phn TASMANIA
An Australian Government Initiative

primary health TASMANIA

Medications in type 2 diabetes: Looking beyond HbA1c

This webinar will start soon.

Medications in type 2 diabetes: Looking beyond HbA1c

Zoom webinar – Tuesday 16 June, 2026

Acknowledgement of traditional owners

We acknowledge the Tasmanian Aboriginal people as the traditional owners and ongoing custodians of the land on which we are meeting today. We pay our respects to Elders past and present.

We would also like to acknowledge Aboriginal people who are joining us today.

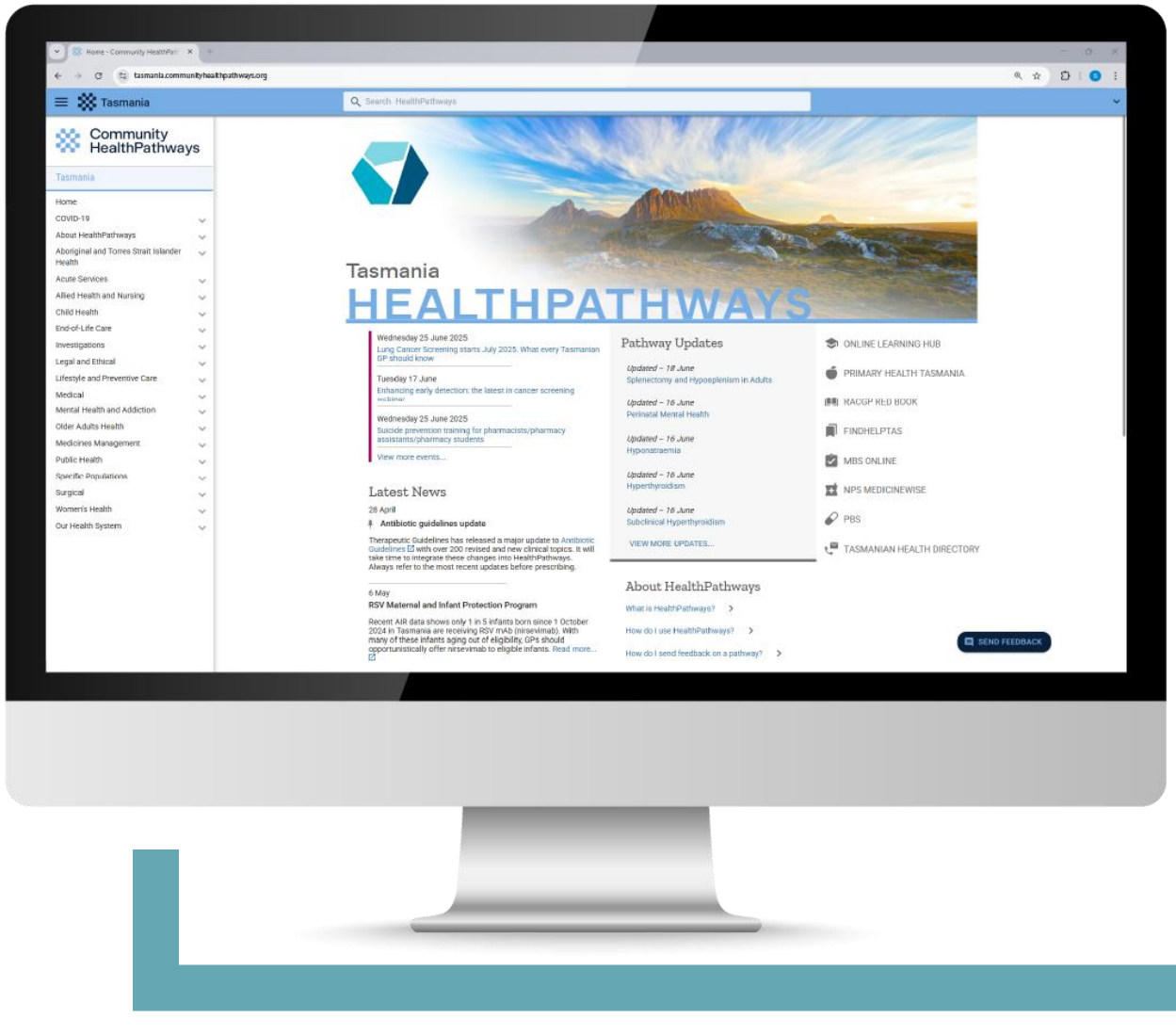
Learning outcomes

After this session, I will be able to:

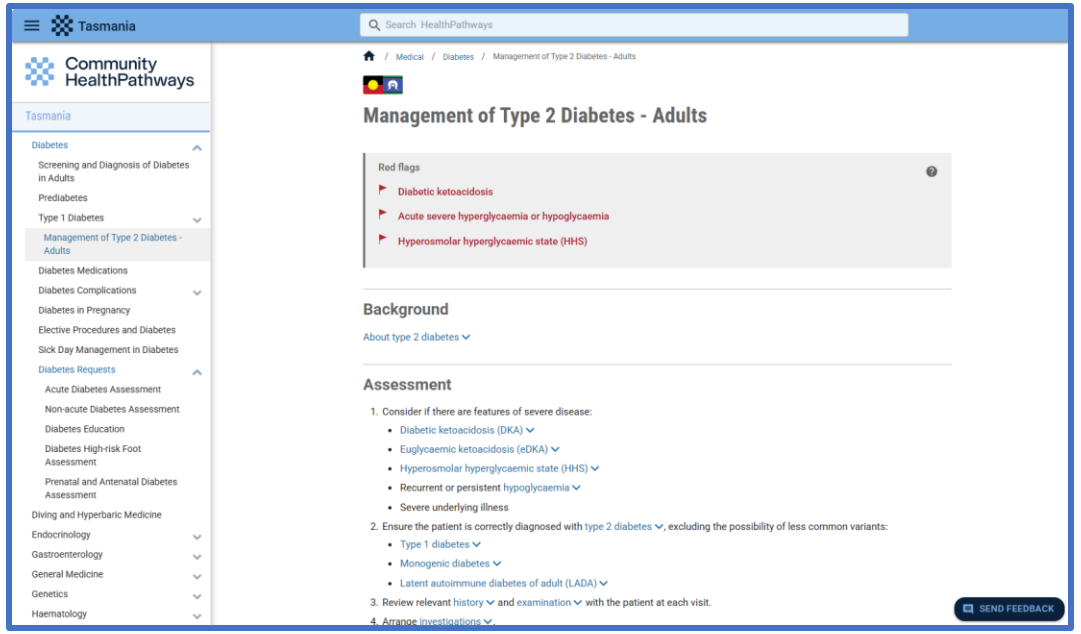
- Describe developments in medications and other treatments as they pertain to diabetes.
- Explain rational use of medications for diabetes to achieve patient goals in accordance with established evidence-based practice.
- Self-reflect on own knowledge and skills in relation to diabetes and act on areas for professional development as identified.

Housekeeping

- Tonight's webinar is being recorded.
- Please use the Zoom Q&A feature to ask questions.
- At the end of the webinar your browser will automatically open a short evaluation survey. We appreciate you taking the time to complete this to help us improve our events program.
- CPD – 1.5 hours Educational Activities (RACGP & ACRRM)
- Please don't forget to register for your next webinar here →



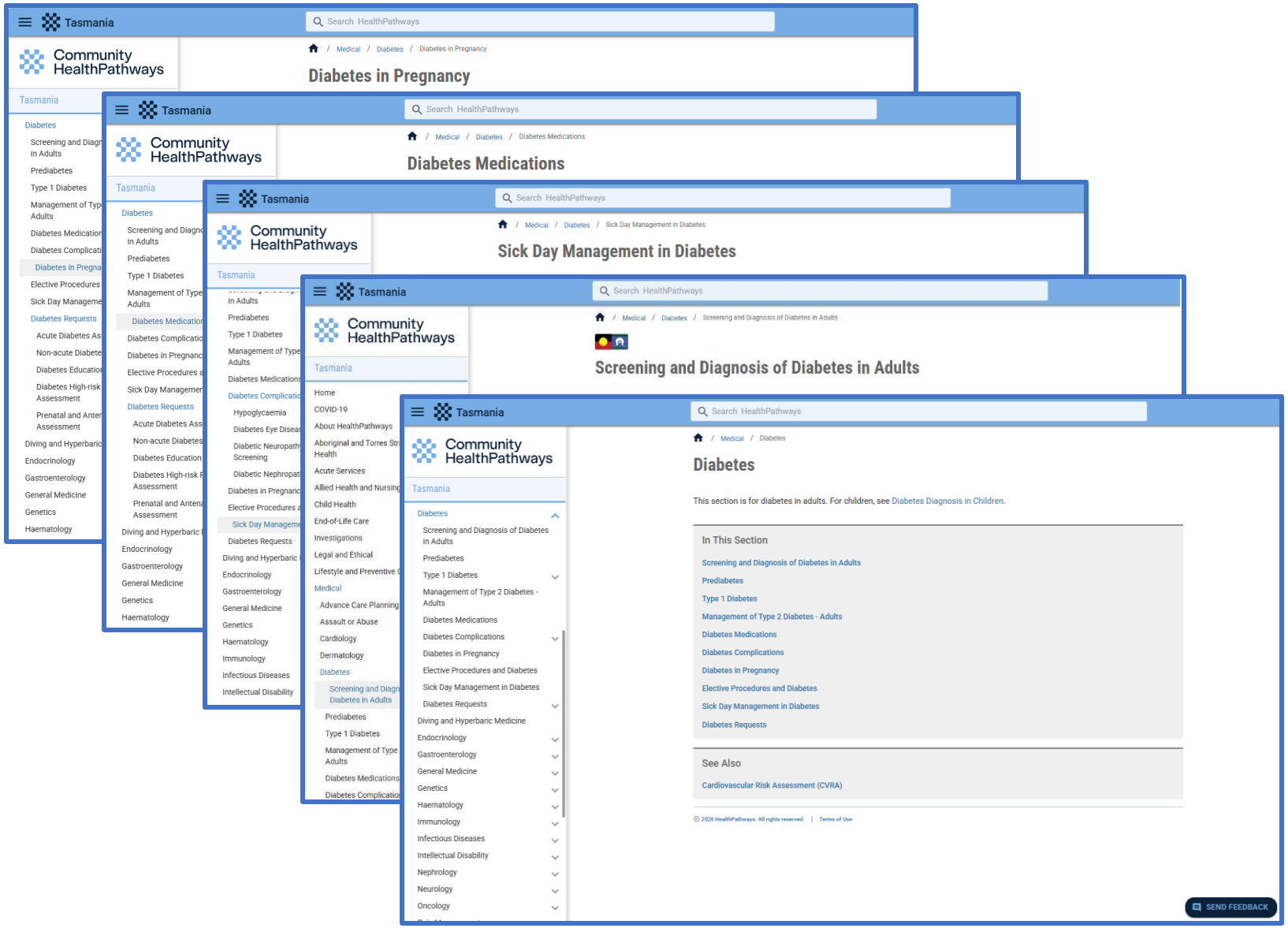
Community HealthPathways



Scan here

To sign in or register, please visit the Tasmania HealthPathways home page.

Diabetes pathways



Presenters

Dr Jonathon Choong

GP Clinical Editor

Primary Health Tasmania

Joanne Gross

Consultant Pharmacist

Tasmanian Health Service

Myles Clarkson Fletcher

Registered Nurse &
Credentialled Diabetes
Educator

Tasmanian Health Service

Liz Kinnersly

Nurse Practitioner &
Credentialled Diabetes
Educator

Diabetes Australia

Case study #1 – Mr AN

- 84 yo male
- PMHx
  - T2DM, diagnosed 2022
  - AF
  - CABG x3 2006
  - HFpEF (EF 40-45% 2023)
  - CKD stage 4
  - PMR 2019
  - Cog imp (MOCA 21/30 2024)
  - Long covid-19 2022
  - Interstitial lung disease
  - Detrusor instability
  - Restless legs
  - AAA repair
  - L spine decompression 2021
  - Bilat THR

Case study #1 – Mr AN

- Medications
  - Bion Tears 0.3%-0.1% eye drops prn
  - Candesartan 4mg d
  - Dutasteride/tamsulosin 500mcg/400mcg d
  - Empagliflozin 10mg d
  - Frusemide 20mg mane/midi
  - Hylo Forte 0.2% Eye drops
  - Melatonin 2mg n
  - Nebivolol 1.25mg n
  - Paracetamol 665mg 2 n
  - Pramipexole 250mcg 0.5 n
  - Prednisolone 4mg d
  - Rivaroxaban 15mg d
  - Rosuvastatin 10mg n

Case study #1 – Mr AN

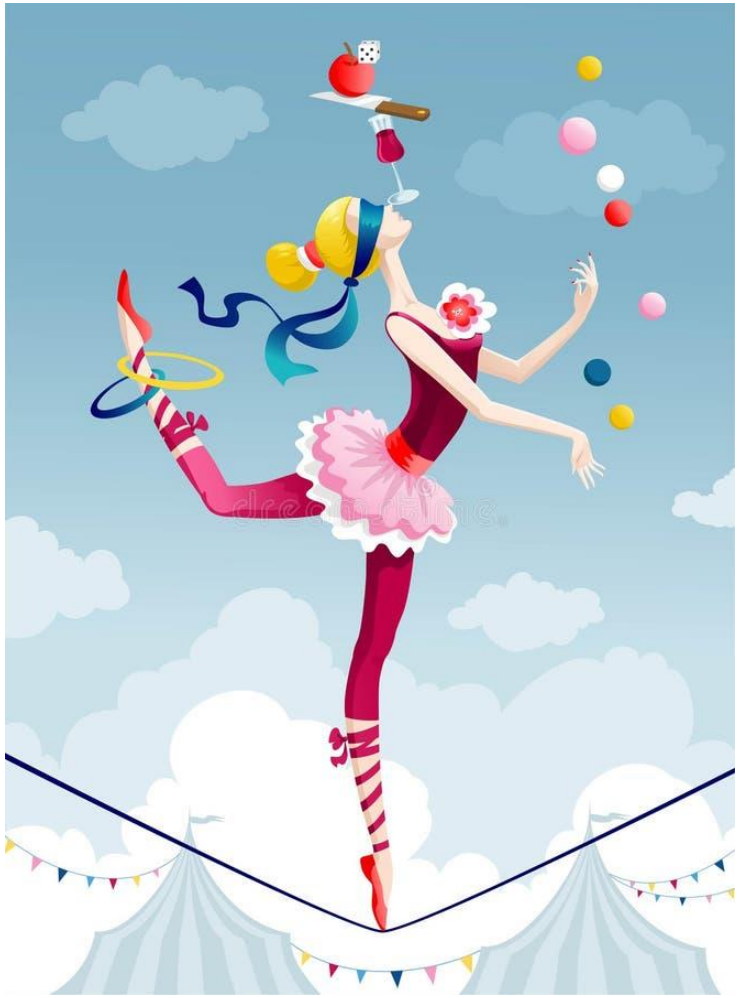
- NKDA
- Non smoker
- No ETOH
- SHx – lives at home with wife, children and grandchildren lives in southern Tasmania

Case study #1 – Mr AN and T2DM

- HMR 2024
  - HbA1c 8.4%, eGFR 37
  - Medications – dapagliflozin 10mg alt days, candesartan 4mg d, rosuvastatin 40mg d
  - Decision made to initiate Metformin XR 1g d
- HMR 2025
  - HbA1c 7.0%, eGFR 25
  - Medications – empagliflozin 10mg d, metformin XR 500mg d, candesartan 16mg d, rosuvastatin 40mg d
  - Metformin stopped during hospital admission, decision made not to restart

Case study #1 – Mr AN other concerns

- Hip and shoulder pain in the context of PMR
- Breathlessness in the context of long covid-19
- Urinary urgency and frequency
- Swollen ankles in the context of heart failure and CKD
- Driver licence in context of cognitive impairment
- Restless legs treatment
- Medication rationalisation
- Hospital admissions – Aug 2025 atypical chest pain, Nov 2025 admitted with pneumonia Cx by AKI, May 2026 admitted post fall in context pre-syncope Cx by AKI



Case study #2 – Ms BP

- 35 yo female, ATSI patient
- PMHx
  - T2DM diagnosed in 2023, GDM 2014
  - Hypertension
  - Dyslipidaemia
  - Hepatitis C infection 2018
  - PTSD
  - Alcohol misuse
  - Current smoker

Case study #2 – Ms BP

- Medications
  - Amitriptyline 50mg 1 tab daily
  - Metformin 500mg mane, 1g nocte
  - Mirena inserted 2024
  - Mirtazapine 45mg 1 tab daily
- Allergies: pethidine
- Current smoker 10-15 cigarettes daily
- ETOH: 5 std drinks daily
- SHx: lives with aunty and her (the patient's) 3 sons

Case study #2 – Ms BP first consultation

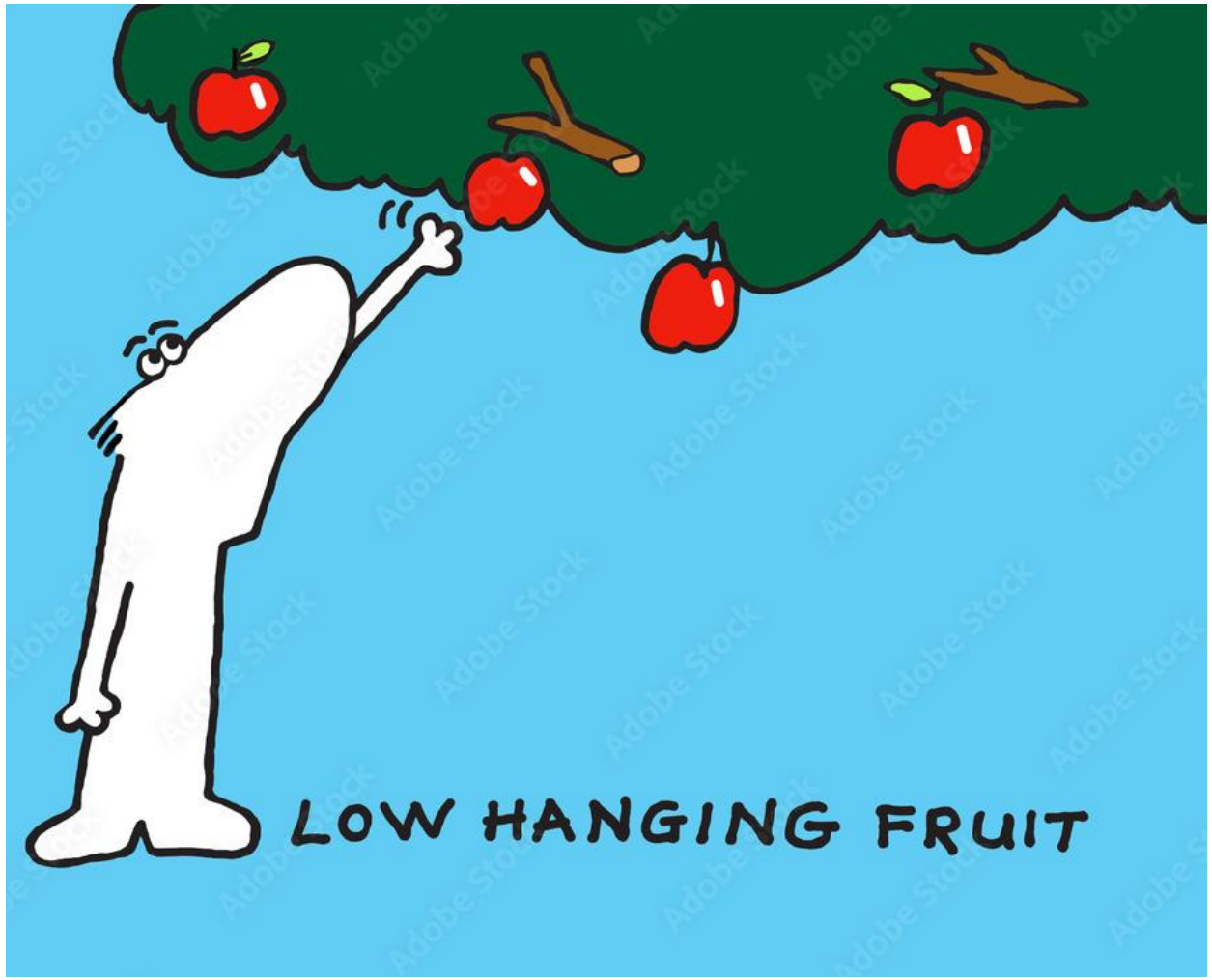
- T2DM
  - ‘Was taking metformin 1g bd but BSLs went down to 3.5 so dose was decrease to 500mg mane/1g nocte’
  - Ran out of medications recently, fingerprick BSLs up to 11
  - Last eye check 10 years ago. Never had podiatry assessment
  - Requests discussion about GLP1 agonist
  - Height 166cm, weight 80.8kg, BMI 29.3
- Hypertension
- Dyslipidaemia

Case study #2 – Ms BP second consultation

- Investigations
  - FBE: normal
  - UEC: Na 138, K 4.7, Cr 79, eGFR 84
  - Fasting glucose: 7.3
  - HbA1c: 5.8%
  - LFT: bili 9, GGT 50, ALT 67, AST 33
  - Lipids: total 6.4, TG 1.7, HDL 1.54, LDL 4.09, total/HDL ratio 4.2
  - Ferritin: 115
  - uACR: 0.5

Case study #2 – Ms BP second consultation

- Metabolic syndrome
  - T2DM
  - Hypertension: home BP systolic 130-153, diastolic 92-103
  - Overweight
  - Dyslipidaemia
- Smoking cessation
- Alcohol use disorder



Therapeutic guidelines overview of T2DM

- Diet, exercise, weight loss
- **Blood glucose lowering medicine** to achieve glycaemic targets
- Minimise risk factors for microvascular and macrovascular complications, especially **CVD prevention**
- Consider **CV and renal benefits** of some blood glucose lowering medicines in patients with atherosclerotic CVD, heart failure or CKD (even if not indicated for glycaemic benefit)
- Practical aspects (alcohol, driving, travel)

MEDICATION MANAGEMENT IN DIABETES

> BGL-LOWERING MEDICINES
> MEDICATION MANAGEMENT REVIEWS (MMRs)
> MEDICATION SAFETY

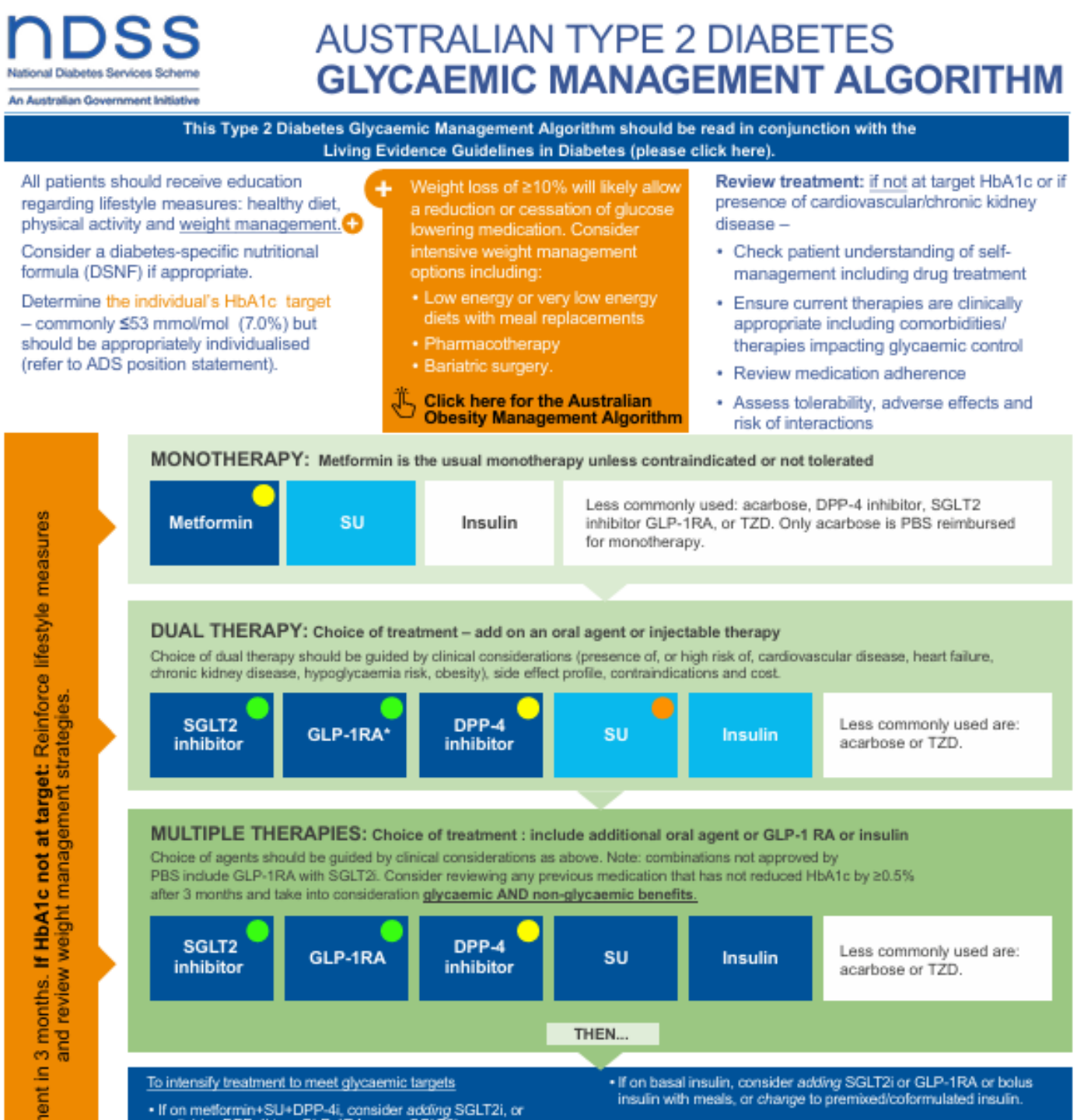
JOANNE GROSS

BPharm(Hons) MPH GCertPharmPrac GCertDMEd MPS CredPharm(MMR)

QUM Pharmacist, Department of Health Tasmania

Independent Consultant Pharmacist (MMRs, Diabetes Education)

TAS Branch President – Pharmaceutical Society of Australia (PSA)



ADS T2DM MANAGEMENT ALGORITHM

- **Metformin** remains first-line medicine
- Early/add-on **SGLT2i** (unless contraindicated or not tolerated)
- Add **GLP1a*** or swap SGLT2i to GLP1a*

  **will depend on PBS criteria or ability to self-fund*
- Ongoing roles for:
  - **Insulin** (as add-on or early)
  - **DPP4i** (with/instead of SGLT2i)
  - SU and others (**acarbose**, **pioglitazone**)

View more detail at Aus living guidelines:

www.diabetessociety.com.au/living-evidence-guidelines-in-diabetes

BLOOD GLUCOSE LOWERING MEDICINES:
CONSIDER ADDITIONAL BENEFITS BEYOND BGLs

	Progression of CKD	ASCVD	Heart failure	Glucose-lowering efficacy	Hypoglycemia risk	Weight effects	Cost	Other key risks / side effects
Metformin	Neutral	Potential benefit	Potential benefit	High	Low	Neutral	Low	GI SEs, metabolic ketoacidosis
SGLT2 inhibitors	Benefit[a]	Benefit[c]	Benefit	Intermediate	Low	Loss	High	Genitourinary infections, euglycaemic ketoacidosis
GLP-1 receptor agonists	Benefit[b]	Benefit[c]	Potential benefit	High	Low	Loss	High	GI SEs, sarcopaenia, mental health
DPP-4 inhibitors	Neutral	Neutral	Potential risk[c] (saxagliptin)	Intermediate	Low	Neutral	High	Pancreatitis, rash
Insulin	Neutral	Neutral	Neutral	Highest	High	Gain	High (analogs) Low (human)	Lipodystrophy
Sulfonylureas	Neutral	Neutral	Neutral	High	High	Gain	Low	
Thiazolidinediones	Neutral	Potential benefit (pioglitazone)	Increased risk	High	Low	Gain	Low	Osteoporosis, bladder cancer
α-Glucosidase inhibitors	Neutral	Neutral	Neutral	Intermediate	Low	Neutral	Low	GI SEs

Neutral

Potential benefit or intermediate glucose-lowering efficacy

Benefit (organ protection, high efficacy, low hypoglycemia risk, weight loss, or low cost)

Potential risk or high cost to patient

Increased risk for adverse effects

From: de Boer I et al. Diabetes management in chronic kidney disease: a consensus report by the American Diabetes Association (ADA) and Kidney Disease: Improving Global Outcomes (KDIGO) 2022 https://www.kidney-international.org/article/S0085-2538(22)00634-2/fulltext

COMBINE AN SGLT2i WITH GLP1a
FOR ADDITIVE CARDIORENAL PROTECTION

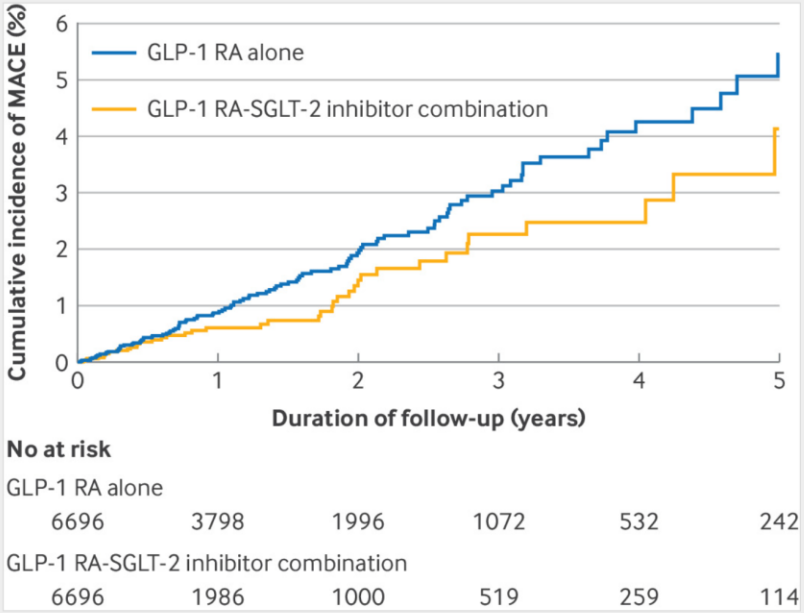


Fig 2
Cumulative incidence curves of major adverse cardiovascular events (MACE) for glucagon-like peptide-1 (GLP-1) receptor agonist (RA)-sodium-glucose cotransporter-2 (SGLT-2) inhibitor combination versus GLP-1 RAs

Download figure | Open in new tab | Download powerpoint

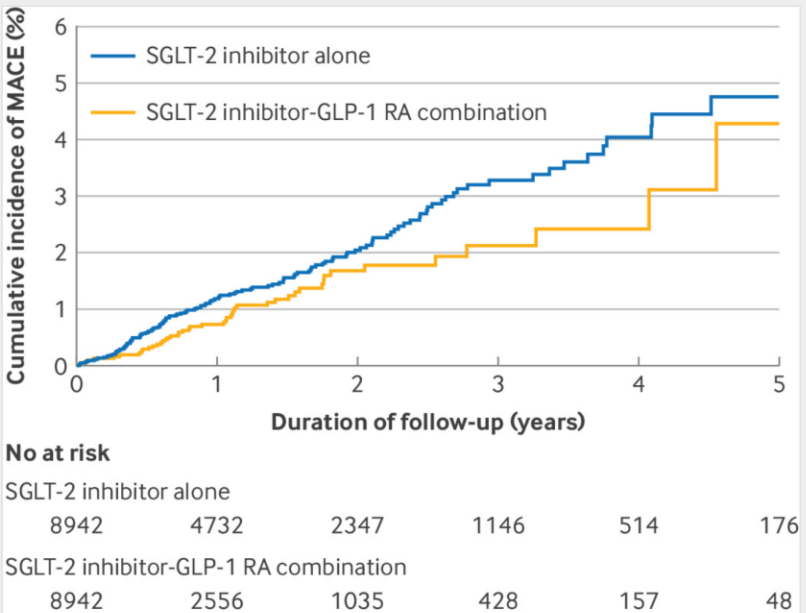


Fig 4
Cumulative incidence curves of major adverse cardiovascular events (MACE) for glucagon-like peptide-1(GLP-1) receptor agonist (RA)-sodium-glucose cotransporter-2 (SGLT-2) inhibitor combination versus SGLT-2 inhibitors

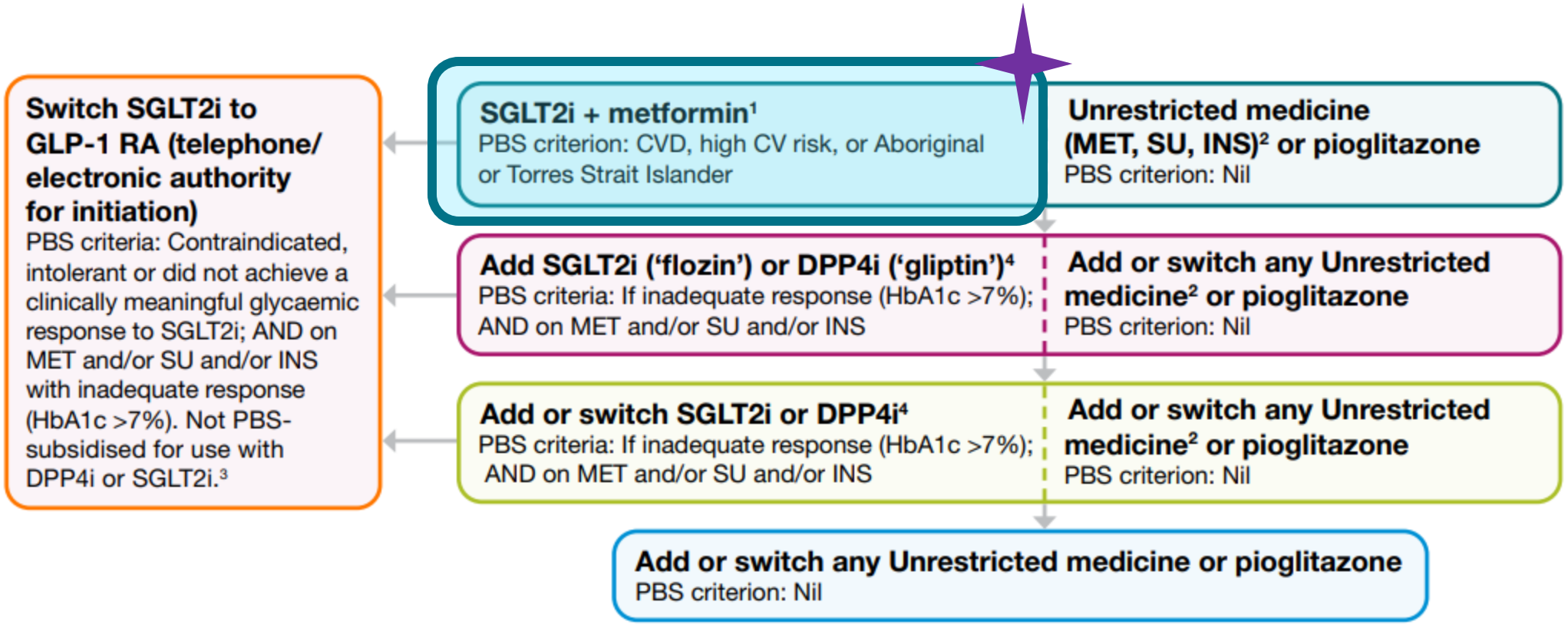
Download figure | Open in new tab | Download powerpoint

From: Simms-Williams N *et al BMJ 2024;385* DOI: https://doi.org/10.1136/bmj-2023-078242

But check:
- PBS eligibility or
- Self-funding options

MAKING THE MOST OF PBS SUBSIDIES

EARLY METFORMIN + SGLT2i THERAPY



https://www.pbs.gov.au/reviews/pbs-restriction-changes-to-type-2-diabetes-mellitus/Attachment-2-PBS-Pharmacotherapy-diagram-April-2025.PDF

MAKING THE MOST OF PBS SUBSIDIES

ADD GLP1a TO SGLT2i vs SWAP SGLT2i TO GLP1a

Switch SGLT2i to GLP-1 RA (telephone/ electronic authority for initiation)
PBS criteria: Contraindicated, intolerant or did not achieve a clinically meaningful glycaemic response to SGLT2i; AND on MET and/or SU and/or INS with inadequate response (HbA1c >7%). Not PBS-subsidised for use with DPP4i or SGLT2i.[3]

If not-PBS eligible, can they self-fund?

SGLT2i + metformin[1]
PBS criterion: CVD, high CV risk, or Aboriginal or Torres Strait Islander

Unrestricted medicine (MET, SU, INS)[2] or pioglitazone
PBS criterion: Nil

Add SGLT2i ('flozin') or DPP4i ('gliptin')[4]
PBS criteria: If inadequate response (HbA1c >7%); AND on MET and/or SU and/or INS

Add or switch any Unrestricted medicine[2] or pioglitazone
PBS criterion: Nil

Add or switch SGLT2i or DPP4i[4]
PBS criteria: If inadequate response (HbA1c >7%); AND on MET and/or SU and/or INS

Add or switch any Unrestricted medicine[2] or pioglitazone
PBS criterion: Nil

Add or switch any Unrestricted medicine or pioglitazone
PBS criterion: Nil

Use SLGT2i CKD, HFrEF or HFpEF PBS codes where possible

Abbreviations: CV—cardiovascular; CVD—cardiovascular disease; DPP4i—dipeptidyl peptidase 4 inhibitor; GLP-1 RA—glucagon-like peptide-1 receptor agonist; INS—insulin; MET—metformin; SGLT2i—sodium-glucose cotransporter 2 inhibitor; SU—sulfonylurea
60-day dispensing: All T2DM medicines are available for 60-day prescriptions, except GLP-1 RAs and insulin.
Fixed dose combinations (FDCs): FDCs of MET + DPP4i, MET + SGLT2i, and SGLT2i + DPP4i, are available on the PBS.

1. Unless contraindicated/intolerant to MET
2. Unrestricted medicines: metformin, sulfonylureas, insulin and acarbose.
3. Use of PBS-subsidised GLP-1 RAs in combination with an SGLT2i is permitted when the patient has T2DM, the SGLT2i is prescribed for an indication other than T2DM (e.g. chronic heart failure or chronic kidney disease), and the patient did not achieve a clinically meaningful glycaemic response to the SGLT2i.
4. Saxagliptin with dapagliflozin FDC requires telephone/electronic authority. Dapagliflozin with sitagliptin FDC and empagliflozin with linagliptin FDC have Authority Required (STREAMLINED) restrictions.

MAKING THE MOST OF PBS SUBSIDIES

SGLT2i FOR CKD (2nd OF 4 PILLARS FOR CKD)

Dapagliflozin or Empagliflozin PBS Streamlined Authority for CKD:

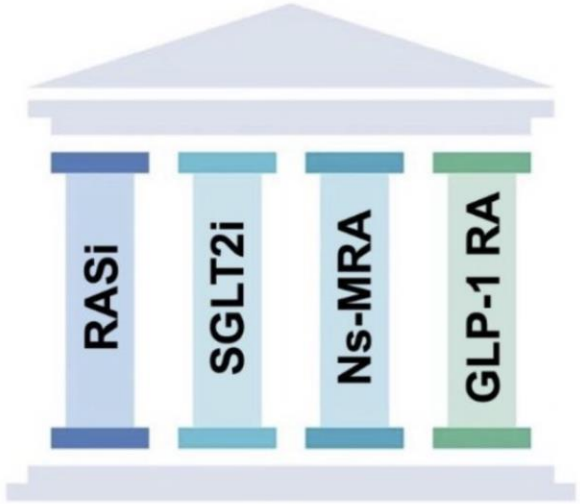
- eGFR 20 to 90 mL/min/1.73m^2

AND

- if eGFR 45 to 90 mL/min/1.73m2, need uACR ≥ 22.6 mg/mmol

OR

- if eGFR 20 to 44 ml/min/1.73m^2, can have normoalbumuria



MEDICATION MANAGEMENT REVIEWS

DMMRs(HMRs)*(ITEM 900)*, RMMRs *(ITEM 903)*

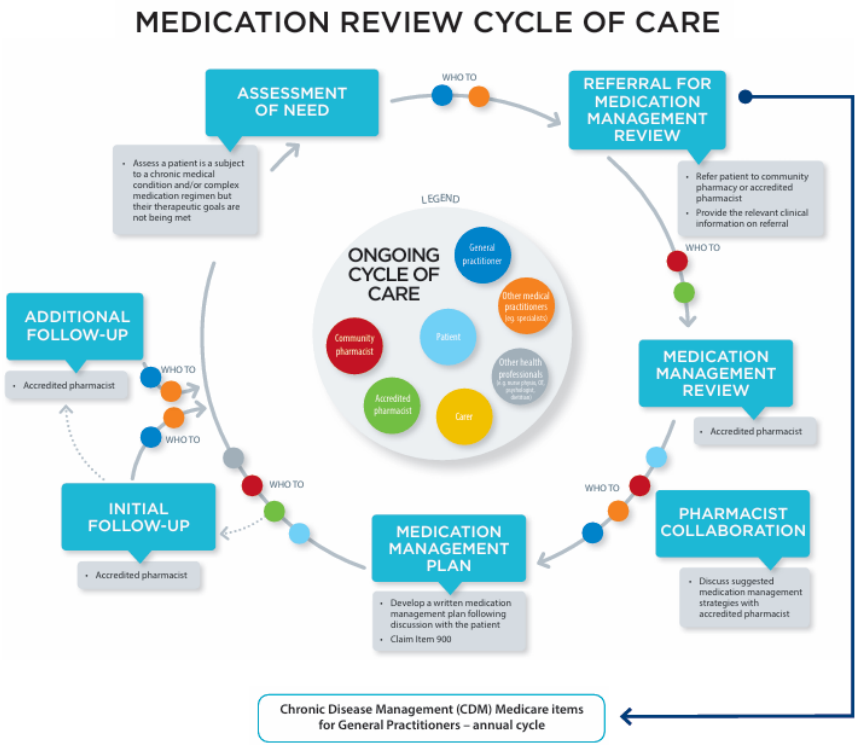
Item 900 (DMMR/HMR) eligibility:

GP assesses patient as:

- having a chronic medical condition OR a complex medication regimen AND
- **not having their therapeutic goals met**

Does <u>not</u> require (but often involves)

- at risk of or experiencing medication-related harm
- polypharmacy

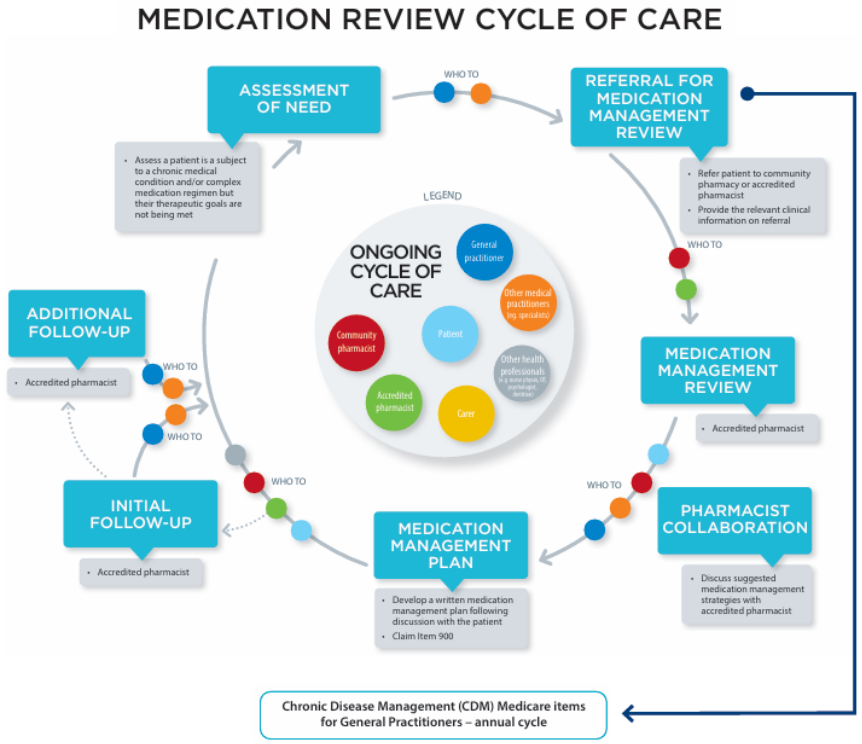


MEDICATION MANAGEMENT REVIEWS

DMMRs(HMRs)*(ITEM 900)*, RMMRs *(ITEM 903)*

MMR-credentialed pharmacist:

- Visits the patient **in their home/RACH***
  - **Pre-approval required for any other location*
- Writes a written report to the GP
- Collaborates with the GP to develop a medication management plan
- May offer up to 2 follow-up visits
  - 1-9 months after 1st visit
  - To offer further support for medication-related problems identified during 1st visit



HOW TO FIND A CREDENTIALED PHARMACIST?

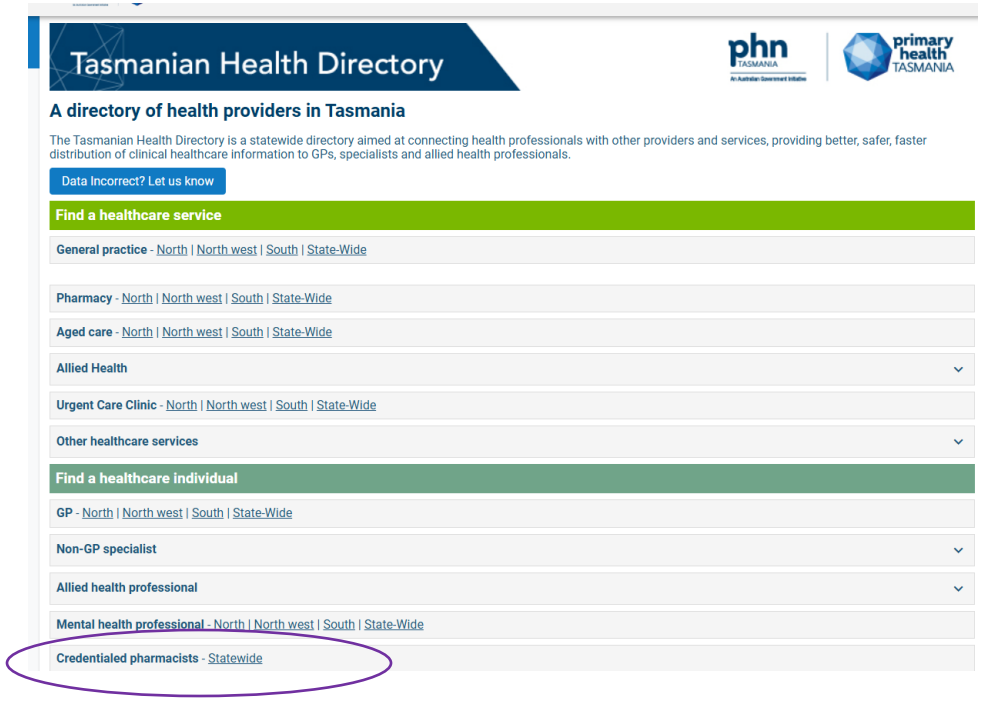
REFER DIRECTLY OR VIA THEIR USUAL COMMUNITY PHARMACY

TASMANIAN HEALTH DIRECTORY:

www.tashealthdirectory.com.au

- FIND A HEALTHCARE INDIVIDUAL
- CREDENTIALED PHARMACISTS

(find CDEs under allied health professional)



PSA CREDENTIALED PHARMACIST DIRECTORY:

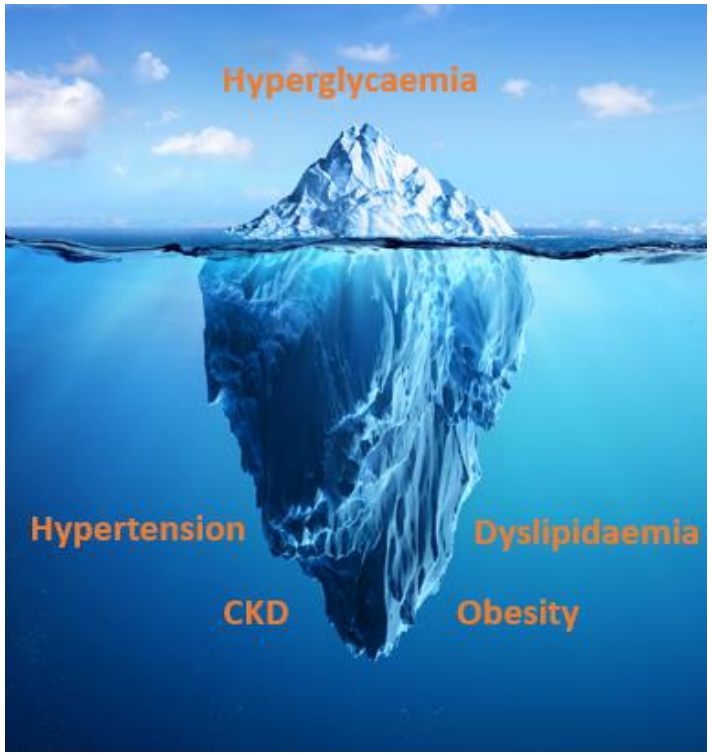
Pharmaceutical Society of Australia

- www.psa.org.au/accreditation-register

If your patients are declined an HMR or waiting for HMRs due to the 30 HMR/month cap, help us to #uncapcare www.psa.org.au/advocacy/uncap-care-fairer-access-to-safe-use-of-medicines/

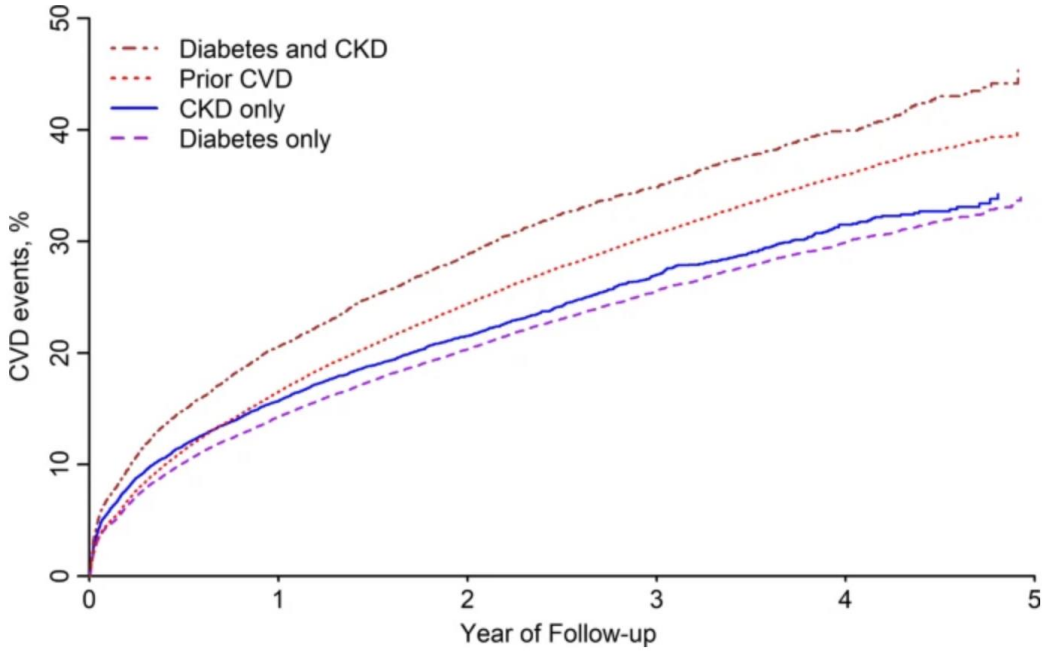
THERAPEUTIC GOALS IN DIABETES:
MUCH MORE THAN MANAGING BGL

- Weight/obesity
- BP/lipids → CVD risk
- CKD
- Management of complications eg neuropathy
- Management of comorbidities eg mental health, arthropathies
- Deprescribing to reduce pill burden



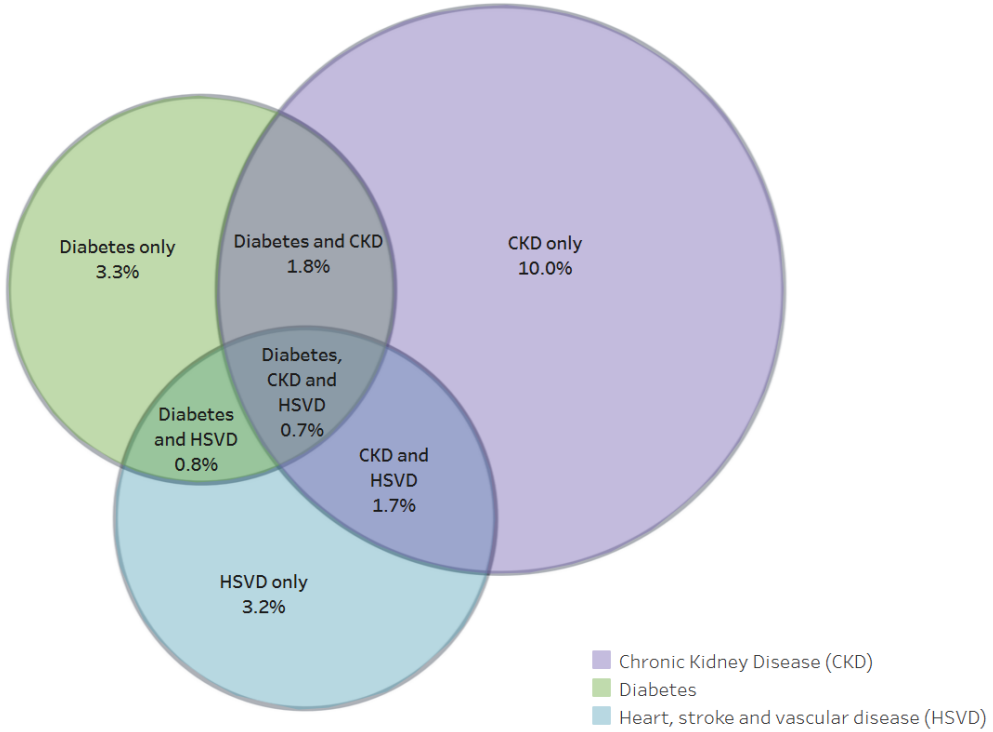
THERAPEUTIC GOAL:
CARDIORENAL RISK REDUCTION

Fig. 1

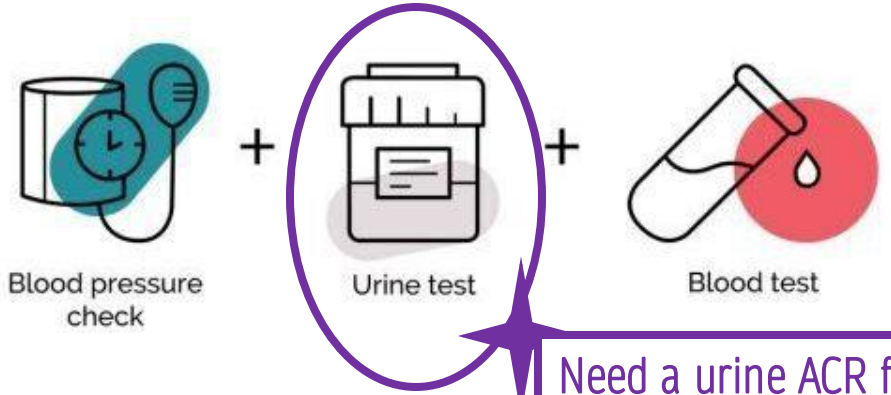


Cumulative incidence of cardiovascular disease events among patients following myocardial infarction. *CKD* chronic kidney disease, *CVD* cardiovascular disease

From: Hubbard D et al. *Cardiovascular Diabetology* 2021:20(58)
DOI: https://doi.org/10.1186/s12933-021-01247-0



From: AIHW analysis of ABS 2025b
www.aihw.gov.au/reports/diabetes/diabetes/contents/comorbidity-of-diabetes

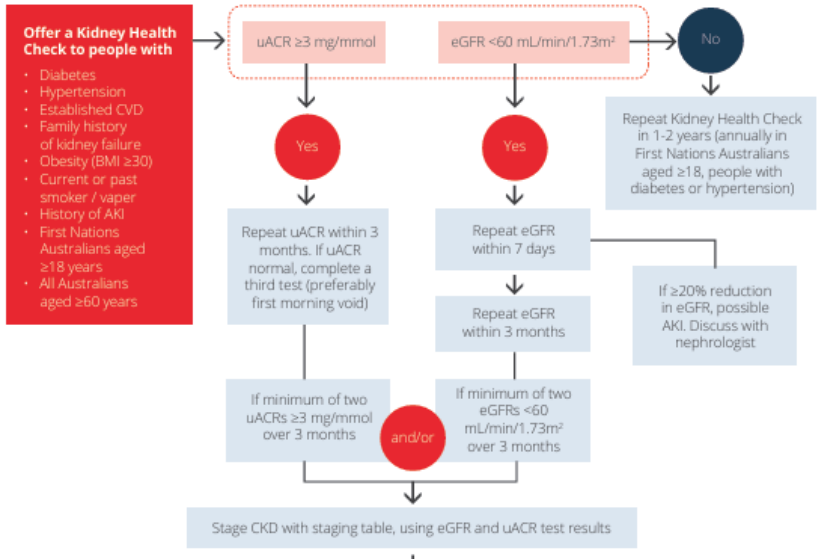


Need a urine ACR for a Kidney Health Check

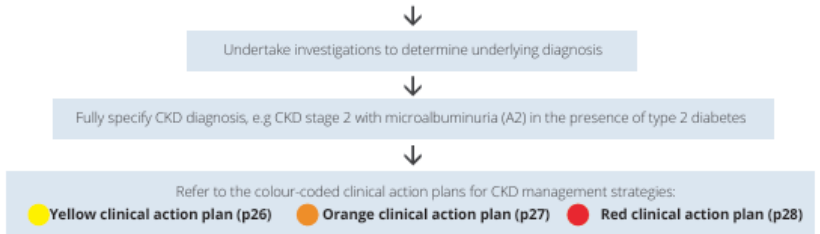
ALBUMINURIA

Calculate and document CKD stage including albuminuria stage to encourage risk reduction strategies

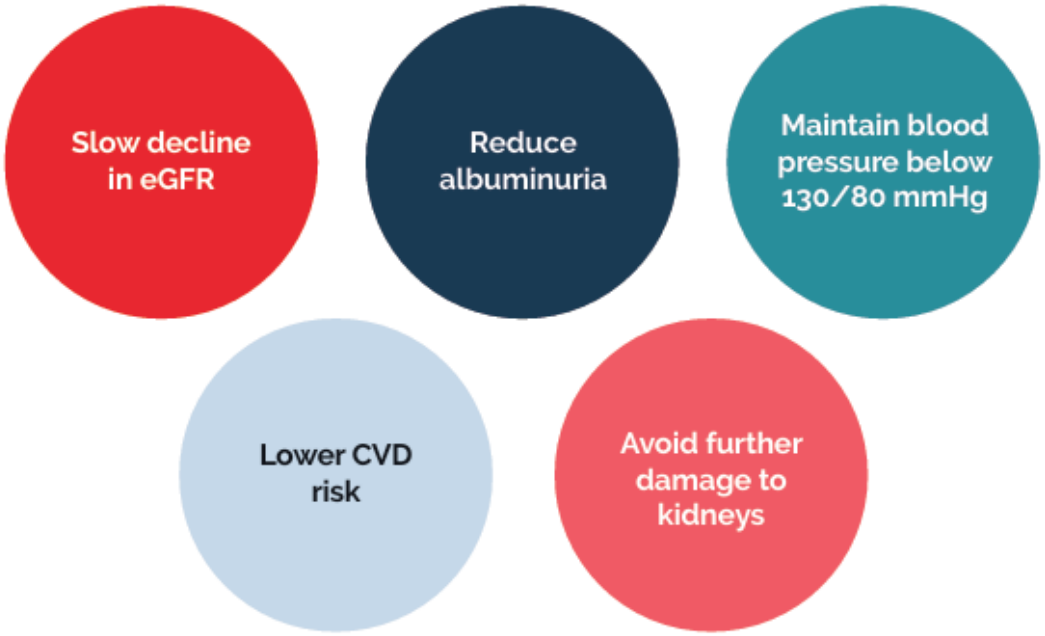
Algorithm for initial detection and diagnosis of CKD



		Albuminuria Stage		
Kidney Function Stage	GFR (mL/min/1.73m²)	Normal (A1) uACR <3.0 mg/mmol	Microalbuminuria (A2) uACR 3.0-30 mg/mmol	Macroalbuminuria (A3) uACR >30 mg/mmol
1	≥90	Not CKD unless haematuria, structural or pathological abnormalities present		
2	60-89			
3a	45-59			
3b	30-44			
4	15-29			
5	<15 or on dialysis			



Key management strategies for all people with CKD



From: Kidney Health Australia CKD Handbook (5th Ed) 2024 https://kidney.org.au/wp-content/uploads/2025/11/KHA-CKD-Handbook-5th-Ed-July2024.pdf

KIDNEY HEALTH CLINICAL ACTION PLANS

Rx: ACEI(or ARB) ± SGLT2i ± nsMRA ± GLP1a

Rx: LIPID-LOWERING THERAPY

Yellow clinical action plan

eGFR ≥60mL/min/1.73m² with microalbuminuria (A2) or
eGFR 45-59 mL/min/1.73m² with normoalbuminuria (A1)

Management goals

- Slow progression of CKD.
  - Slow decline in eGFR.
  - Reduce albuminuria by at least 30%.
- Assess and lower cardiovascular risk.
- Avoid nephrotoxic medications or volume depletion.
- Encourage positive lifestyle changes and self-management practices.

Management strategies

Frequency of review
- Every 12 months

Clinical assessment
- Blood pressure
- Weight and waist circumference
- Smoking/vaping history

Laboratory assessment

Recommended:
- uACR (see page 18)
- eGFR (see page 17)
- Urea, creatinine, and electrolytes
- Full blood count

Also consider:
- Screening for diabetes (fasting blood glucose or HbA1c)
- HbA1c (for people with diabetes)
- Dipstick urinalysis for haematuria detection
- Lipid studies (Trig, Chol, HDLC, LDLC)

Treatment checklist
- Complete investigations to determine underlying cause of CKD.
- Provide advice on positive lifestyle changes (addressing smoking/vaping, nutrition, alcohol use, physical activity, sleep, stress) (see page 29-32).
- Maintain blood pressure consistently below target (see page 52).
- Complete cardiovascular risk assessment (see page 48).
- Prescribe medications to slow CKD progression, e.g., ACE inhibitor or ARB, SGLT2 inhibitor, non-steroidal MRA (see page 34).
- Consider lipid lowering treatment where appropriate (see page 65).
- Optimise glycaemic control (see page 49).
- Avoid nephrotoxic medications or volume depletion (see page 35).
- Discuss contraception with individuals of child-bearing age (see page 37).
- Recommend vaccinations (see page 39).

Orange clinical action plan

eGFR 30-59mL/min/1.73m² with microalbuminuria (A2) or
eGFR 30-44 mL/min/1.73m² with normoalbuminuria (A1)

Management goals

- Slow progression of CKD.
  - Slow decline in eGFR.
  - Reduce albuminuria by at least 30%.
- Assess and lower cardiovascular risk.
- Avoid nephrotoxic medications or volume depletion.
- Encourage positive lifestyle changes and self-management practices.

+

- Early detection and management of complications.
- Adjust medication doses to levels appropriate for kidney function.
- Appropriate referral to a nephrologist when indicated.

Management strategies

Frequency of review
- Every 3-6 months

Clinical assessment
- Blood pressure
- Weight and waist circumference
- Smoking/vaping history

Laboratory assessment

Recommended:
- uACR (see page 18)
- eGFR (see page 17)
- Urea, creatinine, and electrolytes
- Full blood count

Also consider:
- Screening for diabetes (fasting blood glucose or HbA1c)
- HbA1c (for people with diabetes)
- Dipstick urinalysis for haematuria detection
- Lipid studies (Trig, Chol, HDLC, LDLC)
- Iron studies
- Calcium and phosphate
- Parathyroid hormone (6-12 monthly if eGFR <45mL/min/1.73m²)

Treatment checklist
- Complete investigations to determine underlying cause of CKD.
- Provide advice on positive lifestyle changes (addressing smoking/vaping, nutrition, alcohol use, physical activity, sleep, stress) (see page 29-32).
- Maintain blood pressure consistently below target (see page 52).
- Complete cardiovascular risk assessment (see page 48).
- Prescribe medications to slow CKD progression, e.g., ACE inhibitor or ARB, SGLT2 inhibitor, non-steroidal MRA (see page 34).
- Consider lipid lowering treatment where appropriate (see page 65).
- Optimise glycaemic control (see page 49).
- Avoid nephrotoxic medication or volume depletion and adjust doses to levels appropriate for kidney function (see page 35).
- Assess for common issues presenting in CKD (see pages 58-71).
- Appropriate referral to nephrologist when indicated (see page 73).
- Discuss contraception with individuals of child-bearing age (see page 37).
- Recommend vaccinations (see page 39).

Red clinical action plan

Macroalbuminuria irrespective of eGFR or
eGFR <30 mL/min/1.73m² irrespective of albuminuria

Management goals

- Slow progression of CKD.
  - Slow decline in eGFR.
  - Reduce albuminuria by at least 30%.
- Assess and lower cardiovascular risk.
- Avoid nephrotoxic medications or volume depletion.
- Encourage positive lifestyle changes and self-management practices.

+

- Early detection and management of complications.
- Adjust medication doses to levels appropriate for kidney function.
- Appropriate referral to a nephrologist when indicated.

+

- Prepare for kidney replacement therapy if appropriate.
- Prepare for comprehensive conservative care if appropriate.

Management strategies

Frequency of review
- Every 1-3 months

Clinical assessment
- Blood pressure
- Weight and waist circumference
- Smoking/vaping history
- Oedema

Laboratory assessment

Recommended:
- uACR (see page 18)
- eGFR (see page 17)
- Urea, creatinine, and electrolytes
- Full blood count

Also consider:
- Screening for diabetes (fasting blood glucose or HbA1c)
- HbA1c (for people with diabetes)
- Dipstick urinalysis for haematuria detection
- Lipid studies (Trig, Chol, HDLC, LDLC)
- Iron studies
- Calcium and phosphate
- Parathyroid hormone (6-12 monthly if eGFR <45mL/min/1.73m²)

Treatment checklist
- Complete investigations to determine underlying cause of CKD.
- Provide advice on positive lifestyle changes (addressing smoking/vaping, nutrition, alcohol use, physical activity, sleep, stress) (see page 29-32).
- Maintain blood pressure consistently below target (see page 52).
- Address high cardiovascular risk (see page 48).
- Prescribe medications to slow CKD progression as relevant to the persons eGFR, e.g., ACE inhibitor or ARB, SGLT2 inhibitor, non-steroidal MRA (see page 34).*
- Consider lipid lowering treatment where appropriate (see page 65).
- Optimise glycaemic control (see page 49).
- Avoid nephrotoxic medication or volume depletion and adjust doses to levels appropriate for kidney function (see page 35).
- Assess for common issues presenting in CKD (see pages 59-71).
- Appropriate referral to nephrologist when indicated (see page 74).
- Discuss potential progression to kidney failure with patient and treatments.
- Initiate advance care planning (see page 75)
- Discuss contraception with individuals of child-bearing age (see page 37).
- Recommend vaccinations (see page 39).

* Many medications are either contraindicated or require dose adjustments as eGFR declines. We recommend checking individual product information, particularly before prescribing medications in CKD stage 4-5.

Optimise CKD management (4 pillars) + CVD prevention

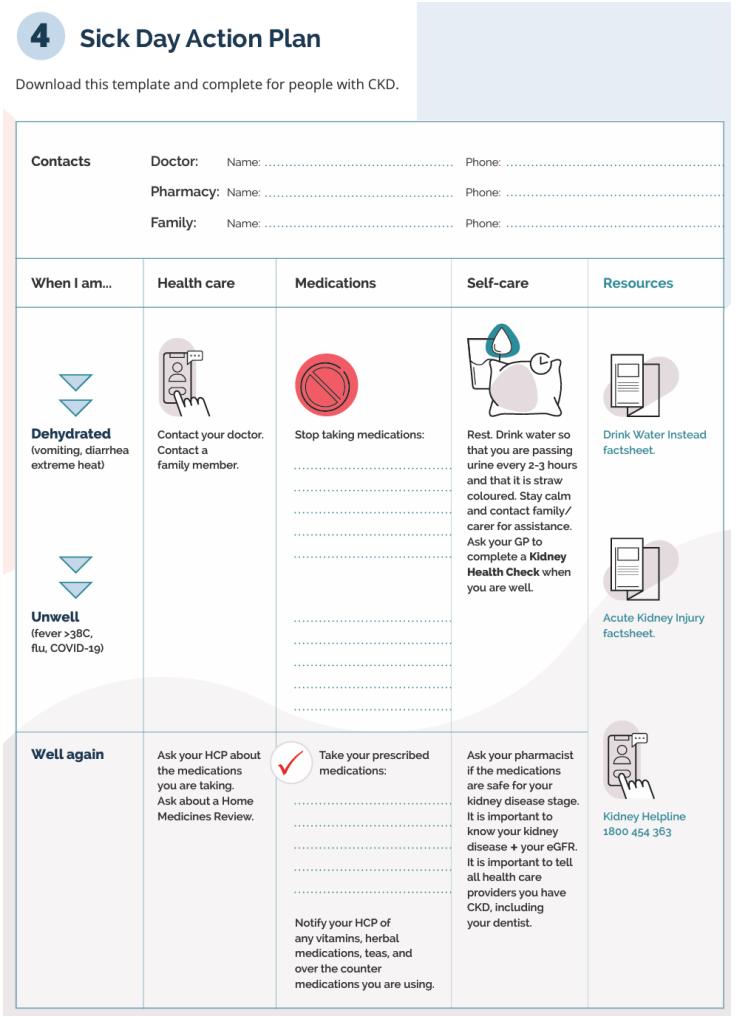
From: Kidney Health Australia CKD Handbook (5th Ed) 2024
https://kidney.org.au/wp-content/uploads/2025/11/KHA-CKD-Handbook-5th-Ed-July2024.pdf

MEDICATION SAFETY: PROTECT THE KIDNEYS

Avoid: nephrotoxic medications

Commonly prescribed drugs that can adversely affect kidney function in CKD	Commonly prescribed drugs that should be avoided temporarily during a sick day (SADMANS)*
• lithium	• **S**ulfonylureas
• aminoglycosides	• **A**CE inhibitors
• NSAIDs/COX-2 inhibitors - beware of the 'triple whammy' (See clinical tip)	• **D**iuretics
	• **M**etformin
	• **A**RBs
	• **N**SAIDs
	• **S**GLT2 inhibitors

* As part of a sick day action plan, it is important that patients are advised to seek guidance from their healthcare professional on temporarily stopping medications during periods of illness.



4 Sick Day Action Plan

Download this template and complete for people with CKD.

Contacts

Doctor: Name: ..... Phone: .....

Pharmacy: Name: ..... Phone: .....

Family: Name: ..... Phone: .....

When I am...	Health care	Medications	Self-care	Resources
Dehydrated (vomiting, diarrhea extreme heat)	Contact your doctor. Contact a family member.	Stop taking medications:	Rest. Drink water so that you are passing urine every 2-3 hours and that it is straw coloured. Stay calm and contact family/ carer for assistance. Ask your GP to complete a **Kidney Health Check** when you are well.	Drink Water Instead factsheet.
Unwell (fever >38C, flu, COVID-19)		.....		Acute Kidney Injury factsheet.
Well again	Ask your HCP about the medications you are taking. Ask about a Home Medicines Review.	Take your prescribed medications: ..... Notify your HCP of any vitamins, herbal medications, teas, and over the counter medications you are using.	Ask your pharmacist if the medications are safe for your kidney disease stage. It is important to know your kidney disease + your eGFR. It is important to tell all health care providers you have CKD, including your dentist.	Kidney Helpline 1800 454 363

From: Kidney Health Australia. Chronic Kidney Disease (CKD) Management in Primary Care. 5th ed. 2024

https://khaprofessionalhub.powerappsportals.com/practicetoolkit/how-to-guides/HPH-KHA-How-To-Sick-Day-Action-Plan.pdf

MEDICATION SAFETY:
PREVENTING MEDICATION-RELATED HARM

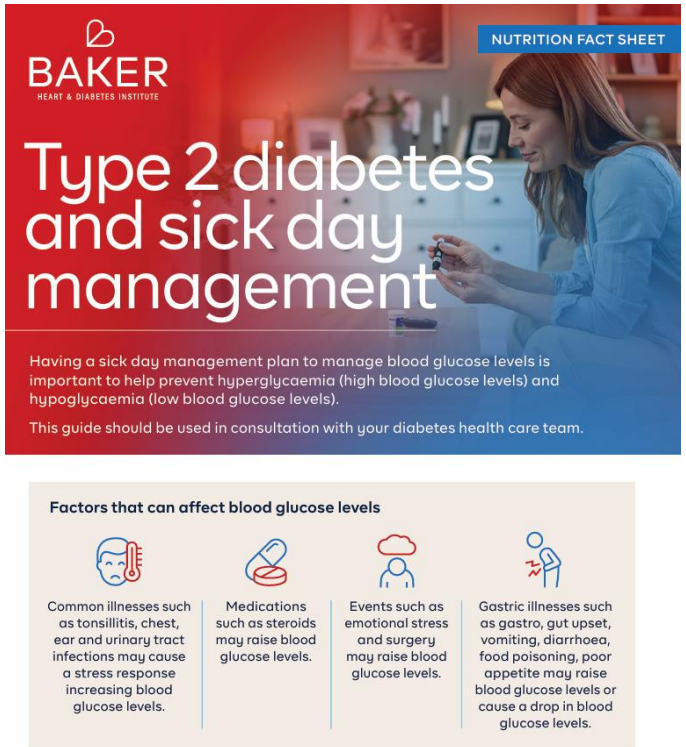
SICK DAY ACTION PLANS

- What is 'sick'?
- How to prevent dehydration
- Increased risk of hyper or hypoglycaemia
- Increased BGL monitoring +/- ketones
- Whether to withhold any medicines
- When to seek help

HYPOGLYCAEMIA

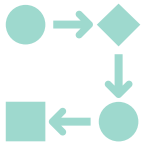
- What are the symptoms of a hypo?
- Check BGL (to see if < 4.0 mmol/L)
- Treating hyperglycaemia (15-15 rule)
- Let treating team know (GP, CDE, pharmacist)
- Severe hypos

SICK DAY MANAGEMENT PLANS
PLENTY TO CHOOSE FROM



- ADEA/NDSS Sick Day Plans (T2DM on insulin, T2DM not on insulin, T1DM various pumps)
- Baker Institute e.g. T2D, T1D
- Diabetes Tasmania / PHT Sick Day Quick Guide
- Kidney Health Australia Sick Day Action Plan
- RACGP Sick Day Management Plan Advice + template
- THS SGLT2i brochure (in HealthPathways)
- QLD Health eg heart failure

SUMMARY



Step-wise approach to blood glucose lowering

Cardiorenal protection

Refer for a DMMR/HMR

Prevent medication-related harm

Type 2 diabetes

Clinical decision-making in a complex disease

Myles Clarkson Fletcher
16 June 2026

Type 2 diabetes is not one disease

2086 Diabetes Volume 69, October 2020

Subtypes of Type 2 Diabetes Determined From Clinical Parameters

Emma Ahlqvist,[1] Rashmi B. Prasad,[1] and Leif Groop[1,2]

Diabetes 2020;69:2086–2093 | https://doi.org/10.2337/db20-0001

DIABETES SYMPOSIUM

Type 2 diabetes (T2D) is defined by a single metabolite, glucose, but is increasingly recognized as a highly heterogeneous disease, including individuals with varying clinical characteristics, disease progression, drug response, and risk of complications. Identification of subtypes with differing risk profiles and disease etiologies at diagnosis could open up avenues for personalized medicine and allow clinical resources to be focused to the patients who would be most likely to develop diabetic complications, thereby both improving patient health and reducing costs for the health sector. More homogeneous populations also offer increased power in experimental, genetic, and clinical studies. Clinical parameters are easily available and reflect relevant disease pathways, including the effects of both genetic and environmental exposures. We used six clinical parameters (GAD autoantibodies, age at diabetes onset, HbA_{1c}, BMI, and measures of insulin resistance and insulin secretion) to cluster adult-onset diabetes patients into five subtypes. These subtypes have been robustly reproduced in several populations and associated with different risks of complications, comorbidities, genetics, and response to treatment. Importantly, the group with severe insulin-deficient diabetes (SIDD) had increased risk of retinopathy and neuropathy, whereas the severe insulin-resistant diabetes (SIRD) group had the highest risk for diabetic kidney disease (DKD) and fatty liver, emphasizing the importance of insulin resistance for DKD and hepatosteatosis in T2D. In conclusion, we believe that subclassification using these highly relevant parameters could provide a framework for personalized medicine in diabetes.

Heterogeneity of Diabetes

Current Classifications for Diabetes

Diabetes is a heterogeneous disease with varying manifestation and risk of complications (1). While diabetes is diagnosed on the basis of a single metabolite, glucose, hyperglycemia can arise due to multiple complex etiological processes that can vary between individuals (2). These processes influence the clinical characteristics, progression, drug response, and development of complications. Diabetes is therefore traditionally divided into different types (Fig. 1A). Type 1 diabetes (T1D) and latent autoimmune diabetes of the adult (LADA) both result from autoimmune destruction of β-cells, often, but not always, reflected by presence of pancreatic autoantibodies in the blood that can be used as a diagnostic marker (3). Identification of such antibodies is a strong indicator that the patient will eventually need insulin treatment to maintain glucose homeostasis (4). Rare monogenic diabetes types, such as maturity-onset diabetes of the young (MODY) and neonatal diabetes, account for about 3% of diabetes diagnosed in individuals <30 years of age. Diagnosis requires sequencing of known monogenic diabetes genes, and the consequences for those affected are life changing, since a correct diagnosis has major implications on choice of treatment (5).

After exclusion of these and a few other subtypes, such as diabetes secondary to steroid use, cystic fibrosis, and hemochromatosis, the remaining patients, 75–85%, are considered to have type 2 diabetes (T2D). Because autoantibodies are not always measured and genetic diagnostics are often not available, the T2D group may include patients with undiagnosed autoimmune or monogenic diabetes. While the two main types of diabetes have been recognized for thousands of years, the names and definitions have changed and there is still no clear cut definition that still allow all patients to be classified as either T1D or T2D (2). Some patients show signs of both autoimmune destruction of β-cells and profound insulin resistance with features of the "insulin resistance syndrome." The large remaining group of true T2D is still highly heterogeneous

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Received 6 May 2020 and accepted 14 July 2020

A six-variable cluster analysis of 14,775 newly diagnosed Swedish patients identified **five reproducible subgroups with distinct complication profiles, disease trajectories, and treatment responses.**

SAID 6% Severe Autoimmune Diabetes	Autoimmune. Needs insulin early. Do not miss LADA.
SIDD 18% Severe Insulin Deficient Diabetes	Secretory failure. Highest retinopathy risk. Early insulin.
SIRD 15% Severe Insulin Resistant Diabetes	Insulin resistance. Highest nephropathy & fatty liver risk. HbA1c misleading.
MOD 22% Mild Obesity-Related Diabetes	Obesity-related. Best prognosis. Weight-loss agents.
MARD 39% Mild Age-Related Diabetes	Age-related. Mild disease. Most primary care T2D. Often over-treated.

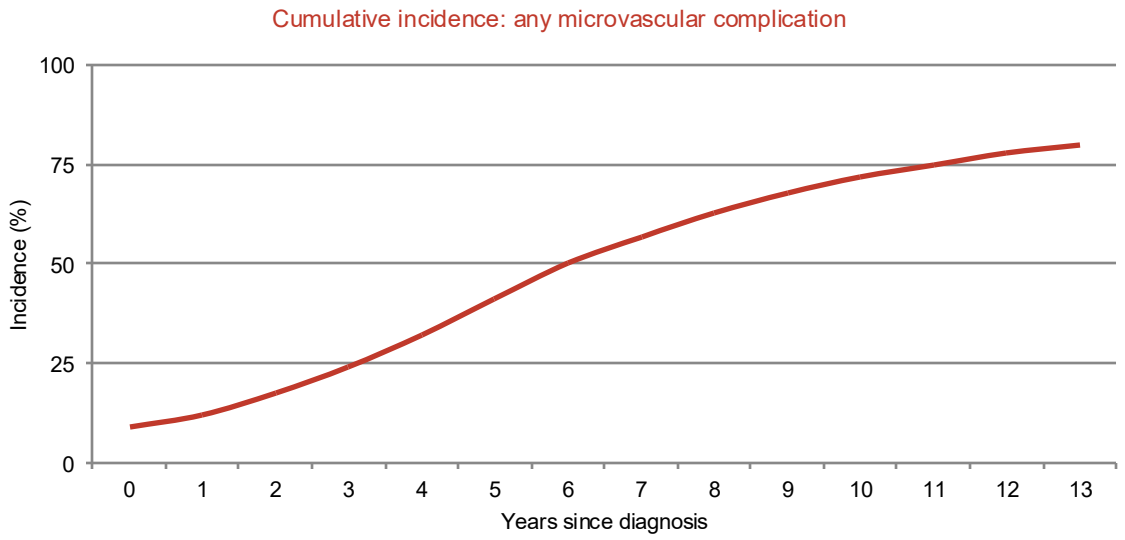
These clusters are illustrative, not a clinical classification tool.
NB: two patients with identical HbA1c 9% may require different management.

Ahlqvist E, Prasad RB, Groop L. Subtypes of Type 2 Diabetes Determined From Clinical Parameters. Diabetes. 2020;69(10):2086–93 | ORIGINAL CLUSTER STUDY: Ahlqvist E et al. Novel subgroups of adult-onset diabetes and their association with outcomes. Lancet Diabetes Endocrinol. 2018;6(5):361–69 | REPLICATION: Zaharia OP et al. Risk of diabetes-associated diseases in subgroups of patients with recent-onset diabetes. Lancet Diabetes Endocrinol. 2019;7(9):684–94 | UK VALIDATION (420,448 patients): Mizani MA et al. BMJ Open Diab Res Care. 2024;12:e004191 | SYSTEMATIC REVIEW (113 studies): Misra S et al. Commun Med. 2023;3:138

Youth-onset T2D: A more aggressive disease

TODAY Study: 500 participants with youth-onset T2D followed for a mean 13.3 years to mean age 26.4 years (NEJM 2021):

80%	67.5%	54.8%	51%
any microvascular complication by 15 yrs	hypertension by 15 years	diabetic kidney disease by 15 years	retinal disease at 7-year follow-up



Clinical implications

- Rapid beta cell decline: secretory failure arrives years, not decades, after diagnosis
- Insulin is not delayed treatment: secretory failure may mandate it within years of diagnosis
- Early aggressive treatment justified: microvascular legacy effect applies
- GLP-1 RA and SGLT2i earlier in the treatment journey, not as step 3 or 4
- Equity: disproportionately affects First Nations, Pacific Islander, and South Asian young Australians: structural risk, not individual behaviour

TODAY Study Group (Bjornstad P et al). N Engl J Med. 2021;385(5):416–26 | Shah AS et al. ISPAD Guidelines 2024: T2D in Children & Adolescents. Horm Res Paediatr. 2024;97:555–83 | ADA Standards of Care 2026, Sec 14. Diabetes Care. 2026;49(Suppl 1):S297–S320

Beta Cell-Centric Framework

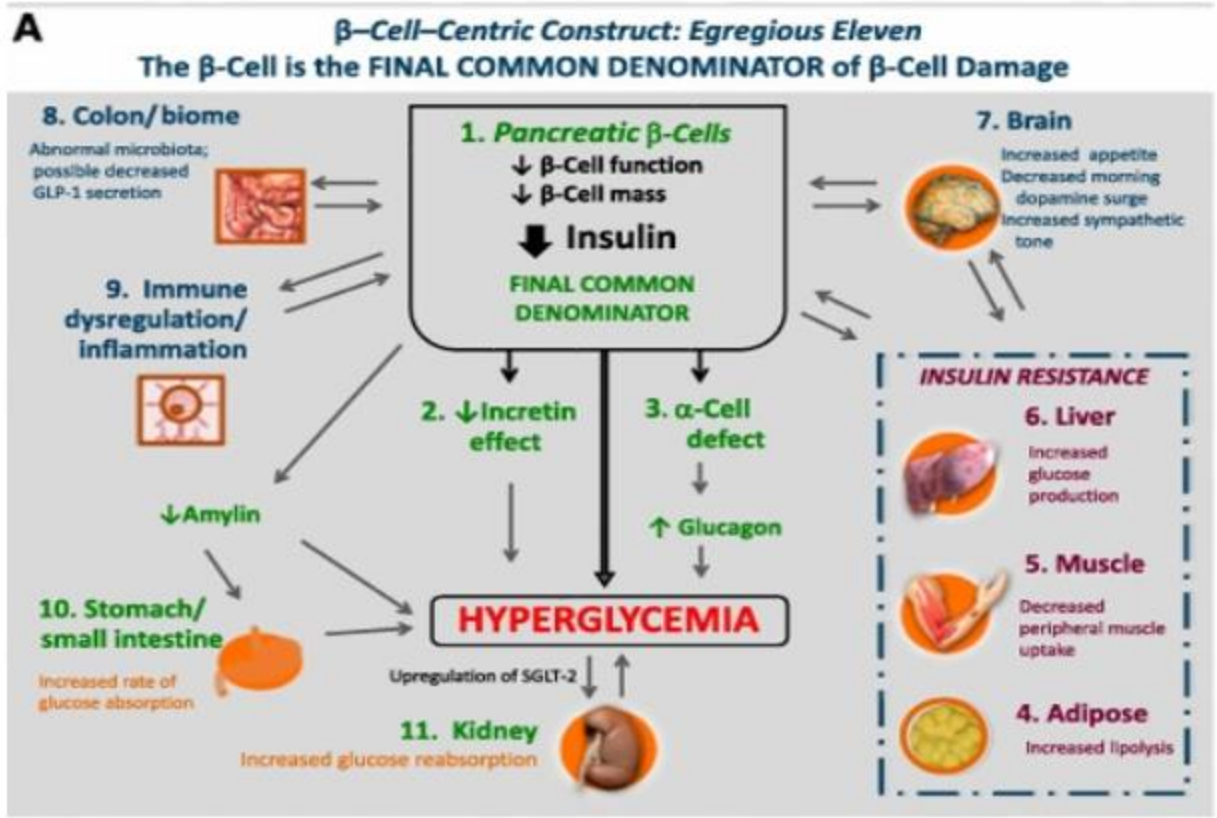


Figure 3—β-Cell–centric construct: the egregious eleven. Dysfunction of the β-cells is the final common denominator in DM. *A*: Eleven currently known mediating pathways of hyperglycemia are shown. Many of these contribute to β-cell dysfunction (liver, muscle, adipose tissue [shown in red to depict additional association with IR], brain, colon/biome, and immune dysregulation/inflammation [shown in blue]), and others result from β-cell dysfunction through downstream effects (reduced insulin, decreased incretin effect, α-cell defect, stomach/small intestine via reduced amylin, and kidney [shown in green]). *B*: Current targeted therapies for each of the current mediating pathways of hyperglycemia. GLP-1, glucagon-like peptide 1; QR, quick release.

- Multiple converging pathways produce beta cell dysfunction and hyperglycaemia.
- **This framework is stable as new agents add intervention points, they do not replace the underlying model.**

Insulin

Not the last resort but the right tool for beta cell failure
NB: comes with risks. Needs flexible, responsive management

Secretory failure: insulin as physiologically correct replacement

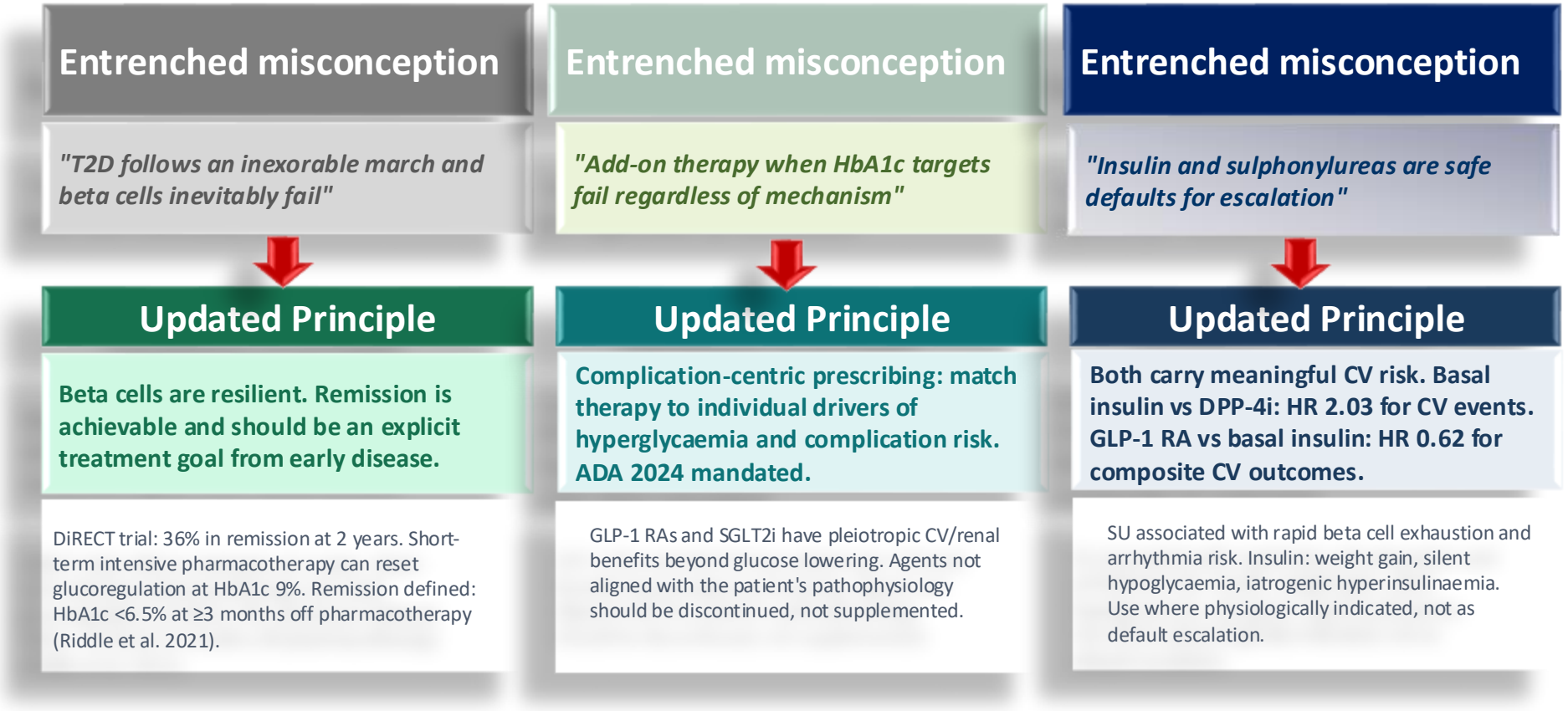
| **Youth-onset:** rapid decline; early insulin appropriate
| **Older adults:** basal insulin for symptom control; don't withhold indefinitely
| **New agents:** act on specific pathways above: same framework, expanded toolkit

✓ Ensure sick day management plan
✓ Hypo plan
✓ Driving risks, alcohol use, kidney function, eating habits
✓ Consider CGM

Schwartz SS et al. Diabetes Care. 2016;39(2):179–86 | Schwartz SS & Herman ME. Front Endocrinol. 2024;15:1394805 | Singh A et al. Compr Physiol. 2025;15:e70003 | DeFronzo RA et al. Am J Physiol. 2020

Updating the Framework

Schwartz & Herman (2024) identify entrenched misconceptions shaping suboptimal T2D care — and the updated principles that should replace them.



Schwartz SS & Herman ME. Front Endocrinol. 2024;15:1394805 | Riddle MC et al. Diabetologia. 2021 | O'Brien MJ et al. JAMA Netw Open. 2018;1:e186125 | Evans M et al. Diabetes Vasc Dis Res. 2023

Applying this in clinic: Proxies for B-Cell status

No HOMA-B or C-peptide required for most decisions. The information is already in the consultation.

Clinical feature	Suggests preserved function	Suggests secretory failure
Duration of T2D	**< 5–8 years**	**> 10–15 years**
Body habitus & weight trend	Overweight or obese; weight stable or increasing	Lean; or unintentional weight loss (not drug-related)
Pattern of hyperglycaemia	Postprandial predominant; fasting BGL near-normal	Elevated fasting BGL; nocturnal hyperglycaemia predominates
Response to secretagogues	Good prior response to SU or GLP-1 RA	Secondary failure: HbA1c rises despite maximum doses
Ketone status	No ketonuria or ketonaemia	Ketonuria or ketonaemia without clear precipitant
C-peptide (if measured)	*Stimulated C-peptide > 600 pmol/L*	*Fasting C-peptide < 200 pmol/L (or < 300 stimulated)*

NB: BMI is not reliably predictable of T2D.
Focus on nutrition rather than weight.

Jones AG & Hattersley AT. The clinical utility of C-peptide measurement in the care of patients with diabetes. Diabet Med. 2013;30(7):803–17 | Shields BM et al. Identifying discriminative features for type 2 diabetes subgroups. Diabet Med. 2018 | Turner RC et al (UKPDS 16). Risk factors for coronary artery disease in non-insulin dependent diabetes mellitus. BMJ. 1998

Beyond HbA1c

HbA1c remains essential , but it is a 90-day average that obscures clinically important variation.

What HbA1c cannot reveal

- Hypoglycaemia: a patient with HbA1c 7.0% may be spending significant time below 3.9 mmol/L nightly
- Glycaemic variability: high coefficient of variation independently associated with CV and cognitive risk
- Haemoglobin variants, anaemia, and renal disease all distort the result
- Time in range: two patients with identical HbA1c can have very different glucose exposure profiles

What matters clinically beyond the number

- Symptomatic hyperglycaemia: thirst, recurrent infections, fatigue, functional decline, delirium in older adults
- Hypoglycaemic episodes: frequency, severity, awareness, and consequences (falls, ED, driving safety)
- Quality of life: treatment burden, number of agents, injections, monitoring demands
- Functional trajectory: is the patient deteriorating despite 'acceptable' HbA1c?

CGM-derived metrics

- Time in Range (TIR): 3.9–10 mmol/L. International consensus target ≥70% (Battelino et al. 2019)
- Time Below Range (TBR): <3.9 mmol/L target <4%; <3.0 mmol/L target <1%
- Time Above Range (TAR): >10 mmol/L — target <25%
- Coefficient of variation (CV): <36% associated with lower hypoglycaemia risk
- GMI (Glucose Management Indicator): HbA1c estimate from CGM mean glucose

Older adults: adjusted targets

- TBR is the priority safety metric, not HbA1c. Time below 3.9 directly linked to falls, arrhythmia, and cognitive events
- TIR 50–70% acceptable in frail or aged care patients; TBR <1% remains non-negotiable
- HbA1c 'at target' can coexist with dangerous nocturnal hypoglycaemia. CGM resolves the ambiguity
- Deprescribing decisions should be informed by TBR data, not HbA1c alone

Battelino T et al. Clinical Targets for Continuous Glucose Monitoring Data Interpretation. Diabetes Care. 2019;42(8):1593–1603 | Ceriello A et al. Glycaemic variability in diabetes: clinical and therapeutic implications. Diabetologia. 2019;62(9):1695–1706 | ADA Standards of Care in Diabetes 2026, Section 6: Glycaemic Goals. Diabetes Care. 2026;49(Suppl 1)

CGM: Safety & clinical decision tool

As a Safety Tool

Hypoglycaemia detection:
Identifies nocturnal, post-exercise, and asymptomatic hypoglycaemia invisible to SMBG. Alarms protect patients with impaired awareness.

Quantifies time below range:
SMBG at arbitrary time points cannot quantify cumulative hypo exposure. CGM-derived TBR changes prescribing decisions.

Reduces fear of hypoglycaemia:
Patients who fear hypoglycaemia run chronically elevated glucose. CGM alarms allow safer titration.

As a Clinical Decision Tool

Insulin titration:
Ambulatory Glucose Profile (AGP) identifies when hyperglycaemia occurs, fasting vs postprandial patterns, and reveals what 4-point SMBG cannot.

Deprescribing decisions:
TBR >1% with HbA1c at target = objective evidence to reduce insulin or cease sulphonylurea. Defensible, patient-centred.

Shared decision-making:
Showing patients their own AGP trace is more motivating and informative than an abstract HbA1c number.

FLASH-UK (Bolinder et al. Lancet 2016): Libre significantly reduced TBR and hypoglycaemia exposure vs SMBG in insulin users. REPLACE-BG (Beck et al. JAMA 2017): CGM non-inferior to SMBG with substantially reduced fingerprick burden in T2D.

The Case for Expanded CGM Access in Insulin-Requiring T2D

A 2026 Australian lifetime economic analysis found RT-CGM to be **dominant** over SMBG in insulin-treated T2D, producing both better outcomes and lower system costs.

Basal insulin (NIIT)

Once-daily long-acting insulin

AUD $5,253

+ 0.319 QALYs

Cost saving per patient over lifetime vs. self-monitoring (SMBG)

Intensive insulin (IIT)

Multiple daily injections

AUD $9,869

+ 0.567 QALYs

Cost saving per patient over lifetime vs. self-monitoring (SMBG)

What drives the savings

Renal complications	**–$14,301 (IIT)**
CV complications	**–$5,638 (IIT)**
Ophthalmic complications	**–$5,078 (IIT)**
Severe hypoglycaemia	**–$3,873 (IIT)**
Severe hyperglycaemia	**–$3,224 (IIT)**

Higher value for younger patients

Modeling age 35 with 1-year T2D duration: net monetary benefit AUD $74,739 (IIT) vs AUD $38,219 at mean age 64.5. Dominance increases substantially with earlier intervention.

Equity imperative: Indigenous Australians

Under conventional treatment, quality-adjusted life expectancy in a 100% Indigenous T2D cohort was 0.85 QALYs lower, with healthcare costs AUD $63,290–88,851 higher than the general T2D population.

NDSS subsidy gap remains

Despite RT-CGM being dominant in both insulin subgroups more effective AND less costly. CGM subsidy for T2D has not been extended. SMBG remains the funded standard of care for insulin-requiring T2D in Australia (June 2026).

https://nadc.net.au/cgm-gp/

Villani M et al. Outcomes of people with severe hypoglycaemia requiring prehospital EMS management. Diabetologia. 2019;62(10):1868–79 — ~50% of patients with severe hypoglycaemia required hospital transport.

Alshannaq H et al. Cost-effectiveness of RT-CGM vs SMBG in insulin-treated T2D in Australia. Adv Ther. 2026;43:615–632. doi:10.1007/s12325-025-03430-1 (IQVIA CORE Diabetes Model v10; lifetime horizon; AUD 2024)

⚠ Note: funded by Dexcom (manufacturer of the Dexcom ONE+). Results were robust across sensitivity analyses.

Bolinder J et al. Lancet. 2016;388:2254–63 (FLASH-UK) | NDSS CGM eligibility criteria. ndss.com.au | Diabetes Australia. CGM Access Position Statement. 2023

CGM for Type 2 Diabetes in Australia: Retail Options (June 2026)

All retail — no NDSS subsidy for T2D

	I-SENS / PHARMACO DIABETES AU **CareSens Air**	ABBOTT **FreeStyle Libre 2 Plus**	DEXCOM / AMSL DIABETES **Dexcom ONE+**
			
Est. retail / month	**~$90–110 / month** Per sensor: $45–55 · 2 sensors	**~$204 / month** Per sensor: $102 · 2 sensors	**~$195 / month** Per sensor: $65 · 3 sensors
Sensor lifespan	**15 days**	**15 days**	**10 days**
Warm-up time	30 min	60 min	30 min
Reading frequency	Every 5 min	Every 1 min (Bluetooth auto)	Every 5 min
MARD (accuracy)	**8.7%**	**~9.2%**	**8.2–8.5%**
Alarms / alerts	High & low alerts	Optional high & low	Customisable high & low (timing + thresholds)
Wear site(s)	Back of upper arm	Back of upper arm	Back of upper arm, abdomen
Age indication	≥18 years	≥2 years	≥2 years
Patient app	CareSens Air app (iOS & Android)	LibreLink app + LibreLinkUp (carer sharing)	Dexcom ONE+ app + Dexcom Clarity app
Clinical software	Sens365 sens365.com	LibreView libreview.com	Dexcom Clarity clarity.dexcom.com/professional
NDSS (T2D)	**Not subsidised for T2D**	**Not subsidised for T2D**	**Not subsidised for T2D**
GP clinic account	Free. GP creates Care Provider account, registers patients. View AGP, TIR, variability. Remote monitoring.	Free. Practice account with Practice ID. Patients connect via ID. AGP, TIR/TBR/TAR, GMI reports. Remote access anytime.	Free. Clinic registers, shares unique code with patient. Auto data upload, no manual sync. AGP, TIR/TBR, patterns. Telehealth-ready.

Sources (June 2026): CareSens Air / Sens365 — pharmacodiabetes.com.au · Libre 2 Plus / LibreView — freestylelibre.com.au ($102/sensor) · Dexcom ONE+ / Clarity — amsldiabetes.com.au ($65/sensor confirmed) · NDSS — ndss.com.au · All clinical software free to register

Key Takeaways

- **T2D is biologically heterogeneous.** Phenotype recognition, particularly beta cell status, is more useful than stepwise HbA1c escalation.
- **The pathophysiology framework is stable.** New treatments add intervention points. They do not replace the underlying model or the clinical reasoning.
  Remission is a realistic treatment goal. Beta cell resilience can be harnessed until late disease. Short-term intensive therapy can reset glucoregulation in many patients (Schwartz & Herman 2024).
- **Insulin is not the last resort.** For secretory failure, youth-onset disease, or older adults needing symptom control, it is the right tool.
- **Barriers to management are structural.** Cost, literacy, social capital, and stigma shape outcomes. Name them. Act on them.
- **HbA1c is necessary but not sufficient.** Time below range is the priority safety metric. Glycaemic variability matters.
- **CGM belongs with insulin.** For at-risk insulin-using patients, it is a safety standard and a clinical decision tool. Advocate for access.

TODAY Study NEJM 2021 · Ahlqvist et al Diabetes 2020 · Schwartz et al Diabetes Care 2016 · ADA 2026 · ISPAD 2024 · RACGP 2023

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Medications in Type 2 diabetes: looking beyond HbA1c . "Referring to endo"

Liz Kinnersly
Nurse Practitioner, Credentialled Diabetes Educator
Diabetes Australia.

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Diabetes Australia acknowledges Aboriginal and Torres Strait Islander peoples as the Traditional Owners and Custodians of this Country.

We pay the utmost respect to them, their cultures and to their Elders, past and present. We extend that respect to the Aboriginal and Torres Strait Islander people here today.

Diabetes Australia is committed to improving health outcomes for all Aboriginal and Torres Strait Islander people affected by diabetes and those at risk

Learning Objectives

- When should patients be referred to an endocrinologist?
- Optimising communication between healthcare team to improve patient care
- Expectations around level of active management prior to referring to endocrinology
- Care coordination for people with complex needs
- Red flag presentations

When to refer to endo ?

≠ T2

< 30 years old

> 7% HbA1c

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HbA1c of 10.2%

AGP Report

13 March 2026 - 26 March 2026 (14 Days)

LibreView

GLUCOSE STATISTICS AND TARGETS

13 March 2026 - 26 March 2026	**14 Days**
Time Sensor Active:	**51%**

Ranges And Targets For	Type 1 or Type 2 Diabetes
Glucose Ranges	**Targets** % of Readings (Time/Day)
Target Range 3.9-10.0 mmol/L	Greater than 70% (16h 48min)
Below 3.9 mmol/L	Less than 4% (58min)
Below 3.0 mmol/L	Less than 1% (14min)
Above 10.0 mmol/L	Less than 25% (6h)
Above 13.9 mmol/L	Less than 5% (1h 12min)

Each 5% increase in time in range (3.9-10.0 mmol/L) is clinically beneficial.

Average Glucose	**16.5** mmol/L
Glucose Management Indicator (GMI)	**10.4% or 90 mmol/mol**
Glucose Variability	**23.6%**

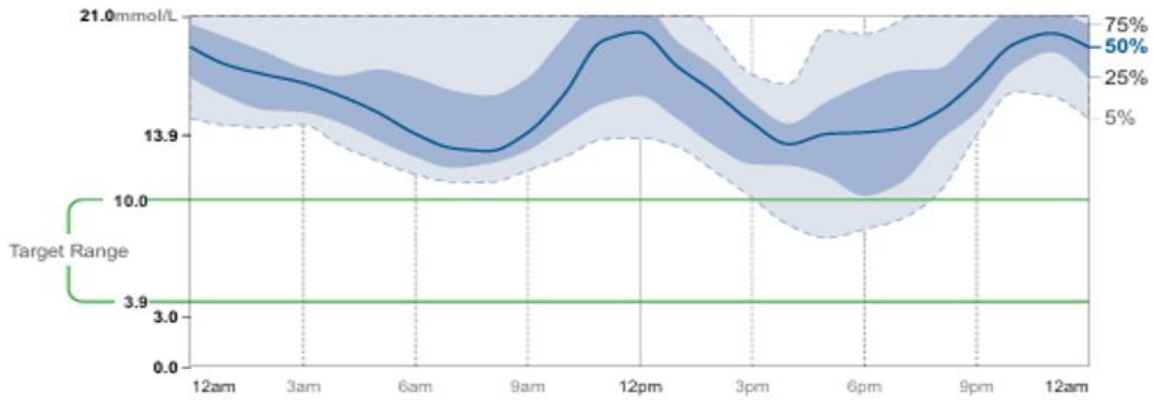
Defined as percent coefficient of variation (%CV); target ≤36%

TIME IN RANGES

Very High	>13.9 mmol/L	**72%**	(17h 16min)
High	10.1 - 13.9 mmol/L	**24%**	(5h 46min)
Target Range	3.9 - 10.0 mmol/L	**4%**	(58min)
Low	3.0 - 3.8 mmol/L	**0%**	(0min)
Very Low	<3.0 mmol/L	**0%**	(0min)

AMBULATORY GLUCOSE PROFILE (AGP)

AGP is a summary of glucose values from the report period, with median (50%) and other percentiles shown as if occurring in a single day.



DAILY GLUCOSE PROFILES

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HbA1c of 7.6%

AGP Report

16 April 2026 - 29 April 2026 (14 Days)

LibreView

GLUCOSE STATISTICS AND TARGETS

16 April 2026 - 29 April 2026	**14 Days**
Time Sensor Active:	**89%**

Ranges And Targets For	Type 1 or Type 2 Diabetes
Glucose Ranges	**Targets** % of Readings (Time/Day)
Target Range 3.9-10.0 mmol/L	Greater than 70% (16h 48min)
Below 3.9 mmol/L	Less than 4% (58min)
Below 3.0 mmol/L	Less than 1% (14min)
Above 10.0 mmol/L	Less than 25% (6h)
Above 13.9 mmol/L	Less than 5% (1h 12min)

Each 5% increase in time in range (3.9-10.0 mmol/L) is clinically beneficial.

Average Glucose	**10.0** mmol/L
Glucose Management Indicator (GMI)	**7.6% or 60 mmol/mol**
Glucose Variability	**28.7%**

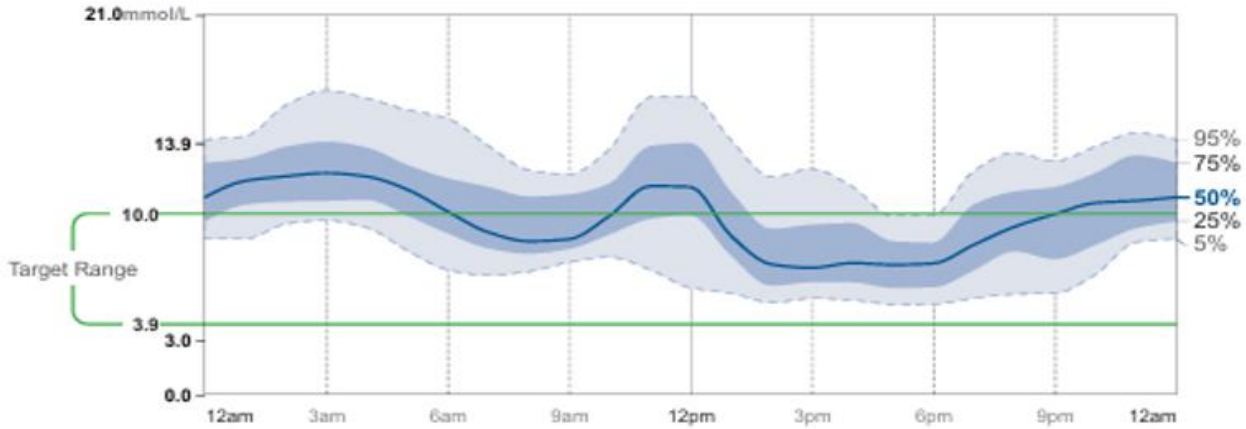
Defined as percent coefficient of variation (%CV); target ≤36%

TIME IN RANGES

Very High	>13.9 mmol/L	**9%**	(2h 10min)
High	10.1 - 13.9 mmol/L	**39%**	(9h 22min)
Target Range	3.9 - 10.0 mmol/L	**52%**	(12h 28min)
Low	3.0 - 3.8 mmol/L	**0%**	(0min)
Very Low	<3.0 mmol/L	**0%**	(0min)

AMBULATORY GLUCOSE PROFILE (AGP)

AGP is a summary of glucose values from the report period, with median (50%) and other percentiles shown as if occurring in a single day.



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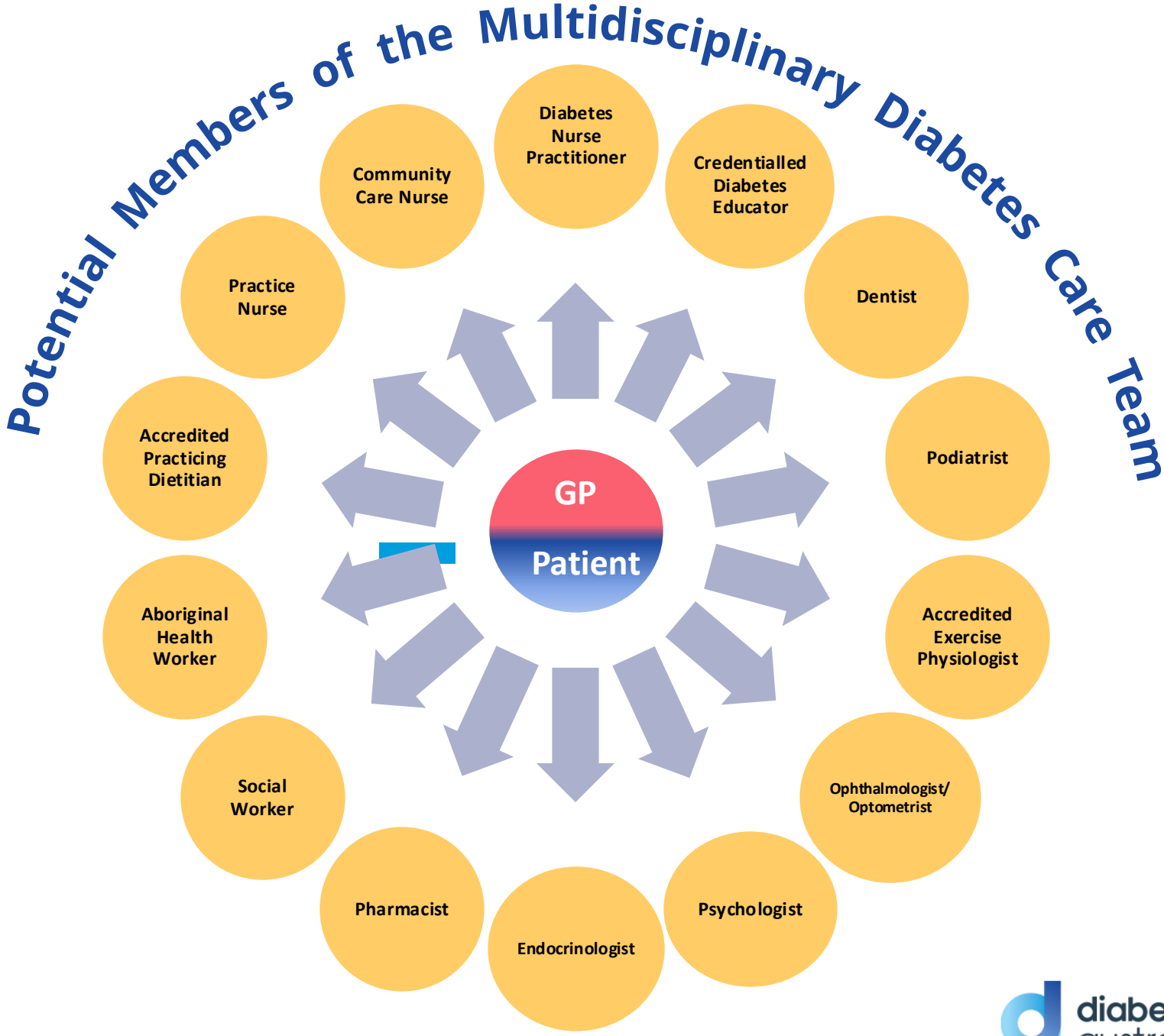
Expectations around level of active management prior to referring to endocrinology

Care Model for People Living with Diabetes in the Community



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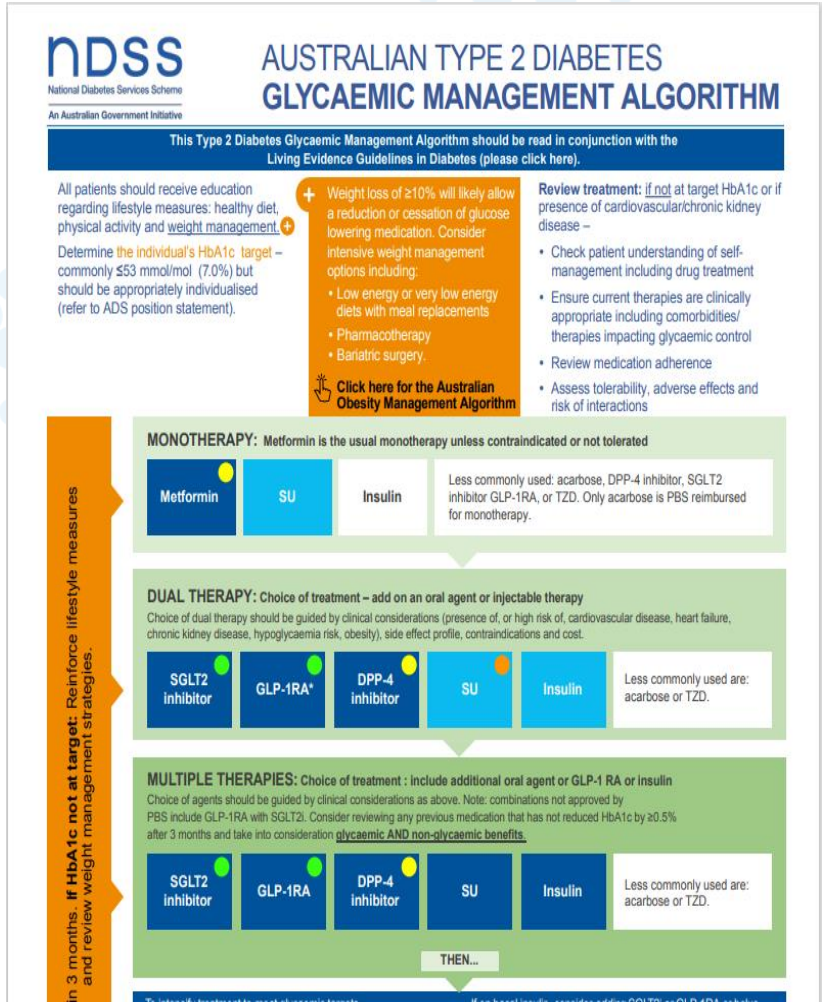
T2DM ADS diabetes guidelines



ndss
National Diabetes Services Scheme
An Australian Government Initiative

AUSTRALIAN TYPE 2 DIABETES
GLYCAEMIC MANAGEMENT ALGORITHM

This Type 2 Diabetes Glycaemic Management Algorithm should be read in conjunction with the Living Evidence Guidelines in Diabetes (please click here).

All patients should receive education regarding lifestyle measures: healthy diet, physical activity and weight management.

Determine the individual's HbA1c target – commonly ≤53 mmol/mol (7.0%) but should be appropriately individualised (refer to ADS position statement).

Weight loss of ≥10% will likely allow a reduction or cessation of glucose lowering medication. Consider intensive weight management options including:
- Low energy or very low energy diets with meal replacements
- Pharmacotherapy
- Bariatric surgery.

Click here for the Australian Obesity Management Algorithm

Review treatment: if not at target HbA1c or if presence of cardiovascular/chronic kidney disease –
- Check patient understanding of self-management including drug treatment
- Ensure current therapies are clinically appropriate including comorbidities/ therapies impacting glycaemic control
- Review medication adherence
- Assess tolerability, adverse effects and risk of interactions

MONOTHERAPY: Metformin is the usual monotherapy unless contraindicated or not tolerated

Metformin | SU | Insulin

Less commonly used: acarbose, DPP-4 inhibitor, SGLT2 inhibitor GLP-1RA, or TZD. Only acarbose is PBS reimbursed for monotherapy.

DUAL THERAPY: Choice of treatment – add on an oral agent or injectable therapy

Choice of dual therapy should be guided by clinical considerations (presence of, or high risk of, cardiovascular disease, heart failure, chronic kidney disease, hypoglycaemia risk, obesity), side effect profile, contraindications and cost.

SGLT2 inhibitor | GLP-1RA* | DPP-4 inhibitor | SU | Insulin

Less commonly used are: acarbose or TZD.

MULTIPLE THERAPIES: Choice of treatment : include additional oral agent or GLP-1 RA or insulin

Choice of agents should be guided by clinical considerations as above. Note: combinations not approved by PBS include GLP-1RA with SGLT2i. Consider reviewing any previous medication that has not reduced HbA1c by ≥0.5% after 3 months and take into consideration glycaemic AND non-glycaemic benefits.

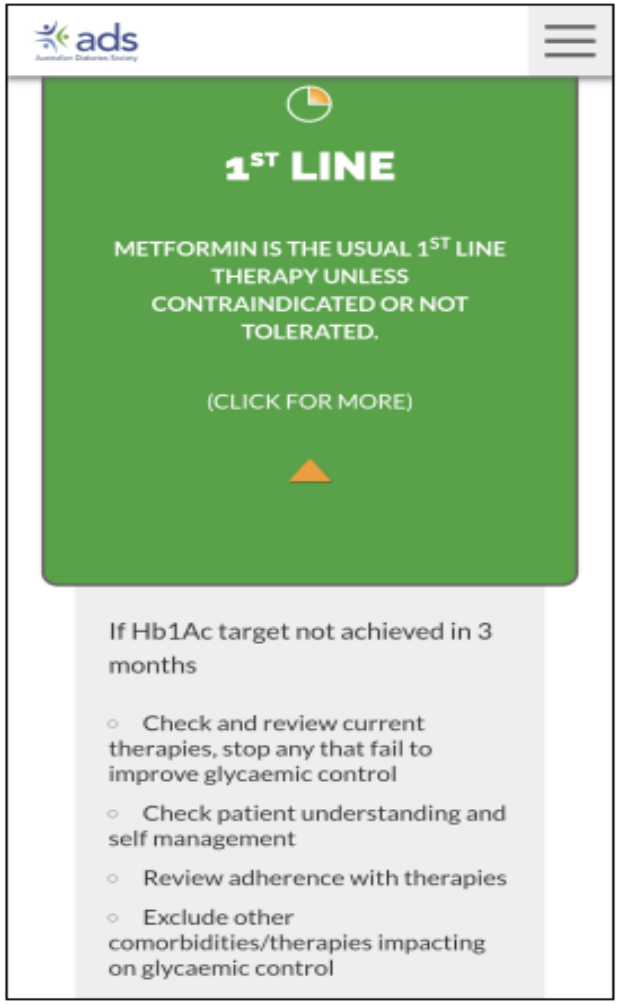
SGLT2 inhibitor | GLP-1RA | DPP-4 inhibitor | SU | Insulin

Less commonly used are: acarbose or TZD.

THEN...

n 3 months. **If HbA1c not at target:** Reinforce lifestyle measures and review weight management strategies.

- Two page document as per left hand, or interactive available for mobile device at https://treatment.diabetessociety.com.au/



ads

1ST LINE

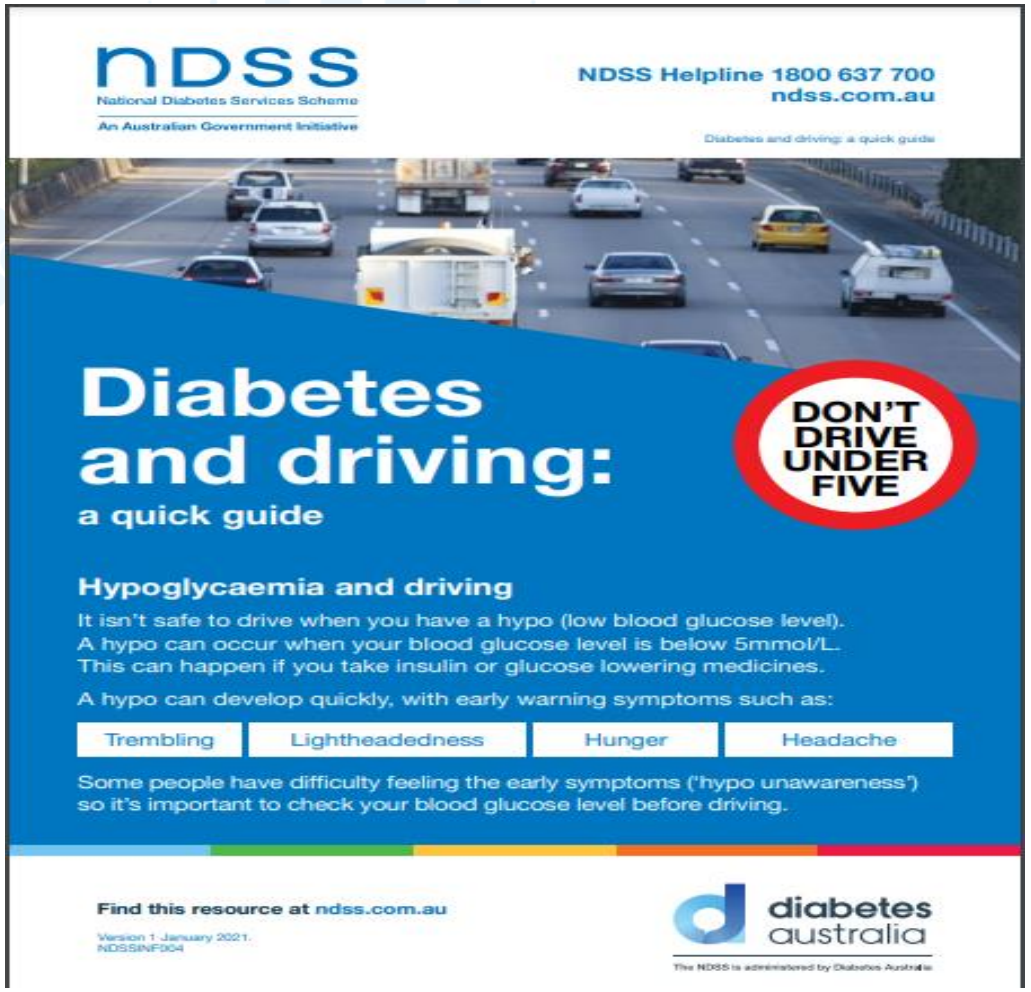
METFORMIN IS THE USUAL 1ST LINE THERAPY UNLESS CONTRAINDICATED OR NOT TOLERATED.

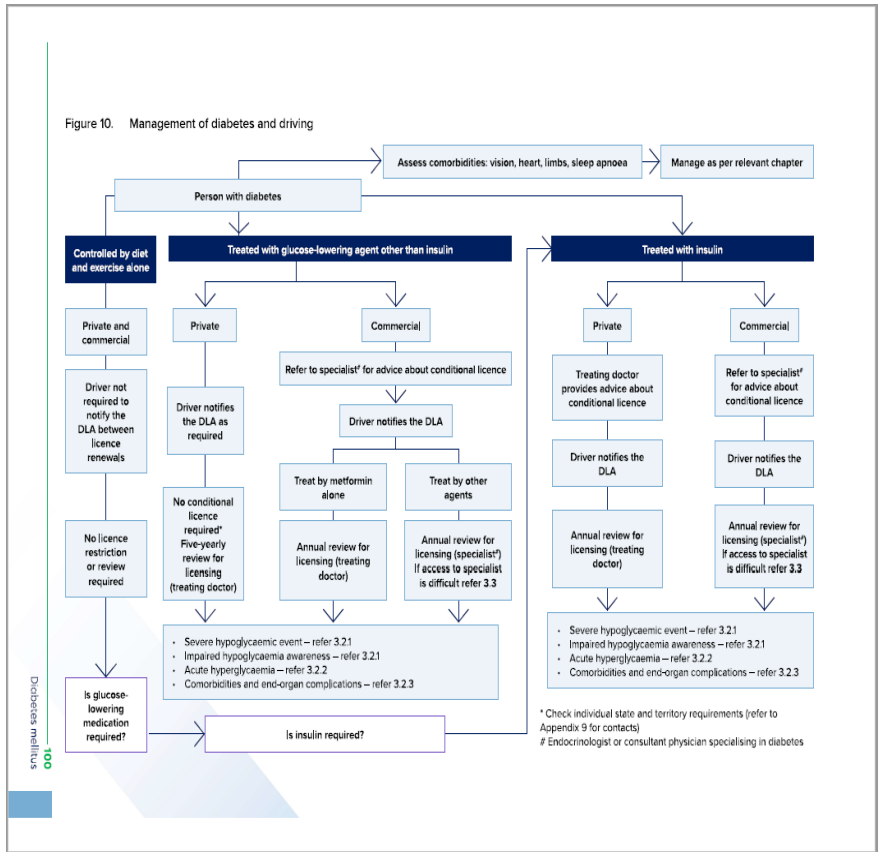
(CLICK FOR MORE)

If Hb1Ac target not achieved in 3 months

- Check and review current therapies, stop any that fail to improve glycaemic control
- Check patient understanding and self management
- Review adherence with therapies
- Exclude other comorbidities/therapies impacting on glycaemic control

Insulin and SU users – hypoglycaemia, and driving obligations





https://austroads.com.au/drivers-and-vehicles/assessing-fitness-to-drive

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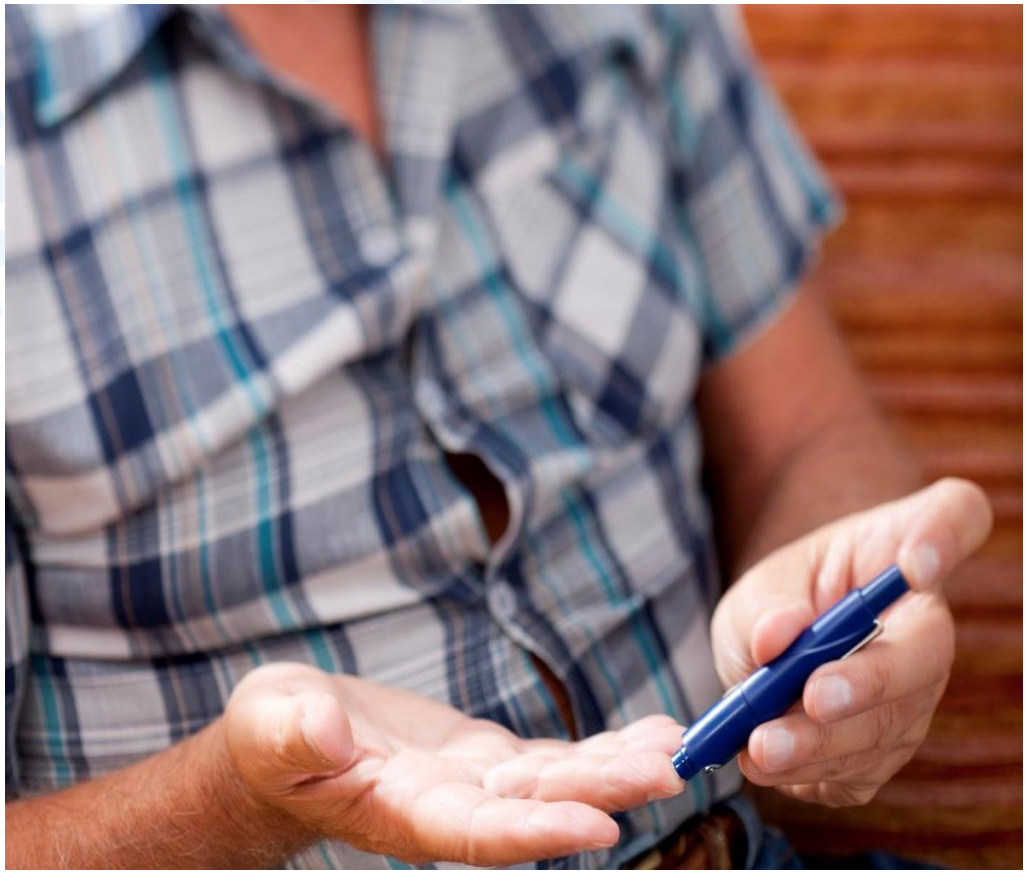
Self Blood Glucose Monitoring



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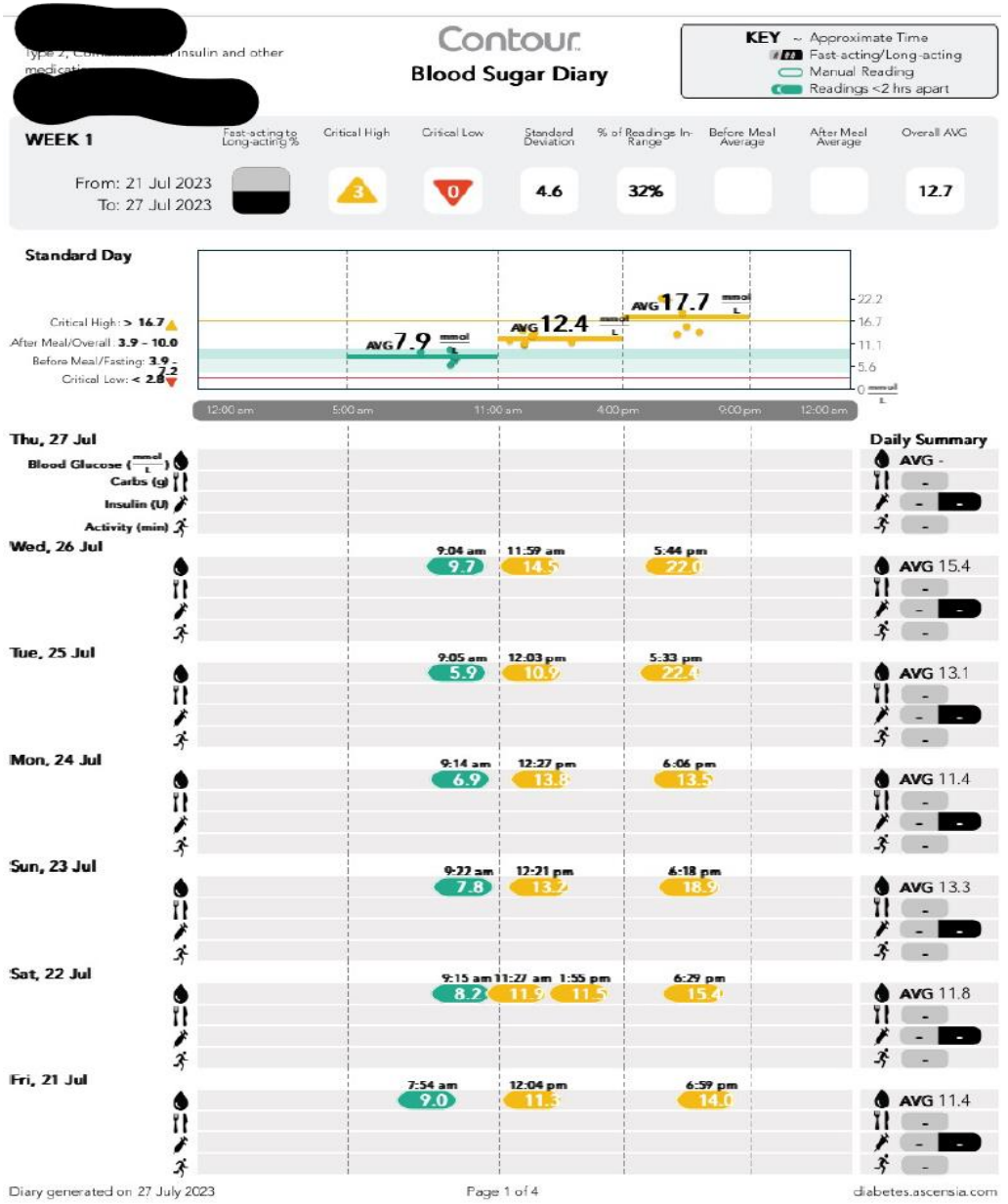
When should self blood glucose measurement be initiated?

- No need for paper diaries if app can be used
- How to use videos on official websites
- Use tech-literate community
- Easy to down-load in clinic



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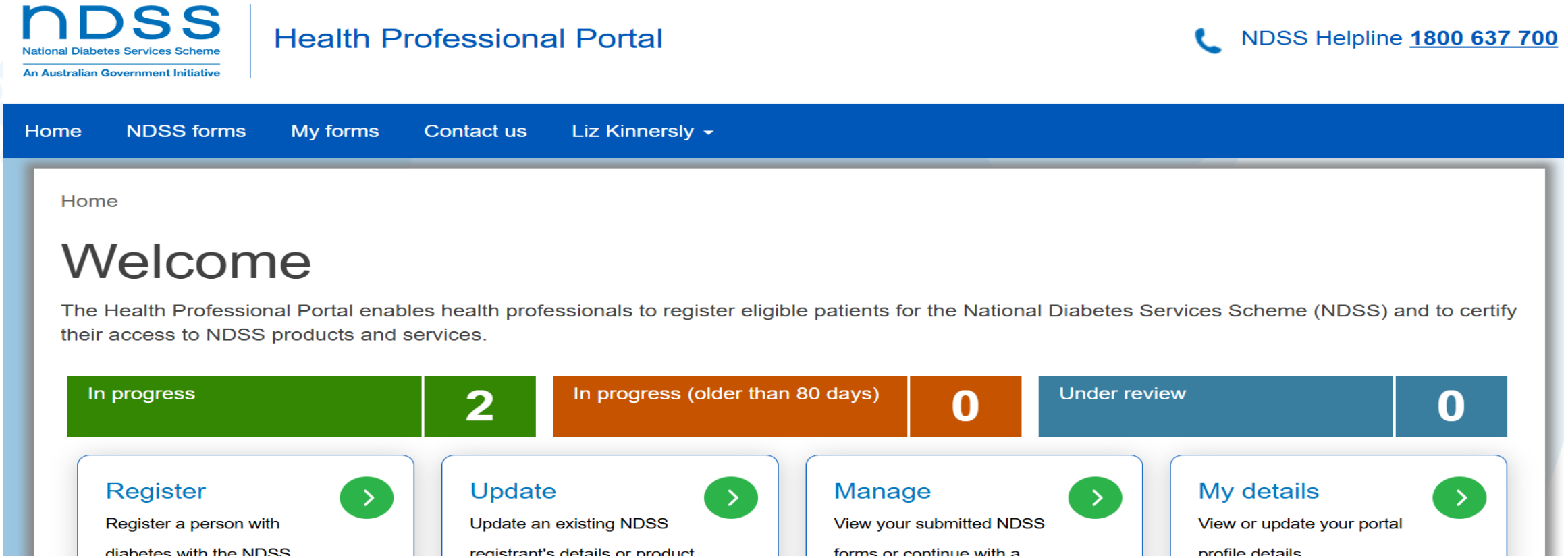
Registration with NDSS – blood glucose strips

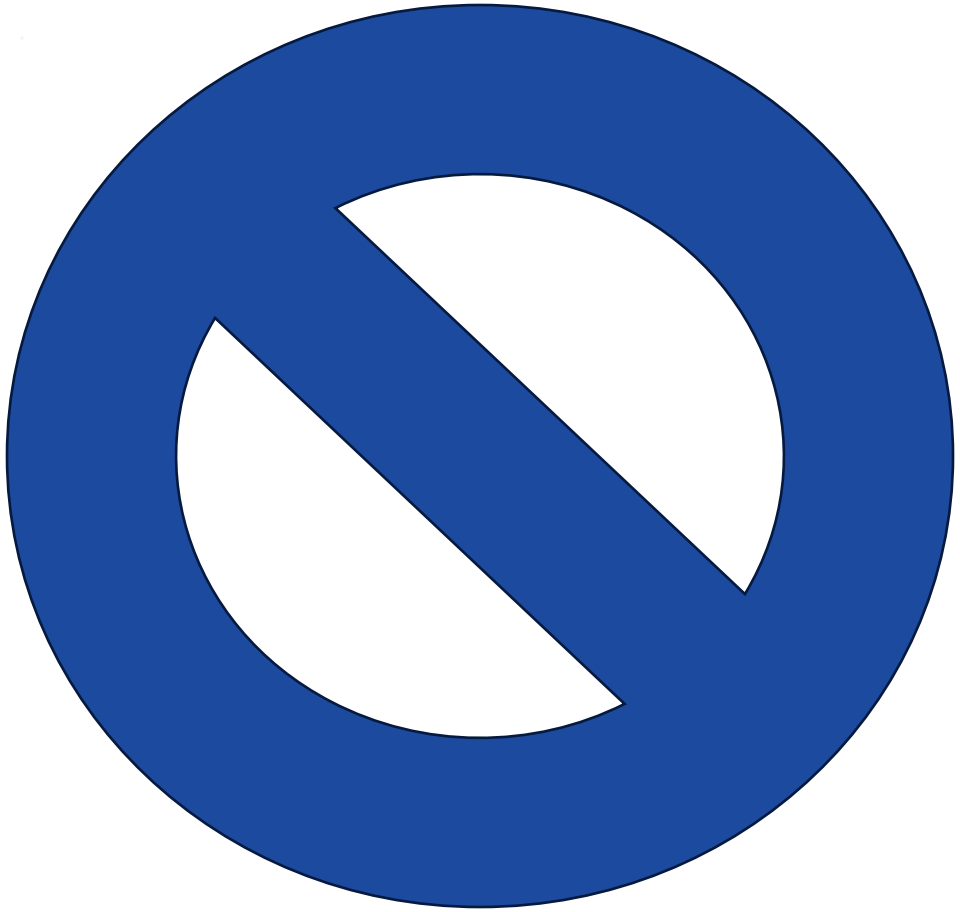


Optimising communication between healthcare teams to improve patient care

MyHealth Record and 1800 medicare

CGM, Apps like BG meters, Ozempic Australia

Care co-ordination for people with complex needs



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Red Flag presentations

Excellent HbA1c on GLM

S AND TARGETS

gust 2025 **14 Days**

76%

Type 1 or Type 2 Diabetes

Targets % of Readings (Time/Day)
Greater than 70% (16h 48min)
Less than 4% (58min)
Less than 1% (14min)
Less than 25% (6h)
Less than 5% (1h 12min)

nge (3.9-10.0 mmol/L) is clinically beneficial.

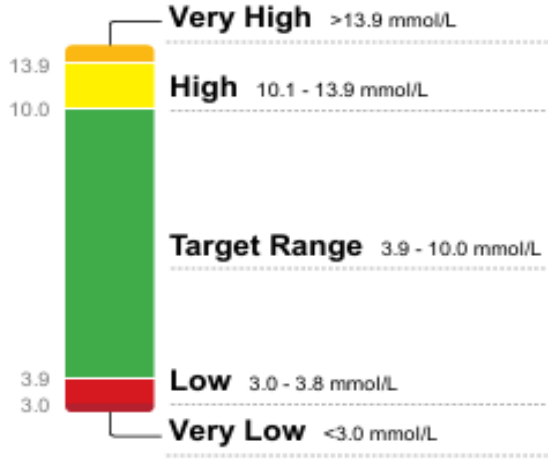
7.3 mmol/L

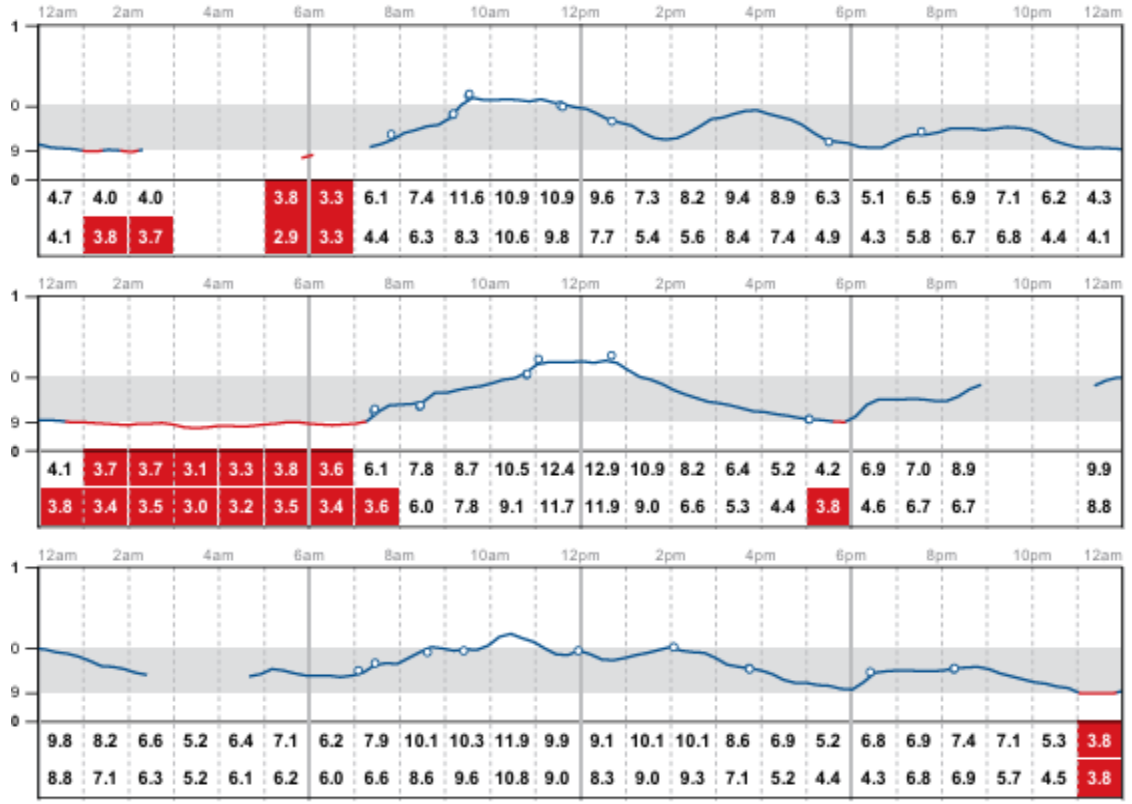
ndicator (GMI) **6.4% or 47 mmol/mol**

36.3%

of variation (%CV); target ≤36%

TIME IN RANGES





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SGLT2i ‘gliflozin’ medications

Position paper from 2018

Euglycaemic DKA – “novel” phenomenon quite old news now

Seems to still be low awareness, anecdotally

Recommendations for Practice

- SGLT2i be ceased at least 3 days pre-operatively (2 days prior to surgery and the day of surgery) or in other physically stressful situations. This may require an increase in other glucose-lowering drugs during this time.
- Strongly consider postponing non-urgent surgery if SGLT2 inhibitors have not been ceased prior to surgery and either blood ketones are >0.6 mmol/L, or HbA1c is >9.0%, as these are indicators of insulin insufficiency and a higher risk of DKA.
- Routinely check both blood glucose and blood ketone levels in the perioperative period if the patient is unwell or is fasting or has limited oral intake, and has been on an SGLT2i prior to surgery.
- If the blood ketone level is >0.6 mmol/L in an unwell pre- or peri-operative patient or >1.5 mmol/L in all other unwell inpatients who have been on an SGLT2i, the treating medical officer and, where relevant, anaesthetist, should be contacted to perform an URGENT VBG to measure the pH
- It is strongly recommended that all patients with DKA are reviewed by an endocrinologist or physician on-call. If required contact your referral tertiary hospital for advice.
- SGLT2i should only be restarted post-operatively when the patient is eating and drinking and close to discharge (usually 3-5 days post-surgery).
- Patients who have day surgery/procedures should only recommence SGLT2i if on full oral intake. It may be prudent to consider delaying recommencement of SGLT2i for a further 24 hours though consideration should also be given to the impact of withholding these agents (and metformin if on combined medication) on glycaemic control.
- Check blood glucose and blood ketone levels if patient has been taking an SGLT2i (prior to or following surgery) and is unwell in the week following surgery.

Could it be T1DM?

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2 stage contact process

Contact us

Diabetes Australia Contact Centre

T: 1800 177 055

E: info@diabetesaustralia.com.au

Our Contact Centre is available Monday to Friday – 8:30am to 4:30pm.

We will be closed from 4.30pm Friday 19 December 2025 (AEDT), reopening 8.00am Monday 5 January 2026 (AEDT).

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Q&A

Final words

- After this webinar ends, your browser will open a link to an evaluation survey – we appreciate your feedback!
- Statements of attendance will be emailed to participants.
- For event queries, please contact events@primaryhealthtas.com.au

Thank you!

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