CKM Guidance

Cardiovascular-Kidney-Metabolic Disease

Preventing Adverse Outcomes in Cardiovascular Kidney Metabolic Conditions

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Please make sure to periodically check for updated content.

Introduction:

The Cardiac, Renal, Diabetes and Stroke National Clinical Networks in conjunction with the Long-Term Conditions and Planning, Funding and Outcome Teams in Te Whatu Ora have developed national consensus best-practice guidance on optimising Cardiovascular-Kidney-Metabolic (CKM) health and reducing associated risk factors in adults.

The prevalence of CKM disease continues to increase and is a major cause of morbidity and mortality in Aotearoa New Zealand. Importantly, CKM disease also creates significant disparities for Māori, Pacific and Indian populations which have not improved over the past 20 years. This has provided the catalyst for Aotearoa New Zealand to develop holistic and people centric consensus guidance which aims to improve outcomes for all people with CKM disease and to eliminate current disparities and achieve equity.

The intent is to have a concise and pragmatic resource for all health professionals working with people who have CKM disease, particularly in community care. The guidance will link into appropriate specialist society detailed guidelines and Health Pathways wherever needed and possible. Our guidance will be updated as evidence and practice evolve. New sections will also be added with time including management of metabolic dysfunction-associated steatotic liver disease, obstructive sleep apnoea and more detailed guidance on interventions for weight loss.

We have included suggestions on the likely best medications to use based on efficacy, tolerance and adherence for prescribers if needed. Tips to improve access and alternatives are provided when these medications are not funded or available. As per all aspects of the guidance, all suggestions are recommendations only and clinical judgement and individualisation of care remain paramount. This link summarises the changes in the guidance from previous care.

Instructions:

The guidance is separated into the multiple sections.

Clicking on the yellow highlighted text will take you to the relevant section of the guidance within this document.

Clicking on a pink highlighted abbreviation will take you to the relevant abbreviation within the abbreviations section of this document.

Clicking on a blue link will open relevant external guidance in a new window for more detailed information.

Contents:

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1. Commitment to equity in CKM care

Te Tiriti O Waitangi and our Shared Obligation

- Our clinical guidance is grounded in the articles and principles of Te Tiriti o Waitangi which forms the foundations of Pae Ora – healthy futures for Māori. We acknowledge the full text of Te Tiriti and the Ritenga Māori Declaration, which affirm Māori rights to tino rangatiratanga, equity, and the protection of cultural identity and mātauranga Māori
- Te Tiriti establishes obligations for the entire health system, including how care is designed, delivered, and experienced by Māori. These obligations are active and enduring, and must shape every aspect of our response to CKM disease, from prevention to treatment to long-term support.

Equity in CKM disease

- While Te Tiriti provides the foundation for Māori health, **equity** is a broader commitment that extends to all groups who experience disadvantage in health outcomes.
- CKM disease drives some of the **deepest and most entrenched health inequities** in Aotearoa, particularly for Māori, Pacific, and Indian populations and the intersection with rurality and disability. These groups experience earlier onset, more severe complications, and higher mortality from CKM conditions. They are also less likely to receive timely, evidence-based, and culturally safe care.
- These inequities are not caused by individual choices or cultural behaviours, but stem from:
    - The historical and ongoing impacts of colonialism as it applies to Māori
    - Institutional racism and implicit bias

    - Unequal access to high-quality, culturally safe healthcare
    - Chronic underfunding of Māori- and Pacific-led models of care

- We also recognise the health needs and strengths of other populations who face structural barriers, including those experiencing socioeconomic hardship.
- To change these outcomes, **our guidance must be implemented with intentionality, accountability, and resourcing** — especially to support Māori and Pacific-led solutions that are by, with, and for communities.

We can all achieve equity in CKM care by:

- Reflecting and challenging how personal, institutional and systemic racism shapes our practice
- Create safe environments where Māori and Pacific whānau feel safe, respected, and understood. Prioritise whakawhanaungatanga and mana-enhancing approaches.
- Support whānau-centred models of care including kaupapa Māori and Pacific providers where available. Enable shared decision-making that honours lived realities, cultural values, and whānau strengths.
- Address social determinants of health wherever possible such as housing, kai/food security, and income, which shape CKM disease and its management.
- Challenging system constraints and continuing to advocate for access and funding to diagnostics, treatments (e.g. GLP1Ra, SGLT2i), and support services to reduce disparities.

We need to achieve equity because it is clinical excellence:

- Every clinical interaction is an opportunity to either uphold or undermine equity.
- We encourage you to use this guidance not only as a technical resource, but as a platform for transformative practice that challenges disparities and improves outcomes for all those most affected with CKM disease.

↑ Back to contents

2. Definition of CKM diseases

- Manifestations of cardiovascular-kidney-metabolic (CKM) disease include:
    - Elevated blood pressure ≥ 130/80 mmHg and hypertension (≥ 140/90 mmHg)
    - Type 2 diabetes - HbA1c ≥ 48 mmol/mol and/or fasting glucose ≥ 7 mmol/L
    - Dyslipidaemia
    - CVD including ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, atrial fibrillation and heart failure
    - Chronic kidney disease (eGFR <60mL/min and/or UACR >3 mg/mmol)
    - Metabolic dysfunction-associated steatotic liver disease (MASLD; previously termed fatty liver disease)

    - Gout
    - Obstructive sleep apnoea

- Manifestations of CKM disease may occur in isolation but typically occur together in CKM syndrome due to their shared pathophysiology and pathophysiological interactions between the cardiovascular (CV), kidney and metabolic systems leading to multiorgan dysfunction and adverse CV and renal outcomes. The most common shared pathophysiology is increased adiposity, particularly abdominal and visceral adiposity. Excess adiposity is defined as any of the following:
    - BMI > 40 kg/m^2 OR
    - BMI > ethnicity-specific threshold for obesity AND one increased anthropometric criteria OR
        - > 35 kg/m^2 in Pacific peoples
        - > 32 kg/m^2 in Māori
        - > 30 kg/m^2 in Europeans
        - > 25 kg/m^2 in South East Asian ethnicity
    - Two increased anthropometric criteria regardless of BMI
    - Anthropometric criteria include:
        - Waist:height ratio > 0.5
        - Increased waist circumference
            - > 88 cm in females and > 102 cm in males of non-South East Asian ethnicity
            - Waist circumference > 80 cm in females and > 90 cm in males of South East Asian ethnicity
        - Waist:hip ratio > 0.86 in women and > 1 in men
        - Body fat percentage by DEXA or bioimpedance > 30% for men and > 42% for women

- Obesity is now characterised as preclinical obesity or clinical obesity:
    - Preclinical obesity is defined as excess adiposity with preserved function of other tissues and organs
        - People with preclinical obesity are high-risk for developing CKM disease and clinical obesity. As such, people with preclinical obesity should be reviewed at least every 5 years to screen for CKM disease and other complications
        - The term healthy obesity should no longer be used given the high lifelong risk of developing CKM disease and other complications such as arthritis and cancer.

- Clinical obesity is defined as excess adiposity impacting the function of tissues, organs and the entire individual including CKM and non-CKM sequelae
    - CKM sequelae include:
        - Blood pressure ≥ 130/80 mmHg
        - Type 2 diabetes - HbA1c ≥ 48 mmol/mol and/or fasting glucose ≥ 7 mmol/L
        - Dyslipidaemia
        - CVD including ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, atrial fibrillation and heart failure
        - Metabolic dysfunction-associated steatotic liver disease (MASLD; previously termed fatty liver disease)
        - Chronic kidney disease (eGFR <60mL/min and/or UACR >3 mg/mmol)
        - Gout
    - Non CKM sequelae include:
        - Joint pain and osteoarthritis
        - Reduced age-adjusted mobility
        - Lymphoedema
        - Raised intracranial hypertension
        - Obstructive sleep apnoea

- Preclinical obesity is typically the beginning of the spectrum of CKM disease and the first stage of CKM syndrome:
    - Stage 1: Preclinical obesity or clinical obesity with non-CKM sequelae
        - Joint pain and osteoarthritis
        - Reduced age-adjusted mobility
        - Lymphoedema
        - Raised intracranial hypertension
        - Obstructive sleep apnoea
    - Stage 2: Metabolic disease AND/OR mild kidney disease without CVD or significant kidney disease including ANY of the following:

        - Type 2 diabetes
        - Elevated blood pressure and hypertension
        - Dyslipidaemia
        - MASLD
        - Gout
        - UACR 3 - 30 mg/mmol
        - eGFR 45 - 60 mL/min

    - Stage 3: Clinical obesity with subclinical CVD OR equivalent CV risk including ANY of the following:
        - 5 year CV risk ≥ 10%
        - Asymptomatic coronary or carotid disease
        - Coronary calcium score ≥ 100
        - Type 2 diabetes and any microvascular or macrovascular complication
        - UACR > 30 mg/mmol
        - eGFR < 45 mL/min

    - Stage 4a: Clinical obesity with overt CVD including ANY of the following:
        - Ischaemic heart disease
        - Cerebrovascular disease
        - Peripheral arterial disease
        - Heart failure
        - Atrial fibrillation

    - Stage 4b: As per stage 4a but eGFR < 15 mL/min or on renal replacement therapy

- **Proactively targeting shared pathophysiology such as obesity and elevated blood pressure is crucial in preventing, delaying and reducing the progression of all manifestations of CKM disease, particularly adverse cardiovascular and renal sequelae, and death.**
- ***The guidance in this section is based on the new international consensus definition of obesity by the Lancet Commission, which can be accessed at www.thelancet.com/commissions-do/clinical-obesity, and the definition of CKM syndrome by the American Heart Association, which can be accessed at www.ahajournals.org/doi/10.1161/CIR.0000000000001186***

↑ Back to contents

3. Clinical assessments in people with CKM disease

- People with any CKM disease should have at least annual assessments to determine whether there has been any progression, complications or development of other CKM disease
    - Manifestations of CKM include:
        - Clinical obesity
        - Elevated blood pressure and hypertension
        - Type 2 diabetes
        - Dyslipidaemia
        - CVD including ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, atrial fibrillation and heart failure
        - Chronic kidney disease (eGFR < 60mL/min and/or UACR > 3 mg/mmol)
        - Metabolic dysfunction-associated steatotic liver disease (MASLD; previously termed fatty liver disease)
        - Gout
        - Obstructive sleep apnoea

- These clinical assessments should include at least:
    - Smoking status and alcohol intake
    - Waist circumference and BMI
    - Seated blood pressure (BP) +/- standing BP
        - Measure sitting/standing or ideally lying/standing if any concerns over postural hypotension e.g. postural symptoms, frail, elderly, multiple BP lowering agents etc.
    - HbA1c +/- fasting glucose
        - Combining fasting glucose with HbA1c prevents the need for another confirmatory test to diagnose diabetes if the HbA1c is > 48 mmol/mol. Fasting glucose is also the preferred diagnostic test if measurement of HbA1c may be unreliable such as:
            - Any haemoglobinopathy e,g, thalassaemia, sickle cell anaemia etc.
            - Altered red cell turnover e.g. bleeding, haemolysis, severe iron deficiency

        - Second and third trimesters of pregnancy
        - Post blood transfusion
    - HbA1c screening may be reduced to every 3 years if < 42 mmol/mol
  - eGFR and urinary ACR
  - Non fasting lipid studies
  - Serum urate if history of gout (may need to ask at assessment)
  - Foot examination and check of retinal photoscreening if known diabetes
  - Epworth sleep score if history suggestive of obstructive sleep apnoea
  - Screen for depression e.g. PHQ-2 score
  - Screen for diabetes distress if known diabetes e.g. DDS2 score
  - Calculation of 5 year CV risk on PREDICT CV risk calculator at diagnosis of CKM and then:
    - Low CV risk (5 year CV risk < 5%) → 5 yearly
    - Moderate CV risk (5 year CV risk 5 – <10%) → yearly
      - May be relaxed to 2 yearly if gout or MASLD alone
    - High CV risk (5 year CV risk ≥ 10%) → no need to repeat as need to optimise treatment

- **It is critical that management commences as soon as reasonably practical if any positive findings:**
  - If BP ≥ 130/80 mmHg start management of elevated blood pressure and hypertension
  - If HbA1c ≥ 48 mmol/mol and start management of type 2 diabetes if diagnosis confirmed
    - The diagnosis of diabetes requires a confirmatory test either with a repeat elevated HbA1c, fasting glucose ≥ 7 mmol/L, a random glucose > 11 mmol/L if symptomatic, or a 2 hour glucose > 11 mmol/L on a 75 g glucose tolerance test. The 2nd test should be done without delay and may be 2 of the same test e.g. 2 x HbA1c measurements.
    - Although > 90% of new diagnoses are type 2 diabetes beware of red flags for other types of diabetes such as:
      - Onset of diabetes at a young age
      - Symptoms of insulin deficiency at or shortly following diagnosis

        - Normal or low BMI at diagnosis
        - Family history of non-type 2 diabetes
        - Pancreatic exocrine deficiency
        - Positive anti-GAD, anti-IA2 and/or anti-ZnT8 antibodies
        - Low C-peptide (fasting < 250 pmol/L; random < 600 pmol/L) with glucose > 8 mmol/L

    - If eGFR < 60mL/min and/or UACR > 3 mg/mmol on ≥ 2 collections over ≥ 3 months → start management of chronic kidney disease
        - Confirmation of albuminuria requires 2 x UACR > 3 mg/mmol over ≥ 3 months to exclude falsely raised ratios due to:
            - Urinary tract infection
            - Intercurrent illness
            - Vigorous physical activity
            - Haematuria
            - Significant hyperglycaemia
            - Orthostatic (postural) proteinuria – particulalry in youth and young adults
            - Idiopathic proteinuria – transient proteinuria of unknown cause
            - False positives are reduced by performing the UACR in the early morning but do not let this be a barrier to any test

    - If excess adiposity then start interventions for weight loss
        - Excess adiposity is defined as:
            - BMI > 40 kg/m^2 OR
            - BMI > ethnicity-specific threshold AND one increased anthropometric criteria OR
                - > 35 kg/m^2 in Pacific peoples
                - > 32 kg/m^2 in Māori
                - > 30 kg/m^2 in Europeans
                - > 25 kg/m^2 in Asian Indians

        - Two increased anthropometric criteria regardless of BMI
        - Anthropometric criteria include:
            - Waist:height ratio > 0.5
            - Increased waist circumference:
                - > 88 cm in females and > 102 cm in males of non-Asian Indians
                - > 80 cm in females and > 90 cm in males of Asian Indians
            - Waist:hip ratio > 0.86 in women and > 1 in men
            - Body fat percentage by DEXA or bioimpedance > 30% for men and > 42% for women

    - If LDL cholesterol > 1.4 mmol/L and/or triglycerides > 1.7 mmol/L determine whether starting management of dyslipidaemia is appropriate
    - If gout and serum urate > 0.36 mmol/L (or > 0.3 mmol/L if tophi) then optimise management of gout
    - If smoking or vaping then discuss cessation
    - If ≥ 3 on PHQ-2 then complete PHQ-9 and consider treatment for depression as appropriate
    - If ≥ 3 on DDS2 then evaluate diabetes distress and consider support as appropriate

- **NB: People with CKM with elevated blood pressure, lipids, urate (if gout) or glucose levels should be reviewed at least every 1-3 months with escalation of treatment until targets are reached.**
    - BP and urate (if gout) levels should ideally be reviewed every month until at target
    - Lipids may also be reviewed monthly as 90% of effects of statin doses are evident within 2 weeks
    - Although it takes 3 months for the full treatment effects on HbA1c, if glucose levels are significantly elevated 1 month after an intervention, then escalation of treatment is almost certainly required

- People with preclinical obesity should be assessed at least every 5 years from 15 years of age to determine whether they have developed clinical obesity i.e. CKM disease or non-CKM sequelae
    - Preclinical obesity is defined as excess adiposity without any other features of CKM disease. Excess adiposity is defined as:
        - BMI > 40 kg/m^2 OR
        - BMI > ethnicity-specific threshold AND one increased anthropometric criteria OR

        - > 35 kg/m^2 in Pacific peoples
        - > 32 kg/m^2 in Māori
        - > 30 kg/m^2 in Europeans
        - > 25 kg/m^2 in Asian Indians
    - Two increased anthropometric criteria regardless of BMI
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        - Waist:hip ratio > 0.86 in women and > 1 in men
        - Body fat percentage by DEXA or bioimpedance > 30% for men and > 42% for women

- Non-CKM manifestations of clinical obesity include:
    - Joint pain and osteoarthritis
    - Reduced age-adjusted mobility
    - Lymphoedema
    - Raised intracranial hypertension
    - Obstructive sleep apnoea

- Assessments should be increased to at least 3 yearly if at least one other risk factor for CKM disease is present:
    - Māori, Pacific, Asian Indian and other non-European ethnicities
    - Socioeconomic deprivation
    - Direct family history of CKM at < 40 years of age
    - Smoker
    - Post transplant

        - History of preeclampsia or gestational diabetes
        - Long term glucocorticoid and/or antipsychotic use
        - Chronic dental and/or peridontal disease
        - Clinical features of insulin resistance e.g. acanthosis nigricans, PCOS etc.

    - **NB: In clinical obesity interventions for weight loss are first line treatment to reverse (if able), prevent, delay and slow progression of complications**

↑ Back to contents

4. Lifestyle management + interventions for weight loss

- Lifestyle changes are the cornerstone of management of CKM disease at all times, but need to be tailored to the individual. In particular, lifestyle changes should not delay urate lowering therapy in gout.
- There are 6 key areas of lifestyle management in CKM disease:
    - Education and support
    - General care
    - Healthy eating
    - Interventions for weight loss
    - Physical activity
    - Healthy sleep
- Education and support
    - Adequate education and support are essential in empowering all individuals and whānau to self-manage their CKM disease and achieve best outcomes.
    - Utilise all members of the multidisciplinary team available to optimise care as required. In addition to the GP and nursing team, many practices now have access within the practice, PHO or community to team members including:
        - Kaiāwhina or health navigator
        - Pharmacist
        - Dietitian
        - Health coach

        - Health improvement practitioner
        - Psychologist
        - Social worker
        - Podiatrist
        - Comprehensive Primary Care Team (CPCT)

    - Utilise locally available relevant courses and programmes e.g. Green Prescription, Diabetes Self-Management Education (DSME) etc.
    - Strongly consider referral to culturally appropriate services e.g. Kaupapa Māori Services or Pacific Health providers if available
    - Strongly consider referral for psychology input if significant distress from CKM disease and/or if depression is present
    - Provide information on community support groups as applicable e.g. Stroke Aotearoa, Heart Foundation, Kidney Health New Zealand/Kidney Society and Diabetes NZ etc.

- General care
    - Smoking cessation remains critical and should be offered yearly if smoking
    - There is increasing evidence that vaping may increase the risk of CV disease, lung disease and heart failure. Consequently, vaping should likely only be seen as an interim measure for stopping smoking
    - Discuss reduction of alcohol intake and other recreational drugs as required
        - There is no safe alcohol limit in CV health.
        - Risk of all recreational drugs including marijuana and methamphetamine are much greater in people with CKM disease
        - Reduction in kava intake is likely important for weight loss
    - Ensure vaccination status is up to date given greater adverse effects of communicable diseases in people with CKM disease
    - Ensure cancer screening as per national recommendations is p to date given greater risk of solid cancers in CKM diseases
    - Screen for depression and treat as required as high risk of depression in CKM diseases
        - PHQ-2 should be part of annual CKM assessment
            - Scores ≥ 3 should prompt further screening with PHQ-9 or other tools

- Screen for diabetes distress if known diabetes
    - DDS2 should be part of annual CKM assessment if known diabetes
        - Scores ≥ 3 on DDS2 highlights need to fully evaluate diabetes distress and consider support as appropriate
- Contraception and pregnancy advice should be discussed in women of childbearing age
    - Risks of pregnancy and contraception will be affected by the stage and degree of CKM disease, but the risks of pregnancy are almost always greater than the risks of contraception.
- Optimise treatment of non-CKM diseases that increase CV risk – these include but are not limited to:
    - Dental and periodontal disease
    - Mental Health Disorders e.g. depression and anxiety
    - Chronic inflammatory conditions e.g. rheumatoid arthritis, SLE, inflammatory bowel disease, psoriasis, HIV/AIDS etc.
    - Respiratory and sleep disorders e.g. COPD , asthma and OSA
    - Endocrine disorders e.g. PCOS, thyroid disease, hypogonadism etc.

- Healthy eating
    - Healthy eating is essential for all people with CKM disease, irrespective of body weight. General principles include:
        - Consider that the unequal distribution of poverty and obesogenic environments means Māori and Pacific populations are exposed to more risk factors for the development of CKM disease
        - Strongly consider referral to a dietitian if available
        - **Reduce processed foods as much as possible** as these are often high in sodium, sugars and fat, and low in fibre
        - Low salt intake - < 2 g of sodium or < 5 g of salt per day
            - Advise not to add salt to food and avoid processed foods
        - Reduction of excess carbohydrates particularly sugary drinks and confectionery
            - Low glycaemic index carbohydrates preferred
        - Reduced animal-sourced saturated and trans fats

        - Aim for at least 5 servings of fruit and vegetables per day and to increase plant-based intake
            - Bananas, potatoes, tomatoes and leafy greens useful sources of increasing potassium intake, which is important in reducing BP + CV risk
        - Maintain protein intake – fish, poultry and nuts useful sources and limit red meat
            - Healthy plate model is useful to demonstrate portion sizes
        - Ensuring at least 30 g of fibre per day – whole grains useful source
        - If history of gout:
            - Whilst the intent is not to make food the central point of discussion, reducing alcohol and high sugar food and drinks may be appropriate, including avoiding foods that have previously triggered gout flares (if any). Advising against traditional kai e.g. kaimoana (seafood) is inappropriate and once urate levels are to target, previous dietary triggers are often well tolerated.
            - Ensure adequate fluid intake (e.g. 2 litres of water per day) if no concerns of fluid overload. Eating regular meals is also likely helpful as flares can be triggered by both fasting and overeating.

- Interventions for weight loss
    - A personalised weight loss plan with evidence-based advice is strongly recommended for all people with CKM disease with excess adiposity. Key targets for weight loss include:
        - Excess adiposity is defined as:
            - BMI > 40 kg/m^2 OR
            - BMI > ethnicity-specific threshold AND one increased anthropometric criteria OR
            - > 35 kg/m^2 in Pacific peoples
            - > 32 kg/m^2 in Māori
            - > 30 kg/m^2 in Europeans
            - > 25 kg/m^2 in Asian Indians
            - Two increased anthropometric criteria regardless of BMI
            - Anthropometric criteria include:

        - Waist:height ratio > 0.5
        - Increased waist circumference
            - > 88 cm in females and > 102 cm in males of non-Asian Indian ethnicity
            - Waist circumference > 80 cm in females and > 90 cm in males of Asian Indian ethnicity
        - Waist:hip ratio > 0.86 in women and > 1 in men
        - Body fat percentage by DEXA or bioimpedance > 30% for men and > 42% for women

- 5% total body weight loss significantly improves the majority of metabolic parameters including glucose levels, blood pressure, and lipid studies. This may allow for a reduction in medications, but typically, greater weight loss is required for remission of CKM disease.
- At least 10-15% total body weight loss is typically required to achieve remission of:
    - Type 2 diabetes
    - OSA
    - Hypertension
- At least 15-20% reduction in total body weight loss is required to achieve remission of:
    - MASLD
    - Heart failure with preserved ejection fraction
- **NB:** It is crucial that conversations about weight loss are conducted in a positive, culturally safe and non-judgmental manner. The focus should be on weight loss for health reasons with ‘medical targets’ rather than societal targets or ‘ideal weight’.
    - Any weight loss is beneficial and will have a positive ‘legacy effect’ lifelong
    - Supportive care is essential because sustained weight loss can be difficult as obesity is typically a lifelong remitting and relapsing disease. Body weight is vigorously defended by increased appetite and reduced mitochondrial energy expenditure through no fault of the patient. Supportive care includes:
        - Whānau-based strategies which are likely more effective
        - Psychology input if any concerns of disordered eating or depression

        - Dietitian input to guide nutritional plan. If dietitian is not available then health coach or health improvement practitioner is a useful alternative
        - Enquiring about food security and utilising social worker, kaiāwhina and health navigator input
        - Referring to local weight loss programmes if suitable and available
        - Considering pharmacotherapy and bariatric surgery for weight loss if possible

- There is currently no conclusive evidence that any nutritional strategy is superior to any other. **Pragmatically the best nutritional strategy for weight loss is the one that works, is nutritionally complete and is sustainable. Current evidence suggests best strategy to achieve long-term weight loss and reductions in CKM disease are:**
    - Very low energy diet with meal replacement e.g. DiRECT style intervention
    - Mediterranean diet
    - Dietary approach to stop hypertension (DASH) diet
        - Modified Mediterranean diet with low salt
    - Plant-based diets
    - Other strategies shown to be safe and effective in the short-term include:
        - Very low carbohydrate or ketogenic diets
        - Intermittent fasting
        - Low glycaemic index diets
        - Commercial weight loss programmes
    - **NB: Choice of strategy is often determined by personal preference, cultural acceptability, tolerability, affordability and nutritional adequacy**
        - **Ensure adequate nutrition in children, pregnancy, breast feeding and in the elderly or anyone at risk of sarcopenia**
            - Remember being mildly overweight is protective if elderly
            - Adequate protein intake and physical activity is important in maintaining muscle mass
            - **Utilise dietitian resources if possible**

- Although nutritional strategies are the cornerstone of management of weight loss, **pharmacotherapy to aid weight loss should be considered if BMI > 27 kg/m^2 with at least one obesity comorbidity and unable to achieve weight loss targets despite not being funded.** Options include:
    - Phenetermine (Duromine) ± topiramate
        - Cheapest agent at ~ $80/month
        - Sympathetic side effects may limit use particularly if CV disease
            - Do not use phentermine if CV disease, arrhythmias, untreated hypertension or thyrotoxicosis, substance abuse, pregnancy, breastfeeding or children
            - Phentermine 15 mg daily appears best dose as similar efficacy with less adverse effects than higher doses
            - NB: Common misconceptions are that phentermine is addictive and can only be used for up to 3 months, which are untrue.
            - Topiramate for weight loss is off-label in NZ but consider if cost an issue
            - Topiramate is typically started at 25-50 mg daily and increased up to doses of 100 mg daily for weight loss. Beware of teratogenic effects of topiramate.
            - Topiramate may also be useful if history of migraines or epilepsy
    - Orlistat (Xenical)
        - Cost is ~ $120/month but not used commonly due to GI adverse effects
            - Best to use orlistat with a low fat diet with doses of 120 mg with main meals
    - Buproprion ± naltrexone (Contrave in combination)
        - Cost of Contrave ~ $250/month
            - Contrave tablets contain 8 mg of naltrexone and 90 mg of bupropion
            - Typically start at 1 tablet per day and increase by 1 tablet per week up to 2 tablets twice daily or maximal tolerated dose.

        - Slow down dose increases if GI adverse effects e.g. nausea
        - GI adverse effects typically dissipate within 2-3 weeks
    - Do not use in pregnancy, breastfeeding, children, uncontrolled hypertension, history of seizures, bipolar disorder, MAOI use or withdrawal of alcohol or benzodiazepines etc.
- Bupropion alone for weight loss is off-label in NZ but can consider if cost an issue. Bupropion ± naltrexone useful if low mood or to help smoking cessation

- GLP1 receptor agonists (GLP1Ra)
    - Likely most effective pharmacological for weight loss. All current GLP1Ra in NZ are subcutaneous injections.
        - Ensure prescribe with 4 or 5 mm BD fine insulin needles
    - Aim to use funded liraglutide (Victoza) or dulaglutide (Trulicity) if possible if type 2 diabetes as expensive to self-fund (currently > $480/month)
    - Liraglutide (Saxenda) was principal GLP1Ra used for weight loss in NZ but Semaglutide (Wegovy) and Tirzepatide (Mounjaro) now available (unfunded), which typically lead to greater weight loss and CV protection than liraglutide.
    - Doses and tips to avoid adverse effects can be found here.
        - Liraglutide (Saxenda) can started at 0.6 mg daily and unlike in diabetes, liraglutide can be titrated to 3 mg daily or maximal tolerated dose
        - Semaglutide (Wegovy) and Tirzepatide (Mounjaro) are now available and typically lead to greater weight loss and CV and renal protection than older GLP1Ra but are not funded
        - Semaglutide can be started at 0.25 mg weekly and increased slowly to 2.4 mg weekly or maximal tolerated dose
        - Tirzepatide can be started at 2.5 mg weekly and increased slowly to 15 mg weekly or maximal tolerated dose
        - Tips should be provided for all GLP1Ra on how to reduce adverse effects:
            - Ensure adequate hydration and stop eating when feeling full

        - Eat smaller meals and avoid alcohol, fatty and spicy foods
        - Slow down dose increases if GI adverse effects
            - GI adverse effects typically dissipate within 2-3 weeks
        - Doses of sulfonylureas may need to be reduced by 50% and doses of insulin by approximately 20% to avoid hypoglycaemia when starting GLP1Ra:
            - Typically only required if baseline HbA1c < 64 mmol/mol
        - Do not use in pregnancy, breastfeeding , children < 10 years of age, significant GI disease or medullary thyroid cancer
        - GLP1Ra should be stopped once eGFR < 15 mL/min

- Metformin may be used ‘off-label’ for weight loss but typically only leads to a maximum average of 2 kg weight loss. Empagliflozin is not recommended for weight loss unless treating underlying type 2 diabetes, renal disease or heart failure.
- Typical approach with pharmacotherapy is to treat for 3 months to determine if ‘responder’, which is defined as ≥ 5% total body weight loss in time period.
    - If ‘non-responder’ → consider different pharmacotherapy for weight loss
    - If ‘responder’ → aim to continue treatment until weight loss plateaus. Can then consider:
        - Dose reduction or cessation but weight regain is common.
        - Continuing current dose to ensure weight stability
            - All pharmacotherapy above safe for at least 3 years
        - Switching to or adding in alternative pharmacotherapy agent
            - Do not use phentermine and bupropion in combination

- Consider bariatric surgery if failure to reach weight loss targets on all other interventions
    - Access to funded bariatric surgery is difficult, and regional variations in indications. But consider if:

        - BMI > 40 kg/m^2 or BMI > 35 kg/m^2 with other features of CKM disease

    - Roux en Y bypass and sleeve gastrectomy appear best procedures for weight loss
        - Appropriate patient selection continues to be critical

- Physical activity
    - Any increase in physical activity is beneficial but current recommendations for non-pregnant adults are:
        - 150 minutes of moderate to high intensity aerobic exercise per week spread over ≥ 3 days each week and no more than 2 consecutive days without exercise
        - At least 2 sessions of resistance exercise at low to moderate intensity per week
        - Sitting for < 30 minutes at a time
        - **NB:** The intensity and duration of the exercise may need to be reduced due to comorbidities such as heart disease and previous stroke etc. Ensure safety including adequate footwear if increased foot risk e.g. peripheral vascular disease. Using body weight for resistance exercise can still be effective.

    - These recommendations are not realistic for all people with CKM disease, but support should be provided to increase physical activity and movement as much as possible given the significant benefits:
        - Physical activity and movement can take on many forms rather than ‘exercise’. For example, housework, gardening, dance, walking around shops, taking the stairs and mowing the lawns etc. are effective and sometimes forgotten forms of physical activity.
        - A 5 – 6 minute brisk walk a day is associated with an additional 4 years of life
        - Adding 500 steps per day is associated with up to 10% reductions in mortality
        - Moving briskly doing everyday activity is associated with up to 50% reductions in CVD
        - Stretching reduces blood pressure and glucose levels

- Healthy sleep
    - Sleep disorders are common in people with CKM disease and are associated with weight gain, high glucose levels, high blood pressure, arrhythmias and cardiovascular disease.
        - Obstructive sleep apnoea (OSA) is the most common sleep disorder in CKM syndrome. Treatment of OSA significantly improves metabolic outcomes and reduces CV events.

- Optimal length of sleep for beneficial effects on body weight and CKM disease appears to be 6 – 8 hours every day. Unfortunately ‘catch-up’ sleep does not fully reverse the deleterious effects of insufficient sleep → beware of the risk of CKM disease in shift workers.
- Strongly consider discussing healthy sleep and sleep hygiene, and screening for OSA and other sleeping disorders when appropriate for all people with CKM disease.

↑ Back to contents

5. Management of elevated blood pressure and hypertension

- Treatment of high blood pressure (BP) is likely the most easily modifiable risk factor in preventing CV and renal sequelae in CKM disease so BP should be measured at every practical opportunity
- An individualised approach is recommended taking into account competing risks and preferences and explored through shared decision making
- **Recommended thresholds for treatment of BP depend whether other CKM disease features are present:**
  - **BP ≥ 140/90 mmHg → prompt confirmation and treat with lifestyle advice + pharmacotherapy in all if appropriate**
    - Ideally elevated BP should be confirmed as soon as possible on validated home or ambulatory BP monitoring. Home monitoring is best done in a structured approach e.g. morning and evening BP measurements at rest for 1 week. If home or ambulatory BP monitoring not available then recommend measurement of BP in clinic with the following:
      - Measure BP after 5 mins seated comfortably in a quiet environment with a validated device with an appropriate cuff size with the cuff at the level of the heart.
      - Measure BP 3 times 1-2 mins apart and average the last 2 readings
      - Often useful to measure BP at both arms and to assess whether postural drop at 1st visit to aid monitoring at future visits.
  - **BP 130 – 139/80 - 89 mmHg → prompt confirmation and treat with lifestyle advice + pharmacotherapy if 5 year CV risk ≥ 10% OR ANY of the following:**
    - Ideally elevated BP should be confirmed as soon as possible on validated home or ambulatory BP monitoring. Home monitoring is best done in a structured approach e.g. morning and evening BP measurements at rest for 1 week. If home or ambulatory BP monitoring not available then recommend measurement of BP in clinic with the following:

        - Measure BP after 5 mins seated comfortably in a quiet environment with a validated device with an appropriate cuff size with the cuff at the level of the heart.
        - Measure BP 3 times 1-2 mins apart and average the last 2 readings
        - Often useful to measure BP at both arms and to assess whether postural drop at 1st visit to aid monitoring at future visits.

    - CVD including ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, atrial fibrillation and heart failure
    - Diabetes with any microvascular or macrovascular complication
    - Familial hypercholesterolaemia
    - Chronic kidney disease (eGFR <60mL/min and/or UACR >3 mg/mmol)
    - NB: Gout, MASLD and OSA all independently increase CV risk and events, but are not currently included in the PREDICT CV risk calculator. Therefore, CV risk is likely underestimated in these conditions and treatment of BP 130-139/80-89 mmHg should still be considered in these conditions if no other indications, particularly if 5 year CV risk 4-5%.

  - **BP 130 - 138/80 - 89 mmHg and 5 year CV risk 5 - < 10% → lifestyle advice + treat underlying condition + consider pharmacotherapy IF ANY of the following:**
    - Gout or auto-immune inflammatory disease
    - MASLD
    - OSA
    - Severe mental illness particularly with antipsychotic use
    - Previous gestational diabetes and/or preeclampsia
    - Direct family history of CVD < 40 years of age
    - Cardiac calcium score ≥ 100

  - **BP 130 - 138/80 - 89 mmHg and 5 year CV risk < 5% → lifestyle advice alone**

- **Target systolic BP is 120 – 129 mmHg in most individuals**
  - A systolic BP < 120 mmHg is not concerning if well tolerated and likely preferable if young or heart failure
  - **Relaxed BP targets to the lowest reasonably and safely achievable BP are appropriate if any of the following:**

        - Frailty and/or limited life expectancy
        - Age ≥ 85 years
        - Symptomatic postural hypotension
            - Measure lying/standing or sitting/standing BP if high risk of postural hypotension and treat to standing BP if > 10 mmHg postural drop.
        - Intolerant of antihypertensives
    - Unlike other features of CKM, treating BP has no ‘metabolic memory or legacy effect’. Therefore, BP targets and treatment should only be relaxed when clinically appropriate
    - Diastolic BP targets are now no longer first-line BP targets due to evidence for the greater benefit of controlling systolic BP. Although it is desirable to have a diastolic BP < 80 mmHg, a systolic BP 120 – 129 mmHg is the primary target.

- Treatment consists of both non-pharmacological and pharmacological management and should ideally be based on several ‘out of office’ or home BP monitoring at rest as BP levels in clinic are typically ≥ 5 mmHg higher. Home monitoring is best done in a structured approach e.g. morning and evening BP measurements at rest for 1 week.
    - If out of office BP monitoring not available then measure BP after 5 mins seated comfortably in a quiet environment with a validated device with an appropriate cuff size with the cuff at the level of the heart. Ideally BP should be measured 3 times 1-2 mins apart with the average of the last 2 readings used.

- Non-pharmacological management is always important and is discussed in detail in lifestyle management. Key points include:
    - Smoking and alcohol cessation
        - Increasing evidence that vaping cessation is also beneficial
    - A low-salt diet with fruit and leafy vegetables to ensure adequate potassium intake
    - Increased physical activity and movement
    - Aiming for > 5% total body weight loss if overweight

- First line pharmacotherapy for lowering BP are ACE inhibitors (ACEi) or angiotensin receptor blockers (ARB), calcium channel blockers (CCB) and thiazide diuretics (TD). Typically, these medications are more effective and better tolerated in low doses in combination, but may be used alone. Cardioselective beta blockers (β-blocker) may be used if ischaemic heart disease, heart failure or atrial fibrillation. Guidance on likely best agents in each to use based on efficacy and tolerance is below.
- Best choice of ACEi, ARB, CCB or TD is dependent on whether renal disease predominates and presence of other comorbidities:

- Chronic kidney disease is present (eGFR <60 mLmin and/or UACR > 3 mg/mmol):
    - Strongly consider ACEi or ARB if no hypotension and check BP in 1 month
    - Titrate ACEi or ARB to maximal tolerated dose
    - Check BP monthly until to target – if BP remains above target add CCB or TD
    - If BP still above target then add other (e.g. TD if CCB previously added)

- No renal or CV disease:
    - Strongly consider low dose ACEi or ARB and CCB in combination
    - Check BP in 1 month → if above target increase dose of combination agents
    - Check BP monthly until to target - if BP remains above target then add TD

- Gout:
    - Consider ACEi or ARB as first-line options. (Note that losartan and dihydropyridine CCBs may serve as helpful adjuncts due to their small uricosuric effect, but they should not replace urate-lowering therapy and the focus should be on the most effective anti-hypertensive)
    - Beware TD typically increase uric acid levels

- Ischaemic heart disease, heart failure or atrial fibrillation:
    - β-blockers may be added at any stage. May need to decrease CCB and/or TD if hypotension
    - In heart failure Entresto (Sacubitril/Valsartan) is preferred renin-angiotensin system inhibitor (rather than ACEi or ARB) but need to meet special authority criteria for funding. Use spironolactone or eplerenone and empagliflozin rather than TD or CCB if risk of hypotension.
        - Current special authority criteria for Entresto requires the following:
            - NHYA/WHO functional II-IV AND
            - LVEF ≤ 35% OR echocardiogram is not reasonably practical AND
            - Other standard treatments of heart failure are optimised
        - Maximise doses of Entresto, β-blocker and spironolactone before addition of other agents.
            - Remember to monitor potassium when using Entresto or spironolactone as per ACEi/ARB below

- Causes of secondary hypertension should be considered if young and/or persistent hypertension despite adherence to ≥ 3 antihypertensive agents
    - Obstructive sleep apnoea is likely the most common cause of secondary hypertension in CKM and is often missed
    - Consider other investigations as appropriate to exclude renal disease, pregnancy, and endocrine conditions such as primary hyperaldosteronism, Cushing's syndrome, thyrotoxicosis and acromegaly.
    - Consider referral to secondary care to discuss addition of other antihypertensives as required
        - Spironolactone likely preferable if heart failure
            - Caution advised if low eGFR and/or baseline hyperkalaemia
        - Alpha blockers likely preferable if prostatic disease

- Likely preferred agents in each of blood pressure lowering medications available in Aotearoa NZ:
    - ACEi and ARB:
        - Ramipril typically preferred ACEi but perindopril useful alternative. Quinapril also useful alternative if combination with TD desirable but often requires twice daily dosing.
            - Usual dose range of ramipril is 2.5 mg – 10 mg daily but may start 1.25 mg daily if elderly and/or risk of hypotension
            - Usual dose range of perindopril is 2 mg - 8 mg daily
            - Usual dose range of quinapril is 2.5 mg – 20 mg once or twice daily. Combination with hydrochlorothiazide available at 10 mg and 20 mg doses of quinapril.
        - Candesartan typically preferred ARB but losartan useful alternative if combination with TD desirable.
            - Usual dose range of candesartan is 8 mg – 32 mg daily but may start at 4 mg daily if elderly and/or risk of hypotension
            - Usual dose range of losartan is 50 mg – 100 mg daily but may start at 12.5 mg - 25 mg daily if elderly and/or risk of hypotension. Combination with hydrochlorothiazide available at 50 mg losartan dose.
        - Check creatinine + electrolytes 2-4 weeks after starting or dose change to ensure no hyperkalaemia or deterioration of renal function
            - K+ < 5.5 mmol/L and up to 30% decrease in creatinine requires no dose change

        - Hyperkalaemia is often spurious due to haemolysis during the delay from collection until analysis in the lab - consider repeating to ensure real.

    - CCB:
        - Amlodipine and felodipine preferred with usual dose range for both 2.5 mg – 5 mg daily
        - 10 mg daily doses associated with greater risk of adverse effects for often little benefit
        - Useful if elevated diastolic BP as vasodilators
    - TD:
        - Chlorthalidone is typically preferred TD but bendroflumethazide useful alternative.
        - Usual dose of chlorthalidone: is 12.5 mg – 25 mg daily and usual dose of bendroflumethazide is 2.5 mg - 5 mg daily. Higher doses typically lead to little additional BP lowering but greatly increase adverse effects.
        - Hydrochlorothiazide is a weak BP lowering agent but may be useful if combination tablets preferable
        - Check eGFR + electrolytes 1-2 weeks after starting to ensure no adverse effects
    - β-blockers – Metoprolol controlled release (CR) or bisoprolol preferred for ischaemic heart disease and rate control
        - Carvedilol likely preferable if congestive heart failure

- ***The guidance in this section is predominantly based on the 2024 European Society of Cardiology Guidelines for the management of elevated blood pressure and hypertension, which can be accessed at www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Elevated-Blood-Pressure-and-Hypertension***

↑ Back to contents

6. Management of dyslipidaemia

- Lipid-lowering therapy should be started if high CV risk (5 year CV risk ≥ 10%) or any of the following irrespective of CV risk with a target LDL cholesterol (LDLc) < 1.4 mmol/L:
    - Previous CV event
    - Established CV disease including known asymptomatic coronary and carotid disease

    - Type 2 diabetes with any microvascular complication e.g. diabetic eye, chronic kidney disease (eGFR < 60 mL/min and/or UACR > 3 mg/mmol)
    - UACR ≥ 30 mg/mmol
    - eGFR < 45 mL/min
    - UACR 3 – 29 mg/mmol and eGFR 45 – 59 mL/min
    - Age > 50 years and UACR > 3 mg/mmol and/or eGFR < 60 mL/min
    - Familial hypercholesterolaemia

- For others lipid-lowering therapy is strongly recommended if moderate CV risk (5 year CV risk 5 -<10%) with a target LDLc < 1.8 mmol/L, particularly if any significant risk factors.
    - Direct family history of CVD < 40 years of age
    - Onset of CKM disease at < 40 years of age
    - Severe mental illness particularly with antipsychotic use
    - Cardiac calcium score ≥ 100
    - Gout
    - MASLD

- Lipid lowering therapy should be still considered if low CV risk (5 year CV risk < 5%) if any risk factors, particularly if 5 year CV risk ≥ 3% and/or LDLc > 4 mmol/L, but is largely driven by patient preference. Statins are generally not recommended if low CV risk without any risk factors.
    - Direct family history of CVD < 40 years of age
    - Onset of CKM disease at < 40 years of age
    - Severe mental illness particularly with antipsychotic use
    - Cardiac calcium score ≥ 100
    - Gout
    - MASLD
    - Previous gestational diabetes and/or preeclampsia

- Lipid lowering therapies
    - Lifestyle management is always important in managing dyslipidaemia, particularly hypertriglyceraemia
        - Even 5% total body weight loss will significantly improve dyslipidaemia if overweight

- Atorvastatin and rosuvastatin are first line pharmacotherapy to achieve LDLc targets in CKM:
    - Rosuvastatin is preferred if previous CV event but mismatch with special authority criteria
        - Current special authority requires previous failures of atorvastatin or simvastatin to reach LDLc target unless Māori or Pacific ethnicity
        - Maximise rosuvastatin use in Māori and Pacific peoples given funded and their high CV risk
        - Other ethnicities may choose to self-fund rosuvastatin without trialling atorvastatin as cost is $12 per week
    - Usual dose range is atorvastatin is 10 – 80 mg daily and rosuvastatin 5 – 40 mg daily
        - At least 40 mg atorvastatin or 10 mg rosuvastatin daily is typically required to achieve a 50% reduction in LDLc
        - Consider starting rosuvastatin 5 mg daily if South East Asian ancestry due to a common genetic polymorphism that increases rosuvastatin levels
        - Recent studies show statins have low and potentially no teratogenicity - so statins should now be used in women of child-bearing age without fear. At present, advice is to still stop statins in pregnancy and breastfeeding, but discuss with secondary care if established cardiovascular disease and/or familial hypercholestraemia as it may be safest to continue statin use.
    - Measure LDLc at least 3 monthly and titrate statin to reach LDLc target or maximal tolerated dose
        - 90% of LDLc lowering evident within 2 weeks so do not need to wait 3 months before titrating
        - Repeat testing may be barrier to optimising treatment so consider starting statin at or rapidly titrating statin to estimated dose to reach LDLc target

Mean % reduction in LDLc for statin dose					
34%	41%	48%	55%	60%	
Atorvastatin	10 mg	20 mg	40 mg	80mg	-
Rosuvastatin	-	5 mg	10 mg	20 mg	40 mg

        - Switch atorvastatin to rosuvastatin if LDLc is above target on 80 mg daily or maximal tolerated dose
        - Maximum dose of rosuvastatin is 10 mg daily once eGFR < 30 mL/min
    - Consider trialling pravastatin if intolerant of low dose atorvastatin and rosuvastatin
        - Statins are typically well tolerated with most adverse effects only minor
        - Many reported adverse effects of statins may be due to the nocebo effect, particularly fatigue and muscle aches. Indeed, statin-induced myopathy predominantly affects the shoulder and hip girdle rather than generalised muscle pain. Rhabdomyolysis and hepatotoxicity are extremely rare.
        - Only 50% of reported mild adverse effects and/or mild derangement of liver enzymes recur on re-trialling the same statin or a different statin. Statins are also useful agents in people with MASLD.
        - Therefore, it is important to determine if true statin intolerance if mild to moderate symptoms due to the significant benefits of statins.
        - The benefits of statins almost always outweigh the rare deleterious effects on hyperglycaemia in people with CKM and should not prevent treatment.
    - Consider ezetimibe 10 mg daily if LDLc above target despite maximal tolerated dose of statin
        - NB: Ezetimibe no longer requires special authority approval
    - Consider alirocumab or inclisiran if LDLc still above target but require SC injection (2-4 weekly or 6 monthly), are not funded and are expensive. They will reduce LDL on average by a further 50% from baseline.
        - Consider bezafibrate if persistently elevated triglycerides (TGs) and LDLc still above target
        - Target TG < 1.7 mmol/L if previous CV event
        - Target TG < 5.7 mmol/L if no previous CV event

- ***The guidance in this section is predominantly based on the European Society of Cardiology Guidelines on Dyslipidaemias (Management of), which can be accessed at www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Dyslipidaemias-Management-of#***

↑ Back to contents

7. Management of hyperglycaemia in type 2 diabetes

- Management of type 2 diabetes is now focussed on preventing, delaying and reducing the progression of CV and renal disease and aiding weight loss if overweight rather than just lowering glucose levels. As a result,

there are new key treatment concepts:

- Lifestyle management and metformin are recommended for all with type 2 diabetes regardless of HbA1c
    - 10-15% total body weight loss (TBWL) is typically required to achieve remission of type 2 diabetes if increased fat mass, but 5% TWBL will significantly improve glucose levels

- If renal disease (UACR > 3 mg/mmol OR eGFR < 60 mL/min), heart failure, CVD OR equivalent risk (5 year CV risk ≥ 10%) add empagliflozin OR a GLP1 receptor agonist (GLP1Ra) regardless of HbA1c
    - There is a mismatch between best practice and the special authority criteria for empagliflozin and GLP1Ra, which states the patient must have heart failure (empagliflozin) or an HbA1c > 53 mmol/mol (both empagliflozin and GLP1Ra) if no heart failure. Self-funding of these agents should be offered but are expensive (approximately $85 per month for empagliflozin and minimum $450 per month for GLP1Ra). Tips to access increase access include:
        - Using the heart failure special authority for empagliflozin if applicable
        - Funding the GLP1Ra under special authority if dual therapy due to the much greater cost
        - Utilising the disability allowance to cover the cost of empagliflozin if able
        - Prescribing half the 25 mg tablet of empagliflozin or 1 tablet of empagliflozin 12.5 mg with metformin (Jardiamet) to halve the cost to approximately $43 per month – please note this is off-label.
        - Checking the cost between pharmacies because there continues to be wide variation
    - Empagliflozin is typically preferred if heart failure or renal disease predominate
    - GLP1Ra may be preferred if significant reduction in HbA1c and/or weight desired
    - Dual empagliflozin/GLP1Ra therapy preferred if HbA1c remains above target on either agent alone. Tips for allowing funded dual therapy include:
        - Using the heart failure special authority for empagliflozin if applicable
        - Funding the GLP1Ra under special authority if dual therapy due to the much greater cost
        - Utilising the disability allowance to cover the cost of empagliflozin if able
        - Prescribing half the 25 mg tablet of empagliflozin or 1 tablet of empagliflozin 12.5 mg with metformin (Jardiamet) to halve the cost to approximately $43 per month – please note this is off-label.
        - Checking the cost between pharmacies because there continues to be wide variation
    - If HbA1c still above target then adding pioglitazone should be considered before other glucose lowering therapies.

- If no renal or CV disease and 5 year CV risk < 10% then treatment is added (not switched) if HbA1c is above target:
    - If weight loss desired → empagliflozin and/or GLP1Ra preferred. Consider acarbose if HbA1c still above target.
    - If weight loss not desired → consider vildagliptin (typically weight neutral and redundant if on GLP1Ra) and pioglitazone (may cause minimal weight gain)
- Sulfonylureas and insulin are still important treatment options but are often used last due to their risk of hypoglycaemia and weight gain (risk is much less for sulfonylureas).
- Target HbA1c for most is < 53 mmol/mol
    - Target HbA1c < 48 mmol/mol preferred in young adults and pre-pregnancy
    - Target HbA1c 55 – 70 mmol/mol may be suitable if high risk of hypoglycaemia or tight glycaemia is not required e.g. life expectancy limited by other conditions
    - **NB:** Targets should always be balanced against risk of hypoglycaemia, **but only insulin and sulphonylureas can cause significant hypoglycaemia**

- Comprehensive guidance on all aspects of the management of type 2 diabetes can be found here. Relevant links to specific areas of the guidance are included in the key points below on starting glucose lowering therapies in CKM:
    - Lifestyle management is important at all stages of type 2 diabetes and CKM disease
    - Metformin is often best tolerated if started at 250 mg – 500 mg with largest meal
        - Titrate metformin to 1 g twice daily or maximal tolerated dose
        - Metformin in combination tablets (e.g. Jardiamet, Galvumet) seems to be better tolerated than metformin alone
        - Doses of metformin need to be reduced once eGFR < 45 mL/min
            - eGFR 30 – 44 mL/min – maximum metformin dose is 1 g daily
            - eGFR – 15 – 29 mL/min – maximum metformin dose is 500 mg daily
            - eGFR < 15 mL/min – stop metformin
    - Empagliflozin is typically started at 10 mg daily alone (Jardiance) or in combination with metformin (Jardiamet)
        - Can increase to 25 mg daily if HbA1c remains above target

- Glucose-lowering effects of empagliflozin reduce once eGFR < 30 mL/min but CV and renal protection persist
- Empagliflozin can be started if eGFR > 20 mL/min but should be stopped if adverse effects occur or dialysis is started.
    - Sick day advice and tips to reduce adverse effects should be provided for all:
        - Withhold empagliflozin in acute illness and 3 days before (including day of) major surgery, bowel prep or low carb diet. Restart when well and eating and drinking normal.
        - Doses of sulfonylureas may need to be reduced by 50% and doses of insulin by approximately 20% to avoid hypoglycaemia when starting empagliflozin – typically only required if baseline HbA1c < 64 mmol/mol.
        - Discuss importance of genital hygiene and reporting changes or concerns
        - Do not use in pregnancy, breastfeeding or children < 10 years of age
        - Do not use in type 1 diabetes, significant alcohol intake, previous diabetic ketoacidosis (DKA) or low carbohydrate diets without specialist advice
        - If symptoms of DKA (e.g. nausea, vomiting, abdominal pain etc.) need to present to GP practice or A+E urgently to ensure blood ketones are < 1.5 mmol/L. DKA needs to be excluded if ketones > 1.5 mmol/L.

◦ Liraglutide is the only currently funded GLP1Ra for new starts

- Start 0.6 mg daily and titrate to 1.8 mg daily or maximal tolerated dose
- Dulaglutide (Trulicity) is still funded if previously funded under special authority
    - Typical dose is 1.5 mg weekly but may be increased to 3 mg and then 4.5 mg weekly if HbA1c remains above target
    - Semaglutide (Wegovy) and Tirzepatide (Mounjaro) will shortly be available and typically lead to greater weight loss and CV protection than older GLP1Ra but are not funded
        - Semaglutide can be started at 0.25 mg weekly and increased slowly to 2.4 mg weekly or maximal tolerated dose
        - Tirzepatide can be started at 2.5 mg weekly and increased slowly to 15 mg weekly or maximal tolerated dose
    - Tips should be provided on how to reduce adverse effects:

        - Ensure adequate hydration and stop eating when feeling full
        - Eat smaller meals and avoid alcohol, fatty and spicy foods
        - Slow down dose increases if GI adverse effects
            - GI adverse effects typically dissipate within 2-3 weeks
        - Doses of sulfonylureas may need to be reduced by 50% and doses of insulin by approximately 20% to avoid hypoglycaemia when starting GLPRa – typically only required if baseline HbA1c < 64 mmol/mol
        - Do not use in pregnancy, breastfeeding or children < 10 years of age
        - GLP1Ra should be stopped once eGFR < 15 mL/min

    - Pioglitazone is started at 15 mg daily if no contraindications
        - Pioglitazone should not be used if history of bladder cancer, high risk of fractures (e.g. osteoporosis) or oedematous conditions (e.g. uncontrolled heart failure and actively treated macular oedema etc.)
        - It may take 16 weeks before the full effects on HbA1c are seen, but pioglitazone may be titrated up to 45 mg as required

- ***The guidance in this section is based on the National Type 2 Diabetes Management Guidance that was developed by the New Zealand Society for the Study of Diabetes (NZSSD) and the Ministry of Health, which can be accessed at www.t2dm.nzssd.org.nz. Our national diabetes guidance is largely based on the joint American Diabetes Association (ADA)/European Association for the Study of Diabetes (EASD) guidelines, which can be accessed at https://link.springer.com/article/10.1007/s00125-022-05787-2.***

↑ Back to contents

8. Management of chronic kidney disease

- Detailed guidance on the investigation and management of CKD can be found here. This link is useful to help determine the cause of albuminuria and/or renal impairment, which is critical for best management. The following guidance is a brief synopsis on the management of albuminuria and renal impairment relevant to CKM disease.
- Chronic kidney disease is defined as an eGFR <60mL/min and/or a urinary albumin:creatinine ratio (UACR) >3 mg/mmol for more than 3 months. Albuminuria, defined as a UACR > 3 mg/mmol, indicates endothelial inflammation. Renal impairment, defined as an eGFR < 60 mL/min, indicates renal damage or scarring. Both are independent risk factors for CV disease irrespective of each other or diabetes.
- Best management of CKD includes:

- **Lifestyle management is always important with an aim for weight loss if overweight**
- **Start ACEi or ARB if blood pressure > 130/80 mmHg OR if any of the following are present if no concerns over hypotension:**
    - UACR > 3 mg/mmol and eGFR > 15 mL/min
    - Diabetes mellitus (any type)
    - Heart failure
- Provide advice on sick day management and titrate ACEi/ARB to maximal tolerated dose
    - Check BP monthly until to target – if BP remains above target add calcium channel blocker (CCB) or thiazide diuretic (TD)
    - If BP still above target then add other (e.g. TD if CCB previously added)
    - ACEi, ARB, CCB and TD use and potential use of spironolactone is discussed in detail in management of elevated blood pressure
- Commence empagliflozin 10 mg daily if any of the following:
    - UACR > 20 mg/mmol and eGFR > 20 mL/min
    - Type 2 diabetes with UACR > 3 mg/mmol and/or eGFR 20 – 60 mL/min
    - Heart failure if eGFR > 20 mL/min at any level of albuminuria
    - eGFR 20 – 44 mL/min at any level of albuminuria
    - **NB:** Only funded at present under special authority if heart failure and/or type 2 diabetes. Although expensive at $85 per month, self-funding should be offered. Tips to enhance access include:
        - Utilising the disability allowance to cover the cost of empagliflozin if able
        - Prescribing half the 25 mg tablet of empagliflozin or 1 tablet of empagliflozin 12.5 mg with metformin (Jardiamet) if type 2 diabetes to halve the cost to approximately $43 per month – please note this is off-label.
        - Checking the cost between pharmacies because there continues to be wide variation
    - Can increase to 25 mg daily if type 2 diabetes and HbA1c remains above target
        - Glucose-lowering effects of empagliflozin reduce once eGFR < 30 mL/min
    - Empagliflozin should only be stopped if adverse effects occur or dialysis is started.

- Sick day advice and tips to reduce adverse effects should be provided for all:
    - Withhold empagliflozin in acute illness and 3 days before (including day of) major surgery, bowel prep or low carb diet. Restart when well and eating and drinking normal.
    - Doses of sulfonylureas may need to be reduced by 50% and doses of inulin by approximately 20% to avoid hypoglycaemia when starting empagliflozin – typically only required when baseline HbA1c < 64 mmol/mol.
    - Discuss importance of genital hygiene
    - Do not use in pregnancy, breastfeeding or children < 10 years of age
    - Do not use in type 1 diabetes, significant alcohol intake, previous diabetic ketoacidosis (DKA) or low carbohydrate diets without specialist advice
    - If symptoms of DKA (e.g. nausea, vomiting, abdominal pain etc.) need to present to GP practice or A+E urgently to ensure blood ketones are < 1.5 mmol/L. DKA needs to be excluded if ketones > 1.5 mmol/L.

- Start lipid lowering therapy if high CV risk (5 year CV risk ≥ 10%) OR if any of the following are present irrespective of calculated CV risk aiming for LDL cholesterol < 1.4 mmol/L:
    - Previous CV event and/or established CV disease
    - Asymptomatic coronary or carotid disease
    - Any type of diabetes with microvascular or macrovascular complication(s)
    - UACR ≥ 30 mg/mmol
    - eGFR < 45 mL/min
    - UACR 3 – 29 mg/mmol and eGFR 45 – 59 mL/min
    - Age > 50 years and UACR > 3 mg/mmol and/or eGFR < 60 mL/min
    - Familial hypercholestraemia

- For others, lipid lowering therapy is strongly recommended if moderate CV risk (5 year CV risk 5-<10%) aiming for LDL cholesterol < 1.8 mmol/L, particularly if any significant risk factors:
    - Direct family history of CVD at < 40 years of age
    - Onset of cardiokidney metabolic disease < 40 years of age
    - Severe mental illness particularly with antipsychotic use
    - Cardiac calcium score ≥ 100

        - Gout and/or autoimmune disease
        - Previous gestational diabetes and/or preeclampsia
        - Metabolic dysfunction-associated steatotic liver disase (MASLD; previously termed fatty liver disease)

    - Lipid lowering therapy should be still considered if low CV risk (5 year CV risk < 5%) if any risk factors, particularly if 5 year CV risk ≥ 3% and/or LDLc > 4 mmol/L, but is largely driven by patient preference.
        - Direct family history of CVD < 40 years of age
        - Onset of CKM disease at < 40 years of age
        - Severe mental illness particularly with antipsychotic use
        - Cardiac calcium score ≥ 100
        - Gout and/or autoimmune inflammatory disease
        - MASLD
        - Previous gestational diabetes and/or preeclampsia

- GLP1 receptor agonist (GLP1Ra) therapy should also be strongly considered if CKM present and type 2 diabetes with an HbA1c above if weight loss is desirable
    - Please click here for more information on GLP1Ra use in diabetes
    - Please click here for more information on GLP1Ra use for weight loss

- Aspirin is important for secondary prevention of CV events in renal disease. However, the risks of aspirin typically outweigh the benefits for primary prevention in people with significant renal disease

- ***The guidance in this section is largely based on the Kidney Disease / Improving Global Outcomes (KDIGO) 2024 Guidelines, which can be accessed at www.kdigo.org/guidelines/ckd-evaluation-and-management/***

↑ Back to contents

9. Management of gout

- Gout is common in people with CKM disease and is an independent risk factor for CV and renal disease. Effective treatment of gout is important in preventing gout flares and reducing CV and renal sequelae.
- Detailed guidance on managing gout can be found here including ensuring the correct diagnosis and management of acute gout flares. The guidance below is a brief synopsis of the management of gout

relevant to CKM disease:

- Start urate lowering pharmacotherapy if any of the following:
  - Recurrent gout flares – especially ≥ 2 flares per year
  - Gouty tophi or chronic gouty arthritis
  - Evidence of joint damage due to gout
  - Urate lowering pharmacotherapy should also be considered if ANY of the following:
    - ≥ 1 episode of gout with onset of gout at a young age or strong family history of gout
    - ≥ 1 episode of gout and serum urate > 0.6 mmol/L
    - ≥ 1 episode of gout and impending significant weight loss e.g. bariatric surgery, total diet replacement strategies etc.

- Allopurinol is first line agent aiming for serum urate levels < 0.36 mmol/L or < 0.3 mmol/L if tophi.
  - Initial dosing ONLY is based on renal function:
    - Initial dosing ONLY is based on renal function:
      - eGFR > 60 mL/min – 100 mg allopurinol daily
      - eGFR 30 – 60 mL/min – 50 mg allopurinol daily
      - eGFR < 30 mL/min – 50 mg allopurinol on alternate days
  - Consider HLA-B*5801 screening in high-risk Asian populations before starting allopurinol to reduce risk of severe hypersensitivity reactions
    - All people of Chinese or Thai descent
    - All Korean people with eGFR < 60 mL/min
  - Ask people to stop allopurinol and to contact the practice if they develop a skin rash
  - Increase dose of allopurinol by 50 – 100 mg every 4 weeks until urate to target
  - Typically recommended cover colchicine when starting allopurinol to prevent gout flares.
    - Consider reducing dose in renal impairment and beware of drug to drug interactions

        - Discuss adverse effects, particularly GI adverse effects and to stop if significant effects occur
        - Ensure safe storage and to keep out of the reach of others as overdose can be fatal as no reversal agent
            - Consider requesting child-proof packaging on prescription if appropriate
        - Continue colchicine for 3-6 months after target serum urate is achieved
    - Once at target, measure serum urate 6 - 12 monthly to ensure still to target
    - Reassure that once target urate reached it may take > 12 months for gout flares to stop and years for tophi to dissolve
- Consider probenecid or febuxostat if intolerant of allopurinol or failure of allopurinol despite ensuring adherence. More information on probenecid and febuxostat can be found here
- Gout is almost always caused by genetic variants and is typically more severe with a younger age of onset in Māori and Pacific Peoples. Recommended lifestyle interventions may be helpful, particularly if dietary triggers, but should not delay starting urate lowering pharmacotherapy or dominate the consultation.
    - Weight loss if overweight
    - Drinking 2 litres of water per day if no concerns over fluid overload
    - Avoiding excess alcohol
    - Reducing high sugar food and drinks
    - Eating regular meals as gout can be triggered by both fasting and overeating
    - Avoid foods that have previously triggered their gout flares (if any).
        - Once urate levels are to target, previous dietary triggers are often well tolerated.
- Acute flares of gout may still occur. If so:
    - Prednisone or colchicine are typically preferred in CKM disease with normal precautions
    - Consider back pocket prescription in case a further gout flare occur
    - Ensure serum urate to target but beware that urate levels may be normal in 50% of acute flares
- People with gout should have their cardiovascular risk factors managed aggressively. If other features of CKM then assessment of cardiovascular risk factors should be performed at least annually to ensure all to target

- **NB:** There continues to be no conclusive evidence supporting urate lowering treatment of asymptomatic hyperuricaemia
- ***The guidance in this section is aligned with the new evidence-based new national Health Pathway for gout***

↑ Back to contents

10. Calculating CV risk and antiplatelet therapy in CKM disease

- The 'traditional cardiovascular risk assessment (CVRA)' no longer applies to people with CKM disease. **People with CKM disease should have their 5 year CV risk calculated using the PREDICT CV risk calculator at diagnosis of CKM disease and then as part of their annual CKM assessments**
- 5 year CV risk is now characterised as:
    - **Low CV risk (5 year CV risk < 5%)**
    - **Moderate CV risk (5 year CV risk 5 – <10%)**
    - **High CV risk (5 year CV risk ≥ 10%) OR any of the following** irrespective of calculated CV risk:
        - Previous CV event
        - Established CV disease including known asymptomatic coronary and carotid disease
        - Type 2 diabetes with any microvascular complication e.g. diabetic eye, chronic kidney disease (eGFR < 60 mL/min and/or UACR > 3 mg/mmol)
        - UACR ≥ 30 mg/mmol
        - eGFR < 45 mL/min
        - UACR 3 – 29 mg/mmol and eGFR 45 – 59 mL/min
        - Age > 50 years and UACR > 3 mg/mmol and/or eGFR < 60 mL/min
        - Familial hypercholesterolaemia
- Management of CKM disease should always be optimised, which may be independent of CV risk. The main role of calculating CV risk in CKM disease is to determine whether treatment is recommended for:
    - Antihypertensives if BP 130 – 139/80 - 89 mmHg in the absence of CV or renal disease, or complications of diabetes
    - Antiplatelet therapy for primary prevention if no diabetes or chronic renal disease
    - Lipid lowering therapy if not high CV risk
    - SGLT2i and/or GLP1Ra in type 2 diabetes if no established renal or CV disease or heart failure

- **CV risk calculations should not influence any other treatment decisions**
    - **High CV risk includes 5 year CV risk ≥ 10% OR any of the following** irrespective of calculated CV risk:
    - Previous CV event
    - Established CV disease including known asymptomatic coronary and carotid disease
    - Type 2 diabetes with any microvascular complication e.g. diabetic eye, chronic kidney disease (eGFR < 60 mL/min and/or UACR > 3 mg/mmol)
    - UACR ≥ 30 mg/mmol
    - eGFR < 45 mL/min
    - UACR 3 – 29 mg/mmol and eGFR 45 – 59 mL/min
    - Age > 50 years and UACR > 3 mg/mmol and/or eGFR < 60 mL/min
    - Familial hypercholesterolaemia

- Additional management and timing of next CV risk calculation based on current CV risk:
    - High CV risk:
        - Start lipid-lowering therapy with a target LDL cholesterol (LDLc) < 1.4 mmol/L
        - Start blood-pressure lowering therapy if BP ≥ 130/80 mmHg
        - Start SGLT2i and/or GLP1Ra if type 2 diabetes regardless of HbA1c
        - Start aspirin 75–150 mg daily for primary prevention if < 70 years of age and benefits appear to outweigh risks:
            - NB: The risks of aspirin in primary prevention likely now outweigh the benefits in people with diabetes, significant renal or liver disease, or significant bleeding risk
            - Tools to help informed individualised decision making include aspirin benefit harm calculators
            - Aspirin and other antiplatelet agents such as clopidogrel and ticragrelor remain important for secondary prevention of CV events for all
        - Onceified as high-risk there is no need to re-calculate CV risk as treatment should always be optimised as per high-risk unless contraindications

- Moderate CV risk:

- **Strongly consider lipid lowering therapy aiming for LDL cholesterol < 1.8 mmol/L, particularly if any significant risk factors:**
    - Direct family history of CVD at < 40 years of age
    - Onset of cardiokidney metabolic disease < 40 years of age
    - Severe mental illness particularly with antipsychotic use
    - Cardiac calcium score ≥ 100
    - Gout and/or autoimmune inflammatory disease
    - Previous gestational diabetes and/or preeclampsia
    - Metabolic dysfunction-associated steatotic liver disase (MASLD; previously termed fatty liver disease)
- Start blood pressure lowering therapy if BP ≥ 140/90 mmHg
- Strongly consider blood pressure lowering therapy and treating underlying condition if BP 130 - 139/80 - 89 mmHg AND ANY of:
    - Gout or auto-immune inflammatory disease
    - MASLD
    - OSA
    - Severe mental illness particularly with antipsychotic use
    - Previous gestational diabetes and/or preeclampsia
    - Direct family history of CVD < 40 years of age
    - Cardiac calcium score ≥ 100
- Start SGLT2i and/or GLP1Ra if type 2 diabetes regardless of HbA1c if any renal impairment or heart failure OR if HbA1c above target despite lifestyle management and metformin and weight loss desirable
- Risks of aspirin for primary prevention typically outweigh the benefits at moderate CV risk
- Recalculate CV risk at next annual CKM assessment
    - May be relaxed to 2 yearly if gout or metabolic liver disease alone

- Low CV risk:
    - Consider lipid lowering therapy if 5 year CV risk ≥ 3% and any significant risk factors are present with a target LDL cholesterol (LDLc) < 1.8 mmol/L, but is largely driven by patient preference.

        - Direct family history of CVD at < 40 years of age
        - Onset of cardiokidney metabolic disease at a young age
        - Severe mental illness particularly with antipsychotic use
        - Cardiac calcium score ≥ 100
        - Gout and/or autoimmune inflammatory disease
        - Metabolic dysfunction-associated steatotic liver disease (previously termed fatty liver disease)
        - Previous gestational diabetes or preeclampsia
    - Start blood-pressure lowering therapy if BP ≥ 140/90 mmHg
    - Start SGLT2i and/or GLP1Ra if type 2 diabetes regardless of HbA1c if any renal impairment or heart failure OR if HbA1c above target despite lifestyle management and metformin and weight loss desirable
    - Aspirin is not recommended for primary prevention in low CV risk
    - Recalculate CV risk in 5 years

↑ Back to contents

11. Screening for CKM disease in the general population

- Optimise opportunistic screening for CKM disease wherever possible
    - Diagnosis of one CKM disease should prompt at least annual screening for other CKM diseases
        - CKM diseases include:
            - Elevated blood pressure
            - Type 2 diabetes
            - Dyslipidaemia
            - CV disease including ischaemic heart disease, cerebrovascular disease, peripheral arterial disease, atrial fibrillation and heart failure
            - Chronic kidney disease

        - Metabolic dysfunction-associated steatotic liver disease (MASLD; previously termed fatty liver disease)
        - Gout
        - Obstructive sleep apnoea

    - Identify and manage CKM disease as early as possible because many people with CKM syndrome have contact with the health system with related presentations (e.g. recurrent skin infections) or incidental findings (e.g. obesity at immunisations) years before management of their CKM disease.

- Otherwise screening for CKM disease in the general population is either at assessments for people with preclinical obesity or via the cardiovascular risk assessment (CVRA). CVRA should now be started at the following ages:
    - Men with any risk factors* - 30 years of age
    - Men without risk factors* – 40 years of age
    - Women with any risk factors* – 40 years of age
    - Women without risk factors* – 50 years of age
    - *Risk factors include:
        - Māori, Pacific, South-East Asian and other non-European ethnicities
        - Socioeconomic deprivation
        - Direct family history of CKM at < 40 years of age
        - Smoker
        - Post transplant
        - History of preeclampsia or gestational diabetes
        - Long term glucocorticoid and/or antipsychotic use
        - Chronic dental and/or peridontal disease
        - Clinical features of insulin resistance e.g. acanthosis nigricans, PCOS etc.

- Screening should include:
    - Seated blood pressure to screen for high blood pressure
    - HbA1c +/- fasting glucose to screen for diabetes

        - Combining fasting glucose with HbA1c prevents the need for another confirmatory test to diagnose diabetes if the HbA1c is > 48 mmol/mol. Fasting glucose is also the preferred diagnostic test if measurement of HbA1c may be unreliable such as:
            - Any haemoglobinopathy e,g, thalassaemia, sickle cell anaemia etc.
            - Altered red cell turnover e.g. bleeding, haemolysis, severe iron deficiency
            - Second and third trimesters of pregnancy
            - Post blood transfusion
    - eGFR and Urinary ACR to screen chronic kidney disease
    - Waist circumference and BMI to screen for increased fat mass
    - Non fasting lipid studies to screen for dyslipidaemia
    - Smoking status

- If any CKM disease is found on screening then optimise treatment of all CKM disease. CVRA is no longer required as CV risk will be calculated as part of their annual CKM assessments
    - Low CV risk (5 year CV risk < 5%) → 5 yearly
    - Moderate CV risk (5 year CV risk 5 – <10%) → yearly
        - May be relaxed to 2 yearly if gout or MASLD alone
    - High CV risk (5 year CV risk ≥ 10%) → no need to repeat CV risk calculation as need to optimise treatment

- If no overt CKM disease is found on CVRA then treatment and follow up is based on 5 year CV risk
    - High CV risk (5 year CV ≥ 10%):
        - Start lipid-lowering therapy aiming for LDL cholesterol < 1.4 mmol/L
        - Strongly consider aspirin 75-150 mg daily if < 70 years and risks appear to outweigh benefits
            - Aspirin benefit harm calculators may aid decision making
        - Review and optimise CV risk factors at least annually. No need to repeat ‘traditional CVRA’
    - Moderate CV risk (5 year CV risk 5 -< 10%):

        - Strongly consider lipid-lowering therapy particularly if direct family history of CKM disease < 40 years of age OR personal history of severe mental illness with antipsychotic use aiming for LDL cholesterol < 1.8 mmol/L
        - Repeat CVRA in 2 years
    - Low CV risk (5 year CV risk < 5%) → repeat CVRA in 5 years

- ***The guidance in this section is predominantly based on the 2018 NZ Cardiovascular Disease Risk Assessment and Management for Primary Care, which can be accessed at www.tewhatuora.govt.nz/publications/cardiovascular-disease-risk-assessment-and-management-for-primary-care, and the 2023 Australian guideline for assessing and managing cardiovascular risk, which can be accessed at www.mja.com.au/journal/2024/220/9/2023-australian-guideline-assessing-and-managing-cardiovascular-disease-risk.***

↑ Back to contents

Abbreviations:

A+E
Accident and Emergency (Also known ER or ED)

ACE
Angiotensin-Converting Enzyme

ACEi
Angiotensin Converting Enzyme Inhibitors

ACR
Albumin:Creatinine Ratio

ARB
Angiotensin Receptor Blocker

β-Blocker
Beta Blocker

BMI
Body Mass Index

BP
Blood Pressure

CCB
Calcium Channel Blocker

CKD
Chronic Kidney Disease

CKM
Cardiovascular-Kidney-Metabolic

CPCT
Comprehensive Primary Care Team

CV
Cardiovascular

CVD
Cardiovascular Disease

CVRA
Cardiovascular Risk Assessment

DASH
Dietary Approaches to Stop Hypertension

DDS2
2-Item Diabetes Distress Screening

DKA
Diabetic Ketoacidosis

DSME
Diabetes Self-Management Education

eGFR
Estimated Glomerular Filtration Rate

GAD
Glutamic Acid Decarboxylase

GI
Gastrointestinal

GLP1Ra
Glucagon-Like Peptide-1 Receptor Agonists

GP
General Practitioner

HbA1c
Glycated Haemoglobin

LDL
Low-Density Lipoprotein

LDLc
Low-Density Lipoprotein Cholesterol

LVEF
Left Ventricular Ejection Fraction

MASLD
Metabolic dysfunction Associated Steatotic Liver Disease (previously termed fatty liver disease)

NHYA
New York Heart Association

OSA
Obstructive Sleep Apnoea

PCOS
Polycystic Ovarian Syndrome

PHO
Primary Health Organisation

PHQ-2
Patient Health Questionnaire 2

PHQ-9
Patient Health Questionnaire 9

SC
Subcutaneous

SGLT2i
Sodium-Glucose Co-Transporter 2 Inhibitors

SLE
Systemic Lupus Erythematosus

TBWL
Total Body Weight Loss

TD
Thiazide Diuretic

TG
Triglyceride

UACR
Urinary Albumin:Creatinine Ratio

WHO
World Health Organisation

ZnT8
Zinc Transporter 8

↑ Back to contents

↑ Back to top