Consider commencing pharmacotherapy to maintain weight, where clinically appropriate
Gastrointestinal symptoms	<ul style="list-style-type: none"> • Reflux • Change in stool frequency 	<ul style="list-style-type: none"> • Prescribe a regular proton pump inhibitor in early post-operative phase • Dietary interventions to assist with stool frequency • Consider surveillance gastroscopy for Barrett's oesophagus at one year, then every 2–3 years (particularly important for people with sleeve gastrectomy or single anastomosis gastric bypass)
Mental health	Potential risk of relapse of psychiatric disorders and/or the emergence of new psychiatric disorders perioperatively, including mood, anxiety, eating and/or substance use disorders	Where relevant, undertake screening for mental health conditions, substance abuse and other addictive or compulsive behaviours with referral pathways (Refer to <i>Appendix 4: Mental health and eating disorder validated assessment tools</i> for a list of validated mental health screening tools)
Medication titration	Altered pharmacokinetics of some medications (e.g. oral contraceptive pill, psychiatric medications) may necessitate dose or formulation changes, or alternative treatment options	<ul style="list-style-type: none"> • Monitor and titrate dose of medications as required • Seek blood levels of psychiatric medications where relevant • Seek pharmacy input as required

Table 11: (Continued)

Post-surgical issue	Risk	Primary healthcare professional actions to consider
Bone health	Potential loss of bone mineral density (osteopenia/osteoporosis) due to nutrient malabsorption (calcium, vitamin D, zinc, copper), menopause, rapid weight loss and mechanical unloading. This may also lead to higher fracture risk	<ul style="list-style-type: none"> • Measure serum PTH, calcium phosphorus, vitamin D, 24-hour urine calcium levels • Lifelong calcium supplementation generally recommended • Vitamin D supplementation should be individualised and guided by serum 25-OH vitamin D levels; interpret in conjunction with corrected calcium and PTH concentrations • Consider DEXA scanning in higher risk people
Pregnancy and contraception	<ul style="list-style-type: none"> • Oral contraception may not be as reliable post-surgery • Increased risk of unplanned pregnancy resulting from weight loss (due to improved fertility) 	<ul style="list-style-type: none"> • Consider alternative contraceptive options that do not rely on absorption (i.e. non oral) including barrier contraception, long-acting reversible contraception such as hormonal implants, hormonal IUDs or copper IUDs • Advise people to use effective contraception to avoid getting pregnant for 12–18 months post-surgery in the interests of maternal and infant health

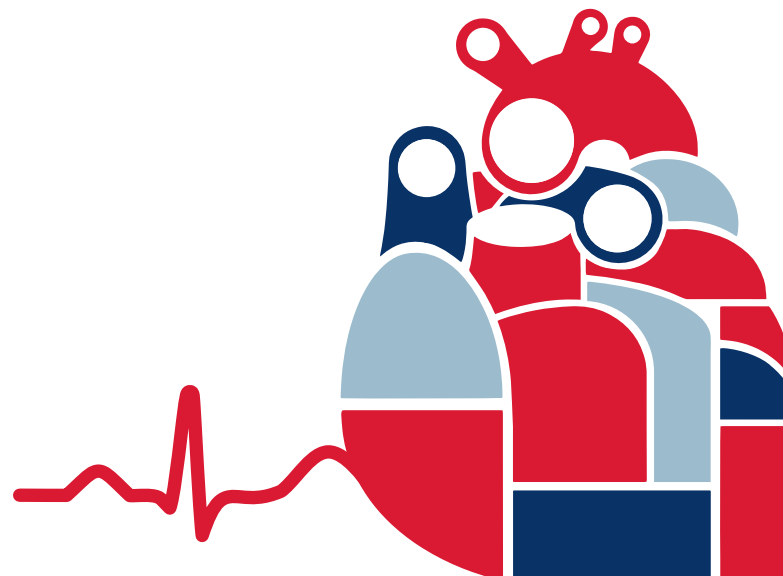
Abbreviations: DEXA, dual-energy x-ray absorptiometry; IUD, intrauterine device; PTH, parathyroid hormone.

Summary of the evidence

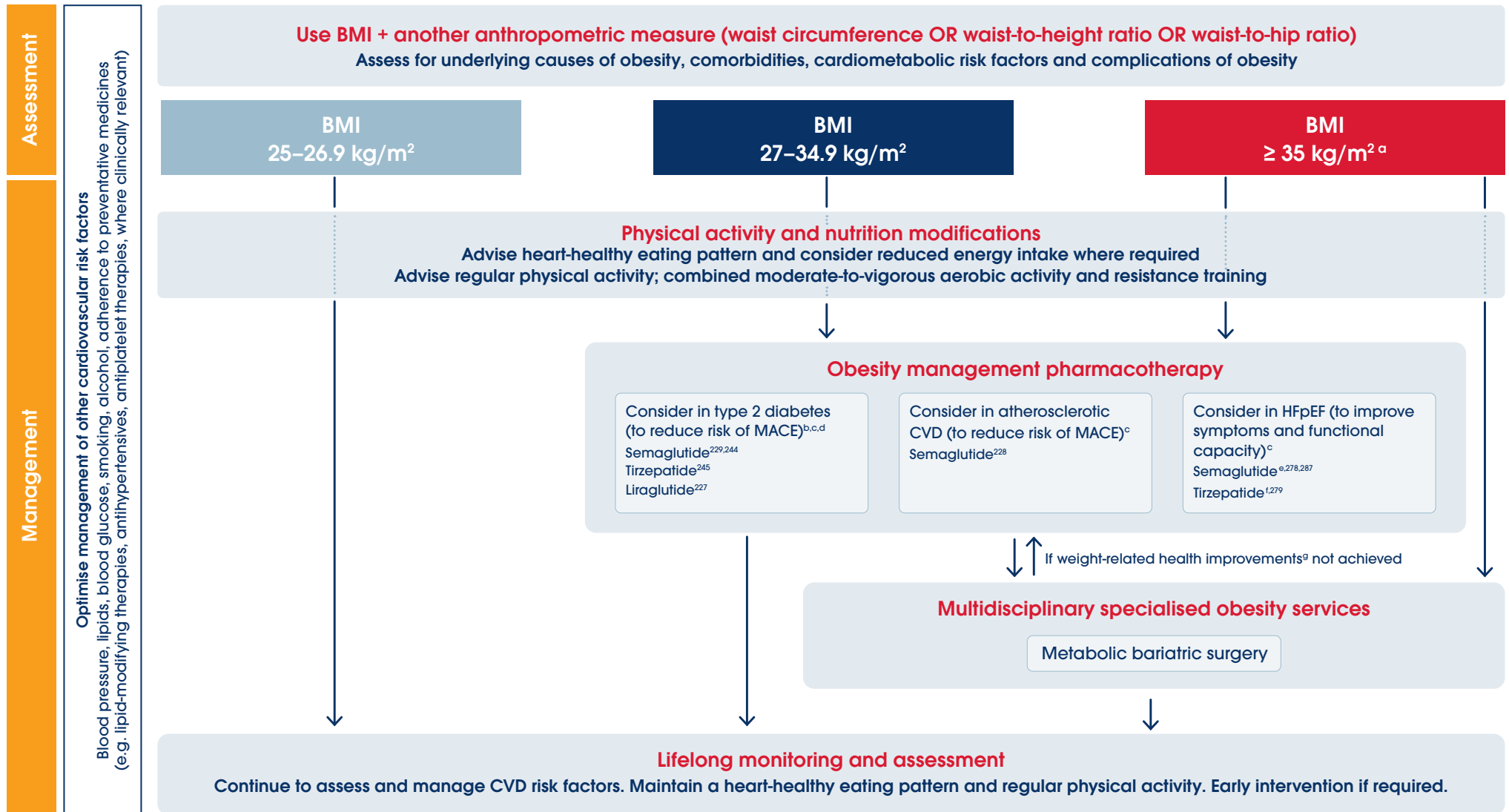
- Evidence from several small RCTs, supported by large observational studies, demonstrates that bariatric surgery is an effective long-term intervention for severe obesity, typically achieving ~25–30% total body weight loss in the first year.^{249,306,341-343}
- Several meta-analyses (of predominantly cohort studies) have shown that metabolic bariatric surgery confers significant improvement in cardiovascular risk factors including blood pressure, glycaemia, lipids and waist circumference, compared with medical management.²⁰⁴⁻²⁰⁶
- Significant reductions in major adverse cardiovascular events of 40–60% have been demonstrated post-bariatric surgery in observational studies. A large meta-analysis of 40 matched cohort studies found that bariatric surgery was associated with a statistically significant reduction in major adverse cardiovascular events, atrial fibrillation, heart failure, myocardial infarction and stroke.¹⁹⁷
- A systematic review and meta-analysis of people with established heart failure who underwent metabolic bariatric surgery were found to have a 72% reduced risk of heart failure exacerbations (RR 0.28; 95% CI 0.13–0.55) compared with those with heart failure who had non-surgical treatment.²⁰³
- Observational cohort studies provide inconsistent evidence regarding the reduction in atrial fibrillation incidence following metabolic bariatric surgery.^{187,344}

Resources and further reading

- National Association of Clinical Obesity Services. Find a service.
- RACGP. Bariatric–metabolic surgery: A guide for the primary care physician.
- Healthdirect. Guide to bariatric surgery. 2024. (For consumers).
- Better Health Channel. Obesity surgery. 2022. (For consumers).
- Australian Journal of General Practice. Bariatric surgery. 2025.
- Australia and New Zealand Metabolic and Obesity Surgery Society. Public bariatric surgery – A national framework. 2020.
- American Society for Metabolic and Bariatric Surgery (ASMBS) and International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO): Indications for metabolic and bariatric surgery. Surgery for obesity and related diseases. 2022.



Management of overweight and obesity in adults with established CVD



Footnotes

- ^a People with BMI ≥ 40 kg/m² may require direct referral for multidisciplinary specialised obesity services.
- ^b Evidence is based on studies conducted in people with type 2 diabetes whereby medication doses are generally lower than those used for weight loss.
- ^c SGLT-2 inhibitors are not TGA-indicated for obesity management but have been shown to have modest weight-lowering effects in these groups (less so than other agents listed here). They have been shown to improve CV outcomes in type 2 diabetes, heart failure, atherosclerotic CVD and CKD.

- ^d Dulaglutide, indicated in type 2 diabetes, has been shown to improve CV outcomes, but is not TGA-indicated for obesity management.
- ^e Based on STEP-HFpEF trial data in people with HFpEF, ejection fraction ≥ 45% and BMI ≥ 30 kg/m².
- ^f Based on SUMMIT trial data in people with HFpEF, ejection fraction ≥ 50% and BMI ≥ 30 kg/m².
- ^g Weight-related health improvements include improvements in blood glucose levels, lipids, blood pressure and liver function.

Section 4:

Lifelong care and follow up

Obesity is a chronic, progressive condition that requires ongoing, multidisciplinary management, particularly in the context of overlapping cardiovascular risk or established disease. Sustained benefits from weight loss, whether achieved through behaviour modifications, pharmacotherapy, surgery or a combination of these, rely on continuous monitoring and support.

Role of primary care

Primary care is critical to the assessment, management and long-term monitoring of people living with overweight and obesity in Australia. General practice teams are ideally placed to provide first-line, person-centred services for obesity management, including support for behaviour modifications, pharmacotherapy and timely referral to multidisciplinary specialised obesity services. Primary care remains an essential conduit between weight specific clinical care pathways and long-term CVD and risk factor management. Existing funding models for chronic disease management and allied health support can be leveraged to support holistic care. These include Medicare-subsidised GP Chronic Condition Management Plans and Home Medicines Reviews.

Ongoing management of cardiovascular risk factors

Lifelong care should include regular assessment of cardiovascular risk factors, nutritional and physical activity status and mental health, as well as strategies to sustain long-term weight loss and prevent weight regain. Continued healthy behaviours, including physical activity and healthy eating, are required not only for obesity management but to improve cardiovascular health outcomes. Alongside established follow-up strategies, longitudinal metabolic monitoring is an emerging area of interest and may provide additional insight into cardiometabolic health beyond weight change alone.

Culturally appropriate models of care that support long-term continuity are also an important consideration, as evidence shows that targeted approaches improve sustained engagement and adherence, contributing to improved cardiovascular outcomes. Telehealth plays an important role, particularly for rural and remote communities, in supporting long-term monitoring and follow up.

For individuals living with established CVD and overweight or obesity, optimising CVD risk factor control is a priority for reducing risk of secondary events. This includes supporting appropriate behaviour modifications – physical activity, nutrition, alcohol reduction and smoking cessation. Attainment of clinical prevention targets (blood pressure, lipids) is critical in this group, aided by adherence to preventative medicines (including anti-thrombotics where appropriate) to reduce risk of secondary cardiovascular events.³⁴⁵ For eligible people, participation in a cardiac rehabilitation program should be encouraged to reduce the risk of future cardiac events, improve quality of life, facilitate exercise and improve uptake of behaviour modifications.

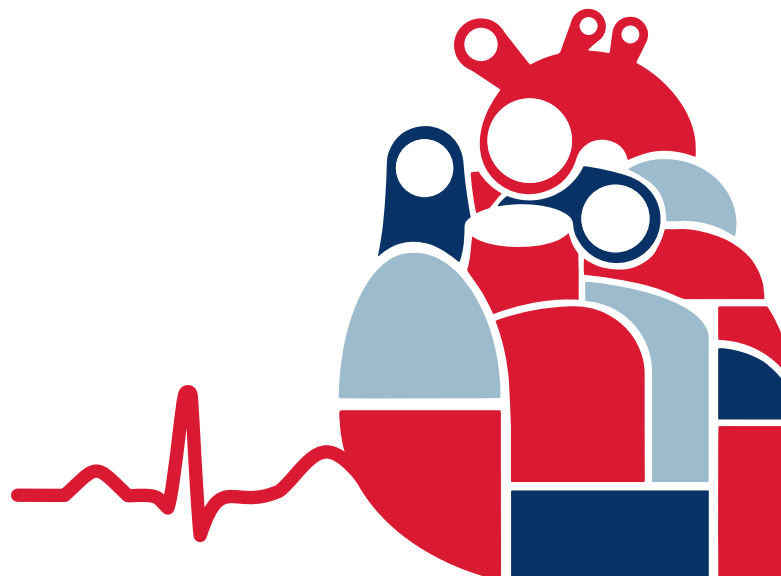
Monitoring for weight loss complications

For people who experience significant and/or rapid weight loss, structured follow up with a multidisciplinary team is essential to prevent or detect complications early and manage potential side effects.

These may include:

- gall stones
- nutritional deficiencies
- muscle mass loss
- psychological impacts.

Weight loss may also positively impact control of cardiometabolic risk factors such as blood glucose and blood pressure, and some medications may require dose adjustment.



Equity considerations for high-priority populations

Culturally and linguistically diverse (CALD) communities

Obesity rates within CALD communities differ significantly between first-generation immigrants and children from CALD backgrounds. First-generation immigrants often arrive in Australia with comparatively better health. However, this advantage tends to diminish over time as 'obesogenic' western dietary patterns and lifestyle behaviours are adopted.³⁴⁶

Over time, adults from CALD backgrounds, along with their offspring, experience a disproportionately higher burden of obesity-related health issues. In certain populations, such as those originating from North Africa and the Middle East, the prevalence of overweight and obesity among adults is estimated to be approximately 40% higher compared with Australian-born adults.³⁴⁷

For people from CALD backgrounds, the problem is further compounded by language and health literacy challenges, culturally incongruent care models, weight stigma and socioeconomic disadvantage.^{84,348} These factors may limit engagement with health services and evidence based treatments.⁸⁴ For some women, cultural or faith-based norms around modesty may affect comfort with physical examination; without culturally safe care, delays in assessment, disengagement, or missed follow-up may occur, increasing long-term cardiovascular risk.³⁴⁹

To improve cardiovascular outcomes and support sustainable behaviour change for obesity management, health services should, where feasible, adopt culturally safe, co-designed models of care that reflect the diversity of the communities they serve. Culturally safe practice builds trust, supports engagement and adherence, and aligns with best practice in primary care. Even in time-constrained settings, culturally safe care can be delivered through small but meaningful actions. These may include:

- documenting preferred language, interpreter requirements and relevant cultural considerations (e.g. culturally specific dietary restrictions like halal or kosher diets) clearly in the medical record, and delivering tailored care accordingly
- offering or linking to culturally appropriate physical activity options, such as women-only programs, community-led walking groups, or culturally-specific exercise initiatives, where available
- providing inclusive materials, signage and culturally relevant visuals in multiple languages to signal safety and respect
- identifying appropriate support for communication, including staff or family members who can assist with brief communication and using professional interpreters for complex discussions or care planning.

Recognition of overlapping identities is important, including among multicultural women, multicultural people living in rural and remote areas, those experiencing socioeconomic disadvantage, and those living with mental health conditions.

Individuals may experience multiple intersecting risk factors simultaneously, and acknowledging these intersections supports more accurate risk assessment and promotes holistic, tailored interventions.

First Nations peoples

In Australia, First Nations peoples have higher rates of morbidity and premature mortality due to cardiovascular and metabolic disease compared with non-Indigenous Australians.^{350,351} Similarly, First Nations peoples experience a higher prevalence of obesity, with 77% of adults living with overweight or obesity compared with 66% of non-Indigenous Australians.² The reasons for these disparities are complex and varied, and include reduced access to healthcare and healthy food, lack of culturally-appropriate health programs and initiatives, intergenerational trauma, lost culture and socioeconomic disadvantage.³⁵²

To effectively manage overweight and obesity in First Nations peoples, approaches must be First Nations-designed and led, rooted in culture and self-determination, and adaptable to the distinct needs of each community.³⁵²

It is important to note that high quality evidence for the most effective obesity management treatments for First Nations peoples is lacking, and further research is needed. In particular, research is needed to better understand the impact of pharmacotherapy for obesity management in First Nations peoples and how these therapies can complement community-based behaviour change programs.³⁵²

Rural and remote communities

Adults living in rural and remote communities face a disproportionately high burden of CVD, with coronary heart disease rates in very remote areas more than double those of major cities.³⁵³ Nearly 70% of adults in these regions are affected by overweight or obesity, compared with 64.4% in metropolitan areas.³⁵³ This increased prevalence is compounded by geographic isolation, limited infrastructure and workforce shortages,³⁵³ along with limited access to fresh and affordable food. Access to metabolic bariatric surgical services in rural and remote communities remains a significant challenge.³¹⁶

Tailored and locally grounded approaches to obesity management that reflect community context, available workforce and existing infrastructure are more likely to be effective in geographically isolated populations compared to generic weight loss advice.³⁵⁴

Telehealth can improve access to care where local services are limited; however, its effectiveness may be constrained by digital infrastructure, access to technology, and a person's digital literacy.³⁵⁵

Case conferencing supports multidisciplinary collaboration in obesity care in rural and remote settings. It enables GPs to coordinate limited local resources with specialist input, which has been shown to improve engagement and treatment outcomes, and mitigate fragmentation of care in geographically isolated populations.³⁵⁴ Digital health platforms that facilitate monitoring and timely communication between people living with overweight or obesity and members of their healthcare team can enable effective case conferencing in rural and remote settings.

Socioeconomic disadvantage

Adults experiencing socioeconomic disadvantage are disproportionately affected by obesity and CVD, with obesity rates 57% higher in the most disadvantaged areas compared with the most advantaged,³⁵⁶ and CVD burden 1.8 times greater.¹ These disparities are driven by structural inequities, limited healthcare access and reduced engagement with traditional health services. Community informed and locally delivered strategies have demonstrated improved cardiovascular outcomes, healthier weight trajectories and enhanced wellbeing in populations experiencing socioeconomic disadvantage.³⁵⁷⁻³⁵⁹

For those experiencing financial hardship, housing instability, or chronic stress, healthcare should focus on what is most feasible and meaningful at the time, with weight loss addressed where appropriate and as circumstances allow. Community integrated models that connect healthcare with services such as schools, housing and employment services foster trust, improve health literacy and empower individuals to take ownership of their health.³⁶⁰

People living with a mental health condition

Mental health conditions* frequently co-occur with obesity and are more prevalent among people living with overweight/obesity.^{361,362} These conditions are often complex, bidirectionally linked and may include depression, mood and anxiety disorders, trauma-related symptoms, eating disorders and neurodivergent conditions such as attention deficit hyperactivity disorder (ADHD) or autism (Figure 7). This underscores

the importance of incorporating mental health screening into obesity care.

In people living with obesity, mental health conditions may arise in response to other comorbidities or obesity-related complications (e.g. chronic pain, obstructive sleep apnoea, CVD or type 2 diabetes) which can contribute to psychological distress and compound the complexity of care.

*A mental disorder defined as a significant impairment of an individual's cognitive, affective and/or relational abilities which may require intervention and may be a recognised, medically diagnosable illness or disorder (World Health Organization, 2025).

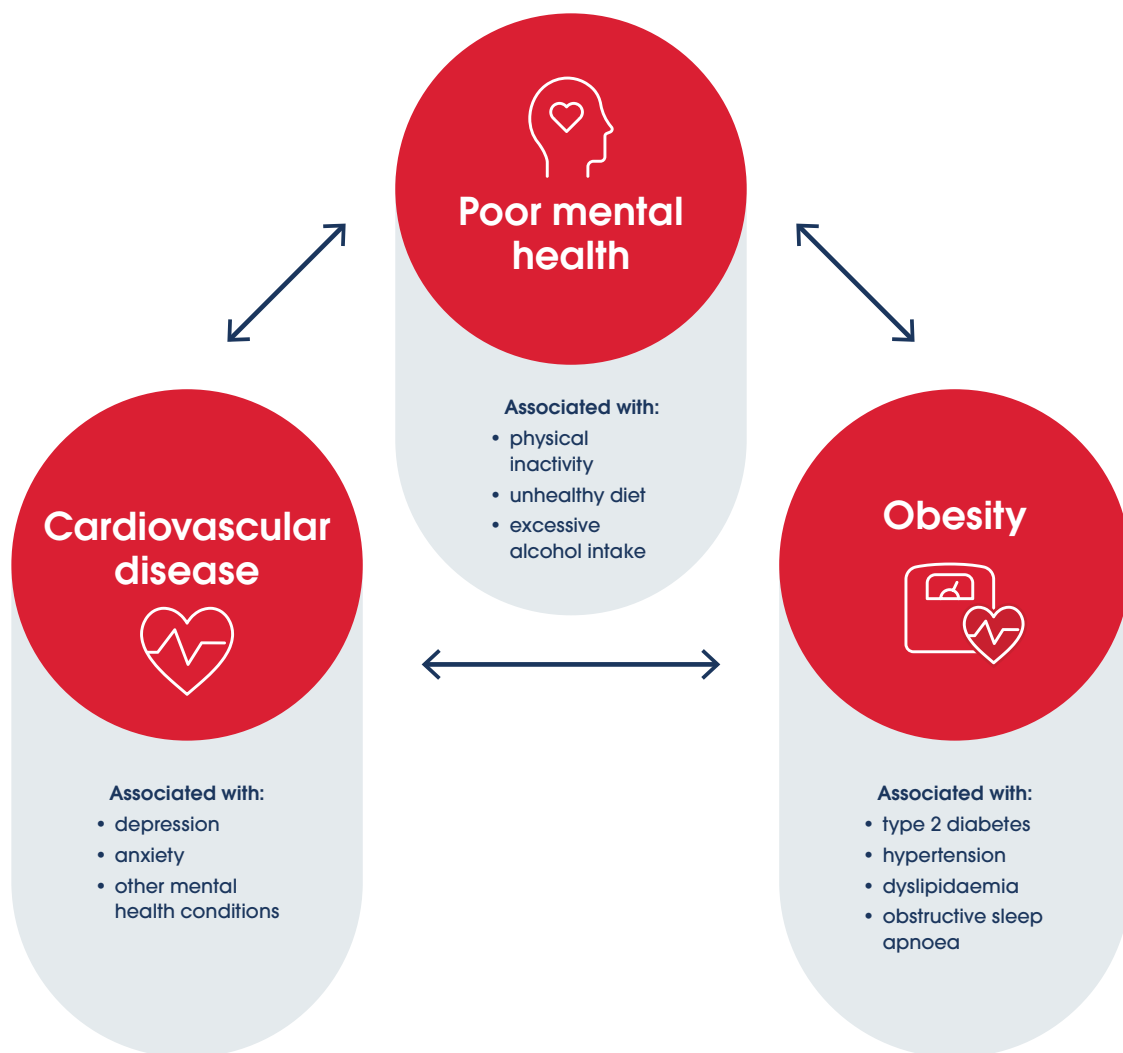


Figure 7: Complex interplay between mental health, obesity and cardiovascular disease.

Routine use of validated tools for mental health and eating disorder assessment (such as the Patient Health Questionnaire (PHQ)-9, Generalized Anxiety Disorder (GAD)-7, Binge Eating Disorder Screening (BEDS)-7 and Eating Disorder Examination Questionnaire (EDE-Q)-6) should be embedded into obesity management protocols (see *Appendix 4: Mental health and eating disorder validated assessment tools* for further details). Healthcare professionals should prioritise early identification and specialist referral for people with a suspected mental health condition.

For people living with obesity who are prescribed psychotropic medications, routine cardiometabolic screening is recommended, as some medicines are associated with increased cardiovascular risk factors and overall cardiovascular risk. Examples include:

- ECG monitoring for medicines that are associated with QTc prolongation (e.g. olanzapine, clozapine and tricyclic antidepressants)^{219,363}
- metabolic monitoring with antipsychotics (weight, lipids, glucose and blood pressure). A higher risk of metabolic side effects, including weight gain, is observed with atypical antipsychotics (such as clozapine, olanzapine and quetiapine).³⁶⁴
- weight monitoring with antidepressants and mood stabilisers. Weight gain is most commonly associated with tricyclic antidepressants, mirtazapine, certain selective serotonin reuptake inhibitors, valproate and lithium.³⁶⁵

When managing individuals with CVD and co-existing mental health conditions, it is important to prioritise stabilisation of severe mental illness before initiating structured obesity interventions. Clinical decision-making should be guided by a person's current mental state and stability, rather than the severity of diagnosis alone when the condition is well managed.³⁶⁶

Women

Whilst similar proportions of women and men are living with obesity in Australia (31% and 33% respectively),³⁶⁷ a closer look beyond BMI classification alone reveals important sex differences in other anthropometric measures. Compared with men of the same BMI, women tend to have a higher percentage of body fat and lower lean muscle mass.³⁶⁸ In Australia, 72% of all women have a waist circumference indicative of an increased risk of cardiometabolic complications compared with 63% of all men.²

Women can experience distinct weight management challenges due to hormonal changes across the life course, including conditions such as polycystic ovarian syndrome, as well as life stages such as pregnancy, perimenopause and menopause.³⁶⁹ These hormonal shifts influence fat distribution and increase cardiometabolic risk. Post-menopausal women are particularly impacted, with higher rates of obesity relative to their male counterparts.³⁷⁰

During menopause, hormonal changes, including oestrogen deficiency, impact insulin resistance, lipid metabolism, energy consumption and body fat distribution.³⁷¹ The latter includes increases in abdominal and visceral adiposity, raising CVD risk.^{86,369} In addition, menopausal symptoms may be more pronounced in women living with obesity, including poor sleep, mood changes and sexual dysfunction.³⁷²

Effectively managing overweight and obesity in women requires a comprehensive, multimodal strategy that takes into account biological differences, unique challenges at various life stages and sociocultural barriers.³⁶⁹ The latter may include limited access to female healthcare professionals (particularly in rural and remote areas), cultural expectations and carer responsibilities.⁸⁷

Emerging evidence suggests incretin-based agents may play a role in the management of women-specific conditions such as polycystic ovarian syndrome, through improvements in weight, insulin resistance and menstrual regularity.³⁷³ However, further sex-specific and reproductive outcome data are needed to inform best practice.³⁷⁴

Section 6:

Other populations and conditions

Children, adolescents and young adults

Obesity in early life is a strong predictor of cardiometabolic disease and premature CVD in adulthood.³⁷⁵ It is associated with early vascular ageing, dyslipidaemia, hypertension, insulin resistance and left ventricular hypertrophy, which increase lifetime risk of CVD.^{376,377} Early assessment and intervention for overweight or obesity in young people are essential to mitigate long-term cardiovascular morbidity.³⁷⁸ The management of overweight and obesity in children and adolescents should focus on multicomponent interventions that combine dietary strategies to improve diet quality and reduce consumption of highly processed foods, alongside increased physical activity and reduced sedentary behaviour.

Psychological, behavioural and social determinants should form part of a holistic approach. This should include emphasis on family-based behaviour approaches and coordinated multidisciplinary care delivered in a respectful, dignified and non-judgemental manner that avoids blame or stigmatisation. Interventions should be tailored to the developmental stage of the child or adolescent. Obesity management medications and metabolic bariatric surgery may be considered as therapeutic options in adolescents and young adults, alongside behaviour modifications. The upcoming NHMRC Clinical Practice Guidelines will provide further information about managing overweight and obesity in children, adolescents and young adults.

Older adults

The management of overweight and obesity in older adults with, or at high risk of, CVD should balance the cardiometabolic benefits of intentional weight reduction against the potential risks of sarcopenia, frailty and functional decline.^{8,106} Management strategies should be individualised, multidisciplinary and conservative, emphasising modest weight loss targets, high-quality nutrition to prevent malnutrition, and the important role of resistance and aerobic physical activity to preserve physical function and muscle mass.³⁷⁹

Sarcopenic obesity is defined as the co-existence of excess adiposity and low skeletal muscle mass and/or function.³⁸⁰ Sarcopenic obesity confers higher CVD risk than obesity defined by BMI alone. Measures of adiposity distribution (particularly central/visceral fat) alongside assessments of muscle mass and strength, can provide more clinically relevant information in this group.³⁸¹ Sarcopenia risk is increased in individuals with obesity related complications, including heart failure, prolonged injury or physical inactivity, and during periods of rapid weight loss.^{382,383} Evidence indicates that resistance and strength based exercise interventions are effective in mitigating muscle loss and improving functional outcomes, with particularly strong benefits observed in post menopausal women and those with sarcopenic obesity.^{382,384}

Obesity management medications and metabolic bariatric surgery may be considered in older adults, alongside behaviour modifications, when cardiometabolic risk is high and the potential benefits outweigh the age-related risks.³⁸⁵ However, a careful assessment of comorbidities and polypharmacy is required, alongside consideration of the person's preferences and priorities.³⁸⁶ The upcoming NHMRC Clinical Practice Guidelines will provide further information about managing overweight and obesity in older adults.

Obstructive sleep apnoea

Obstructive sleep apnoea (OSA) is a type of sleep disorder characterised by abnormal breathing patterns that reduce quality of sleep. Obesity, particularly central obesity, is a major risk factor for OSA, and OSA is more common in people with obesity.³⁸⁷ OSA causes intermittent hypoxia and negative intrathoracic pressure, leading to acute surges in blood pressure and heart rate and chronic changes such as left ventricular hypertrophy and autonomic dysfunction.³⁸⁸ Left untreated, OSA significantly increases the risk of stroke, coronary heart disease, atrial fibrillation³⁸⁹ and cardiovascular mortality.^{182,390}

Neck circumference, waist-to-hip ratio, body fat percentage and visceral adiposity index strongly predict OSA, with even small increases in waist-to-hip ratio increasing risk. Early identification and management of OSA are essential to mitigate long-term cardiovascular risk. Referral for polysomnography or home sleep apnoea testing should be considered in people with overweight/obesity and accompanying OSA symptoms such as snoring, daytime sleepiness, non-restorative sleep or morning headaches and hypertension. Continuous positive airway pressure (CPAP) therapy may improve symptoms and quality of life and reduce risk of major adverse cardiovascular events, although its role in secondary prevention remains uncertain.^{391,392}

Dementia

Emerging evidence links midlife obesity and its associated cardiometabolic risk factors, including hypertension, dyslipidaemia and insulin resistance, to an increased risk of cognitive decline and dementia later in life.^{393,394} These associations are thought to reflect shared pathophysiological mechanisms, including vascular dysfunction, inflammation and metabolic dysregulation, which accelerate neurodegeneration.³⁹⁵

The 2024 Lancet Commission on dementia prevention identifies obesity, hypertension, type 2 diabetes and elevated LDL-C as key modifiable midlife risk factors for dementia, emphasising that reducing vascular and metabolic damage across the life course has substantial potential to lower dementia risk.³⁹⁶ Effective clinical management of obesity and cardiovascular risk factors is likely to have significant benefits for cognitive health, including a potential reduction in dementia risk. Accordingly, weight and cardiometabolic health should be regarded as important components of strategies for long-term cognitive wellbeing.

References

1. Australian Institute of Health and Welfare. Heart, stroke and vascular disease: Australian facts. 2025. Accessed 30 Mar 2026. www.aihw.gov.au/reports/heart-stroke-vascular-diseases/hsvd-facts
2. Australian Institute of Health and Welfare. Overweight and obesity. 2024. Accessed 30 Mar 2026. www.aihw.gov.au/reports/overweight-obesity/overweight-and-obesity
3. World Obesity Federation. World Obesity Atlas. 2023. Accessed 30 Mar 2026. <https://data.worldobesity.org/publications/?cat=19>
4. Afshin A, Forouzanfar MH, Reitsma MB, et al. Health effects of overweight and obesity in 195 countries over 25 years. *N Engl J Med*. 2017;377(1):13-27. doi:10.1056/NEJMoa1614362
5. World Health Organization. Obesity and overweight. 2025. Accessed 30 Mar 2026. <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>
6. Australian Institute of Health and Welfare. *Australian Burden of Disease Study 2024*. 2024. www.aihw.gov.au/reports/burden-of-disease/australian-burden-of-disease-study-2024
7. Bays HE, Kirkpatrick CF, Maki KC, et al. Obesity, dyslipidemia, and cardiovascular disease: A joint expert review from the Obesity Medicine Association and the National Lipid Association 2024. *J Clin Lipidol*. 2024;18(3):e320-e350. doi:10.1016/j.jacl.2024.04.001
8. Powell-Wiley TM, Poirier P, Burke LE, et al. Obesity and Cardiovascular Disease: A Scientific Statement From the American Heart Association. *Circulation*. 2021;143(21):e984-e1010. doi:10.1161/cir.0000000000000973
9. Rubino F, Cummings DE, Eckel RH, et al. Definition and diagnostic criteria of clinical obesity. *Lancet Diabetes Endocrinol*. 2025;13(3):221-262. doi:10.1016/s2213-8587(24)00316-4
10. World Health Organization. *Health service delivery framework for prevention and management of obesity*. 2023. <https://www.who.int/publications/i/item/9789240073234>
11. Wen Z, Sun W, Wang H, et al. Comparison of the effectiveness and safety of GLP-1 receptor agonists for type 2 diabetes mellitus patients with overweight/obesity: A systematic review and network meta-analysis. *Diabetes Res Clin Pract*. 2025;222:111999. doi:10.1016/j.diabres.2025.111999
12. Rivera FB, Cruz LLA, Magalong JV, et al. Cardiovascular and renal outcomes of glucagon-like peptide 1 receptor agonists among patients with and without type 2 diabetes mellitus: A meta-analysis of randomized placebo-controlled trials. *Am J Prev Cardiol*. 2024;18:100679. doi:10.1016/j.ajpc.2024.100679
13. Hill B, de la Piedad Garcia X, Rathbone J, et al. Supporting healthcare professionals to reduce weight stigma. *Aust J Gen Pract*. 2024;53:682-685. doi:10.31128/AJGP-07-23-6906
14. National Health and Medical Research Council. *DRAFT Clinical practice guidelines for the management of overweight and obesity for adults, adolescents and children in Australia*. 2025. https://www.deakin.edu.au/__data/assets/pdf_file/0023/116924/Draft-Guidelines-Full-Guidelines.pdf
15. Commonwealth of Australia. *The National Obesity Strategy 2022-2032*. 2022. <https://www.health.gov.au/resources/publications/national-obesity-strategy-2022-2032?language=en>
16. Sidhu SK, Aleman JO, Heffron SP. Obesity duration and cardiometabolic disease. *Arterioscler Thromb Vasc Biol*. 2023;43(10):1764-1774. doi:10.1161/atvbaha.123.319023
17. European Society of Cardiology. *ESC Scientific Documents Policy*. 2021. Accessed 17 Mar 2026. <https://www.escardio.org/guidelines/scientific-documents/scientific-document-policy/>
18. Murad MH, Asi N, Alsawas M, Alahdab F. New evidence pyramid. *Evid Based Med*. 2016;21(4):125. doi:10.1136/ebmed-2016-110401

19. Welsh A, Hammad M, Piña IL, Kulinski J. Obesity and cardiovascular health. *Eur J Prev Cardiol.* 2024;31(8):1026-1035. doi:10.1093/eurjpc/zwae025
20. Shah A, Davarci O, Chaffari P, Avenatti E. Obesity as a disease: A primer on clinical and physiological insights. *Methodist Debakey Cardiovasc J.* 2025;21(2):4-13. doi:10.14797/mdcvj.1515
21. Kibret KT, Strugnell C, Backholer K, Peeters A, Tegegne TK, Nichols M. Life-course trajectories of body mass index and cardiovascular disease risks and health outcomes in adulthood: Systematic review and meta-analysis. *Obes Rev.* 2024;25(4):e13695. doi:10.1111/obr.13695
22. Jayedi A, Rashidy-Pour A, Khorshidi M, Shab-Bidar S. Body mass index, abdominal adiposity, weight gain and risk of developing hypertension: a systematic review and dose-response meta-analysis of more than 2.3 million participants. *Obes Rev.* 2018;19(5):654-667. doi:10.1111/obr.12656
23. Tan L, Long LZ, Ma XC, et al. Association of body mass index trajectory and hypertension risk: A systematic review of cohort studies and network meta-analysis of 89,094 participants. *Front Cardiovasc Med.* 2022;9:941341. doi:10.3389/fcvm.2022.941341
24. Quek J, Chan KE, Wong ZY, et al. Global prevalence of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis in the overweight and obese population: a systematic review and meta-analysis. *Lancet Gastroenterol Hepatol.* 2023;8(1):20-30. doi:10.1016/s2468-1253(22)00317-x
25. Lopez-Jimenez F, Almahmeed W, Bays H, et al. Obesity and cardiovascular disease: mechanistic insights and management strategies. A joint position paper by the World Heart Federation and World Obesity Federation. *Eur J Prev Cardiol.* 2022;29(17):2218-2237. doi:10.1093/eurjpc/zwac187
26. Australian Chronic Disease Prevention Alliance. *Australian guideline for assessing and managing cardiovascular disease risk 2023.* 2023. <https://www.cvdcheck.org.au>
27. Liu X, Ling J, Wu Y, et al. Association between metabolically healthy obesity and atrial fibrillation: A systematic review and meta-analysis of longitudinal studies. *Diabetes Metab Syndr.* 2025;19(4):103228. doi:10.1016/j.dsx.2025.103228
28. Wu G, Wu J, Lu Q, Cheng Y, Mei W. Association between cardiovascular risk factors and atrial fibrillation. *Front Cardiovasc Med.* 2023;10:1110424. doi:10.3389/fcvm.2023.1110424
29. Zheng Y, Xie Z, Li J, et al. Meta-analysis of metabolic syndrome and its individual components with risk of atrial fibrillation in different populations. *BMC Cardiovasc Disord.* 2021;21(1):90. doi:10.1186/s12872-021-01858-1
30. Asad Z, Abbas M, Javed I, Korantzopoulos P, Stavrakis S. Obesity is associated with incident atrial fibrillation independent of gender: A meta-analysis. *J Cardiovasc Electrophysiol.* 2018;29(5):725-732. doi:10.1111/jce.13458
31. Aune D, Sen A, Schlesinger S, et al. Body mass index, abdominal fatness, fat mass and the risk of atrial fibrillation: a systematic review and dose-response meta-analysis of prospective studies. *Eur J Epidemiol.* 2017;32(3):181-192. doi:10.1007/s10654-017-0232-4
32. Mongraw-Chaffin ML, Peters SAE, Huxley RR, Woodward M. The sex-specific association between BMI and coronary heart disease: a systematic review and meta-analysis of 95 cohorts with 1.2 million participants. *Lancet Diabetes Endocrinol.* 2015;3(6):437-449. doi:10.1016/s2213-8587(15)00086-8
33. Hsueh YW, Yeh TL, Lin CY, et al. Association of metabolically healthy obesity and elevated risk of coronary artery calcification: a systematic review and meta-analysis. *PeerJ.* 2020;8:e8815. doi:10.7717/peerj.8815
34. Mahajan R, Stokes M, Elliott A, et al. Complex interaction of obesity, intentional weight loss and heart failure: a systematic review and meta-analysis. *Heart.* 2020;106(1):58-68. doi:10.1136/heartjnl-2019-314770

35. Aune D, Sen A, Norat T, et al. Body mass index, abdominal fatness, and heart failure incidence and mortality: A systematic review and dose-response meta-analysis of prospective studies. *Circulation*. 2016;133(7):639-49. doi:10.1161/circulationaha.115.016801
36. Song P, Rudan D, Zhu Y, et al. Global, regional, and national prevalence and risk factors for peripheral artery disease in 2015: an updated systematic review and analysis. *The Lancet Global Health*. 2019;7(8):e1020-e1030. doi:10.1016/S2214-109X(19)30255-4
37. Wang X, Huang Y, Chen Y, et al. The relationship between body mass index and stroke: a systemic review and meta-analysis. *J Neurol*. 2022;269(12):6279-6289. doi:10.1007/s00415-022-11318-1
38. Ma LZ, Sun FR, Wang ZT, et al. Metabolically healthy obesity and risk of stroke: a meta-analysis of prospective cohort studies. *Ann Transl Med*. 2021;9(3):197. doi:10.21037/atm-20-4387
39. Dwivedi AK, Dubey P, Cistola DP, Reddy SY. Association between obesity and cardiovascular outcomes: updated evidence from meta-analysis studies. *Curr Cardiol Rep*. 2020;22(4):25. doi:10.1007/s11886-020-1273-y
40. Dugani SB, Hydoub YM, Ayala AP, et al. Risk factors for premature myocardial infarction: A systematic review and meta-analysis of 77 studies. *Mayo Clin Proc Innov Qual Outcomes*. 2021;5(4):783-794. doi:10.1016/j.mayocpiqo.2021.03.009
41. Chen H, Deng Y, Li S. Relation of body mass index categories with risk of sudden cardiac death: A systematic review and meta-analysis. *Int Heart J*. 2019;60(3):624-630. doi:10.1536/ihj.18-155
42. Aune D, Schlesinger S, Norat T, Riboli E. Body mass index, abdominal fatness, and the risk of sudden cardiac death: a systematic review and dose-response meta-analysis of prospective studies. *Eur J Epidemiol*. 2018;33(8):711-722. doi:10.1007/s10654-017-0353-9
43. Neeland IJ, Ross R, Després JP, et al. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *Lancet Diabetes Endocrinol*. 2019;7(9):715-725. doi:10.1016/s2213-8587(19)30084-1
44. Xue R, Li Q, Geng Y, Wang H, Wang F, Zhang S. Abdominal obesity and risk of CVD: a dose-response meta-analysis of thirty-one prospective studies. *Br J Nutr*. 2021;126(9):1420-1430. doi:10.1017/s0007114521000064
45. Cordola Hsu AR, Xie B, Peterson DV, et al. Metabolically Healthy/Unhealthy Overweight/Obesity Associations With Incident Heart Failure in Postmenopausal Women. *Circulation: Heart Failure*. 2021;14(4):e007297. doi:10.1161/CIRCHEARTFAILURE.120.007297
46. Weeldreyer NR, De Guzman JC, Paterson C, Allen JD, Gaesser GA, Angadi SS. Cardiorespiratory fitness, body mass index and mortality: a systematic review and meta-analysis. *Br J Sports Med*. 2025;59(5):339-346. doi:10.1136/bjsports-2024-108748
47. Barry VW, Caputo JL, Kang M. The Joint Association of Fitness and Fatness on Cardiovascular Disease Mortality: A Meta-Analysis. *Prog Cardiovasc Dis*. 2018;61(2):136-141. doi:10.1016/j.pcad.2018.07.004
48. Geraci C, Geraci G, Buonauro A, Morello V, La Rocca F, Esposito R. Gaps in Current Cardiometabolic Risk Assessment: A Review Supporting the Development of the C.O.R.E. Indicator Model. *Journal of Clinical Medicine*. 2026;15(2):617. doi:10.3390/jcm15020617
49. Zhang X, Zhu J, Kim JH, Sumerlin TS, Feng Q, Yu J. Metabolic health and adiposity transitions and risks of type 2 diabetes and cardiovascular diseases: a systematic review and meta-analysis. *Diabetol Metab Syndr*. 2023;15(1):60. doi:10.1186/s13098-023-01025-w
50. Eckel N, Meidtner K, Kalle-Uhlmann T, Stefan N, Schulze MB. Metabolically healthy obesity and cardiovascular events: A systematic review and meta-analysis. *Eur J Prev Cardiol*. 2016;23(9):956-66. doi:10.1177/2047487315623884

51. Nowak MM, Niemczyk M, Gołębiewski S, Pączek L. Impact of Body Mass Index on All-Cause Mortality in Adults: A Systematic Review and Meta-Analysis. *J Clin Med*. 2024;13(8):2305. doi:10.3390/jcm13082305
52. Wang M, Wei X, Zhao M. Association of body mass index with clinical outcomes in patients with acute coronary syndrome: A systematic review and meta-analysis. *Turk Gogus Kalp Damar Cerrahisi Derg*. 2024;32(1):1-8. doi:10.5606/tgkdc.dergisi.2024.24405
53. Wattanachayakul P, Yanpiset P, Wannaphut C, et al. Association between obesity paradox in the all-cause mortality among patients with cardiac resynchronization therapy device. *Pacing Clin Electrophysiol*. 2024;47(11):1464-1473. doi:10.1111/pace.15069
54. Liu SH, Lin YZ, Han S, Jin YZ. The obesity paradox in ST-segment elevation myocardial infarction patients: A meta-analysis. *Ann Noninvasive Electrocardiol*. 2023;28(2):e13022. doi:10.1111/anec.13022
55. De Paola L, Mehta A, Pana TA, et al. Body Mass Index and Mortality, Recurrence and Readmission after Myocardial Infarction: Systematic Review and Meta-Analysis. *J Clin Med*. 2022;11(9):2581. doi:10.3390/jcm11092581
56. Hoffman H, Cote JR, Wood J, Jalal MS, Draytsel DY, Gould GC. The Influence of Body Mass Index on Outcomes in Patients Undergoing Mechanical Thrombectomy for Anterior Circulation Large Vessel Occlusion: Institutional Experience and Meta-analysis. *Neurocrit Care*. 2024;40(2):654-663. doi:10.1007/s12028-023-01801-6
57. Yasmin F, Moeed A, Zaidi F, Adil M, Surani S, Alraies MC. Impact of obesity on outcomes of transcatheter aortic valve implantation in patients with aortic stenosis: a systematic review and meta-analysis of real-world data. *Am J Cardiovasc Dis*. 2025;15(2):85-99. doi:10.62347/vtye4110
58. An KR, Sandner S, Redfors B, et al. Association between overweight and obesity with coronary artery bypass graft failure: an individual patient data analysis of clinical trials. *Eur J Cardiothorac Surg*. 2024;65(6):ezae221. doi:10.1093/ejcts/ezae221
59. Seo J, Li W, Safiriyu I, et al. A Meta-Analysis on the Impact of High BMI in Patients Undergoing Transcatheter Aortic Valve Replacement. *Journal of Cardiovascular Development and Disease*. 2022;9(11):386. doi:10.3390/jcdd9110386
60. Mei X, Hu S, Mi L, Zhou Y, Chen T. Body mass index and all-cause mortality in patients with percutaneous coronary intervention: A dose-response meta-analysis of obesity paradox. *Obes Rev*. 2021;22(2):e13107. doi:10.1111/obr.13107
61. Zomer E, Gurusamy K, Leach R, et al. Interventions that cause weight loss and the impact on cardiovascular risk factors: a systematic review and meta-analysis. *Obes Rev*. 2016;17(10):1001-11. doi:10.1111/obr.12433
62. Ma C, Avenell A, Bolland M, et al. Effects of weight loss interventions for adults who are obese on mortality, cardiovascular disease, and cancer: systematic review and meta-analysis. *BMJ*. 2017;359:j4849. doi:10.1136/bmj.j4849
63. Lemstra M, Bird Y, Nwankwo C, Rogers M, Moraros J. Weight loss intervention adherence and factors promoting adherence: a meta-analysis. *Patient Preference and Adherence*. 2016;10:1547-1559. doi:10.2147/PPA.S103649
64. Ndumele CE, Rangaswami J, Chow SL, et al. Cardiovascular-Kidney-Metabolic Health: A Presidential Advisory From the American Heart Association. *Circulation*. 2023;148(20):1606-1635. doi:10.1161/cir.0000000000001184
65. Tsai MK, Kao JT, Wong CS, et al. Cardiovascular-kidney-metabolic syndrome and all-cause and cardiovascular mortality: A retrospective cohort study. *PLoS Med*. 2025;22(6):e1004629. doi:10.1371/journal.pmed.1004629
66. Theodorakis N, Nikolaou M. From Cardiovascular-Kidney-Metabolic Syndrome to Cardiovascular-Renal-Hepatic-Metabolic Syndrome: Proposing an Expanded Framework. *Biomolecules*. 2025;15(2)doi:10.3390/biom15020213

67. Theodorakis N, Nikolaou M. Integrated Management of Cardiovascular-Renal-Hepatic-Metabolic Syndrome: Expanding Roles of SGLT2is, GLP-1RAs, and GIP/GLP-1RAs. *Biomedicines*. 2025;13(1):135. doi:10.3390/biomedicines13010135
68. Alberga AS, Nagpal TS, Patton I. A Call for Weight Stigma Reduction in Physical Activity, Exercise, and Sport Settings. *Journal of Physical Activity and Health*. 2025;22(8):875-877. doi:10.1123/jpah.2025-0221
69. Prunty A, Hahn A, O'Shea A, Edmonds S, Clark MK. Associations among enacted weight stigma, weight self-stigma, and multiple physical health outcomes, healthcare utilization, and selected health behaviors. *International Journal of Obesity*. 2023;47(1):33-38. doi:10.1038/s41366-022-01233-w
70. Figueroa DG, Murley WD, Parker JE, Hunger JM, Tomiyama AJ. Weight stigma and mental health symptoms: mediation by perceived stress. *Front Psychiatry*. 2025;16:1587105. doi:10.3389/fpsy.2025.1587105
71. Alimoradi Z, Golboni F, Griffiths MD, Broström A, Lin CY, Pakpour AH. Weight-related stigma and psychological distress: A systematic review and meta-analysis. *Clin Nutr*. 2020;39(7):2001-2013. doi:10.1016/j.clnu.2019.10.016
72. Wu YK, Berry DC. Impact of weight stigma on physiological and psychological health outcomes for overweight and obese adults: A systematic review. *J Adv Nurs*. 2018;74(5):1030-1042. doi:10.1111/jan.13511
73. Puhl RM, Heuer CA. Obesity stigma: important considerations for public health. *Am J Public Health*. 2010;100(6):1019-28. doi:10.2105/ajph.2009.159491
74. Pearl RL, Wadden TA, Hopkins CM, et al. Association between weight bias internalization and metabolic syndrome among treatment-seeking individuals with obesity. *Obesity (Silver Spring)*. 2017;25(2):317-322. doi:10.1002/oby.21716
75. Sutin AR, Stephan Y, Terracciano A. Weight Discrimination and Risk of Mortality. *Psychol Sci*. 2015;26(11):1803-11. doi:10.1177/0956797615601103
76. Phelan SM, Burgess DJ, Yeazel MW, Hellerstedt WL, Griffin JM, van Ryn M. Impact of weight bias and stigma on quality of care and outcomes for patients with obesity. *Obes Rev*. 2015;16(4):319-26. doi:10.1111/obr.12266
77. Puhl RM, Lessard LM, Himmelstein MS, Foster GD. The roles of experienced and internalized weight stigma in healthcare experiences: Perspectives of adults engaged in weight management across six countries. *PLoS One*. 2021;16(6):e0251566. doi:10.1371/journal.pone.0251566
78. Rubino F, Puhl RM, Cummings DE, et al. Joint international consensus statement for ending stigma of obesity. *Nat Med*. 2020;26(4):485-497. doi:10.1038/s41591-020-0803-x
79. Mihaileanu FV, Fadgyas Stanculete M, Gherman C, et al. Beyond the Physical: Weight Stigma and the Bariatric Patient Journey. *J Clin Med*. 2025;14(2):543. doi:10.3390/jcm14020543
80. Philip SR, Standen EC, Schueler J, Fields SA, Phelan SM. Weight bias in mental health settings: a scoping review. *Front Psychiatry*. 2025;16:1596625. doi:10.3389/fpsy.2025.1596625
81. Mekonnen A, Vasilevski V, Chapman A, et al. Barriers and enablers to effective weight management for people living with overweight and obesity: A rapid scoping review. *Obesity Reviews*. 2025;26(3):e13858. doi:10.1111/obr.13858
82. Pearl RL, Sheynblyum M. How Weight Bias and Stigma Undermine Healthcare Access and Utilization. *Current Obesity Reports*. 2025;14(1):11. doi:10.1007/s13679-025-00605-3
83. Gilbert O, Gulati M, Gluckman TJ, et al. 2025 Concise Clinical Guidance: An ACC Expert Consensus Statement on Medical Weight Management for Optimization of Cardiovascular Health. *JACC*. 2025;86(7):536-555. doi:10.1016/j.jacc.2025.05.024
84. Kelly R, Hatzikiriakidis K, Kuswara K. Inequities in obesity: Indigenous, culturally and linguistically diverse, and disability perspectives. *Public Health Research & Practice*. 2022;32(3):3232225. doi:10.17061/phrp3232225

85. Afzal M, Siddiqi N, Ahmad B, et al. Prevalence of Overweight and Obesity in People With Severe Mental Illness: Systematic Review and Meta-Analysis. *Front Endocrinol (Lausanne)*. 2021;12:769309. doi:10.3389/fendo.2021.769309
86. Palacios S, Chedraui P, Sanchez-Borrego R, et al. Management of obesity in menopause. *Climacteric*. 2024;27(4):357-363. doi:10.1080/13697137.2024.2374760
87. Golestani R, Farahani FK, Peters P. Exploring barriers to accessing health care services by young women in rural settings: a qualitative study in Australia, Canada, and Sweden. *BMC Public Health*. 2025;25(1):213. doi:10.1186/s12889-025-21387-2
88. Offer S, Alexander E, Barbara K, Hemmingsson E, Flint SW, Lawrence BJ. The association between childhood trauma and overweight and obesity in young adults: the mediating role of food addiction. *Eat Weight Disord*. 2022;27(8):3257-3266. doi:10.1007/s40519-022-01454-y
89. Baez AS, Ortiz-Whittingham LR, Tarfa H, et al. Social determinants of health, health disparities, and adiposity. *Prog Cardiovasc Dis*. 2023;78:17-26. doi:10.1016/j.pcad.2023.04.011
90. Laddu D, Neeland IJ, Carnethon M, et al. Implementation of Obesity Science Into Clinical Practice: A Scientific Statement From the American Heart Association. *Circulation*. 2024;150(1):e7-e19. doi:10.1161/cir.0000000000001221
91. Busetto L, Dicker D, Frühbeck G, et al. A new framework for the diagnosis, staging and management of obesity in adults. *Nature Medicine*. 2024;30(9):2395-2399. doi:10.1038/s41591-024-03095-3
92. Royal Australian College of General Practitioners. *Guidelines for preventive activities in general practice (10th edition)*. 2025. <https://www.racgp.org.au/clinical-resources/clinical-guidelines/key-racgp-guidelines/view-all-racgp-guidelines/preventive-activities-in-general-practice/about-the-red-book>
93. World Health Organization. *Waist circumference and waist-hip ratio: report of a WHO expert consultation*. 2008. <https://www.who.int/publications/i/item/9789241501491>
94. National Health and Medical Research Council. *DRAFT Clinical practice guidelines for the management of overweight and obesity for adults, adolescents and children in Australia*. 2025. https://www.deakin.edu.au/___data/assets/pdf_file/0023/116924/Draft-Guidelines-Full-Guidelines.pdf
95. Atkins JL, Wannamethee SG. Sarcopenic obesity in ageing: cardiovascular outcomes and mortality. *Br J Nutr*. 2020;124(10):1102-1113. doi:10.1017/s0007114520002172
96. Erdoğan O, Erdoğan T, Önür NH, Özkök S, Karan MA, Bahat G. Beyond BMI: central obesity measures and cardiovascular risk in late life. *Aging Clin Exp Res*. 2025;37(1):287. doi:10.1007/s40520-025-03197-z
97. Taylor RW, Brooking L, Williams SM, et al. Body mass index and waist circumference cutoffs to define obesity in indigenous New Zealanders. *Am J Clin Nutr*. 2010;92(2):390-7. doi:10.3945/ajcn.2010.29317
98. Reed RM, Whyte MB, Goff LM. Cardiometabolic disease in Black African and Caribbean populations: an ethnic divergence in pathophysiology? *Proc Nutr Soc*. 2025;84(3):316-326. doi:10.1017/s0029665123004895
99. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet*. 2004;363(9403):157-63. doi:10.1016/s0140-6736(03)15268-3
100. Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. *Obes Rev*. 2012;13(3):275-86. doi:10.1111/j.1467-789X.2011.00952.x
101. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0.5 could be a suitable global boundary value. *Nutr Res Rev*. 2010;23(2):247-69. doi:10.1017/s0954422410000144

102. Kondalsamy-Chennakesavan S, Hoy WE, Wang Z, et al. Anthropometric measurements of Australian Aboriginal adults living in remote areas: Comparison with nationally representative findings. *American Journal of Human Biology*. 2008;20(3):317-324. doi:10.1002/ajhb.20729
103. Munshi A, Garg R. Obesity and Menopause: Redefining Obesity - New Guidelines. *J Midlife Health*. 2025;16(1):1-5. doi:10.4103/jmh.jmh_24_25
104. Aragaki AK, Manson JE, LeBlanc ES, et al. Development and Validation of Body Mass Index–Specific Waist Circumference Thresholds in Postmenopausal Women. *Annals of Internal Medicine*. 2025;178(8):1073-1084. doi:10.7326/ANNALS-24-00713
105. Anker SD, Linong J, Kindel T, et al. iCARDIO Alliance Global Implementation Guidelines for the Management of Obesity 2025. Vol. 3. 2025. *Global Cardiology*. Accessed 4 Aug 2025. <https://icardioalliance.org/icardioalliance-cvd-in-obesity-guidelines/>
106. Koskinas KC, Van Craenenbroeck EM, Antoniadou C, et al. Obesity and cardiovascular disease: an ESC clinical consensus statement. *Eur Heart J*. 2024;45(38):4063-4098. doi:10.1093/eurheartj/ehae508
107. Neeland IJ, Poirier P, Després JP. Cardiovascular and Metabolic Heterogeneity of Obesity: Clinical Challenges and Implications for Management. *Circulation*. 2018;137(13):1391-1406. doi:10.1161/circulationaha.117.029617
108. Australian Government Department of Health, Disability and Ageing. Medicare Benefits Schedule - Note DN.1.18 Accessed 17 Mar 2026. <https://www9.health.gov.au/mbs/fullDisplay.cfm?type=note&q=DN.1.18&qf=noteID>
109. Alberti KG, Zimmet P, Shaw J. Metabolic syndrome—a new world-wide definition. A Consensus Statement from the International Diabetes Federation. *Diabet Med*. 2006;23(5):469-80. doi:10.1111/j.1464-5491.2006.01858.x
110. Department of Health and Aged Care. *Body mass index (BMI) and waist measurement*. 2021. <https://www.health.gov.au/topics/overweight-and-obesity/bmi-and-waist>
111. World Health Organization. Global database on body mass index: BMI classification 2006. Accessed 1 Apr 2026. <http://www.assessmentpsychology.com/icbmi.htm>
112. Alberti KG, Zimmet P, Shaw J. The metabolic syndrome—a new worldwide definition. *Lancet*. 2005;366(9491):1059-62. doi:10.1016/s0140-6736(05)67402-8
113. Ashwell M, Gibson S. Waist to height ratio is a simple and effective obesity screening tool for cardiovascular risk factors: Analysis of data from the British National Diet And Nutrition Survey of adults aged 19-64 years. *Obes Facts*. 2009;2(2):97-103. doi:10.1159/000203363
114. Wharton S, Lau DCW, Vallis M, et al. Obesity in adults: a clinical practice guideline. *CMAJ*. 2020;192(31):E875-e891. doi:10.1503/cmaj.191707
115. Mulvihill C, Parretti HM, Aswani N. Overweight and obesity management: summary of updated NICE guidance. *BMJ*. 2025;391:r2286. doi:10.1136/bmj.r2286
116. Mallik R, Carpenter J, Zalin A. Assessment of obesity. *Clin Med (Lond)*. 2023;23(4):299-303. doi:10.7861/clinmed.2023-0148
117. Caleyachetty R, Thomas GN, Toulis KA, et al. Metabolically Healthy Obese and Incident Cardiovascular Disease Events Among 3.5 Million Men and Women. *J Am Coll Cardiol*. 2017;70(12):1429-1437. doi:10.1016/j.jacc.2017.07.763
118. Forhan M, Gill SV. Obesity, functional mobility and quality of life. *Best Pract Res Clin Endocrinol Metab*. 2013;27(2):129-37. doi:10.1016/j.beem.2013.01.003

119. Rhynehart A, Dunlevy C, Hayes K, O'Connell J, O'Shea D, O'Malley E. The Association of Physical Function Measures With Frailty, Falls History, and Metabolic Syndrome in a Population With Complex Obesity. Original Research. *Frontiers in Rehabilitation Sciences*. 2021;2: 716392. doi:10.3389/fresc.2021.716392
120. Byker Shanks C, Bruening M, Yaroch AL. BMI or not to BMI? debating the value of body mass index as a measure of health in adults. *International Journal of Behavioral Nutrition and Physical Activity*. 2025;22(1):23. doi:10.1186/s12966-025-01719-6
121. Wu Y, Li D, Vermund SH. Advantages and Limitations of the Body Mass Index (BMI) to Assess Adult Obesity. *International Journal of Environmental Research and Public Health*. 2024;21(6):757. doi:10.3390/ijerph21060757.
122. Sweatt K, Garvey WT, Martins C. Strengths and Limitations of BMI in the Diagnosis of Obesity: What is the Path Forward? *Current Obesity Reports*. 2024;13(3):584-595. doi:10.1007/s13679-024-00580-1
123. Poirier P, Lavie CJ, Connelly K. Practical Implications of Obesity for Cardiovascular Diagnostics. *Can J Cardiol*. 2025;41(9):1679-1698. doi:10.1016/j.cjca.2025.07.006
124. Matta M, Harb SC, Cremer P, Hachamovitch R, Ayoub C. Stress testing and noninvasive coronary imaging: What's the best test for my patient? *Cleve Clin J Med*. 2021;88(9):502-515. doi:10.3949/ccjm.88a.20068
125. Picano E, Pierard L, Peteiro J, et al. The clinical use of stress echocardiography in chronic coronary syndromes and beyond coronary artery disease: a clinical consensus statement from the European Association of Cardiovascular Imaging of the ESC. *Eur Heart J Cardiovasc Imaging*. 2024;25(2):e65-e90. doi:10.1093/ehjci/jead250
126. Constantino MF CF, D'Addeo G. Diagnostic modalities for ischemic heart disease: evaluating the role of stress echocardiography, cardiac CT, and myocardial perfusion scintigraphy. *Exploration of Cardiology*. 2025;3:101243. doi:10.37349/ec.2025.101243
127. Perry AS, Dooley EE, Master H, Spartano NL, Brittain EL, Pettee Gabriel K. Physical Activity Over the Lifecourse and Cardiovascular Disease. *Circulation Research*. 2023;132(12):1725-1740. doi:10.1161/CIRCRESAHA.123.322121
128. Lichtenstein AH, Appel LJ, Vadiveloo M, et al. 2021 Dietary Guidance to Improve Cardiovascular Health: A Scientific Statement From the American Heart Association. *Circulation*. 2021;144(23):e472-e487. doi:10.1161/CIR.0000000000001031
129. Chan BS, Yu DSF, Wong CWY, Li PWC. Multi-modal interventions outperform nutritional or exercise interventions alone in reversing metabolic syndrome: a systematic review and network meta-analysis. *European Journal of Preventive Cardiology*. 2025;zwaf167:In press. doi:10.1093/eurjpc/zwaf167
130. O'Sullivan DJ, Bearne LM, Harrington JM, Cardoso JR, McVeigh JG. The effectiveness of social prescribing in the management of long-term conditions in community-based adults: A systematic review and meta-analysis. *Clin Rehabil*. 2024;38(10):1306-1320. doi:10.1177/02692155241258903
131. Htun HL, Teshale AB, Cumpston MS, et al. Effectiveness of social prescribing for chronic disease prevention in adults: a systematic review and meta-analysis of randomised controlled trials. *Journal of Epidemiology and Community Health*. 2023;77(4):265. doi:10.1136/jech-2022-220247
132. Xu X, Zeng L, Jha V, et al. Potassium-Enriched Salt Substitutes: A Review of Recommendations in Clinical Management Guidelines. *Hypertension*. 2024;81(3):400-414. doi:10.1161/HYPERTENSIONAHA.123.21343
133. Cheng M, Zhu C, Liu H, Pu C, Hou Y. Effects of calorie restriction therapy on health-related outcomes in patients with heart failure, a systematic review and meta-analysis. *Nutr Metab Cardiovasc Dis*. 2025;35(1):103756. doi:10.1016/j.numecd.2024.09.026
134. Harris L, McGarty A, Hutchison L, Ells L, Hankey C. Short-term intermittent energy restriction interventions for weight management: a systematic review and meta-analysis. *Obesity Reviews*. 2018;19(1):1-13. doi:10.1111/obr.12593

135. McKechnie T, Lee Y, Dionne J, et al. Very low energy diets prior to bariatric surgery may reduce postoperative morbidity: a systematic review and meta-analysis of randomized controlled trials. *Front Nutr.* 2023;10:1211575. doi:10.3389/fnut.2023.1211575
136. Royal Australian College of General Practitioners. *VLED for type 2 diabetes. Handbook of non-drug interventions.* 2020. <https://www.racgp.org.au/clinical-resources/clinical-guidelines/handi/handi-interventions/nutrition/vled-for-type-2-diabetes>
137. Ezzati A, Rosenkranz SK, Phelan J, Logan C. The Effects of Isocaloric Intermittent Fasting vs Daily Caloric Restriction on Weight Loss and Metabolic Risk Factors for Noncommunicable Chronic Diseases: A Systematic Review of Randomized Controlled or Comparative Trials. *Journal of the Academy of Nutrition and Dietetics.* 2023;123(2):318-329.e1. doi:10.1016/j.jand.2022.09.013
138. Silverii GA, Cresci B, Benvenuti F, Santagiuliana F, Rotella F, Mannucci E. Effectiveness of intermittent fasting for weight loss in individuals with obesity: A meta-analysis of randomized controlled trials. *Nutr Metab Cardiovasc Dis.* 2023;33(8):1481-1489. doi:10.1016/j.numecd.2023.05.005
139. Wang B, Wang C, Li H. The impact of intermittent fasting on body composition and cardiometabolic outcomes in overweight and obese adults: a systematic review and meta-analysis of randomized controlled trials. *Nutrition Journal.* 2025;24(1):120. doi:10.1186/s12937-025-01178-6
140. Semnani-Azad Z, Khan TA, Chiavaroli L, et al. Intermittent fasting strategies and their effects on body weight and other cardiometabolic risk factors: systematic review and network meta-analysis of randomised clinical trials. *BMJ.* 2025;389:e082007. doi:10.1136/bmj-2024-082007
141. Schroor MM, Joris PJ, Plat J, Mensink RP. Effects of Intermittent Energy Restriction Compared with Those of Continuous Energy Restriction on Body Composition and Cardiometabolic Risk Markers - A Systematic Review and Meta-Analysis of Randomized Controlled Trials in Adults. *Advances in Nutrition.* 2024;15(1)doi:10.1016/j.advnut.2023.10.003
142. Lei L, Huang J, Zhang L, Hong Y, Hui S, Yang J. Effects of low-carbohydrate diets versus low-fat diets on metabolic risk factors in overweight and obese adults: A meta-analysis of randomized controlled trials. *Front Nutr.* 2022;9:935234. doi:10.3389/fnut.2022.935234
143. Yao Y, Huang V, Seah V, Kim JE. Impact of Quantity and Type of Dietary Protein on Cardiovascular Disease Risk Factors Using Standard and Network Meta-analyses of Randomized Controlled Trials. *Nutr Rev.* 2025;83(3):e814-e828. doi:10.1093/nutrit/nuae086
144. George ES, Kucianski T, Mayr HL, Moschonis G, Tierney AC, Itsiopoulos C. A Mediterranean Diet Model in Australia: Strategies for Translating the Traditional Mediterranean Diet into a Multicultural Setting. *Nutrients.* 2018;10(4):In press. doi:10.3390/nu10040465
145. Jayedi A, Soltani S, Abdolshahi A, Shab-Bidar S. Healthy and unhealthy dietary patterns and the risk of chronic disease: an umbrella review of meta-analyses of prospective cohort studies. *Br J Nutr.* 2020;124(11):1133-1144. doi:10.1017/s0007114520002330
146. Hajjar M, Rezazadeh A. The Recommended Food Score and Healthy Nordic Food Index in cardiovascular disease and stroke: A systematic review. *ARYA Atheroscler.* 2020;16(5):248-257. doi:10.22122/arya.v16i5.2067
147. Heart Foundation. *Dietary Position Statement: Heart Healthy Eating Patterns.* 2019. https://assets.contentstack.io/v3/assets/blf8a393bb3b76c0ede/bltfdaee65e5c5af0/659e0be9a0eb30216a4ff37d/Nutrition_Position_Statement_-_HHEP_FINAL-3.pdf
148. Ge L, Sadeghirad B, Ball GDC, et al. Comparison of dietary macronutrient patterns of 14 popular named dietary programmes for weight and cardiovascular risk factor reduction in adults: systematic review and network meta-analysis of randomised trials. *BMJ.* 2020;369:m696. doi:10.1136/bmj.m696

149. Cross V, Stanford J, Gómez-Martín M, Collins CE, Robertson S, Clarke ED. Do Personalized Nutrition Interventions Improve Dietary Intake and Risk Factors in Adults With Elevated Cardiovascular Disease Risk Factors? A Systematic Review and Meta-analysis of Randomized Controlled Trials. *Nutr Rev*. 2025;83(7):e1709-e1721. doi:10.1093/nutrit/nuae149
150. Adler AJ, Taylor F, Martin N, Gottlieb S, Taylor RS, Ebrahim S. Reduced dietary salt for the prevention of cardiovascular disease. *Cochrane Database Syst Rev*. 2014;2014(12):CD009217. doi:10.1002/14651858.CD009217.pub3
151. Huang L, Trieu K, Yoshimura S, et al. Effect of dose and duration of reduction in dietary sodium on blood pressure levels: systematic review and meta-analysis of randomised trials. *BMJ*. 2020;368:m315. doi:10.1136/bmj.m315
152. Sacks FM, Bray GA, Carey VJ, et al. Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. *N Engl J Med*. 2009;360(9):859-73. doi:10.1056/NEJMoa0804748
153. Johnston BC, Kanters S, Bandayrel K, et al. Comparison of weight loss among named diet programs in overweight and obese adults: a meta-analysis. *JAMA*. 2014;312(9):923-33. doi:10.1001/jama.2014.10397
154. Astbury NM, Piernas C, Hartmann-Boyce J, Lapworth S, Aveyard P. A systematic review and meta-analysis of the effectiveness of meal replacements for weight loss. *Obesity Reviews*. 2019;20(4):569-587. doi:10.1111/obr.12816
155. Strain T, Dempsey PC, Wijndaele K, et al. Quantifying the Relationship Between Physical Activity Energy Expenditure and Incident Type 2 Diabetes: A Prospective Cohort Study of Device-Measured Activity in 90,096 Adults. *Diabetes Care*. 2023;46(6):1145-1155. doi:10.2337/dc22-1467
156. Iso-Markku P, Kujala UM, Knittle K, Polet J, Vuoksima E, Waller K. Physical activity as a protective factor for dementia and Alzheimer's disease: systematic review, meta-analysis and quality assessment of cohort and case-control studies. *Br J Sports Med*. 2022;56(12):701-709. doi:10.1136/bjsports-2021-104981
157. Garcia L, Pearce M, Abbas A, et al. Non-occupational physical activity and risk of cardiovascular disease, cancer and mortality outcomes: a dose-response meta-analysis of large prospective studies. *Br J Sports Med*. 2023;57(15):979-989. doi:10.1136/bjsports-2022-105669
158. Australian Government Department of Health, Disability and Ageing. 24 hour movement guidelines for all Australians. 2026. Accessed 18 Mar 2026. <https://www.health.gov.au/topics/physical-activity/24-hour-movement-guidelines-for-all-australians>
159. Bull FC, Al-Ansari SS, Biddle S, et al. World Health Organization 2020 guidelines on physical activity and sedentary behaviour. *Br J Sports Med*. 2020;54(24):1451-1462. doi:10.1136/bjsports-2020-102955
160. Reed JL, Pipe AL. The talk test: a useful tool for prescribing and monitoring exercise intensity. *Curr Opin Cardiol*. 2014;29(5):475-80. doi:10.1097/hco.0000000000000097
161. Sá KMM, da Silva GR, Martins UK, et al. Resistance training for postmenopausal women: systematic review and meta-analysis. *Menopause*. 2023;30(1):108-116. doi:10.1097/gme.0000000000002079
162. Capel-Alcaraz AM, García-López H, Castro-Sánchez AM, Fernández-Sánchez M, Lara-Palomo IC. The Efficacy of Strength Exercises for Reducing the Symptoms of Menopause: A Systematic Review. *Journal of Clinical Medicine*. 2023;12(2):548. doi:10.3390/jcm12020548
163. Hu C, Xia Y, Zeng D, Ye M, Mei T. Effect of resistance circuit training on comprehensive health indicators in older adults: a systematic review and meta-analysis. *Sci Rep*. 2024;14(1):8823. doi:10.1038/s41598-024-59386-9
164. Islam H, Gibala MJ, Little JP. Exercise Snacks: A Novel Strategy to Improve Cardiometabolic Health. *Exerc Sport Sci Rev*. 2022;50(1):31-37. doi:10.1249/jes.0000000000000275

165. Rodríguez MÁ, Quintana-Cepedal M, Cheval B, Thøgersen-Ntoumani C, Crespo I, Olmedillas H. Effect of exercise snacks on fitness and cardiometabolic health in physically inactive individuals: systematic review and meta-analysis. *British Journal of Sports Medicine*. 2026;60(2):133-141. doi:10.1136/bjsports-2025-110027
166. Ding D, Nguyen B, Nau T, et al. Daily steps and health outcomes in adults: a systematic review and dose-response meta-analysis. *The Lancet Public Health*. 2025;10(8):e668-e681. doi:10.1016/S2468-2667(25)00164-1
167. Angelucci A, Canali S, Aliverti A. Digital technologies for step counting: between promises of reliability and risks of reductionism. *Front Digit Health*. 2023;5:1330189. doi:10.3389/fdgth.2023.1330189
168. Oppert JM, Bellicha A, van Baak MA, et al. Exercise training in the management of overweight and obesity in adults: Synthesis of the evidence and recommendations from the European Association for the Study of Obesity Physical Activity Working Group. *Obes Rev*. 2021;22 (Suppl 4):e13273. doi:10.1111/obr.13273
169. Johnson NA, Sultana RN, Brown WJ, Bauman AE, Gill T. Physical activity in the management of obesity in adults: A position statement from Exercise and Sport Science Australia. *Journal of Science and Medicine in Sport*. 2021;24(12):1245-1254. doi:10.1016/j.jsams.2021.07.009
170. Locatelli JC, Costa JG, Haynes A, et al. Incretin-Based Weight Loss Pharmacotherapy: Can Resistance Exercise Optimize Changes in Body Composition? *Diabetes Care*. 2024;47(10):1718-1730. doi:10.2337/dci23-0100
171. Memel Z, Gold SL, Pearlman M, Muratore A, Martindale R. Impact of GLP- 1 Receptor Agonist Therapy in Patients High Risk for Sarcopenia. *Curr Nutr Rep*. 2025;14(1):63. doi:10.1007/s13668-025-00649-w
172. Binmahfoz A, Dighriri A, Gray C, Gray SR. Effect of resistance exercise on body composition, muscle strength and cardiometabolic health during dietary weight loss in people living with overweight or obesity: a systematic review and meta-analysis. *BMJ Open Sport Exerc Med*. 2025;11(3):e002363. doi:10.1136/bmjsem-2024-002363
173. Wang H, Cheng R, Xie L, Hu F. Comparative efficacy of exercise training modes on systemic metabolic health in adults with overweight and obesity: a network meta-analysis of randomized controlled trials. *Front Endocrinol (Lausanne)*. 2023;14:1294362. doi:10.3389/fendo.2023.1294362
174. Wewege MA, Thom JM, Rye K-A, Parmenter BJ. Aerobic, resistance or combined training: A systematic review and meta-analysis of exercise to reduce cardiovascular risk in adults with metabolic syndrome. *Atherosclerosis*. 2018;274:162-171. doi:10.1016/j.atherosclerosis.2018.05.002
175. Batrakoulis A, Jamurtas AZ, Metsios GS, et al. Comparative Efficacy of 5 Exercise Types on Cardiometabolic Health in Overweight and Obese Adults: A Systematic Review and Network Meta-Analysis of 81 Randomized Controlled Trials. *Circ Cardiovasc Qual Outcomes*. 2022;15(6):e008243. doi:10.1161/circoutcomes.121.008243
176. Ahmadi MN, Hamer M, Gill JMR, et al. Brief bouts of device-measured intermittent lifestyle physical activity and its association with major adverse cardiovascular events and mortality in people who do not exercise: a prospective cohort study. *Lancet Public Health*. 2023;8(10):e800-e810. doi:10.1016/s2468-2667(23)00183-4
177. Ohayon M, Wickwire EM, Hirshkowitz M, et al. National Sleep Foundation's sleep quality recommendations: first report. *Sleep Health*. 2017;3(1):6-19. doi:10.1016/j.sleh.2016.11.006
178. Briançon-Marjollet A, Weiszenstein M, Henri M, Thomas A, Godin-Ribuot D, Polak J. The impact of sleep disorders on glucose metabolism: endocrine and molecular mechanisms. *Diabetol Metab Syndr*. 2015;7:25. doi:10.1186/s13098-015-0018-3
179. Figorilli M, Velluzzi F, Redolfi S. Obesity and sleep disorders: A bidirectional relationship. *Nutr Metab Cardiovasc Dis*. 2025;35(6):104014. doi:10.1016/j.numecd.2025.104014
180. Qasrawi SO, BaHammam AS. Role of Sleep and Sleep Disorders in Cardiometabolic Risk: a Review and Update. *Current Sleep Medicine Reports*. 2024;10(1):34-50. doi:10.1007/s40675-024-00276-x

181. St-Onge MP, Grandner MA, Brown D, et al. Sleep duration and quality: impact on lifestyle behaviors and cardiometabolic health: A Scientific Statement from the American Heart Association. *Circulation*. 2016;134(18) doi:10.1161/cir.0000000000000444
182. Direksunthorn T. Sleep and Cardiometabolic Health: A Narrative Review of Epidemiological Evidence, Mechanisms, and Interventions. *Int J Gen Med*. 2025; 18:5831-5843. doi: 10.2147/IJGM.S563616
183. Henst RHP, Pienaar PR, Roden LC, Rae DE. The effects of sleep extension on cardiometabolic risk factors: A systematic review. *J Sleep Res*. 2019;28(6):e12865. doi:10.1111/jsr.12865
184. St-Onge M-P, Aggarwal B, Fernandez-Mendoza J, et al. Multidimensional Sleep Health: Definitions and Implications for Cardiometabolic Health: A Scientific Statement From the American Heart Association. *Circulation: Cardiovascular Quality and Outcomes*. 2025;18(5):e000139. doi:10.1161/HCQ.0000000000000139
185. Creasy SA, Ostendorf DM, Blankenship JM, et al. Effect of sleep on weight loss and adherence to diet and physical activity recommendations during an 18-month behavioral weight loss intervention. *Int J Obes (Lond)*. 2022;46(8):1510-1517. doi:10.1038/s41366-022-01141-z
186. Wright DR, Guo J, Hernandez I. A Prescription for Achieving Equitable Access to Antiobesity Medications. *JAMA Health Forum*. 2023;4(4):e230493. doi:10.1001/jamahealthforum.2023.0493
187. van Veldhuisen SL, Gorter TM, van Woerden G, et al. Bariatric surgery and cardiovascular disease: a systematic review and meta-analysis. *Eur Heart J*. 2022;43(20):1955-1969. doi:10.1093/eurheartj/ehac071
188. Dhar D, Packer J, Michalopoulou S, et al. Assessing the evidence for health benefits of low-level weight loss: a systematic review. *International Journal of Obesity*. 2025;49(2):254-268. doi:10.1038/s41366-024-01664-7
189. Ryan DH, Yockey SR. Weight Loss and Improvement in Comorbidity: Differences at 5%, 10%, 15%, and Over. *Curr Obes Rep*. 2017;6(2):187-194. doi:10.1007/s13679-017-0262-y
190. Liu L, Li Z, Ye W, et al. Safety and effects of anti-obesity medications on weight loss, cardiometabolic, and psychological outcomes in people living with overweight or obesity: a systematic review and meta-analysis. *EClinicalMedicine*. 2025;79:103020. doi:10.1016/j.eclinm.2024.103020
191. Yin Y, Zhang M, Cao Q, et al. Efficacy of GLP-1 Receptor Agonist-Based Therapies on Cardiovascular Events and Cardiometabolic Parameters in Obese Individuals Without Diabetes: A Meta-Analysis of Randomized Controlled Trials. *J Diabetes*. 2025;17(4):e70082. doi:10.1111/1753-0407.70082
192. Abdrabou Abouelmagd A, Abdelrehim AM, Bashir MN, et al. Efficacy and safety of retatrutide, a novel GLP-1, GIP, and glucagon receptor agonist for obesity treatment: a systematic review and meta-analysis of randomized controlled trials. *Baylor University Medical Center Proceedings*. 2025;38(3):291-303. doi:10.1080/08998280.2025.2456441
193. Yates N, South T-L. Injectable drugs for weight management. *Australian Prescriber*. 2025;48(6):197-202. doi:10.18773/austprescr.2025.052
194. Thomsen RW, Mailhac A, Løhde JB, Pottegård A. Real-world evidence on the utilization, clinical and comparative effectiveness, and adverse effects of newer GLP-1RA-based weight-loss therapies. *Diabetes Obes Metab*. 2025;27 Suppl 2(Suppl 2):66-88. doi:10.1111/dom.16364
195. le Roux CW, Done N, Brnabic AJM, et al. Comparative effectiveness of tirzepatide and semaglutide for obesity management in US clinical practice: a 6-month retrospective cohort study. *J Endocrinol Invest*. 2026;49(2):413-423. doi:10.1007/s40618-025-02792-1
196. Budini B, Luo S, Tam M, et al. Trajectory of weight regain after cessation of GLP-1 receptor agonists: a systematic review and nonlinear meta-regression. *eClinicalMedicine*. doi:10.1016/j.eclinm.2026.103796

197. Cui B, Wang G, Li P, et al. Disease-specific mortality and major adverse cardiovascular events after bariatric surgery: a meta-analysis of age, sex, and BMI-matched cohort studies. *Int J Surg*. 2023;109(3):389-400. doi:10.1097/js9.0000000000000066
198. Pontiroli AE, Ceriani V, Tagliabue E. Compared with Controls, Bariatric Surgery Prevents Long-Term Mortality in Persons with Obesity Only Above Median Age of Cohorts: a Systematic Review and Meta-Analysis. *Obes Surg*. 2020;30(7):2487-2496. doi:10.1007/s11695-020-04530-3
199. Chandrakumar H, Khatun N, Gupta T, Graham-Hill S, Zhyvotovska A, McFarlane SI. The Effects of Bariatric Surgery on Cardiovascular Outcomes and Cardiovascular Mortality: A Systematic Review and Meta-Analysis. *Cureus*. 2023;15(2):e34723. doi:10.7759/cureus.34723
200. Tang B, Zhang Y, Wang Y, Wang X, An Z, Yu X. Effect of bariatric surgery on long-term cardiovascular outcomes: a systematic review and meta-analysis of population-based cohort studies. *Surg Obes Relat Dis*. 2022;18(8):1074-1086. doi:10.1016/j.soard.2022.05.007
201. Zhou X, Yu J, Li L, et al. Effects of Bariatric Surgery on Mortality, Cardiovascular Events, and Cancer Outcomes in Obese Patients: Systematic Review and Meta-analysis. *Obes Surg*. 2016;26(11):2590-2601. doi:10.1007/s11695-016-2144-x
202. Doumouras AG, Wong JA, Paterson JM, et al. Bariatric Surgery and Cardiovascular Outcomes in Patients With Obesity and Cardiovascular Disease. *Circulation*. 2021;143(15):1468-1480. doi:10.1161/CIRCULATIONAHA.120.052386
203. Berger S, Meyre P, Blum S, et al. Bariatric surgery among patients with heart failure: a systematic review and meta-analysis. *Open Heart*. 2018;5(2):e000910. doi:10.1136/openhrt-2018-000910
204. Goodarzi S, Shafiee A, Rafiei MA, et al. Bariatric Surgery vs. Low-energy Diet and Cardiometabolic Factors: a Systematic Review and Meta-analysis. *Obes Surg*. 2025:In press. doi:10.1007/s11695-025-07952-z
205. Yan Y, Sha Y, Yao G, et al. Roux-en-Y Gastric Bypass Versus Medical Treatment for Type 2 Diabetes Mellitus in Obese Patients: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *Medicine (Baltimore)*. 2016;95(17):e3462. doi:10.1097/md.0000000000003462
206. Pipek LZ, Moraes WAF, Nobetani RM, et al. Surgery is associated with better long-term outcomes than pharmacological treatment for obesity: a systematic review and meta-analysis. *Sci Rep*. 2024;14(1):9521. doi:10.1038/s41598-024-57724-5
207. Duarte-Guerra LS, Villares JF, Santo MA, Lotufo-Neto F, Wang Y-P. Relationship between psychiatric disorders and loss weight among patients underwent metabolic and bariatric surgery: A reassessment observational study after nine years. *Clinics*. 2024;79:100517. doi:10.1016/j.clinsp.2024.100517
208. Canetti L, Bachar E, Bonne O. Deterioration of mental health in bariatric surgery after 10 years despite successful weight loss. *European Journal of Clinical Nutrition*. 2016;70(1):17-22. doi:10.1038/ejcn.2015.112
209. Herpertz S, Müller A, Burgmer R, Crosby RD, de Zwaan M, Legenbauer T. Health-related quality of life and psychological functioning 9 years after restrictive surgical treatment for obesity. *Surgery for Obesity and Related Diseases*. 2015;11(6):1361-1370. doi:10.1016/j.soard.2015.04.008
210. Royal Australian College of General Practitioners. Obesity prevention and management 2025. Accessed <https://www.racgp.org.au/advocacy/position-statements/view-all-position-statements/clinical-and-practice-management/obesity-prevention-and-management>
211. Pharmaceutical Benefits Advisory Committee. Meeting Outcomes November 2025. Accessed <https://www.pbs.gov.au/industry/listing/elements/pbac-meetings/pbac-outcomes/2025-11/pbac-web-outcomes-11-2025.pdf>

212. Aly A TM, Brown W. Bariatric surgery: a call for greater access to coordinated surgical and specialist care in the public health system. *Med J Aust.* 2022;217(5):228-231. doi:10.5694/mja2.51673
213. Ansari HUH, Qazi SU, Sajid F, et al. Efficacy and Safety of Glucagon-Like Peptide-1 Receptor Agonists on Body Weight and Cardiometabolic Parameters in Individuals With Obesity and Without Diabetes: A Systematic Review and Meta-Analysis. *Endocr Pract.* 2024;30(2):160-171. doi:10.1016/j.eprac.2023.11.007
214. Zheng Z, Zong Y, Ma Y, et al. Glucagon-like peptide-1 receptor: mechanisms and advances in therapy. *Signal Transduction and Targeted Therapy.* 2024;9(1):234. doi:10.1038/s41392-024-01931-z
215. Deanfield J, Lincoff AM, Kahn SE, et al. Semaglutide and cardiovascular outcomes by baseline and changes in adiposity measurements: a prespecified analysis of the SELECT trial. *Lancet.* 2025;406(10516):2257-2268. doi:10.1016/S0140-6736(25)01375-3
216. Masson W, Lobo M, Nogueira JP, Rodriguez-Granillo AM, Barbagelata LE, Siniawski D. Anti-inflammatory effect of semaglutide: updated systematic review and meta-analysis. *Front Cardiovasc Med.* 2024;11:1379189. doi:10.3389/fcvm.2024.1379189
217. Min T, Bain SC. The Role of Tirzepatide, Dual GIP and GLP-1 Receptor Agonist, in the Management of Type 2 Diabetes: The SURPASS Clinical Trials. *Diabetes Ther.* 2021;12(1):143-157. doi:10.1007/s13300-020-00981-0
218. Samms RJ, Sloop KW. A Contemporary Rationale for Agonism of the GIP Receptor in the Treatment of Obesity. *Diabetes.* 2025;74(8):1326-1333. doi:10.2337/dbi24-0026
219. Australian Medicines Handbook. 2025. Accessed 13 Jan 2026. amhonline.amh.net.au
220. Nauck MA, Quast DR, Wefers J, Meier JJ. GLP-1 receptor agonists in the treatment of type 2 diabetes - state-of-the-art. *Mol Metab.* 2021;46:101102. doi:10.1016/j.molmet.2020.101102
221. Ali A, Siddiqui AA, Usman MS, Shahid I, Khan MS, Perswani P. Effect of glucagon-like peptide 1 receptor agonists on systolic blood pressure in patients with obesity, with or without diabetes: A systematic review and network meta-analysis. *Clin Obes.* 2025;15(4):e70012. doi:10.1111/cob.70012
222. Mullur N, Morissette A, Morrow NM, Mulvihill EE. GLP-1 receptor agonist-based therapies and cardiovascular risk: a review of mechanisms. *J Endocrinol.* 2024;263(1)doi:10.1530/joe-24-0046
223. Therapeutic Goods Administration. *Phentermine sandoz (phentermine)*. 2022. www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/pdf?OpenAgent=&id=CP-2022-PI-01212-1&d=20260112172310101
224. Therapeutic Goods Administration. *Prolistat (orlistat)*. 2021. www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/pdf?OpenAgent=&id=CP-2022-PI-01672-1
225. Therapeutic Goods Administration. *Naltrexone/bupropion Product Information*. 2025. https://www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/pdf?OpenAgent=&id=CP-2019-PI-01018-1&d=20260504172310101
226. Padwal R, Li SK, Lau DC. Long-term pharmacotherapy for overweight and obesity: a systematic review and meta-analysis of randomized controlled trials. *Int J Obes Relat Metab Disord.* 2003;27(12):1437-46. doi:10.1038/sj.ijo.0802475
227. Marso SP, Daniels GH, Brown-Frandsen K, et al. Liraglutide and Cardiovascular Outcomes in Type 2 Diabetes. *New Engl J Med.* 2016;375(4):311-322. doi:10.1056/NEJMoa1603827
228. Lincoff AM, Brown-Frandsen K, Colhoun HM, et al. Semaglutide and Cardiovascular Outcomes in Obesity without Diabetes. *New Engl J Med.* 2023;389(24):2221-2232. doi:10.1056/NEJMoa2307563
229. Marso SP, Bain SC, Consoli A, et al. Semaglutide and Cardiovascular Outcomes in Patients with Type 2 Diabetes. *New Engl J Med.* 2016;375(19):1834-1844. doi:doi:10.1056/NEJMoa1607141

230. Lilly's Mounjaro (tirzepatide); a GIP/GLP-1 dual agonist, demonstrated cardiovascular protection in landmark head-to-head trials, reinforcing its benefit in patients with type 2 diabetes and heart disease. <https://investor.lilly.com/news-releases/news-release-details/lillys-mounjaro-tirzepatide-gipglp-1-dual-agonist-demonstrated>
231. Therapeutic Goods Administration. *Wegovy (Novo Nordisk Pharmaceuticals Pty Ltd)*. 2024. <https://www.tga.gov.au/resources/prescription-medicines-registrations/wegovy-novo-nordisk-pharmaceuticals-pty-ltd-1>
232. Therapeutic Goods Administration. *Mounjaro (Eli Lilly Australia Pty Ltd)*. 2024. <https://www.tga.gov.au/resources/prescription-medicines-registrations/mounjaro-eli-lilly-australia-pty-ltd-0>
233. Therapeutic Goods Administration. *Contrave (naltrexone hydrochloride and bupropion hydrochloride)*. 2025. www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/pdf?OpenAgent=&id=CP-2019-PI-01018-1
234. Therapeutic Goods Administration. *Liraglutide sandoz (liraglutide)*. 2025. www.ebs.tga.gov.au/ebs/picmi/picmirepository.nsf/pdf?OpenAgent=&id=CP-2025-PI-01388-1
235. Wilding John PH, Batterham Rachel L, Calanna S, et al. Once-Weekly Semaglutide in Adults with Overweight or Obesity. *New Engl J Med*. 2021;384(11):989-1002. doi:10.1056/NEJMoa2032183
236. Lewis KH, Fischer H, Ard J, et al. Safety and Effectiveness of Longer-Term Phentermine Use: Clinical Outcomes from an Electronic Health Record Cohort. *Obesity (Silver Spring)*. 2019;27(4):591-602. doi:10.1002/oby.22430
237. Kelley DE, Bray GA, Pi-Sunyer FX, et al. Clinical efficacy of orlistat therapy in overweight and obese patients with insulin-treated type 2 diabetes: A 1-year randomized controlled trial. *Diabetes Care*. 2002;25(6):1033-41. doi:10.2337/diacare.25.6.1033
238. Greenway FL, Fujioka K, Plodkowski RA, et al. Effect of naltrexone plus bupropion on weight loss in overweight and obese adults (COR-1): a multicentre, randomised, double-blind, placebo-controlled, phase 3 trial. *Lancet*. 2010;376(9741):595-605. doi:10.1016/s0140-6736(10)60888-4
239. Davies MJ, Bergenstal R, Bode B, et al. Efficacy of Liraglutide for Weight Loss Among Patients With Type 2 Diabetes: The SCALE Diabetes Randomized Clinical Trial. *JAMA*. 2015;314(7):687-699. doi:10.1001/jama.2015.9676
240. Aronne LJ, Sattar N, Horn DB, et al. Continued Treatment With Tirzepatide for Maintenance of Weight Reduction in Adults With Obesity: The SURMOUNT-4 Randomized Clinical Trial. *JAMA*. 2024;331(1):38-48. doi:10.1001/jama.2023.24945
241. Zhao L, Cheng Z, Lu Y, et al. Tirzepatide for Weight Reduction in Chinese Adults With Obesity: The SURMOUNT-CN Randomized Clinical Trial. *JAMA*. 2024;332(7):551-560. doi:10.1001/jama.2024.9217
242. Jastreboff Ania M, Aronne Louis J, Ahmad Nadia N, et al. Tirzepatide Once Weekly for the Treatment of Obesity. *New Engl J Med*. 2022;387(3):205-216. doi:10.1056/NEJMoa2206038
243. Rubino DM, Greenway FL, Khalid U, et al. Effect of Weekly Subcutaneous Semaglutide vs Daily Liraglutide on Body Weight in Adults With Overweight or Obesity Without Diabetes: The STEP 8 Randomized Clinical Trial. *JAMA*. 2022;327(2):138-150. doi:10.1001/jama.2021.23619
244. Perkovic V, Tuttle Katherine R, Rossing P, et al. Effects of Semaglutide on Chronic Kidney Disease in Patients with Type 2 Diabetes. *New Engl J Med*. 2024;391(2):109-121. doi:10.1056/NEJMoa2403347
245. Nicholls Stephen J, Pavo I, Bhatt Deepak L, et al. Cardiovascular Outcomes with Tirzepatide versus Dulaglutide in Type 2 Diabetes. *New Engl J Med*. 2025;393(24):2409-2420. doi:10.1056/NEJMoa2505928
246. Pi-Sunyer X, Astrup A, Fujioka K, et al. A Randomized, Controlled Trial of 3.0 mg of Liraglutide in Weight Management. *New Engl J Med*. 2015;373(1):11-22. doi:10.1056/NEJMoa1411892
247. Singh N, Stewart RAH, Benatar JR. Intensity and duration of lifestyle interventions for long-term weight loss and association with mortality: a meta-analysis of randomised trials. *BMJ Open*. 2019;9(8):e029966. doi:10.1136/bmjopen-2019-029966

248. Padwal R, Li SK, Lau DC. Long-term pharmacotherapy for obesity and overweight. *Cochrane Database Syst Rev.* 2004;2003(3):Cd004094. doi:10.1002/14651858.CD004094.pub2
249. van Rijswijk A-S, van Olst N, Schats W, van der Peet DL, van de Laar AW. What Is Weight Loss After Bariatric Surgery Expressed in Percentage Total Weight Loss (%TWL)? A Systematic Review. *Obesity Surgery.* 2021;31(8):3833-3847. doi:10.1007/s11695-021-05394-x
250. Therapeutic Goods Administration. Wegovy (Novo Nordisk Pharmaceuticals Pty Ltd)
251. Wharton S, Freitas P, Hjelmæsæth J, et al. Once-weekly semaglutide 7.2 mg in adults with obesity (STEP UP): a randomised, controlled, phase 3b trial. *Lancet Diabetes Endocrinol.* 2025;13(11):949-963. doi:10.1016/s2213-8587(25)00226-8
252. Therapeutic Goods Administration. *Mounjaro (Eli Lilly Australia Pty Ltd)*. 2023. <https://www.tga.gov.au/resources/auspmd/mounjaro>
253. West S, Scragg J, Aveyard P, et al. Weight regain after cessation of medication for weight management: systematic review and meta-analysis. *BMJ.* 2026;392:e085304. doi:10.1136/bmj-2025-085304
254. Mozaffarian D, Agarwal M, Aggarwal M, et al. Nutritional priorities to support GLP-1 therapy for obesity: A joint advisory from the American College of Lifestyle Medicine, the American Society for Nutrition, the Obesity Medicine Association, and the Obesity Society. *Obesity Pillars.* 2025;15:100181. doi:10.1016/j.obpill.2025.100181
255. Schoretsanitis G, Weiler S, Barbui C, Raschi E, Gastaldon C. Disproportionality Analysis From World Health Organization Data on Semaglutide, Liraglutide, and Suicidality. *JAMA Network Open.* 2024;7(8):e2423385-e2423385. doi:10.1001/jamanetworkopen.2024.23385
256. Therapeutic Goods Administration. GLP-1 RAs: warnings aligned over potential risk of suicidal thoughts or behaviours. 2025. <https://www.tga.gov.au/news/safety-updates/glp-1-ras-warnings-aligned-over-potential-risk-suicidal-thoughts-or-behaviours>
257. Australian Government Department of Health, Disability and Ageing. Medicines containing GLP-1 and dual GIP/GLP-1 receptor agonists: Medicines Safety Update 2025. Accessed 7 Apr 2026. <https://www.tga.gov.au/news/safety-updates/medicines-containing-glp-1-and-dual-gipglp-1-receptor-agonists>
258. Therapeutic Goods Administration. Counterfeit (fake) medicines and medical devices. 2024. <https://www.tga.gov.au/resources/consumer-information-and-resources/buying-medicines-and-medical-devices-online/counterfeit-fake-medicines-and-medical-devices#case-study>
259. Damluji AA, Alfaraidhy M, AlHajri N, et al. Sarcopenia and Cardiovascular Diseases. *Circulation.* 2023;147(20):1534-1553. doi:10.1161/circulationaha.123.064071
260. Neeland IJ, Linge J, Birkenfeld AL. Changes in lean body mass with glucagon-like peptide-1-based therapies and mitigation strategies. *Diabetes, Obesity and Metabolism.* 2024;26(S4):16-27. doi:10.1111/dom.15728
261. Silver HJ, Olson D, Mayfield D, et al. Effect of the glucagon-like peptide-1 receptor agonist liraglutide, compared to caloric restriction, on appetite, dietary intake, body fat distribution and cardiometabolic biomarkers: A randomized trial in adults with obesity and prediabetes. *Diabetes Obes Metab.* 2023;25(8):2340-2350. doi:10.1111/dom.15113
262. Linge J, Birkenfeld AL, Neeland IJ. Muscle Mass and Glucagon-Like Peptide-1 Receptor Agonists: Adaptive or Maladaptive Response to Weight Loss? *Circulation.* 2024;150(16):1288-1298. doi:10.1161/CIRCULATIONAHA.124.067676
263. Christensen S, Robinson K, Thomas S, Williams DR. Dietary intake by patients taking GLP-1 and dual GIP/GLP-1 receptor agonists: A narrative review and discussion of research needs. *Obesity Pillars.* 2024;11:100121. doi:10.1016/j.obpill.2024.100121

264. Gerstein HC, Colhoun HM, Dagenais GR, et al. Dulaglutide and cardiovascular outcomes in type 2 diabetes (REWIND): a double-blind, randomised placebo-controlled trial. *Lancet*. 2019;394(10193):121-130. doi:10.1016/s0140-6736(19)31149-3
265. Liu X, Chen Y, Liu T, Cai L, Yang X, Mou C. The effects of Sodium-glucose cotransporter 2 inhibitors on adipose tissue in patients with type 2 diabetes: A meta-analysis of randomized controlled trials. Systematic Review. *Frontiers in Endocrinology*. 2023;14:1115321. doi:10.3389/fendo.2023.1115321
266. McGuire DK, Shih WJ, Cosentino F, et al. Association of SGLT2 Inhibitors With Cardiovascular and Kidney Outcomes in Patients With Type 2 Diabetes: A Meta-analysis. *JAMA Cardiology*. 2021;6(2):148-158. doi:10.1001/jamacardio.2020.4511
267. Van Dril E, Allison M, Schumacher C. Deprescribing in type 2 diabetes and cardiovascular disease: Recommendations for safe and effective initiation of glucagon-like peptide-1 receptor agonists in patients on insulin therapy. *American Heart Journal Plus: Cardiology Research and Practice*. 2022;17:100163. doi:10.1016/j.ahjo.2022.100163
268. Bethel MA, Diaz R, Castellana N, Bhattacharya I, Gerstein HC, Lakshmanan MC. HbA(1c) Change and Diabetic Retinopathy During GLP-1 Receptor Agonist Cardiovascular Outcome Trials: A Meta-analysis and Meta-regression. *Diabetes Care*. 2021;44(1):290-296. doi:10.2337/dc20-1815
269. Cai CX, Hribar M, Baxter S, et al. Semaglutide and Nonarteritic Anterior Ischemic Optic Neuropathy. *JAMA Ophthalmology*. 2025;143(4):304-314. doi:10.1001/jamaophthol.2024.6555
270. Chen J-W, Yu FT-E, Chen H-A, Huang T-F, Chen H-S, Wu T-E. Glucagon-like peptide-1 receptor agonists and the risk of nonarteritic anterior ischemic optic neuropathy: Evidence from a global real-world cohort. *Diabetes & Metabolism*. 2026;52(2):101740. doi:https://doi.org/10.1016/j.diabet.2026.101740
271. Therapeutic Goods Administration. *ACM meeting statement, Meeting 53, 3 October 2025*. 2025. <https://www.tga.gov.au/resources/publication/meeting-statements/acm-meeting-statement-meeting-53-3-october-2025>
272. Sundaresh S, Saliba S, Ziyadeh F, Mauer Y, Valencia WM. Managing obesity in older adults. *Cleve Clin J Med*. 2025;92(11):686-692. doi:10.3949/ccjm.92a.24094
273. Thapaliya K, Sweeting A, Black KI, Poprzeczny A, Mazza D, Grzeskowiak LE. Incidence of GLP-1 receptor agonist use by women of reproductive age attending general practices in Australia, 2011-2022: a retrospective open cohort study. *Med J Aust*. 2025;223(7):365-371. doi:10.5694/mja2.70026
274. Muller DRP, Stenvers DJ, Malekzadeh A, Holleman F, Painter RC, Siegelaar SE. Effects of GLP-1 agonists and SGLT2 inhibitors during pregnancy and lactation on offspring outcomes: a systematic review of the evidence. Review. *Front Endocrinol*. 2023;14:1215356. doi:10.3389/fendo.2023.1215356
275. Skelley JW, Swearengen K, York AL, Glover LH. The impact of tirzepatide and glucagon-like peptide 1 receptor agonists on oral hormonal contraception. *J Am Pharm Assoc (2003)*. 2024;64(1):204-211.e4. doi:10.1016/j.japh.2023.10.037
276. Australian Government Department of Health, Disability and Ageing. *Updated contraception advice for Mounjaro (tirzepatide)*. 2025. <https://www.tga.gov.au/news/safety-updates/updated-contraception-advice-mounjaro-tirzepatide>
277. Kittleson MM, Benjamin EJ, Blumer V, et al. 2025 ACC Scientific Statement on the Management of Obesity in Adults With Heart Failure. *JACC*. 2025;86(20):1953-1975. doi:10.1016/j.jacc.2025.05.008
278. Kosiborod MN, Abildstrøm SZ, Borlaug BA, et al. Semaglutide in Patients with Heart Failure with Preserved Ejection Fraction and Obesity. *New Engl J Med*. 2023;389(12):1069-1084. doi:10.1056/NEJMoa2306963

279. Packer M, Zile MR, Kramer CM, et al. Tirzepatide for Heart Failure with Preserved Ejection Fraction and Obesity. *N Engl J Med*. 2025;392(5):427-437. doi:10.1056/NEJMoa2410027
280. Adamson C, Kondo T, Jhund PS, et al. Dapagliflozin for heart failure according to body mass index: the DELIVER trial. *Eur Heart J*. 2022;43(41):4406-4417. doi:10.1093/eurheartj/ehac481
281. Perkovic V, Tuttle KR, Rossing P, et al. Effects of Semaglutide on Chronic Kidney Disease in Patients with Type 2 Diabetes. *N Engl J Med*. 2024;391(2):109-121. doi:10.1056/NEJMoa2403347
282. Wang Y, Zhou Y, Wang Z, Ni Y, Prud'homme GJ, Wang Q. Efficacy of GLP-1-based Therapies on Metabolic Dysfunction-associated Steatotic Liver Disease and Metabolic Dysfunction-associated Steatohepatitis: A Systematic Review and Meta-analysis. *J. Clin. Endocrinol. Metab*. 2025;110(10):2964-2979. doi:10.1210/clinem/dgaf336
283. Abushamat LA, Shah PA, Eckel RH, Harrison SA, Barb D. The Emerging Role of Glucagon-Like Peptide-1 Receptor Agonists for the Treatment of Metabolic Dysfunction-Associated Steatohepatitis. *Clin. Gastroenterol. Hepatol*. 2024;22(8):1565-1574. doi:10.1016/j.cgh.2024.01.032
284. Sanyal AJ, Newsome PN, Kliers I, et al. Phase 3 Trial of Semaglutide in Metabolic Dysfunction-Associated Steatohepatitis. *N Engl J Med*. 2025;392(21):2089-2099. doi:10.1056/NEJMoa2413258
285. Krüger N, Schneeweiss S, Desai RJ, et al. Cardiovascular outcomes of semaglutide and tirzepatide for patients with type 2 diabetes in clinical practice. *Nature Medicine*. 2025;32:342-352. doi:10.1038/s41591-025-04102-x
286. Lam CSP, Rodriguez A, Aminian A, et al. Tirzepatide for reduction of morbidity and mortality in adults with obesity: rationale and design of the SURMOUNT-MMO trial. *Obesity*. 2025;33(9):1645-1656. doi:10.1002/oby.24332
287. Kosiborod MN, Petrie MC, Borlaug BA, et al. Semaglutide in Patients with Obesity-Related Heart Failure and Type 2 Diabetes. *New Engl J Med*. 2024;390(15):1394-1407. doi:10.1056/NEJMoa2313917
288. Sundaram V. TRANSFORM-AF: targeting metabolic therapy with GLP-1 receptor agonism for secondary prevention in atrial fibrillation. *Presented at Heart Rhythm Society (HRS) 2025 Annual Scientific Sessions*. 2025.
289. Jastreboff AM, Kaplan LM, Frías JP, et al. Triple-Hormone-Receptor Agonist Retatrutide for Obesity - A Phase 2 Trial. *N Engl J Med*. 2023;389(6):514-526. doi:10.1056/NEJMoa2301972
290. Rosenstock J, Frías J, Jastreboff AM, et al. Retatrutide, a GIP, GLP-1 and glucagon receptor agonist, for people with type 2 diabetes: a randomised, double-blind, placebo and active-controlled, parallel-group, phase 2 trial conducted in the USA. *Lancet*. 2023;402(10401):529-544. doi:10.1016/s0140-6736(23)01053-x
291. le Roux CW, Wharton S, Bozkurt B, et al. Survodutide for treatment of obesity: Baseline characteristics of participants in a randomized, double-blind, placebo-controlled, phase 3 trial (SYNCHRONIZE™-1). *Diabetes Obes Metab*. 2026;28(1):337-346. doi:10.1111/dom.70196
292. Boehringer Ingelheim. *Data shows nearly 19% weight loss in people with overweight or obesity in Boehringer Ingelheim and Pharma Phase II trial with survodutide*. 2023. <https://www.boehringer-ingelheim.com/human-health/metabolic-diseases/obesity/phase-ii-clinical-trial-weight-loss-results>
293. Boehringer Ingelheim. *Phase III studies to investigate survodutide for people living with obesity and overweight, with and without diabetes, cardiovascular disease and chronic kidney disease*. 2023. <https://www.boehringer-ingelheim.com/human-health/metabolic-diseases/phase-3-studies-survodutide-obesity-and-overweight>
294. Wharton S, Lingvay I, Bogdanski P, et al. Oral Semaglutide at a Dose of 25 mg in Adults with Overweight or Obesity. *N Engl J Med*. 2025;393(11):1077-1087. doi:10.1056/NEJMoa2500969
295. McGuire Darren K, Marx N, Mulvagh Sharon L, et al. Oral Semaglutide and Cardiovascular Outcomes in High-Risk Type 2 Diabetes. *New Engl J Med*. 2025;392(20):2001-2012. doi:10.1056/NEJMoa2501006

296. Wharton S, Aronne LJ, Stefanski A, et al. Orforglipron, an Oral Small-Molecule GLP-1 Receptor Agonist for Obesity Treatment. *N Engl J Med*. 2025;393(18):1796-1806. doi:10.1056/NEJMoa2511774
297. Horn DB, Ryan DH, Kis SG, et al. Orforglipron, an oral small-molecule GLP-1 receptor agonist, for the treatment of obesity in people with type 2 diabetes (ATTAIN-2): a phase 3, double-blind, randomised, multicentre, placebo-controlled trial. *Lancet*. 2025;406(10522):2927-2944. doi:10.1016/S0140-6736(25)02165-8
298. Rosenstock J, Hsia S, Ruiz LN, et al. Orforglipron, an Oral Small-Molecule GLP-1 Receptor Agonist, in Early Type 2 Diabetes. *New England Journal of Medicine*. 2025;393(11):1065-1076. doi:10.1056/NEJMoa2505669
299. Garvey WT, Blüher M, Contreras CKO, et al. Coadministered Cagrilintide and Semaglutide in Adults with Overweight or Obesity. *New Engl J Med*. 2025;393(7):635-647. doi:10.1056/NEJMoa2502081
300. Davies MJ, Bajaj HS, Broholm C, et al. Cagrilintide–Semaglutide in Adults with Overweight or Obesity and Type 2 Diabetes. *New Engl J Med*. 2025;393(7):648-659. doi:10.1056/NEJMoa2502082
301. Billings LK, Hsia S, Bays H, et al. Eloralintide, a selective amylin receptor agonist for the treatment of obesity: a 48-week phase 2, multicentre, double-blind, randomised, placebo-controlled trial. *Lancet*. 2025;406(10520):2631-2643. doi:10.1016/S0140-6736(25)02155-5
302. Garvey WT, Blüher M, Osorto Contreras CK, et al. Coadministered Cagrilintide and Semaglutide in Adults with Overweight or Obesity. *N Engl J Med*. 2025;393(7):635-647. doi:10.1056/NEJMoa2502081
303. Jastreboff AM, Ryan DH, Bays HE, et al. Once-Monthly Maridebart Cafraglutide for the Treatment of Obesity — A Phase 2 Trial. *New Engl J Med*. 2025;393(9):843-857. doi:doi:10.1056/NEJMoa2504214
304. Department of Health , Disability and Ageing. Medicare Benefits Schedule Bariatric Procedures (Items 31569 to 31581; TN.9.29) Accessed 19 Mar 2026. <https://www9.health.gov.au/mbs/fullDisplay.cfm?type=item&q=ItemID&q=20791>
305. Schauer Philip R, Bhatt Deepak L, Kirwan John P, et al. Bariatric Surgery versus Intensive Medical Therapy for Diabetes — 5-Year Outcomes. *New Engl J Med*. 376(7):641-651. doi:10.1056/NEJMoa1600869
306. Salminen P, Helmiö M, Ovaska J, et al. Effect of Laparoscopic Sleeve Gastrectomy vs Laparoscopic Roux-en-Y Gastric Bypass on Weight Loss at 5 Years Among Patients With Morbid Obesity: The SLEEVEPASS Randomized Clinical Trial. *JAMA*. 2018;319(3):241-254. doi:10.1001/jama.2017.20313
307. Sjöström L, Peltonen M, Jacobson P, et al. Bariatric Surgery and Long-term Cardiovascular Events. *JAMA*. 2012;307(1):56-65. doi:10.1001/jama.2011.1914
308. Hamilton Health Sciences Corporation. Bariatric Surgery for the Reduction of Cardiovascular Events Feasibility Trial (BRAVE) 2022. Accessed 19 Mar 2026. <https://clinicaltrials.gov/study/NCT04226664>
309. Kenkre JS, Gesell S, Keller A, Milani RM, Scholtz S, Barley EA. Alcohol Misuse post Metabolic and Bariatric Surgery: A Systematic Review of Longer-term Studies with Focus on new Onset Alcohol use Disorder and Differences Between Surgery Types. *Curr Obes Rep*. 2024;13(3):596-616. doi:10.1007/s13679-024-00577-w
310. Butt M, Eisler RA, Hu A, Rogers AM, Rigby A. Incidence of Substance Use Disorder Following Bariatric Surgery: A Retrospective Cohort Study. *Obes Surg*. 2023;33(3):890-896. doi:10.1007/s11695-022-06400-6
311. Sherf Dagan S, Goldenshluger A, Globus I, et al. Nutritional Recommendations for Adult Bariatric Surgery Patients: Clinical Practice. *Adv Nutr*. 2017;8(2):382-394. doi:10.3945/an.116.014258
312. Mechanick JI, Apovian C, Brethauer S, et al. Clinical Practice Guidelines for the Perioperative Nutrition, Metabolic, and Nonsurgical Support of Patients Undergoing Bariatric Procedures – 2019 Update: Cosponsored by American Association of Clinical Endocrinologists/American College of Endocrinology, The Obesity Society, American Society for Metabolic and Bariatric Surgery, Obesity Medicine Association, and American Society of Anesthesiologists. *Obesity*. 2020;28(4):O1-O58. doi:10.1002/oby.22719

313. Queensland Health. Nutrition Education Materials Online (NEMO). Diet strategies for managing dumping syndrome after bariatric surgery. 2024. Accessed 19 Mar 2026. https://www.health.qld.gov.au/__data/assets/pdf_file/0014/1327001/bari-surg-dumping-syndrome.pdf
314. Scarpellini E, Arts J, Karamanolis G, et al. International consensus on the diagnosis and management of dumping syndrome. *Nat Rev Endocrinol*. 2020;16(8):448-466. doi:10.1038/s41574-020-0357-5
315. Heber D, Greenway FL, Kaplan LM, Livingston E, Salvador J, Still C. Endocrine and nutritional management of the post-bariatric surgery patient: an Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab*. 2010;95(11):4823-43. doi:10.1210/jc.2009-2128
316. Australian & New Zealand Metabolic and Obesity Surgery Society. *Public bariatric surgery - A national framework*. 2020. https://anzmoss.com.au/wp-content/uploads/Public-Bariatric-Surgery-Framework_EXEC-SUM-ONLY.pdf
317. Goubar T, Goubar C, Fenton-Lee D, Stefanidis A, Macdonald PS, Rushworth RL. A Population-Based Study of Bariatric Surgery Trends in Australia: Variations Reflect Continuing Inequities in Access to Surgery. *Obesity Surgery*. 2025;35(3):1026-1035. doi:10.1007/s11695-025-07699-7
318. Lee PC. Bariatric–metabolic surgery: A guide for the primary care physician. *Aust J Gen Pract*. 2017;46:465-471.
319. Salminen P, Grönroos S, Helmiö M, et al. Effect of Laparoscopic Sleeve Gastrectomy vs Roux-en-Y Gastric Bypass on Weight Loss, Comorbidities, and Reflux at 10 Years in Adult Patients With Obesity: The SLEEVEPASS Randomized Clinical Trial. *JAMA Surgery*. 2022;157(8):656-666. doi:10.1001/jamasurg.2022.2229
320. Vieira de Sousa JP, Santos-Sousa H, Vieira S, et al. Assessing Nutritional Deficiencies in Bariatric Surgery Patients: A Comparative Study of Roux-en-Y Gastric Bypass versus Sleeve Gastrectomy. *J Pers Med*. 2024;14(6) doi:10.3390/jpm14060650
321. Thiel AM, Plamper A, Kroll J, Alizai PH, Schmitz SM, Rheinwald KP. Long-term outcomes of sleeve gastrectomy in a patient group with mainly high BMI: a single-center study. *Updates Surg*. 2025;77(7):2077-2085. doi:10.1007/s13304-025-02076-6
322. Australian Bariatric Surgery Registry. *Annual Report. 2023*. https://www.monash.edu/__data/assets/pdf_file/0007/3757876/Bariatric_Surgery_Registry_Annual_Report_2023.pdf
323. Lee PC, Dixon J. Bariatric–metabolic surgery: A guide for the primary care physician. *Aust J Gen Pract*. 2017;46:465-471.
324. Kermansaravi M, Mahawar KK, Davarpanah Jazi AH, Eghbali F, Kabir A, Pazouki A. Revisional surgery after one anastomosis/mini gastric bypass: A narrative review. *J Res Med Sci*. 2020;25:62. doi:10.4103/jrms.JRMS_727_19
325. Verhoeff K, Mocanu V, Zalasky A, et al. Evaluation of Metabolic Outcomes Following SADI-S: a Systematic Review and Meta-analysis. *Obesity Surgery*. 2022;32(4):1049-1063. doi:10.1007/s11695-021-05824-w
326. Spinos D, Skarentzos K, Esagian SM, Seymour KA, Economopoulos KP. The Effectiveness of Single-Anastomosis Duodenoileal Bypass with Sleeve Gastrectomy/One Anastomosis Duodenal Switch (SADI-S/OADS): an Updated Systematic Review. *Obesity Surgery*. 2021;31(4):1790-1800. doi:10.1007/s11695-020-05188-7
327. Axer S, Al-Tai S, Ihle C, Alwan M, Hoffmann L. Perioperative Safety and 1-Year Outcomes of Single-Anastomosis Duodeno-Ileal Bypass (SADI) vs. Biliopancreatic Diversion with Duodenal Switch (BPD/DS): A Randomized Clinical Trial. *Obesity Surgery*. 2024;34(9):3382-3389. doi:10.1007/s11695-024-07421-z
328. Shilton H. Bariatric surgery. *Aust J Gen Pract*. 2025;54(4):202-206. doi:10.31128/AJGP-10-24-7432
329. O’Kane M, Parretti HM, Hughes CA, et al. Guidelines for the follow-up of patients undergoing bariatric surgery. *Clinical Obesity*. 2016;6(3):210-224. doi:10.1111/cob.12145

330. Akharaekpanya T, Nevins E, Brown J. Biochemical Monitoring and Micronutrient Replacement Following Bariatric Surgery. *British Journal of Surgery*. 2023;110(S7):znad258.198. doi:10.1093/bjs/znad258.198
331. Bhatti JA, Nathens AB, Thiruchelvam D, Grantcharov T, Goldstein BI, Redelmeier DA. Self-harm emergencies after bariatric surgery: a population-based cohort study. *JAMA Surgery*. 2016;151(3):226-232. doi:10.1001/jamasurg.2015.3414
332. Azam H, Shahrestani S, Phan K. Alcohol use disorders before and after bariatric surgery: a systematic review and meta-analysis. *Ann Transl Med*. 2018;6(8):148. doi:10.21037/atm.2018.03.16
333. Padwal R, Brocks D, Sharma AM. A systematic review of drug absorption following bariatric surgery and its theoretical implications. *Obes Rev*. 2010;11(1):41-50. doi:10.1111/j.1467-789X.2009.00614.x
334. Stein EM, Silverberg SJ. Bone loss after bariatric surgery: causes, consequences, and management. *Lancet Diabetes Endocrinol*. 2014;2(2):165-174. doi:10.1016/S2213-8587(13)70183-9
335. Shawe J, Ceulemans D, Akhter Z. Pregnancy after bariatric surgery: Consensus recommendations for periconception, antenatal and postnatal care. *Obes Rev*. 2019;20(11):1507-1522. doi:10.1111/obr.12927
336. Parrott J, Frank L, Rabena R, Craggs-Dino L, Isom KA, Greiman L. American Society for Metabolic and Bariatric Surgery Integrated Health Nutritional Guidelines for the Surgical Weight Loss Patient - 2016 Update: Micronutrients. *Surg Obes Relat Dis*. 2017;13(5):727-741. doi:10.1016/j.soard.2016.12.018
337. Qumseya BJ, Qumsiyeh Y, Ponniah SA. Barrett's esophagus after sleeve gastrectomy: a systematic review and meta-analysis. *Gastrointest Endosc*. 2021;93(2):343-352. doi:10.1016/j.gie.2020.08.008
338. Vosburg RW, Nimeri A, Azagury D, et al. American Society for Metabolic and Bariatric Surgery literature review on risk factors, screening recommendations, and prophylaxis for marginal ulcers after metabolic and bariatric surgery. *Surg Obes Relat Dis*. 2025;21(2):101-108. doi:10.1016/j.soard.2024.10.013
339. Karam L, Paccou J. Management of Adverse Skeletal Effects Following Bariatric Surgery Procedures in People Living with Obesity. *Curr Osteoporos Rep*. 2025;23(1):11. doi:10.1007/s11914-025-00902-9
340. Kim J, Brethauer S. Metabolic bone changes after bariatric surgery. *Surg Obes Relat Dis*. 2015;11(2):406-11. doi:10.1016/j.soard.2014.03.010
341. Salminen P, Grönroos S, Helmiö M, et al. Effect of Laparoscopic Sleeve Gastrectomy vs Roux-en-Y Gastric Bypass on Weight Loss, Comorbidities, and Reflux at 10 Years in Adult Patients With Obesity: The SLEEVEPASS Randomized Clinical Trial. *JAMA Surg*. 2022;157(8):656-666. doi:10.1001/jamasurg.2022.2229
342. Schauer PR, Kashyap SR, Wolski K, et al. Bariatric Surgery versus Intensive Medical Therapy in Obese Patients with Diabetes. *New Engl J Med* 2012;366(17):1567-1576. doi:10.1056/NEJMoa1200225
343. Sjöström L, Narbro K, Sjöström CD, et al. Effects of Bariatric Surgery on Mortality in Swedish Obese Subjects. *New Engl J Med* 2007;357(8):741-752. doi:10.1056/NEJMoa066254
344. Chokesuwattanaskul R, Thongprayoon C, Bathini T, et al. Incident atrial fibrillation in patients undergoing bariatric surgery: a systematic review and meta-analysis. *Intern Med J*. 2020;50(7):810-817. doi:10.1111/imj.14436
345. Brieger D, Cullen L, Briffa T, et al. National Heart Foundation of Australia & Cardiac Society of Australia and New Zealand: Comprehensive Australian Clinical Guideline for Diagnosing and Managing Acute Coronary Syndromes 2025. *Heart Lung Circ*. 2025;34(4):309-397. doi:10.1016/j.hlc.2025.02.102
346. Cyril S, Green J, Nicholson JM, Agho K, Renzaho AM. Exploring Service Providers' Perspectives in Improving Childhood Obesity Prevention among CALD Communities in Victoria, Australia. *PLoS One*. 2016;11(10):e0162184. doi:10.1371/journal.pone.0162184

347. Menigoz K, Nathan A, Turrell G. Ethnic differences in overweight and obesity and the influence of acculturation on immigrant bodyweight: evidence from a national sample of Australian adults. *BMC Public Health*. 2016;16(1):932. doi:10.1186/s12889-016-3608-6
348. Olani AB, Eastwood K, Finn J, Clark RA, Bray JE. Prehospital treatment-seeking for acute coronary syndrome in culturally and linguistically diverse immigrant populations: a scoping review. *Eur J Cardiovasc Nurs*. 2025;24(2):207-217. doi:10.1093/eurjcn/zvae122
349. Balla S, Gomez SE, Rodriguez F. Disparities in Cardiovascular Care and Outcomes for Women From Racial/Ethnic Minority Backgrounds. *Curr Treat Options Cardiovasc Med*. 2020;22(12):75. doi:10.1007/s11936-020-00869-z
350. Australian Bureau of Statistics. *Causes of death, Australia 2024*. 2025. <https://www.abs.gov.au/statistics/health/causes-death/causes-death-australia/latest-release>
351. Australian Institute of Health and Welfare. *Australian Burden of Disease Study: impact and causes of illness and death in Aboriginal and Torres Strait Islander people 2018*. 2022. <https://www.aihw.gov.au/reports/burden-of-disease/illness-death-indigenous-2018/summary>
352. Lan NSR, Ford J, Gregory L, Jones G, Dwivedi G, Yeap BB. Interventions to prevent or treat obesity in adult Indigenous Australians: A systematic review. *Obes Res Clin Pract*. 2025;19(2):85-93. doi:10.1016/j.orcp.2025.04.003
353. National Rural Health Alliance. *Rural Health in Australia Snapshot 2025*. 2025. <https://www.ruralhealth.org.au/wp-content/uploads/2025/02/NRHA-Rural-Health-in-Australia-Snapshot-2025.pdf>
354. Summers R, Lea J, East L. An exploration of extreme obesity and weight loss management for adults in rural, remote, and regional areas: a systematic review. *Contemporary Nurse*. 2024;60(1):54-66. doi:10.1080/10376178.2024.2304712
355. Bradford NK, Caffery LJ, Smith AC. Telehealth services in rural and remote Australia: a systematic review of models of care and factors influencing success and sustainability. *Rural Remote Health*. 2016;16(4):3808.
356. Broerse J, Maple J-L, Klepac P, Gormilovic B, Macklin S, Calder R. *Australia's Health Tracker by Socioeconomic Status* 2021. <https://www.vu.edu.au/sites/default/files/mitchell-institute-australias-health-tracker-by-socioeconomic-status-ses.pdf>.
357. Brewer LC, Jenkins S, Hayes SN, et al. Community-Based, Cluster-Randomized Pilot Trial of a Cardiovascular Mobile Health Intervention: Preliminary Findings of the FAITH! Trial. *Circulation*. 2022;146(3):175-190. doi:10.1161/circulationaha.122.059046
358. Seguin RA, Eldridge G, Graham ML, Foltz SC, Nelson ME, Strogatz D. Strong Hearts, healthy communities: a rural community-based cardiovascular disease prevention program. *BMC Public Health*. 2016;16(1):86. doi:10.1186/s12889-016-2751-4
359. Seguin RA, Paul L, Foltz SC, et al. Strong Hearts, Healthy Communities: A Community-Based Randomized Trial for Rural Women. *Obesity (Silver Spring)*. 2018;26(5):845-853. doi:10.1002/oby.22158
360. White-Williams C, Rossi LP, Bittner VA, et al. Addressing Social Determinants of Health in the Care of Patients With Heart Failure: A Scientific Statement From the American Heart Association. *Circulation*. 2020;141(22):e841-e863. doi:10.1161/cir.0000000000000767
361. Leutner M, Dervic E, Bellach L, Klimek P, Thurner S, Kautzky A. Obesity as pleiotropic risk state for metabolic and mental health throughout life. *Translational Psychiatry*. 2023;13(1):175. doi:10.1038/s41398-023-02447-w
362. Gerardo G, Peterson N, Goodpaster K, Heinberg L. Depression and Obesity. *Curr Obes Rep*. 2025;14(1):5. doi:10.1007/s13679-024-00603-x

363. Scherf-Clavel M, Zebner J, Hommers L, Deckert J, Menke A, Unterecker S. Nortriptyline serum concentration as a predictor for cardiac risk in amitriptyline-treated patients. *Eur J Clin Pharmacol.* 2020;76(1):73-80. doi:10.1007/s00228-019-02766-2
364. Mazereel V, Detraux J, Vancampfort D, van Winkel R, De Hert M. Impact of Psychotropic Medication Effects on Obesity and the Metabolic Syndrome in People With Serious Mental Illness. *Front Endocrinol (Lausanne).* 2020;11:573479. doi:10.3389/fendo.2020.573479
365. Mouawad M, Nabipur L, Agrawal DK. Impact of Antidepressants on Weight Gain: Underlying Mechanisms and Mitigation Strategies. *Arch Clin Biomed Res.* 2025;9(3):183-195.
366. Bueno H, Deaton C, Farrero M, et al. European Society of Cardiology Clinical Consensus Statement on mental health and cardiovascular disease. *Eur Heart J.* 2025;46(41):4156-4225. doi:10.1093/eurheartj/ehaf191
367. Australian Bureau of Statistics. *Waist circumference and BMI.* 2023. www.abs.gov.au/statistics/health/health-conditions-and-risks/waist-circumference-and-bmi/2022
368. Schorr M, Dichtel LE, Gerweck AV, et al. Sex differences in body composition and association with cardiometabolic risk. *Biol Sex Differ.* 2018;9(1):28. doi:10.1186/s13293-018-0189-3
369. Ayesha H, Nasser SA, Ferdinand KC, Carranza Leon BG. Sex-specific factors influencing obesity in women: Bridging the gap between science and clinical practice. *Circ Res.* 2025;136(6):594-605. doi:10.1161/circresaha.124.325535
370. Knight MG, Anekwe C, Washington K, Akam EY, Wang E, Stanford FC. Weight regulation in menopause. *Menopause.* 2021;28(8):960-965. doi:10.1097/gme.0000000000001792
371. Opoku AA, Abushama M, Konje JC. Obesity and menopause. *Best Pract Res Clin Obstet Gynaecol.* 2023;88:102348. doi:10.1016/j.bpobgyn.2023.102348
372. Allen JT, Zahler-Miller C, Lough M, Titus H. Management of obesity in the menopause transition and postmenopausal period. *Surg Obes Relat Dis.* 2026;22(5): 496-506. doi:10.1016/j.soard.2026.01.015
373. Lin S, Deng Y, Huang J, et al. Efficacy and safety of GLP-1 receptor agonists on weight management and metabolic parameters in PCOS women: a meta-analysis of randomized controlled trials. *Sci Rep.* 2025;15(1):16512. doi:10.1038/s41598-025-99622-4
374. Waite M, Khoo B, Tan T. GLP-1 receptor agonists, reproductive health, and the evidence gap in young women. *Lancet obstet. gynaecol. women's health.* 2025;1(4):e261-e262. doi:10.1016/S3050-5038(25)00149-9
375. Qiu Z, Liu Z, Zhang N, et al. Association between early age body mass index and the risk of adulthood cardiovascular diseases: a systematic review and meta-analysis. *Int J Obes (Lond).* 2025;49(5):766-775. doi:10.1038/s41366-024-01710-4
376. McGill HC, Jr., McMahan CA, Herderick EE, et al. Obesity accelerates the progression of coronary atherosclerosis in young men. *Circulation.* 2002;105(23):2712-8. doi:10.1161/01.cir.0000018121.67607.ce
377. Umer A, Kelley GA, Cottrell LE, Giacobbi P, Jr., Innes KE, Lilly CL. Childhood obesity and adult cardiovascular disease risk factors: a systematic review with meta-analysis. *BMC Public Health.* 2017;17(1):683. doi:10.1186/s12889-017-4691-z
378. Hampl SE, Hassink SG, Skinner AC, et al. Clinical practice guideline for the evaluation and treatment of children and adolescents with obesity. *Pediatrics.* 2023;151(2):e2022060640. doi:10.1542/peds.2022-060640
379. Villareal DT, Apovian CM, Kushner RF, Klein S. Obesity in older adults: technical review and position statement of the American Society for Nutrition and NAASO, The Obesity Society. *Am J Clin Nutr.* 2005;82(5):923-34. doi:10.1093/ajcn/82.5.923

380. Donini LM, Busetto L, Bischoff SC, et al. Definition and diagnostic criteria for sarcopenic obesity: ESPEN and EASO consensus statement. *Clin Nutr*. 2022;41(4):990-1000. doi:10.1016/j.clnu.2021.11.014
381. Batsis JA, Villareal DT. Sarcopenic obesity in older adults: aetiology, epidemiology and treatment strategies. *Nat Rev Endocrinol*. 2018;14(9):513-537. doi:10.1038/s41574-018-0062-9
382. Prokopoulos K, Isanejad M, Akpan A, et al. Exercise and nutritional interventions on sarcopenia and frailty in heart failure: a narrative review of systematic reviews and meta-analyses. *ESC Heart Fail*. 2022;9(5):2787-2799. doi:10.1002/ehf2.14052
383. Mirzai S, Carbone S, Batsis JA, Kritchevsky SB, Kitzman DW, Shapiro MD. Sarcopenic obesity and cardiovascular disease: An overlooked but high-risk syndrome. *Curr Obes Rep*. 2024;13(3):532-544. doi:10.1007/s13679-024-00571-2
384. Buckinx F, Aubertin-Leheudre M. Sarcopenia in menopausal women: current perspectives. *Int J Womens Health*. 2022;14:805-819. doi:10.2147/ijwh.S340537
385. De Luca M, Shikora S, Eisenberg D, et al. Scientific Evidence for the Updated Guidelines on Indications for Metabolic and Bariatric Surgery (IFSO/ASMBS). *Obes Surg*. 2024;34(11):3963-4096. doi:10.1007/s11695-024-07370-7
386. Nadolsky K, Garvey WT, Agarwal M, et al. American Association of Clinical Endocrinology Consensus Statement: Evaluation and treatment of obesity/adiposity-based chronic disease – 2025 update. *Endocr Pract*. 2025;31(11):1351-1394. doi:10.1016/j.eprac.2025.07.017
387. Deng H, Duan X, Huang J, et al. Association of adiposity with risk of obstructive sleep apnea: a population-based study. *BMC Public Health*. 2023;23(1):1835. doi:10.1186/s12889-023-16695-4
388. Bradley TD, Floras JS. Obstructive sleep apnoea and its cardiovascular consequences. *Lancet*. 2009;373(9657):82-93. doi:10.1016/s0140-6736(08)61622-0
389. Linz D, McEvoy RD, Cowie MR, et al. Associations of obstructive sleep apnea with atrial fibrillation and continuous positive airway pressure treatment: a review. *JAMA Cardiol*. 2018;3(6):532-540. doi:10.1001/jamacardio.2018.0095
390. Loke YK, Brown JW, Kwok CS, Niruban A, Myint PK. Association of obstructive sleep apnea with risk of serious cardiovascular events: a systematic review and meta-analysis. *Circ Cardiovasc Qual Outcomes*. 2012;5(5):720-8. doi:10.1161/circoutcomes.111.964783
391. Sánchez-de-la-Torre M, Gracia-Lavedan E, Benitez ID, et al. Adherence to CPAP treatment and the risk of recurrent cardiovascular events: A meta-analysis. *JAMA*. 2023;330(13):1255-1265. doi:10.1001/jama.2023.17465
392. McEvoy RD, Antic NA, Heeley E, et al. CPAP for prevention of cardiovascular events in obstructive sleep apnea. *N Engl J Med*. 2016;375(10):919-31. doi:10.1056/NEJMoa1606599
393. Pedditzi E, Peters R, Beckett N. The risk of overweight/obesity in mid-life and late life for the development of dementia: a systematic review and meta-analysis of longitudinal studies. *Age Ageing*. 2016;45(1):14-21. doi:10.1093/ageing/afv151
394. Morys F, Dadar M, Dagher A. Association between midlife obesity and its metabolic consequences, cerebrovascular disease, and cognitive decline. *J Clin Endocrinol Metab*. 2021;106(10):e4260-e4274. doi:10.1210/clinem/dgab135
395. Wong Zhang DE, Tran V, Vinh A, et al. Pathophysiological links between obesity and dementia. *Neuromolecular Med*. 2023;25(4):451-456. doi:10.1007/s12017-023-08746-1
396. Livingston G, Huntley J, Liu KY, et al. Dementia prevention, intervention, and care: 2024 report of the Lancet Commission. *Lancet*. 2024;404(10452):572-628. doi:10.1016/S0140-6736(24)01296-0

Appendix 1

Contributors to the clinical consensus statement

The National Heart Foundation of Australia ('Heart Foundation') developed the Obesity and cardiovascular disease (CVD) clinical consensus statement ('the consensus statement') in collaboration with a network of leading Australian experts and consumers. A national multidisciplinary taskforce ('the Obesity and CVD Taskforce'; 'the Taskforce') was convened to guide the development of the consensus statement, with representation from cardiology, endocrinology/diabetology, bariatric surgery, general practice, First Nations health, epidemiology, dietetics, exercise physiology, pharmacy, public health, and consumers with lived experience. Four topic-specific expert subgroups were formed to develop the related recommendations, practice points and narratives (Figure 1).

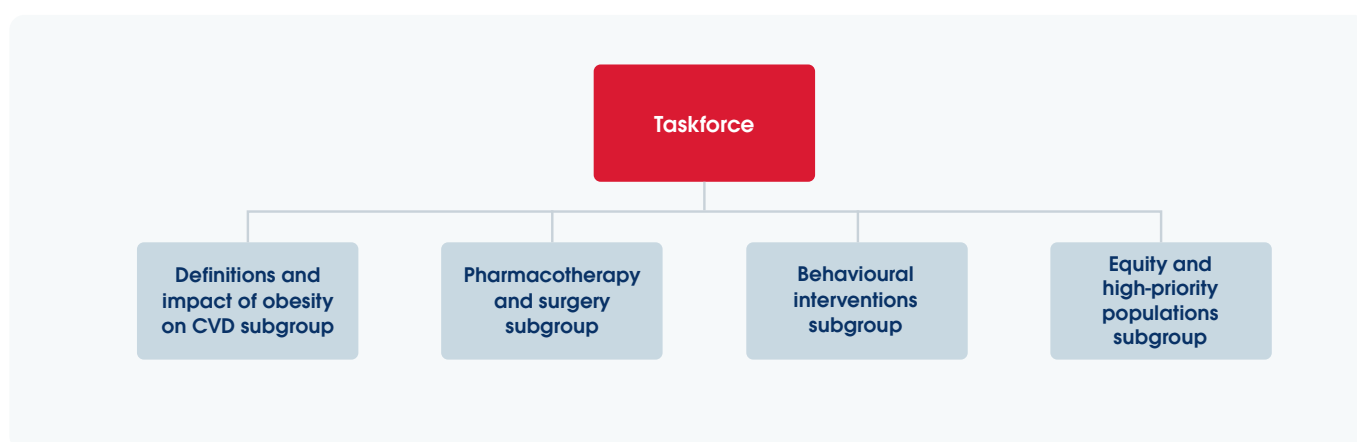


Figure 1: Obesity and CVD clinical consensus statement – expert subgroups

Obesity and CVD Taskforce

Establishment of the Taskforce

In June 2025, the Heart Foundation convened a Taskforce to guide the development of a new clinical consensus statement on obesity and CVD. The roles and responsibilities of the Taskforce included:

- endorsing the proposed clinical scope
- reviewing and providing feedback on the commissioned evidence review and environmental scan
- providing expert clinical input into the development of the consensus statement, including evidence-based recommendations and relevant practice points
- ensuring the consensus statement reflects current best practice and aligns with other major guideline recommendations, where appropriate
- assisting with writing, review and finalisation of the consensus statement.

Taskforce members

Name	Position
Prof Louise Baur	Professor of Child & Adolescent Health, University of Sydney
A/Prof Harsha Chandraratna	Consultant Surgeon, Obesity Surgery WA
Prof Clara Chow	Cardiologist; Academic Director, Westmead Applied Research Centre
Prof Clare Collins	Laureate Professor, Nutrition and Dietetics, College of Health, Medicine & Wellbeing, University of Newcastle
Dr Paresh Dawda	General Practitioner; Director and Principal, Prestantia Health
Prof Sandra Eades	Chief Medical Advisor - First Nations Health, National Heart Foundation of Australia; Deputy Dean, University of Melbourne
Prof Elif Ekinci (Co-chair)	Academic Endocrinologist; Head of Diabetes, Austin Health; Head of Department of Medicine, University of Melbourne
A/Prof Samantha Hocking	Endocrinologist, Royal Prince Alfred Hospital; Clinical Academic, University of Sydney
Prof Garry Jennings (Co-chair)	Chief Medical Advisor, National Heart Foundation of Australia
Mr Ray Kelly	Exercise Physiologist and Exercise Scientist; Casual Academic and Indigenous Researcher, University of Technology Sydney
Dr Mark Mellor	Specialist GP and Obesity Doctor, Perth Weight Clinic
Ms Tammy Merton	Consumer advocate
A/Prof Adam Nelson	Academic Cardiologist; Clinical Lecturer, University of Adelaide
Prof Rajesh Puranik	Consultant Cardiologist, Royal Prince Alfred Hospital; Clinical Associate Professor, University of Sydney
Prof Jonathan Shaw	Consultant Endocrinologist; Deputy Director (Clinical and Population Health), Baker Heart and Diabetes Institute
Dr Terri-Lynne South	General Practitioner and Dietitian; Director, Lifestyle Metabolic
Dr Irene Um	Pharmacist; Senior Lecturer, School of Pharmacy, The University of Sydney
Prof Emerita Alison Venn	Epidemiologist; Adjunct Professor, Menzies Institute for Medical Research, University of Tasmania
Mr Andrew Wilson	Engagement/Advocacy Lead & Board Member, Weight Issues Network
Prof Andrew Wilson	Professor and Co-Director of the Leeder Centre for Health Policy, Economics and Data, University of Sydney
Dr Anna Wood	Physician in Endocrinology and General Medicine, Royal Darwin Hospital; Senior Research Fellow, Menzies School of Health Research
Prof Sophia Zoungas	Clinical Endocrinologist; Head, School of Public Health and Preventive Medicine, Monash University

Expert subgroups

The Taskforce members were each assigned to one of four topic-specific expert subgroups:

Subgroup	Focus
Definitions and impact of obesity on CVD	Definition of and diagnostic tools for clinical overweight and obesity to support risk assessment for the Australian population; impact of weight loss on CVD risk and clinical management of people with overweight or obesity and CVD.
Pharmacotherapy and surgery	Impact of weight loss on cardiovascular (CV) risk (including role of pharmacological and surgical interventions for obesity).
Behavioural interventions	Impact of behaviour change – diet and physical activity to support weight loss and improve CV outcomes or risk factors.
Equity and high-priority populations	Ensure that recommendations consider high priority groups; i.e. adults living with overweight or obesity from culturally and linguistically diverse communities, First Nations peoples, people living in rural and remote areas, people experiencing socio-economic disadvantage and people living with a mental health condition.

Definitions and impact of obesity on CVD subgroup members

Name	Position
Prof Rajesh Puranik (Chair)	Consultant Cardiologist, Royal Prince Alfred Hospital; Clinical Associate Professor, University of Sydney
A/Prof Adam Nelson	Academic Cardiologist; Clinical Lecturer, University of Adelaide
Prof Jonathan Shaw	Consultant Endocrinologist; Deputy Director (Clinical and Population Health), Baker Heart and Diabetes Institute
Prof Sophia Zoungas	Clinical Endocrinologist; Head, School of Public Health and Preventive Medicine, Monash University

Pharmacotherapy and surgery subgroup members

Name	Position
Prof Elif Ekinci (Chair)	Academic Endocrinologist; Head of Diabetes, Austin Health; Head of Department of Medicine, University of Melbourne
A/Prof Harsha Chandraratna	Consultant Surgeon, Obesity Surgery WA
Prof Clara Chow	Cardiologist; Academic Director, Westmead Applied Research Centre
Dr Paresh Dawda	General Practitioner; Director and Principal, Prestantia Health
A/Prof Samantha Hocking	Endocrinologist, Royal Prince Alfred Hospital; Clinical Academic, University of Sydney
Dr Irene Um	Pharmacist; Senior Lecturer, School of Pharmacy, The University of Sydney
Prof Andrew Wilson	Professor and Co-Director of the Leeder Centre for Health Policy, Economics and Data, University of Sydney

Behavioural interventions subgroup members

Name	Position
Dr Terri-Lynne South (Chair)	General Practitioner and Dietitian; Director, Lifestyle Metabolic
Prof Louise Baur	Professor of Child & Adolescent Health, University of Sydney
Prof Clare Collins	Laureate Professor, Nutrition and Dietetics, College of Health, Medicine & Wellbeing, University of Newcastle
Mr Ray Kelly	Exercise Physiologist and Exercise Scientist; Casual Academic and Indigenous Researcher, University of Technology Sydney
Mr Andrew Wilson	Engagement/Advocacy Lead & Board Member, Weight Issues Network

Equity and high-priority populations subgroup members

Name	Position
Dr Mark Mellor (Chair)	Specialist GP and Obesity Doctor, Perth Weight Clinic
Prof Sandra Eades	Chief Medical Advisor - First Nations Health, National Heart Foundation of Australia; Deputy Dean, University of Melbourne
Ms Tammy Merton	Consumer advocate
Prof Emerita Alison Venn	Epidemiologist; Adjunct Professor, Menzies Institute for Medical Research, University of Tasmania
Mr Andrew Wilson	Engagement/Advocacy Lead & Board Member, Weight Issues Network
Dr Anna Wood	Physician in Endocrinology and General Medicine, Royal Darwin Hospital; Senior Research Fellow, Menzies School of Health Research

Heart Foundation project team

Name	Position
Natalie Raffoul	Senior Manager Healthcare Programs and Clinical Strategy
Christine Sarie	Project Management Coordinator, Healthcare Programs
Emily Bradburn	Senior Healthcare Programs Officer, Patient Support Lead
Kelly Donnelly	Senior Healthcare Programs Officer
Jasmine Just	Senior Healthcare Programs Officer, Clinical Communications
Tina Garcia	Senior Medical Writer

Appendix 2

Developing the clinical consensus statement

The Heart Foundation led the development of the consensus statement in collaboration with expert members of the Taskforce. The statement was based on a review of published literature and an environmental scan, and was developed through a structured process to distil practical guidance based on evidence-informed expert consensus. It addresses common clinical challenges and highlights rapidly emerging evidence, with a strong focus on clinical management to support informed, evidence-based care.

Clinical scope

The clinical scope of the consensus statement was prepared by the Heart Foundation project team with direction and input from the Taskforce. The scope was reviewed, refined and signed off by the Taskforce.

The Taskforce agreed on the following 14 clinical scoping questions:

1. How should clinical obesity and overweight be defined and characterised to support accurate diagnosis and risk assessment for the Australian population?
2. What is the association between overweight or obesity and the risk of experiencing a CV event – in the primary and secondary prevention setting – in adults?
3. In adults living with overweight or obesity, how do structured weight management interventions – compared to standard care or no intervention – impact CV outcomes?
4. What is the impact of overweight or obesity on the diagnosis, clinical management and outcomes for people living with or at high risk of CVD?
5. What are the unique clinical considerations for the management of obesity or overweight in people living with or at high risk of CVD?
6. In adults living with overweight or obesity and CVD or at high risk of CVD, what nutritional interventions support weight loss and improve CV outcomes or risk factors?
7. In adults living with overweight or obesity and CVD or at high risk of CVD, what physical activity interventions (including type, time and intensity) support weight loss and improve CV outcomes or risk factors?
8. In adults living with overweight or obesity and CVD or at high risk of CVD, which pharmacological therapies are effective in achieving weight loss and improving CV outcomes and risk factors?
9. What is the impact of bariatric surgery (focusing on the common techniques in Australia) on CVD outcomes, morbidity and long-term or sustained improvements in complications of obesity?
10. In adults living with overweight or obesity from culturally and linguistically diverse communities, how do tailored or integrated management strategies improve CV outcomes, weight reduction and overall health outcomes?

- | | |
|-----|---|
| 11. | In First Nations people living with overweight or obesity, how do tailored or integrated management strategies improve CV outcomes, weight reduction and overall health outcomes? |
| 12. | In adults living with overweight or obesity who have mental health conditions, how do tailored or integrated management strategies improve CV outcomes, weight reduction and overall health outcomes? |
| 13. | In adults living with overweight or obesity from rural and remote communities, how do tailored or integrated management strategies improve CV outcomes, weight reduction and overall health outcomes? |
| 14. | In adults living with overweight or obesity from low socioeconomic status, how do tailored or integrated management strategies improve CV outcomes, weight reduction and overall health outcomes? |

Out of scope

The Taskforce agreed that the following areas were out of scope for the consensus statement:

- detailed advice for children and adolescents
- very detailed recommendations on behaviour modifications (e.g. nutrition and physical activity)
- detailed system/environment level factors (e.g. food supply chain, food availability, built environment)
- detailed advice on eating disorders
- detailed advice on surgical procedures
- detailed consideration of complementary medicines
- detailed or new economic analyses.

Evidence review and environmental scan

To address the clinical scoping questions identified above, a targeted evidence review and accompanying environmental scan were independently commissioned. Dr Shelley Keating from the University of Queensland undertook this work.

Unlike the Heart Foundation's clinical guidelines, this consensus statement did not apply the Grading of Recommendations Assessment, Development and Evaluation (GRADE) methodology or involve undertaking new systematic reviews. It also did not seek to address broader policy, environmental or social issues. The statement does not intend to replace the forthcoming National Health and Medical Research Council's *Clinical Practice Guidelines for the Management of Overweight and Obesity for Adults, Adolescents and Children in Australia*. Rather, the consensus statement complements them with more detailed guidance to address the specific clinical considerations in people living with overweight/obesity with established CVD, or at high risk of CVD.

See supporting document *Evidence Review and Environmental Scan: Clinical Management of Obesity and Overweight in the Context of Cardiovascular Health (2025)*

Developing the recommendations

The consensus statement was developed through a rigorous and transparent process, underpinned by published evidence and expert clinical judgment. The multidisciplinary membership of the Taskforce ensured a breadth of perspective and expertise. Clinical recommendations and/or narrative pieces were developed to address each of the clinical scoping questions.

Evidence-to-recommendation template

The evidence-to-recommendation template provides a structured framework for translating evidence into clinical advice. It includes dedicated sections where the following are documented:

- Recommendations and their strength of advice
- Rationale for the recommendations, narrative summary and practice points
- A brief summary of benefits and harms, weighing desirable and undesirable outcomes
- Certainty of evidence, including relevance and missing data
- Preferences and values, reflecting variability in an individual's priorities
- Resources and other considerations, such as out-of-pocket costs, feasibility in the Australian context and accessibility across diverse communities

Strength of advice

To support the consistency and integrity of the recommendations, a structured framework for strength of advice was specifically developed to guide the evaluation of evidence and formulation of advice (Table 1). This framework was adapted from the European Society of Cardiology's Scientific Documents Policy.¹

Based on evidence quality and consensus among the Taskforce members, the strength of advice is both evidence-based and practical. Generally, the more high-quality studies and robust research designs included in an analysis, the stronger its evidence rating. Systematic reviews, meta-analyses and randomised controlled trials typically represent the highest level of evidence due to their rigorous methodology and strong indications of effectiveness.²

Table 1. Strength of advice hierarchy

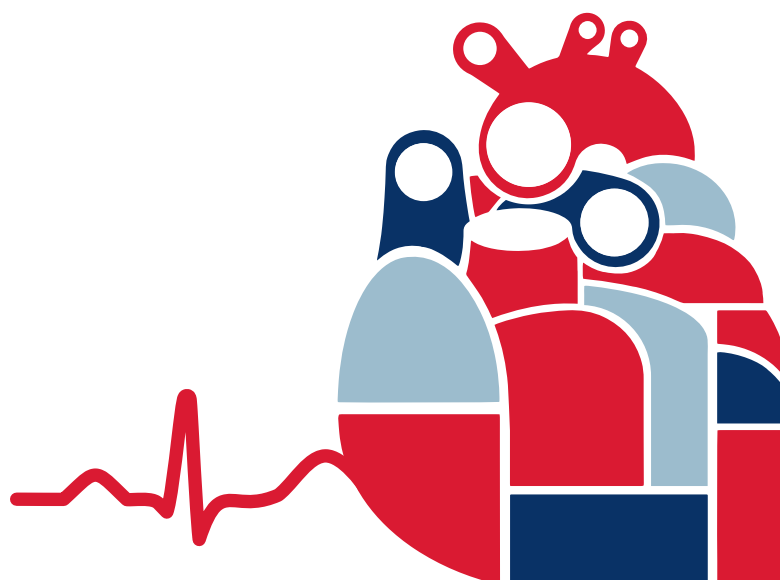
<p>A. Strong advice, based on robust published evidence* and supported by expert consensus.** High confidence that benefits clearly outweigh risks. Supported by consistent robust high-quality evidence and expert consensus.</p>	● ● ●
<p>B. Moderate advice, based on some published evidence* and supported by expert consensus.** Benefits likely outweigh risks, but with some uncertainty. Evidence may not be as strong or expert opinion may not reach unanimous consensus.</p>	● ● ○
<p>C. May be appropriate, based on published evidence* and expert consensus.** Insufficient evidence in this area. Recommendation based primarily on expert consensus.</p>	● ○ ○
<p>D. Area of uncertainty. Insufficient evidence or consensus to support a clear position. Clinical practice may vary in this area.</p>	○ ○ ○

* Systematic reviews, meta-analyses and randomised controlled trials constitute the highest level of evidence. Observational studies constitute a lower level of evidence and are considered less robust. Case studies and grey literature constitute the lowest level of evidence.

** While formal voting thresholds were not specified, recommendations were retained only where there was strong majority support and no substantive unresolved objections.

1 European Society of Cardiology. ESC Scientific Documents Policy. 2021. <https://www.escardio.org/guidelines/scientific-documents/scientific-document-policy/>

2 Murad MH, Asi N, Alsawas M, Alahdab F. New evidence pyramid. *BMJ Evid Based Med*. 2016;21:125-127. doi: 10.1136/ebmed-2016-110401



Drafting the consensus statement

The Heart Foundation project team brought together the evidence-to-recommendation templates signed off by each subgroup to draft the consensus statement. The first draft was reviewed and approved by the Taskforce prior to targeted public consultation.

Following consultation, the consensus statement was updated and the final draft was approved by the Taskforce.

Targeted consultation

Targeted public consultation was conducted between 16 February and 9 March 2026, to seek feedback on the draft consensus statement prior to finalisation. The draft was circulated to a targeted group of stakeholders, including experts in cardiology, endocrinology and diabetology, bariatric surgery, general practice, First Nations health, epidemiology, nutrition and dietetics, exercise physiology, physiotherapy, pharmacy, public health and research, as well as representatives from peak health bodies, government and consumer peak bodies. Consultation was conducted within a defined scope, focusing on the clarity, balance, and clinical applicability of the draft.

A total of 833 individual pieces of feedback were received via an online form from 34 organisations and 13 individual contributors. All feedback was initially reviewed by the project team to determine whether it was in or out of scope. For feedback that was considered in scope, the project team determined whether it should be accepted, accepted with amendment, escalated to the Taskforce, noted, considered for implementation or rejected (giving rationale). This process was guided by the Taskforce Co-Chairs and broader Taskforce at regular meetings. Feedback considered outside the agreed scope was noted but not incorporated. Key changes to the statement resulting from targeted consultation were deliberated by the Taskforce, and the final document was approved following incorporation of feedback.

Summary of consultation feedback

Overall support and value of the statement

Respondents broadly welcomed the development of a national, evidence-informed consensus statement addressing the intersection of obesity and CVD. Many described the document as comprehensive, timely, and clinically valuable, particularly its recognition of obesity as a complex, chronic condition and its emphasis on multidisciplinary care, equity considerations, and weight stigma.

General feedback

Overall, consultation feedback was highly engaged and constructive, with strong support for the statement's intent and scope. Suggested revisions largely focused on improving balance, clarity, equity, and real-world applicability, while preserving the evidence-informed, consensus-based foundation of the document.

Defining overweight and obesity

Feedback suggested:

- stronger positioning of waist-based measures and metabolic markers
- clearer articulation of obesity as a chronic disease rather than solely a risk factor.

Weight stigma and high-priority populations

Feedback suggested:

- the inclusion of weight stigma, but recommended strengthening access and equity considerations, as well as practical guidance for healthcare professionals
- the inclusion of high-priority populations, with requests for more detailed guidance on practical implementation and the inclusion of women-specific information.

Nutrition

Feedback suggested:

- the need for additional information to address the specific nutritional considerations for people taking obesity management pharmacotherapy and post metabolic bariatric surgery, including protein adequacy and lean mass preservation for people during weight loss
- the inclusion of information on other dietary approaches (e.g. carbohydrate-reduced approaches for people with metabolic dysfunction)
- stronger recognition of the central role of Accredited Practising Dietitians within multidisciplinary care.

Physical activity

Key themes from the feedback included:

- the need to acknowledge potential barriers to physical activity, including obesity-related pain, disability, deconditioning, and stigma
- request for the inclusion of guidance for the specific physical activity requirements for people on incretin-based therapies to preserve lean muscle mass
- the need to strengthen the reference to physiotherapists, exercise physiologists, and allied healthcare professionals in assessment, rehabilitation, and safe progression of activity, particularly for people with CVD and complex comorbidity.

Pharmacotherapy

Common themes included:

- avoiding class-based language where evidence is agent-specific
- clarifying recommendations to better align with trial populations, dosages and outcomes
- greater transparency around access, cost, Pharmaceutical Benefits Scheme status, and real-world adherence
- need to address lean muscle mass loss, nutritional risk, and weight regain following treatment discontinuation, with informed consent and ongoing monitoring emphasised
- stronger safety and monitoring guidance.

Metabolic bariatric surgery

Key feedback included:

- the importance of clearly articulating the rationale underpinning eligibility thresholds for metabolic bariatric surgery
- opportunities to further emphasise multidisciplinary care and the role of lifelong follow-up
- the value of including tailored dietary information for people who have undergone metabolic bariatric surgery
- the importance of describing care pathways, supporting timely referral, and ensuring language and evidence presentation are contemporary and aligned with best practice.

Appendix 3

Conflicts of interest

The Heart Foundation and members of the Taskforce acknowledge the importance of both transparency and appropriate management of conflicts of interest.

Conflicting interests were considered within a framework of both:

- i. the relationship (direct or indirect) of the participating individual to any third party with interest in the topic under consideration during the development process of the consensus statement
- ii. the nature (financial and non-financial) of the potential conflict.

Conflicting interests among the subgroups required appropriate management to ensure clinical recommendations were not compromised. Processes employed by the Heart Foundation project team aimed to ensure the integrity of the Taskforce and to strike an appropriate balance between the existence of interests in a topic under review and the expertise required to make sound and meaningful recommendations.

Conflicts of interests were managed in the following ways:

- All Taskforce members were required to disclose potential conflicts of interest at the commencement of membership, and to update the Taskforce and their subgroups during the project if there were any changes to this declaration.
- Conflict of interest declarations were revisited at each Taskforce and subgroup meeting to ensure new disclosures were recorded and considered.
- In circumstances where a conflict of interest was identified and deemed significant, it was managed by ensuring that the member had limited involvement in the deliberation of the evidence (with the possibility of bias noted), or in discussions on the wording, structure, intent or formulation of the clinical recommendation relevant to disclosure of a conflict.

All conflict-of-interest declarations were regularly reviewed by the project team (Table 2).

Table 2. Conflict of interest register

Taskforce member name	Involvement in project	High level benefit (Y/N)?	Low level benefit (Y/N)?	If yes, details	No benefit but position held, or investment in an activity that does not provide cash incentive
Louise Baur	Taskforce member, Public health professional	N	Y	Low level benefit Speaker fees (non-personal): Speakers bureau, Lilly; Speakers bureau, Novo Nordisk (funds directed to institutional cost centre). Commissioning/funding research (non-personal): Australian PI of ACTION Teens Study (Novo Nordisk sponsored; conducted in 2021).	Holding office (personal): Past-President, World Obesity Federation (2024–2026).
Harsha Chandraratna	Taskforce member, Bariatric surgeon	N	Y	Low level benefit Speaker fees (personal): Paid lecture fees. Travel, accommodation and conference registration (personal): Provided in connection with attendance at overseas meetings.	N
Clara Chow	Taskforce member, Cardiologist	Y	Y	High level benefit Professor of Medicine, Academic Director (WARC, USyd), Cardiologist – Westmead Hospital Low level benefit Membership of Advisory Boards (personal): WSLHD Board; Heart Foundation Board. Speaker fees (personal): previously received speakers' fees including Novartis; Limbic; Eli Lilly; Novo Nordisk; Amgen. Expenses/hospitality: Novartis; Limbic; Eli Lilly; Novo Nordisk; Amgen (conference and advisory meeting attendance abroad). Commissioning/funding research: NHMRC Investigator grant recipient; investigator on MRFF/NHMRC grants; previously received grants from NSW Health; Australian Digital Health Agency; Google. Other support (non-personal): The George Institute patent applications for low fixed-dose combination products (no direct financial interests).	
Clare Collins	Taskforce member, Nutrition and dietetics	N	Y	Low level benefit Member of Research Advisory Committee, Health and Well-being Queensland (personal) and co-investigator on a co-funded pilot study with University of Newcastle and Honeysuckle Health testing addition of telehealth Medical Nutrition Therapy to Healthy Weight For Life telehealth coaching (non-personal). Amgen-sponsored meeting travel support.	Opinions informed by personal evaluation of research studies and role as a researcher and clinician (Accredited Practising Dietitian) and commonly conveyed via the media. Host of a podcast Nutrition Science Bites and leader of the No Money No Time website.

Taskforce member name	Involvement in project	High level benefit (Y/N)?	Low level benefit (Y/N)?	If yes, details	No benefit but position held, or investment in an activity that does not provide cash incentive
Paresh Dawda	Taskforce member, GP	Y	Y	<p>High level benefit Directorship/work (personal): Director in Prestantia Health (healthcare consultancy services); Director at Next Practice Canberra Pty Ltd (general practice).</p> <p>Low level benefit Speaker fees (personal): BMJ IHI Quality Forum conference organising committee – complimentary access</p>	
Sandra Eades	Taskforce member, Public health professional	N	N	N/A	N
Elif Ekinici	Taskforce member, Co-Chair & Endocrinologist/diabetologist	N	Y	<p>Low level benefit Membership of advisory boards (institutional): Eli Lilly; Boehringer Ingelheim; Novo Nordisk (advisory board fees paid to institution for diabetes research). Membership of advisory boards (institutional): CSL (fees donated to the university; no personal remuneration). Other: Novo Nordisk provided direct payment in circumstances where payment to the institution was not possible.</p>	N
Samantha Hocking	Taskforce member, Endocrinologist/diabetologist	N	Y	<p>Low level benefit (Personal): Lilly; Seqirus; Novo Nordisk. Commissioning/funding of research (non-personal): Amgen; Astra Zeneca; Lilly; Novo Nordisk; Pfizer; Harmony Biosciences; Impact Obesity; Endogenex; Spruce Biosciences. Eli Lilly advisory board regarding SURPASS CVOT.</p>	Holding office (non-personal): President NACOS; Member of Council ANZOS
Garry Jennings	Taskforce member, Co-chair & Cardiologist	N	N	N/A	Trustee & Director Baker Foundation; Honorary Professor University of Sydney; Honorary Professor Monash University; Honorary Cardiologist Alfred Health; Life Governor & Cardiologist Baker Heart & Diabetes institute; Board Member Global Cardiovascular Research Funders Forum; AHA Journal Associate Editor Hypertension; Board Member Hypertension Australia

Taskforce member name	Involvement in project	High level benefit (Y/N)?	Low level benefit (Y/N)?	If yes, details	No benefit but position held, or investment in an activity that does not provide cash incentive
Ray Kelly	Taskforce member, Exercise physiologist	N	N	N/A	
Mark Mellor	Taskforce member, GP	Y	Y	<p>High level benefit Founder and clinical lead of Perth Weight Clinic. Clinical lead Cardiometabolic services, WA.</p> <p>Low level benefit Membership of advisory boards (personal): Novo Nordisk; Eli Lilly. Speaker fees (personal): Novo Nordisk; Eli Lilly (including presentations relating to semaglutide (Wegovy) and tirzepatide). Expenses/hospitality (personal): Novo Nordisk; Eli Lilly. Steering committee membership: Boehringer Ingelheim (general practitioner education). Other: Online media sponsored by Nestlé; webinar funded by the WA Primary Health Alliance.</p>	Holding office (personal): WA lead RACGP obesity specific interest group (2022 appointed); Chair of advocacy panel for equitable access to funded treatment for people with obesity (Mar 2025 on). Clinical lead Perth Weight Clinic and Cardiometabolic services.
Tammy Merton	Taskforce member, Lived experience representative	N	N	N/A	
Adam Nelson	Taskforce member, Cardiologist	N	Y	<p>Low level benefit Membership of Advisory Boards (personal): Astra Zeneca; Boehringer Ingelheim; Eli Lilly; GSK; Novartis; Novo Nordisk; Pfizer. Speaker fees (personal): Amgen; Astra Zeneca; Boehringer Ingelheim; CSL; Eli Lilly; GSK; Novartis; Novo Nordisk. Expenses/hospitality (personal): Eli Lilly; Novo Nordisk. Commissioning/funding of research (non-personal): Amgen; Boehringer Ingelheim; Eli Lilly.</p> <p>Additional notes Current grant: AMGEN – GOAL ASIA implementation trial (AUD \$1.2 million).</p>	
Rajesh Puranik	Taskforce member, Cardiologist	N	Y	<p>Low level benefit Speaker fees (personal): Astra Zeneca meeting, Dec 2023 (\$1852.50).</p>	Holding office: Board member, CSANZ.
Jonathan Shaw	Taskforce member, Endocrinologist/diabetologist	N	Y	<p>Low level benefit Membership of Advisory Boards of Novo Nordisk, Eli Lilly, Sanofi and Astra Zeneca. Speaker fees for Eli Lilly; Roche; Astra Zeneca; Novo Nordisk; Boehringer Ingelheim; GSK.</p>	Vice President of the Australian Diabetes Society (personal)

Taskforce member name	Involvement in project	High level benefit (Y/N)?	Low level benefit (Y/N)?	If yes, details	No benefit but position held, or investment in an activity that does not provide cash incentive
Terri-Lynne South	Taskforce member, GP	Y	Y	<p>High level benefit Director of Lifestyle Metabolic - a private clinic treating clients with obesity and associated conditions (2022 on).</p> <p>Low level benefit Speaker fees (personal): Novo Nordisk; Eli Lilly; iNOVA; Nestlé. Advisory boards (personal): Novo Nordisk; Eli Lilly; Boehringer Ingelheim. Travel, accommodation and conference registration (personal): Novo Nordisk. Commissioning/funding of research: Nestlé. Educational grant: Impact Obesity. Advisory role: Impact Obesity.</p>	<p>Holding office (personal):</p> <ul style="list-style-type: none"> • Chair RACGP obesity specific interest group (2022 appointed) • Advisor TOC (The Obesity Collective) 2022 onwards • Committee member NACOS (National Association Clinical Obesity Services) 2022 onwards • Member of advocacy panel for equitable access to funded treatment for people with obesity (Mar 2025 on) • Advisor: Impact Obesity 2022 onwards
Irene Um	Taskforce member, Pharmacist/ pharmacologist	N	N	N/A	N
Alison Venn	Taskforce member, Epidemiologist	N	N	N/A	Past and ongoing research roles as an epidemiologist investigating relationships between obesity and cardiovascular disease risk, and the health service use and outcomes of bariatric surgery. Currently Adjunct Professor at the Menzies Institute for Medical Research, University of Tasmania and formerly Institute Director (2016-22). Menzies receives funding from the Heart Foundation. Member of the Heart Foundation Tasmania Advisory Board (2025).
Andrew Wilson	Taskforce member, Lived experience representative	N	N	N/A	Obesity Australia Board Member (three years tenure - non financial) and Impact Obesity Advisory Group (nearly two years - non-financial).

Taskforce member name	Involvement in project	High level benefit (Y/N)?	Low level benefit (Y/N)?	If yes, details	No benefit but position held, or investment in an activity that does not provide cash incentive
Andrew Wilson	Taskforce member, Public health professional	N	N	N/A	<p>Member, NSW Health Expert Advisory Group on Type 2 Diabetes Prevention.</p> <p>Chair, Health Technology Assessment (HTA) Review Implementation Advisory Group, Commonwealth Department of Health (no direct relationship to obesity treatment or CVD prevention).</p> <p>Chair, Pharmaceutical Benefits Advisory Committee (PBAC), including meetings with pharmaceutical companies marketing obesity medicines (e.g. Novo Nordisk, Eli Lilly); no benefits received.</p> <p>Chief Investigator / Co-author, NHMRC Medicines Intelligence CRE, including authorship of a manuscript under review on the use of weight-reduction pharmacotherapies in Australia.</p> <p>Lead Investigator, NHMRC Prevention Partnership Centre, involving research on obesity and prevention, jointly funded by NHMRC and government agencies (no industry involvement).</p> <p>Current grant holder, MRFF-funded research on ethnicity and cardiovascular disease (no industry funding; not focused on obesity).</p> <p>Other grant involvement, NHMRC- and Heart Foundation-funded projects on the prevention of cardiovascular disease (no industry sponsorship).</p>
Anna Wood	Taskforce member, Endocrinologist/diabetologist	N	N	N/A	N/A
Sophia Zoungas	Taskforce member, Endocrinologist/diabetologist	N	Y	<p>Low level benefit</p> <p>Speaker fees for sponsored educational events (non-personal): payment to institution. Expenses/hospitality for meetings/conferences (non-personal): payment to institution.</p>	N/A

Appendix 4

Mental health and eating disorder validated assessment tools

Tool	Items/format	Purpose/notes
Depression		
PHQ9	9 items	Screens and monitors severity of depression; widely validated in primary care.
DASS21	21 items (3 subscales)	Measures depression, anxiety and stress; brief and validated.
K10	10 items	Assesses psychological distress; recommended in Australian GP settings.
WHO5	5 items	Short wellbeing index; useful for monitoring emotional wellbeing.
Anxiety		
GAD7	7 items	Screens for generalised anxiety disorder; validated for primary care.
DASS21 (Anxiety subscale)	7 items	Measures anxiety symptoms alongside depression and stress.
K10	10 items	Captures general distress including anxiety.
Eating disorders		
SCOFF	5 items	Rapid screening for eating disorders; widely used in GP settings.
BEDS7	7 items	Screens for binge eating disorder.
InsideOut Screener	Variable	Australian developed tool for eating disorder risk assessment.
Eating disorder tool finder	https://hub.eatingdisordersresearch.org.au/tools	
Neurodivergence		
ASRS (Adult attention deficit hyperactivity disorder Self Report Scale)	18 items (short form available)	Screens for ADHD in adults; validated for primary care.
AQ10	10 items	Brief screening for autism spectrum traits; recommended.

