All things bright and shiny in CKD

s

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Renal Society of Australasia

Advancing the care of people with kidney disease

Conflict of Interest

- Speaker fees from Vantive and Astra Zeneca



Acknowledgement

I would like to respectfully acknowledge Traditional Custodians of the land on which we meet.

I would also like to pay respect to elders past, present and First Nations people attending today.



Objectives

- Guideline directed management therapy
- 4 pillars of pharmacological treatments for CKD
- IgA treatments - promising research
- Novel Therapies
- KDIGO management of anaemia and CKD guidelines

Chronic Disease Prevalence in Australian Adults

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Chronic Kidney Disease (CKD):

~1 in 7 adults (14.2%) show CKD biomarkers (e.g. eGFR/albuminuria)

Around 1.0% (~264,200 adults) report diagnosed CKD

Aboriginal and Torres Strait Islander peoples 4 times as likely to die from CKD as non-Indigenous Australians

Diabetes:

6.6% of adults have diagnosed diabetes (~1 in 15)

Approximately 1.3 million Australians living with diabetes (2021 estimate)

Disproportionate impact on vulnerable populations

Heart Failure:

~144,000 adults (~0.7%) self-report living with heart failure

Estimated true prevalence may be closer to 1–2% due to underdiagnosis = ~300,000

~ 1.5% total hospital admissions in 2021-2022

Key Takeaways:

CKD is common, especially early stages that often go undiagnosed

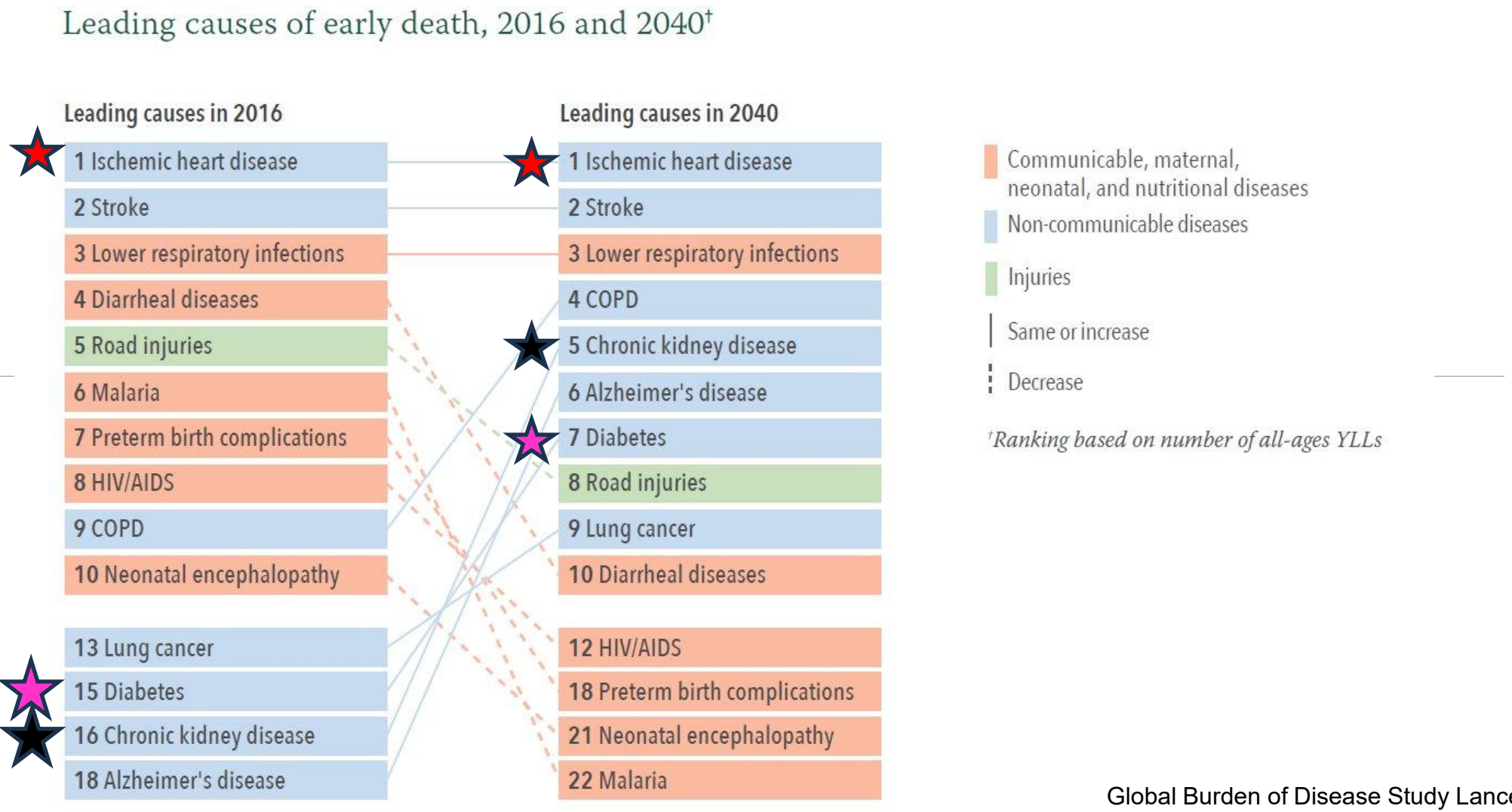
Diabetes is a major driver of CKD

Heart failure is under-recognised; real-world burden likely higher than reported

These conditions frequently co-exist and require integrated management in primary care

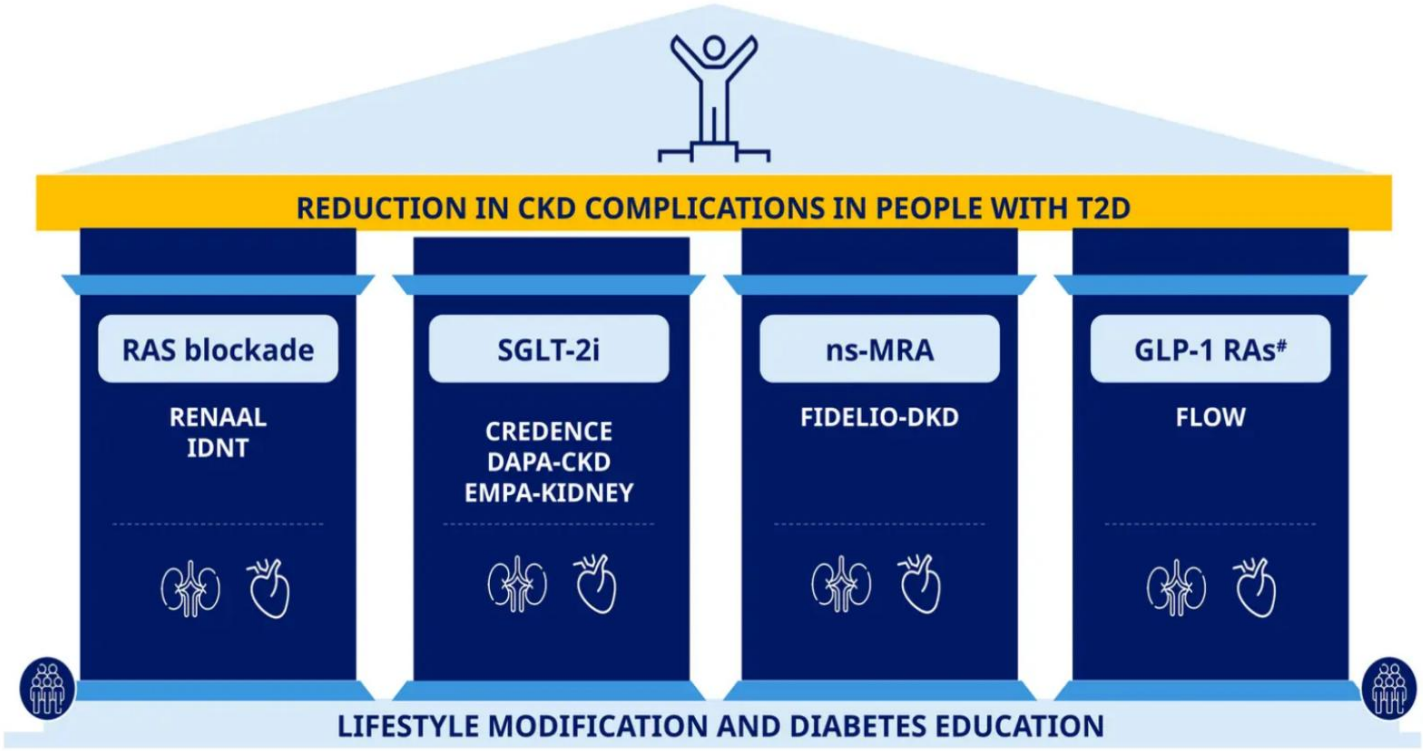
(ABS, 2023)

Chronic Illness and the Shift in Global Mortality Patterns



Global Burden of Disease Study Lancet 2018

4 Pillars of Cardio Renal protection in CKD



 # The definitive place in the treatment of people with T2D and CKD remains to be established. Semaglutide is the only GLP-1RA demonstrating effects in a dedicated kidney outcomes trial. CKD, chronic kidney disease; CVOTs, cardiovascular outcomes trials; GLP-1 RAs, glucagon-like peptide-1 receptor agonists; ns-MRA, non-steroidal mineralocorticoid receptor antagonists; RAS, Renin–angiotensin system; SGLT-2i, sodium-glucose cotransporter-2 inhibitor; T2D, type 2 diabetes. Figure design adapted from: 2023 ADA, Standards of Medical Care in Diabetes - 2023: Diabetes Care, December 2022, Vol.46, Supplement 1; Agarwal R et al, Nephrology Dialysis Transplantation (2023) 38: 253–257

Impact of Guidelines Directed Management Therapy in New Zealand

Renal Society of Australasia

Only **funded SGLT2 inhibitor** is:
Empagliflozin (brand name Jardiance)
Also available as the combination product Empagliflozin with Metformin (Jardiamet)

For **type 2 diabetes**, empagliflozin requires Special Authority approval. Eligibility generally includes:
Type 2 diabetes, and
Inadequate glycaemic control despite standard therapy, and
Increased cardiovascular or kidney risk (for example established cardiovascular disease, diabetic kidney disease, heart failure, or elevated cardiovascular risk).

Heart failure
Chronic heart failure with reduced ejection fraction (HFrEF)

Chronic kidney disease (CKD)
Unlike Australia and some other countries, New Zealand does **not yet have broad funded access to SGLT2 inhibitors solely for CKD** without diabetes or heart failure..

Impact of Guidelines Directed Management Therapy in New Zealand

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Proposed August 2026 changes

- Lowering the cardiovascular risk threshold from **15% to 10%**
- Removing the ethnicity-based eligibility criterion

Heart Failure Funding

No consideration of HFpEF a yet

The biggest difference between New Zealand and Australia is that **Australia treats SGLT2 inhibitors much more as standard cardiorenal therapy, whereas New Zealand still limits funding through Special Authority criteria.**

GLP-1s – dulaglutide and liraglutide – Special Authority – for glycaemic control. No access to semaglutide / terzepitide _ GLP-1- GIP

Guideline Directed Medical Therapy

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Aim of GDMT is to :

- **Slow CKD progression**
- **Reduce albuminuria**
- **Lower cardiovascular risk**
- **Delay dialysis or transplantation**
- **Improve survival or quality of life**

Core Components of GDMT

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Blood Pressure Control

- Target < 130/80 (or in KDIGO guidelines 120/80) – first line ACE/ARBs

RAAS blockage

- Foundational therapy
- Slows CKD progression

- CV benefit

SGLT2 inhibitors

- Slow eGFR decline
- Reduce albuminuria
- Reduce heart failure admissions
- Reduce dialysis /transplant /death risk
- Landmark trails – DAPA CKD, EMPA-Kidney

Core Components of GDMT

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Glycaemic Management in Diabetic CKD

- Avoid hyperglycaemic kidney injury
- Reduce CV events
- Minimise hypoglycaemia
- Individualise HbA1C targets – depending on vintage

Use medications with CV and CKD benefit

- GLTP-1 s (LEADER, SuSTAIN-6 FLOW - shows renal and CV benefits)
- SGLT2's

Core components of GDMT

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Non-steroidal Mineralocorticioid Receptor Antagonists

- Finerenone – targets inflammation , fibrosis pathways
- Slows CKD progression, and reduces CV events – FIGARO – DKD
- Currently only used in diabetic CKD

Lipid Management

- Reduce CV risk by using statin
- Explore AUS CVD Risk Calculator Assessing and managing cardiovascular disease risk - 2023 Australian guideline and calculator

Lifestyle and Supportive Therapy

- Sodium restriction
- Smoking cessation
- Exercise, weight, nutrition, avoid nephrotoxins, vaccination,

ACE inhibitor or ARB	Statin (+/- ezetimibe)	SGLT2 inhibitor*	Non-steroidal MRA*	GLP-1 RA*
• First-line treatment. • Up-titrate to maximum tolerated dose.	Consider use in: • People with CKD and a CVD risk ≥10% and • First Nations Australians with CKD and a CVD risk ≥5%.	• Use in people with CKD and proteinuria, with/without diabetes*. • **Do not initiate** if **eGFR <20mL/min/ 1.73m^2).**	• Indicated for use in people with CKD (with albuminuria) associated with type 2 diabetes. • **Do not initiate** if **eGFR <25mL/min/ 1.73m^2** or when **K+ >5.0mmol/L.**	• Indicated for use in people with CKD if they also have type 2 diabetes. • Do not use in people with advanced kidney disease.

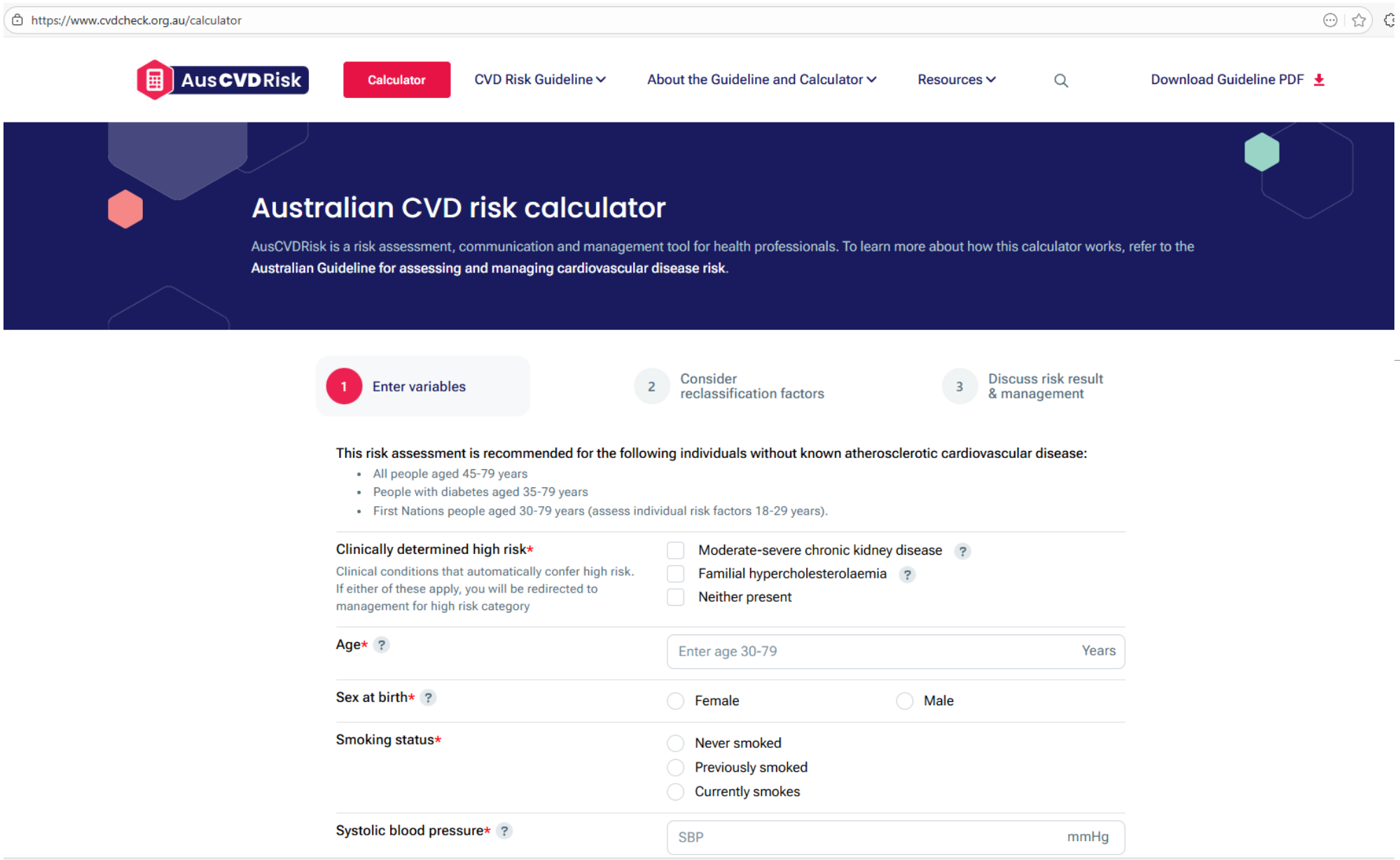
** Refer to product information for eligibility criteria and dosing*

Refer to pages **34-35** in the CKD Handbook

Chronic Kidney Disease (CKD) Management in Primary Care (5th edition). Kidney Health Australia, Melbourne, 2024.

New Australian CVD Risk Calculator

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Mechanism of action- SGLT2

Block the sodium-glucose co-transporter in the proximal convoluted tubule of the kidney,

Improving ventricular loading conditions and reducing preload *via* diuretic and natriuretic SGLT-2 inhibitors

Reduce plasma volume by selectively reducing interstitial fluid, followed by reduced preload, decreased arterial stiffness, and blood pressure, which, in turn, would reduce afterload, leading to an improved coronary circulation

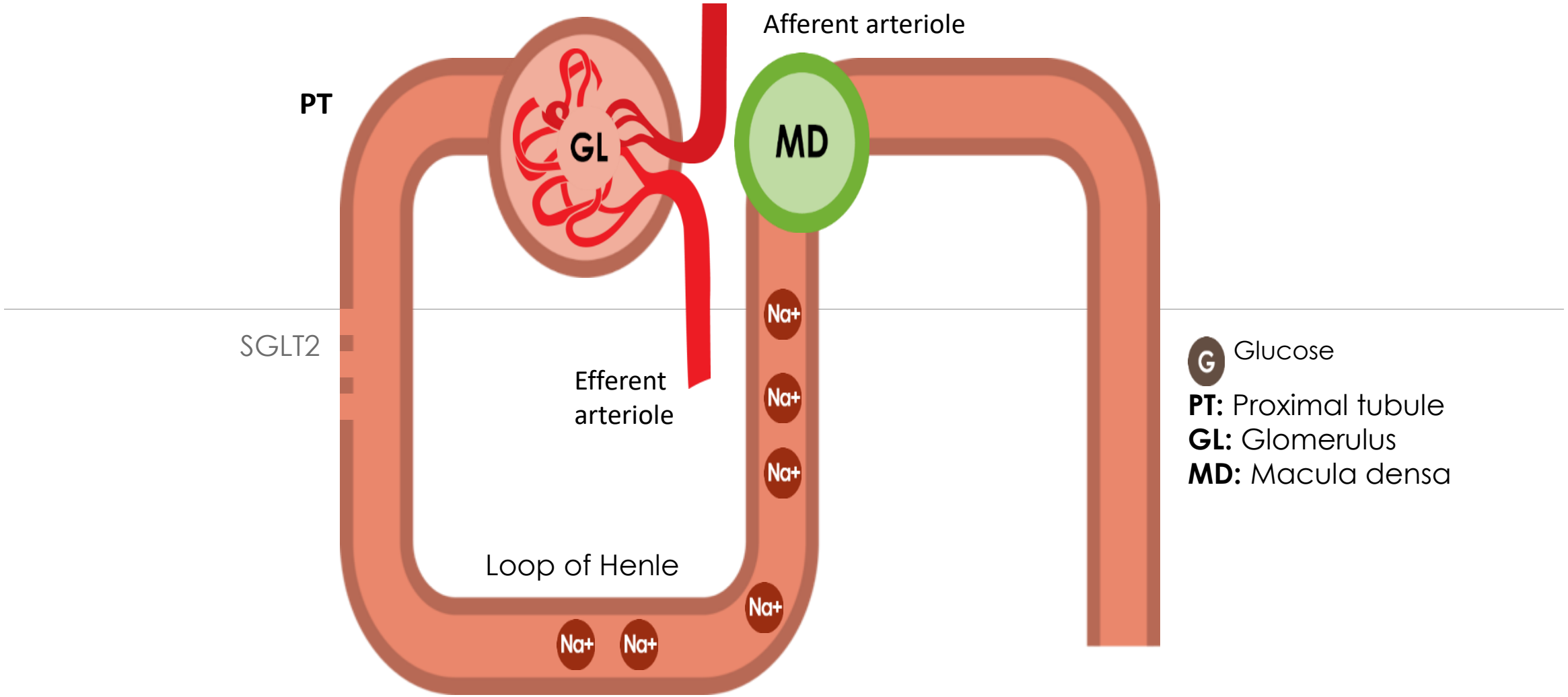
SGLT-2 inhibitors can also downregulate the activity of Na+/H+ exchanger 3 in the proximal tubule, through which, promote natriuresis and reduce heart failure

Also reduce total fat mass and regional adipose tissue distribution

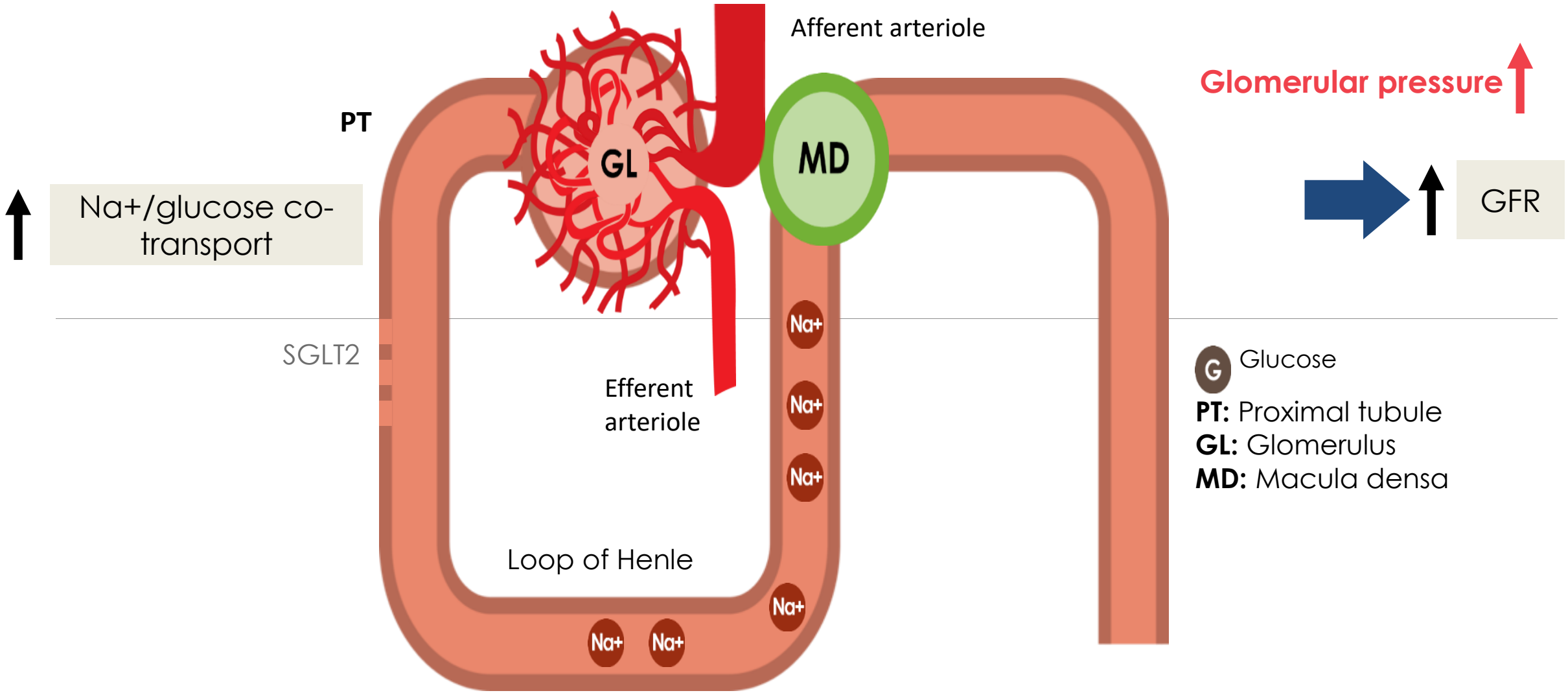
The mechanism of SLGT2's are still not fully explored especially in relation to ↓ CV death

(Gallo et al., 2015; Sattar et al., 2026; Verma et al., 2017; Lytvyn et al., 2017, Baartscheer at al., 2017, Bolinder et al., 2012)

Normal renal blood flow

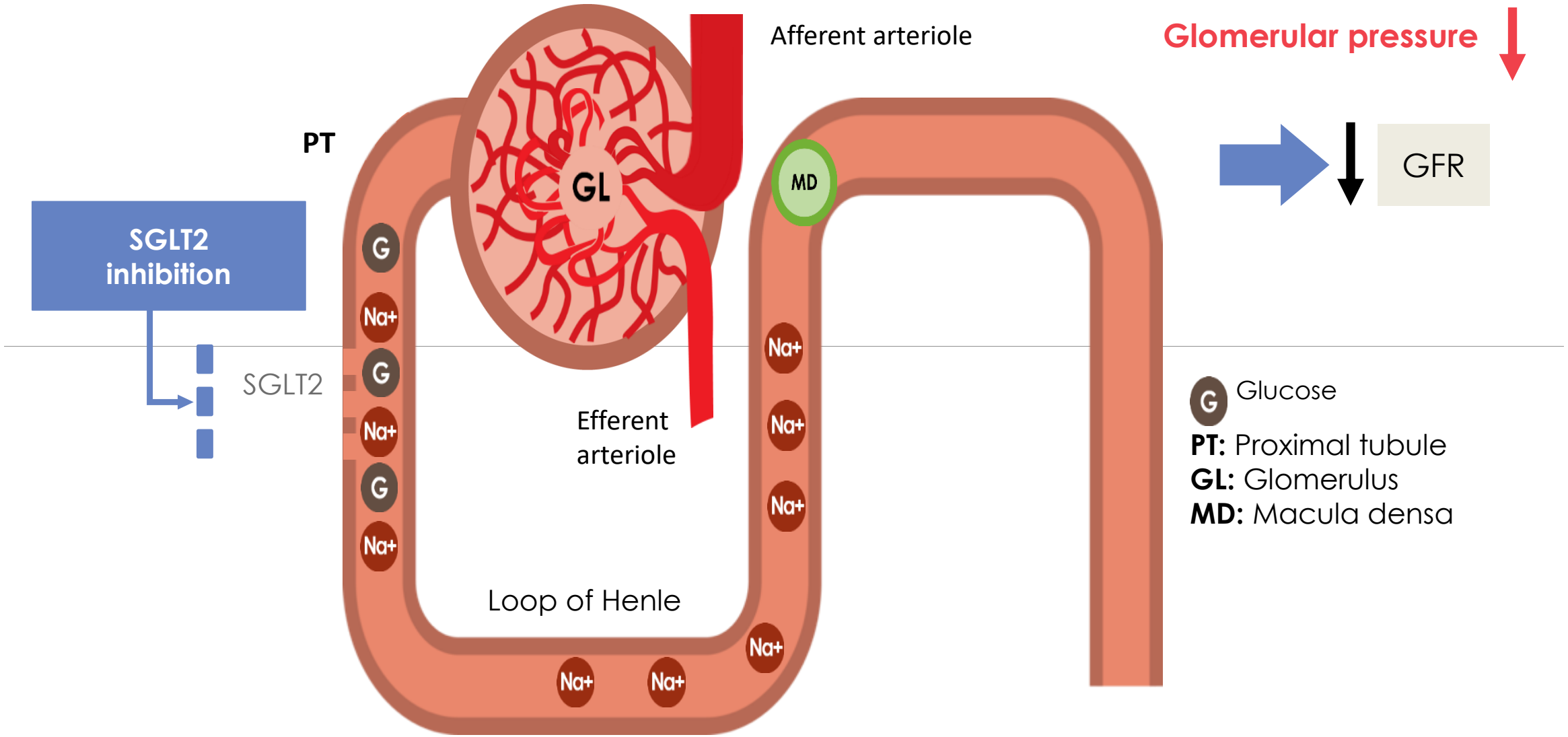


Hyperglycemia increases glomerular pressure

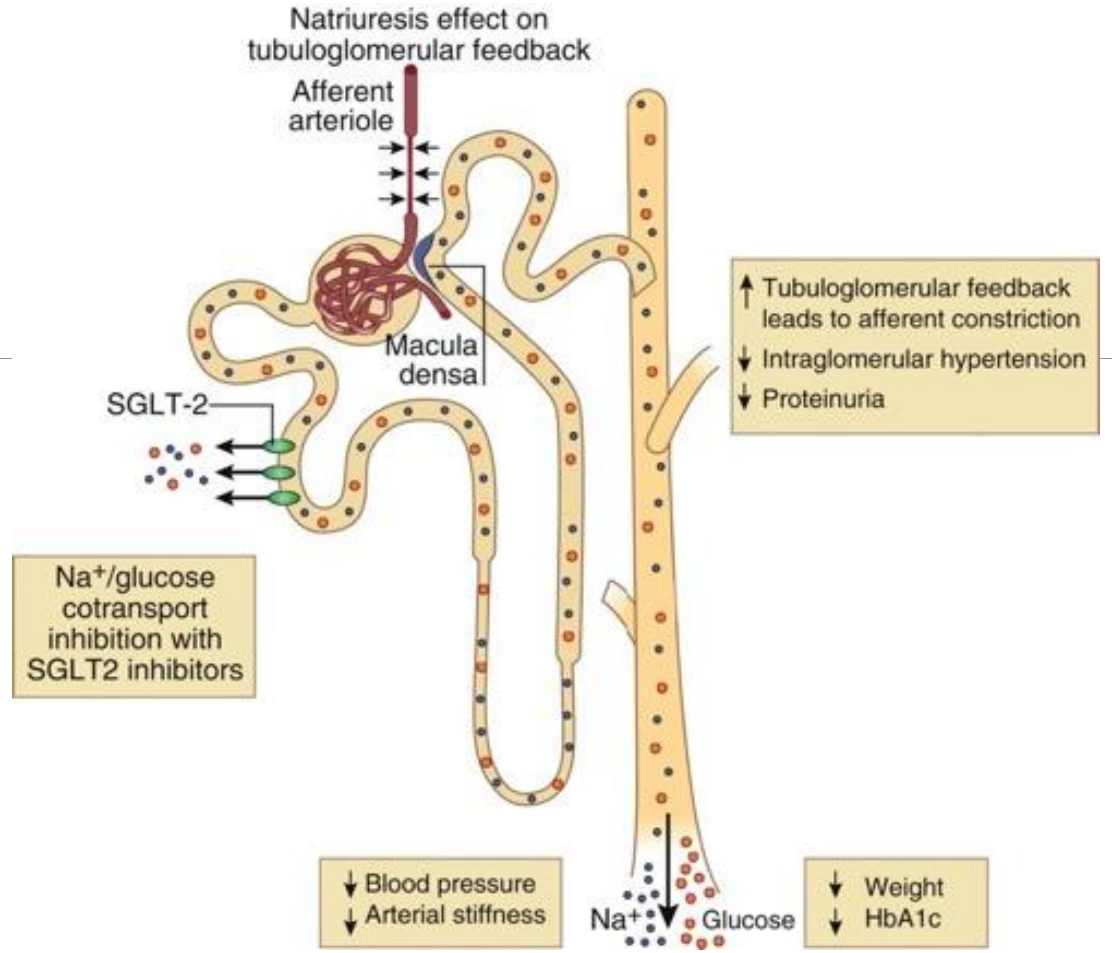


SGLT2 inhibitors reduce glomerular pressure

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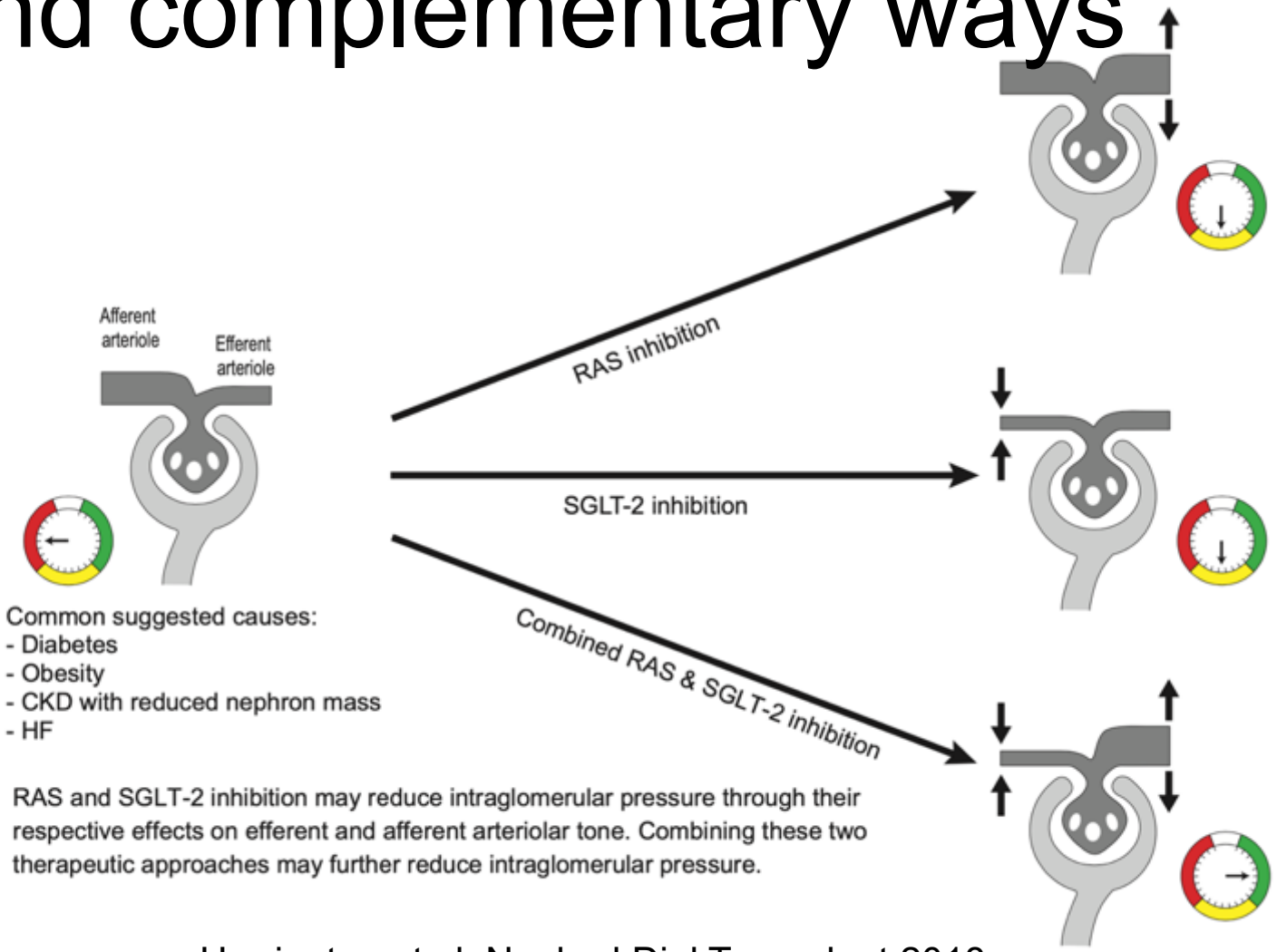


SGLT2 inhibitors work at the proximal tubule & enhance urinary sodium/glucose excretion



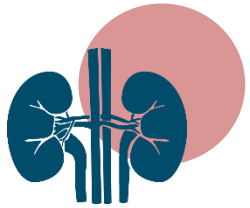
(Heerspink et al., 2018)

ACEi/ARB & SGLT2i work in parallel and complementary ways

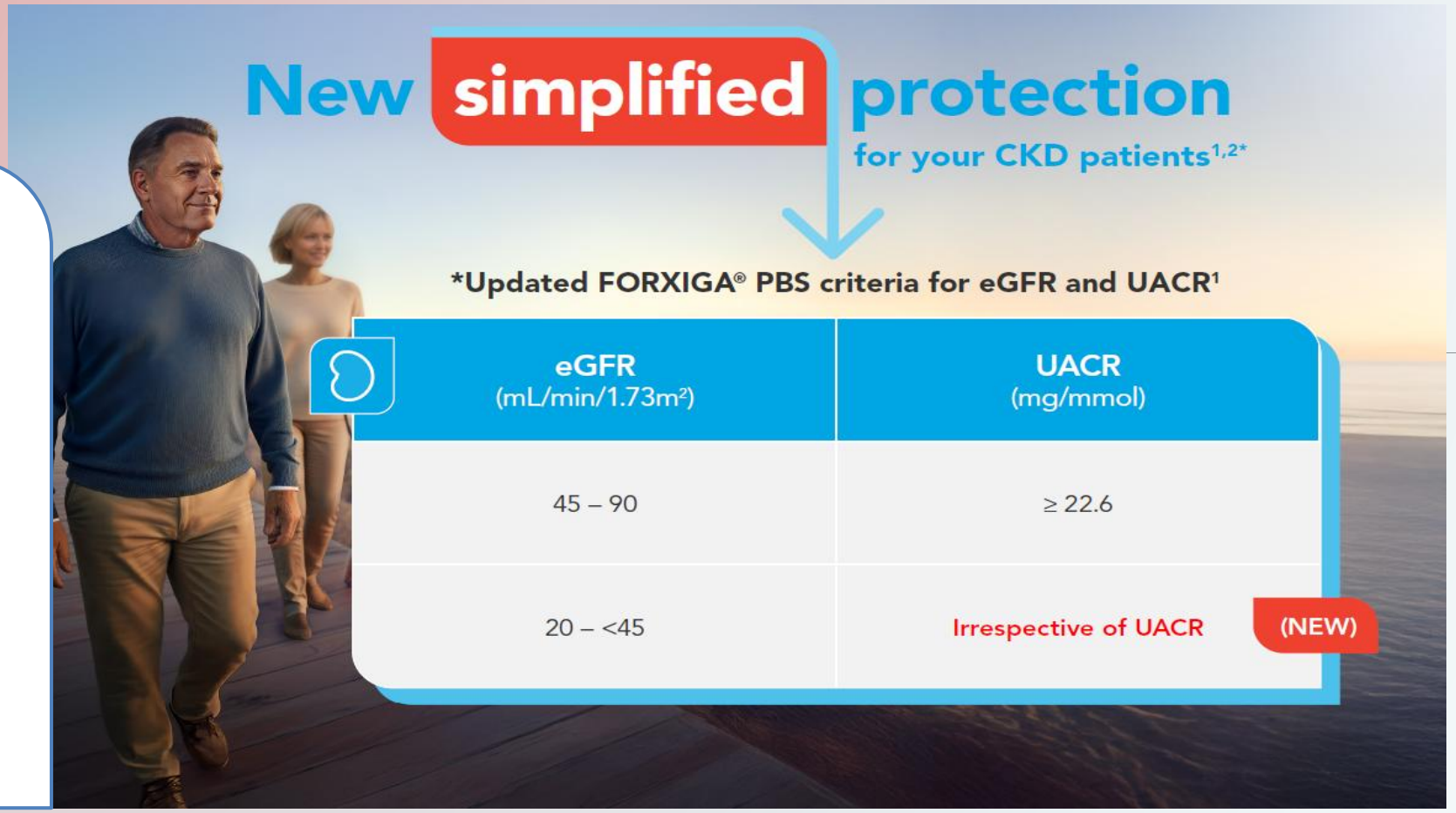


Herrington et al. Nephrol Dial Transplant 2018

FORXIGA® is indicated to reduce the risk of progressive decline in kidney function in adults with proteinuric chronic kidney disease (CKD Stage 2, 3 or 4)[2]



For patients with an eGFR between 20 – <45 mL/min/1.73m, FORXIGA® treatment can now be initiated irrespective of UACR

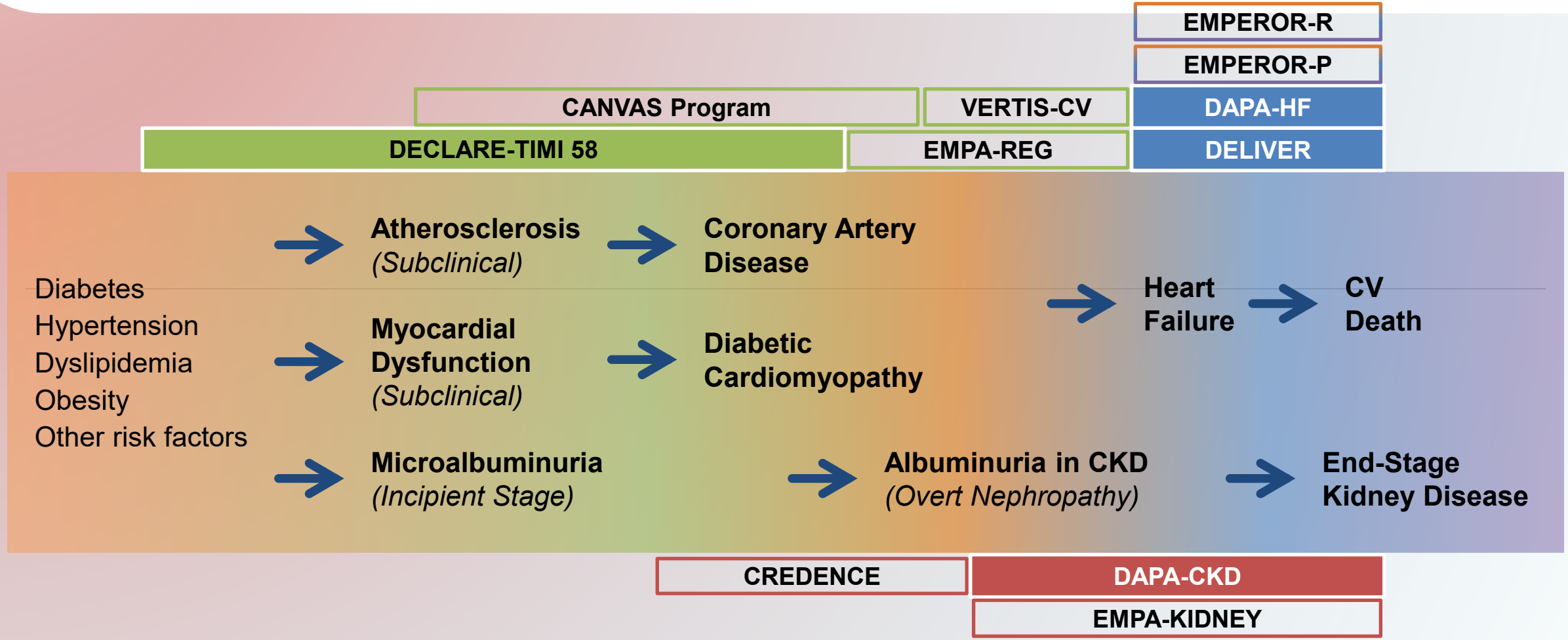


Please refer to PBS website for full Authority Information. Due to limited experience, Initiating treatment below eGFR 25 mL/min/1.73m2 is not recommended.[2]

CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; PBS, Pharmaceutical Benefits Scheme; UACR, urinary albumin-to-creatinine ratio.

References: 1. The Pharmaceutical Benefits Scheme (PBS). PBS website. https://www.pbs.gov.au. **2.** FORXIGA® Approved Product Information.

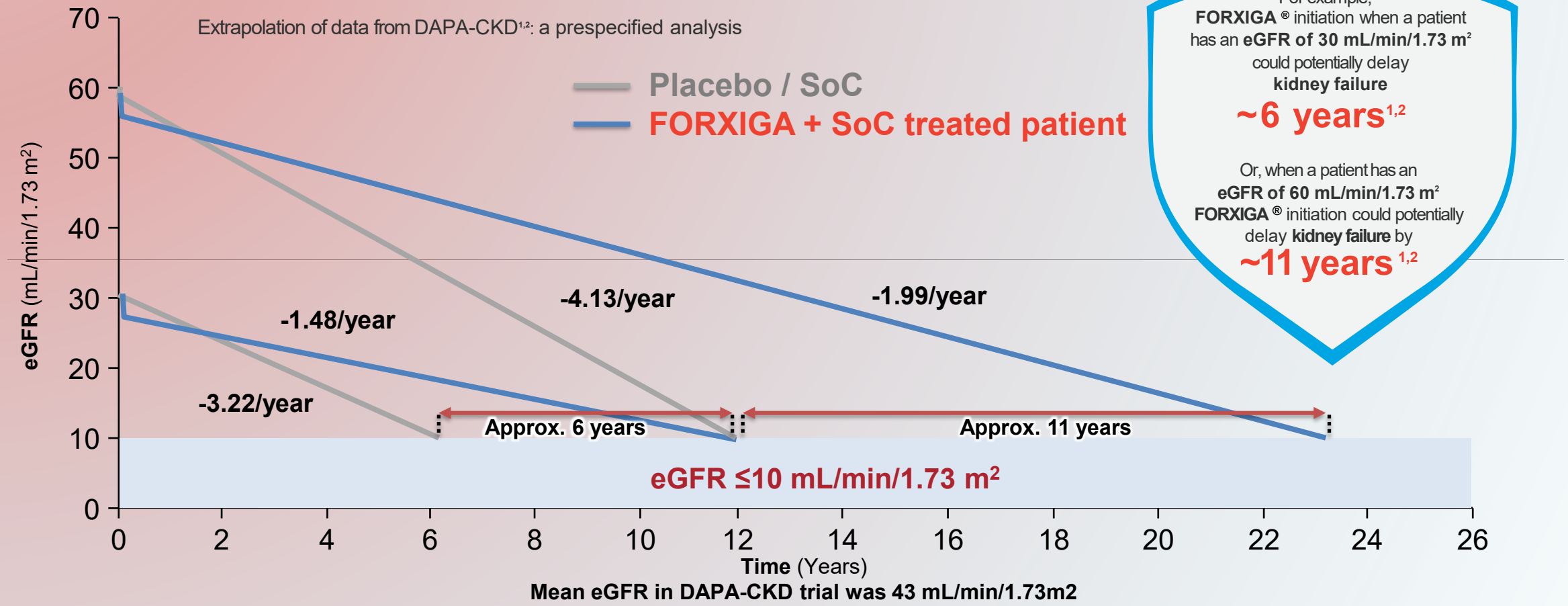
SGLT2is have established a body of clinical evidence from primary prevention T2D patients to those with established CV and/or renal disease[1–5]



Adapted from González-Albarrán O et al. 2022.[1]
Please note canagliflozin and ertugliflozin are not registered in Australia.
CKD, chronic kidney disease; CV, cardiovascular; HF, heart failure; SGLT2i, sodium-glucose co-transporter inhibitor; T2D, type 2 diabetes
References: 1. González-Albarrán O *et al. Diabetes Ther.* 2022;13(Suppl 1):35–49. **2.** Davies MJ *et al. Cardiovasc Diabetol.* 2022;21(1):144. **3.** The EMPA-KIDNEY Collaborative Group *et al. N Engl J Med.* 2023;388(2):117–127. **4.** Anker *SD et al. N Engl J Med.* 2021;385(16):1451–1461. **5.** Solomon *SD et al. N Engl J Med.* 2022;387(12):1089–1098.

Help delay dialysis—initiate SGLT2is earlier[1,2]

Projections from the DAPA-CKD suggests that starting FORXIGA® at a higher eGFR could delay kidney failure for longer vs waiting to start at a lower eGFR[1,2]



Mean eGFR in DAPA-CKD trial was 43 mL/min/1.73m2

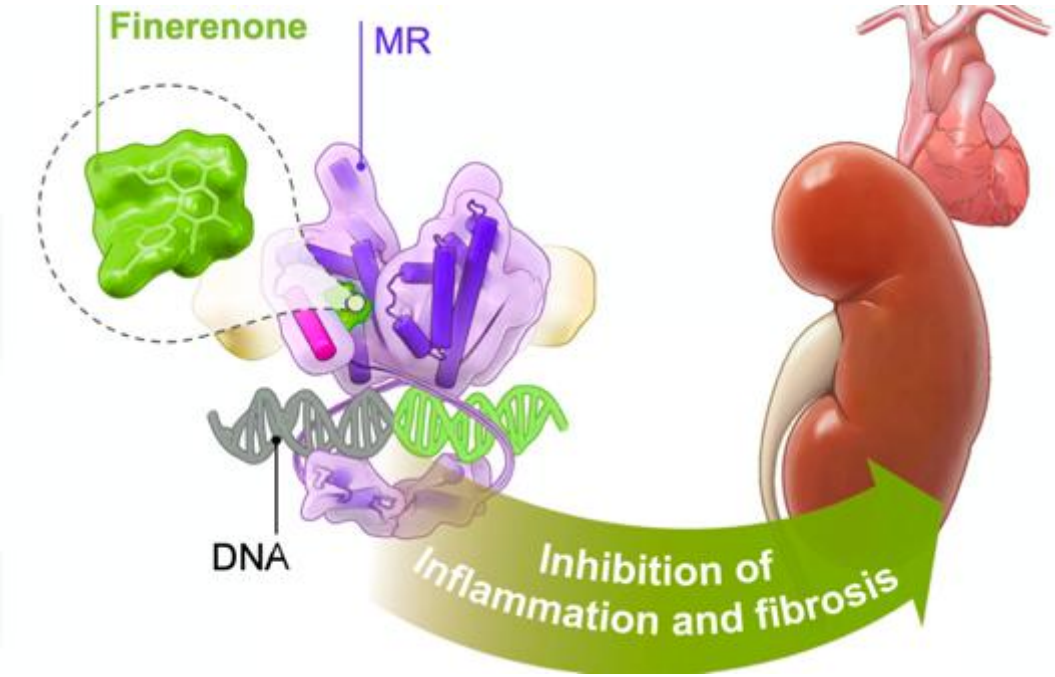
For example,
FORXIGA ® initiation when a patient has an **eGFR of 30 mL/min/1.73 m²** could potentially delay **kidney failure**
~6 years[1,2]

Or, when a patient has an **eGFR of 60 mL/min/1.73 m²** **FORXIGA ®** initiation could potentially delay **kidney failure** by
~11 years [1,2]

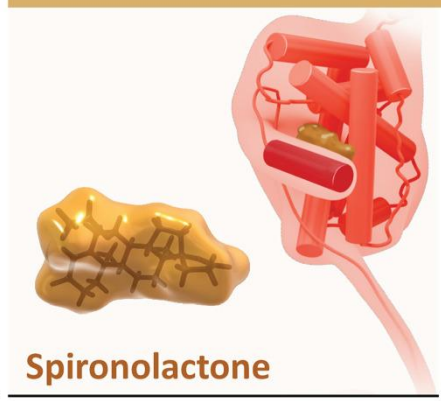
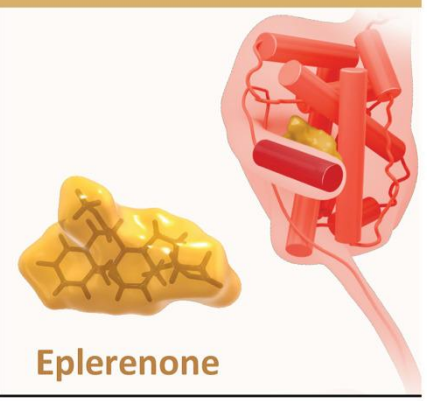
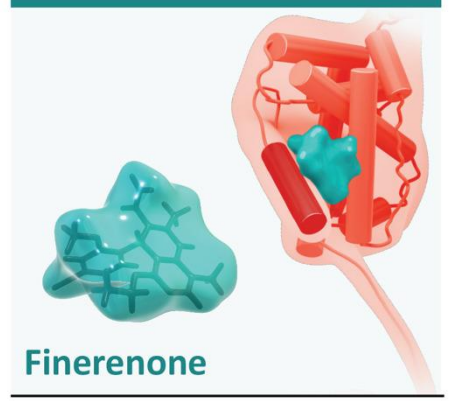
Adapted from Madero M, et al.2
Extrapolated data provides insightful evidence, but robust conclusions should not be deduced on the specific time frame delayed in the absence of clinical studies. Estimates do not consider competing risk of death. Assume that adherence is maintained, that benefits are sustained over time, and that there are no longer-term adverse effects.1,2

References
1. Heerspink HJL et al. Lancet Diabetes Endocrinol. 2021;9(11):743–754.
2. Madero M et al. Kidney Med. 2024;6(8):100851.

Finerenone

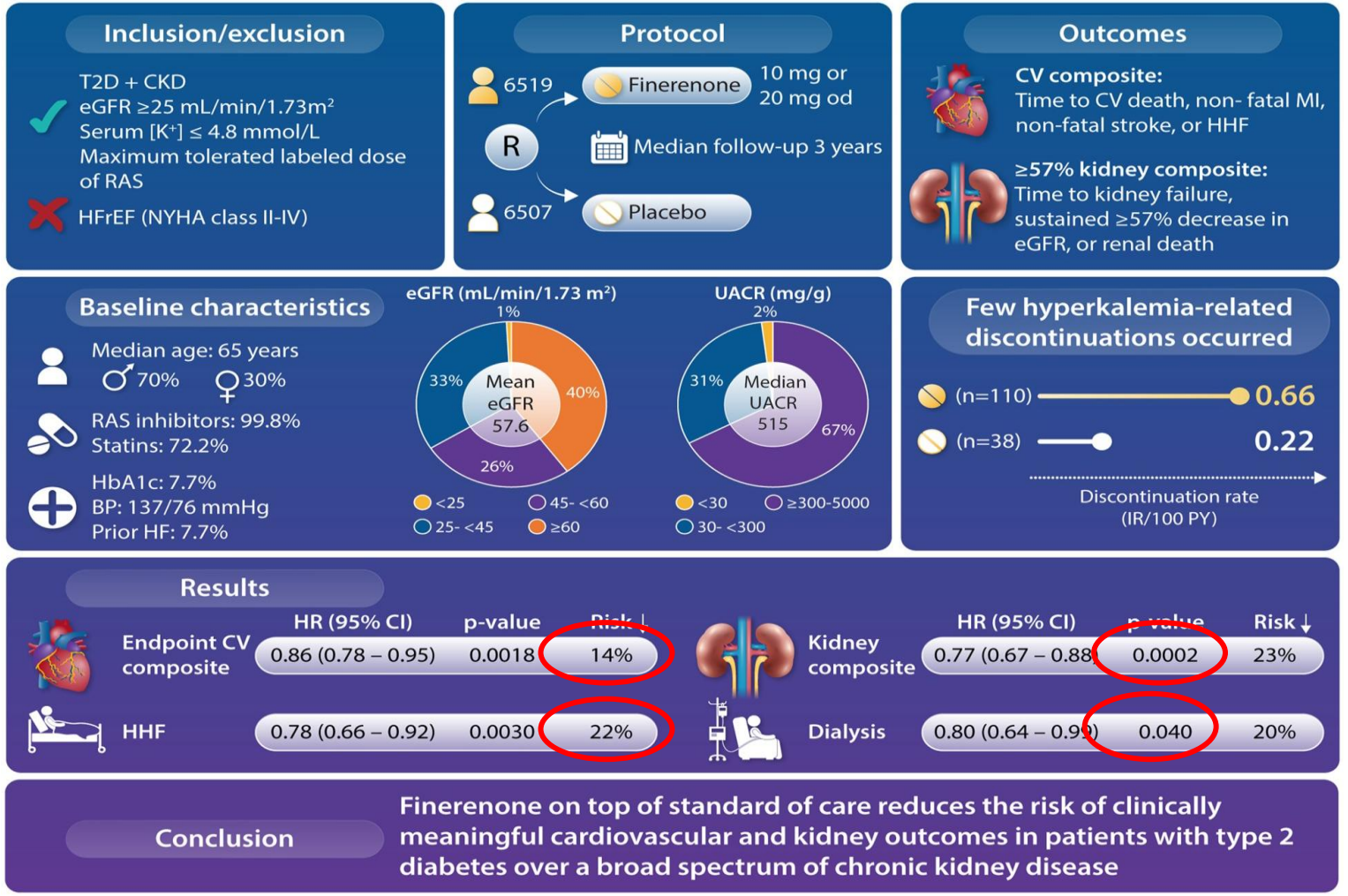


- Finerenone is a novel, selective, **non-steroidal Minerocortiod Receptor Antagonist (nsMRA)** that inhibits inflammation and fibrosis which may contribute to CV/renal protection
- In preclinical models:
  - Greater anti-inflammatory and antifibrotic effects than steroidal MRAs
  - No affinity for androgen and progesterone receptor

	Steroidal MRAs		Finerenone
	 Spironolactone	 Eplerenone	 Finerenone
Structural properties	Flat (steroidal)	Flat (steroidal)	Bulky (non-steroidal)
Potency to MR	+++	+	+++
Selectivity to MR	+	++	+++
CNS penetration	+	+	–
Sexual side effects	++	(+)	–
Half-life	>20 h**	4–6 h**	2–3 h*
Active metabolites	++	–	–
Effect on BP	+++	++	+

- Finerenone less risk of hyperkalaemia than spironolactone

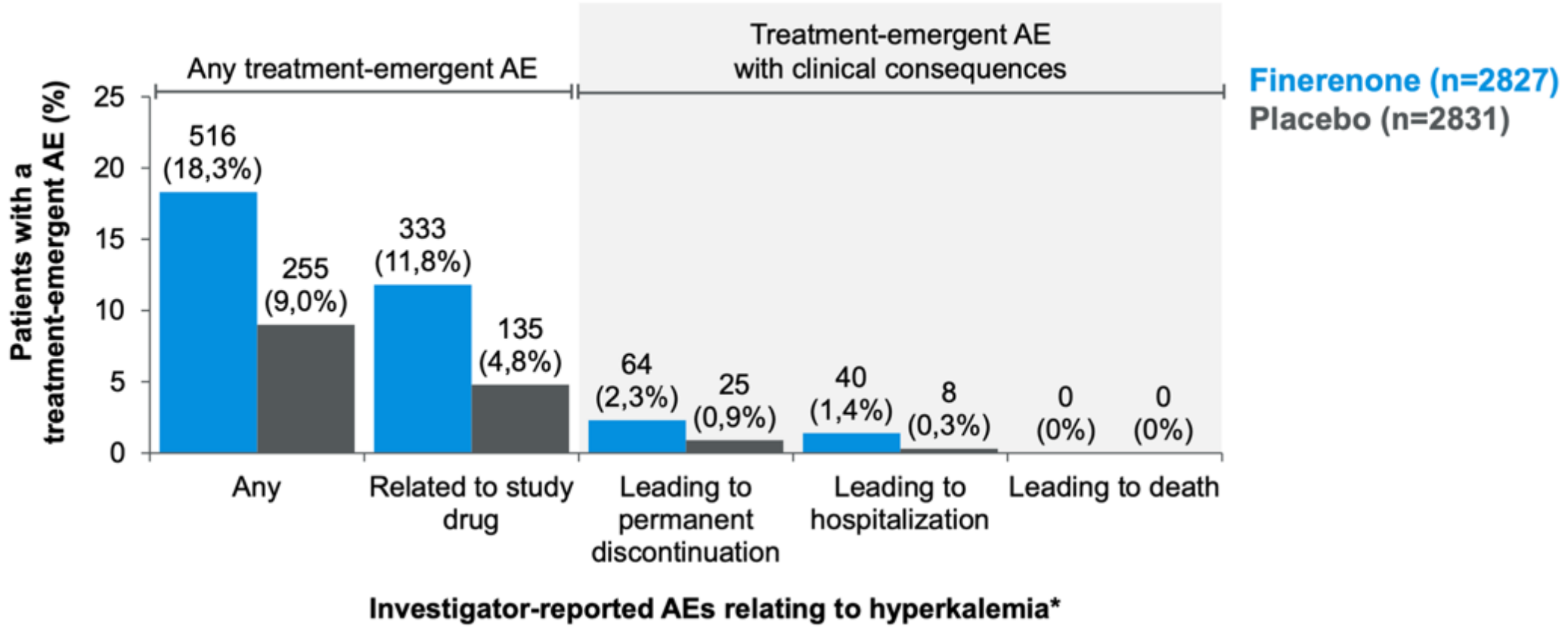
Finerenone reduced the risk of clinically important cardiovascular and kidney outcomes versus placebo across ...



Eur Heart J, Volume 43, Issue 6, 7 February 2022, Pages 474–484, https://doi.org/10.1093/eurheartj/ehab777

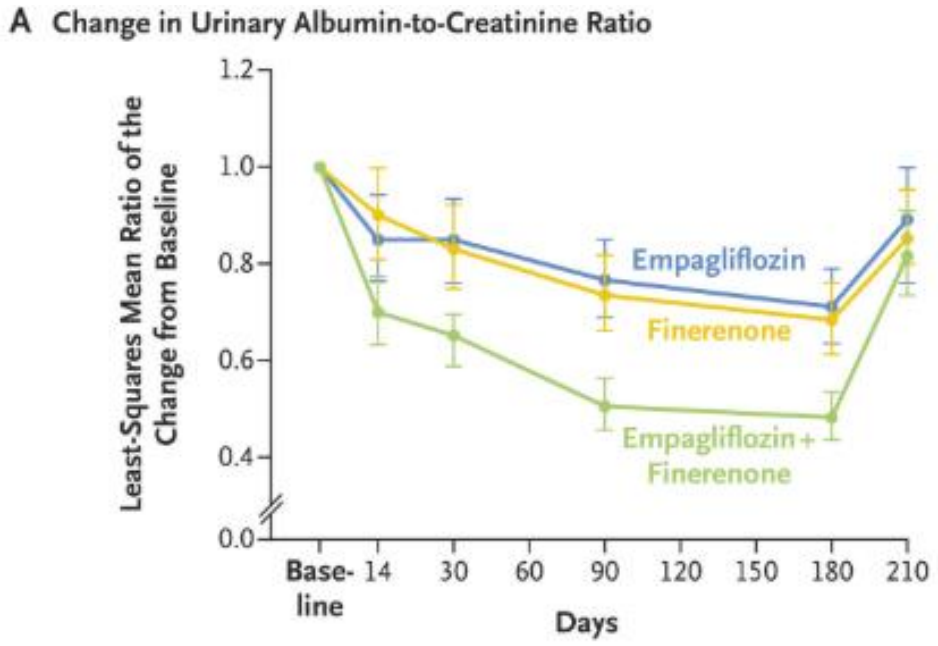
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Finerenone and risk of hyperkalemia

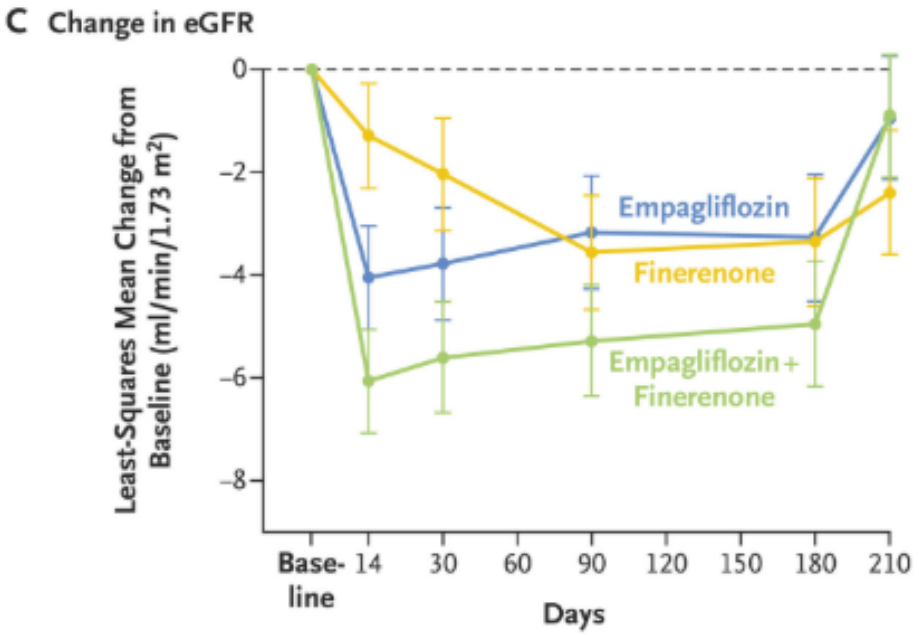


Bakris et al. NEJM 2020

CONFIDENCE – Finerenone with SGLT2 in CKD and T2DM



No. of Patients						
Finerenone	258	247	248	237	236	227
Empagliflozin	261	254	252	246	238	232
Empagliflozin + finerenone	265	248	253	248	240	238



No. of Patients						
Finerenone	262	250	251	243	239	234
Empagliflozin	265	258	255	249	242	243
Empagliflozin + finerenone	269	253	261	254	243	253

JAMA

QUESTION What is the effect of treatment with finerenone on patients with chronic kidney disease due to glomerular diseases?

CONCLUSION In this analysis, treatment with finerenone slowed loss of kidney function and may have an important role in preserving kidney function in patients with glomerular diseases.

© AMA

POPULATION



541 Males
362 Females

Adults with a clinical diagnosis of glomerular disease

Mean age: **51** years

LOCATION



24 Countries and regions

INTERVENTION



1584 Patients with nondiabetic kidney disease randomized

903 Patients with glomerular disease analyzed



446
Finerenone
10 mg or 20 mg of finerenone taken orally once a day

457
Placebo
Matching placebo taken orally once a day

PRIMARY OUTCOME

Total estimated glomerular filtration rate (eGFR) slope, the annual rate of change in eGFR from baseline to month 32

FINDINGS

Total eGFR slope

Finerenone
−3.50 mL/min/1.73 m² per year

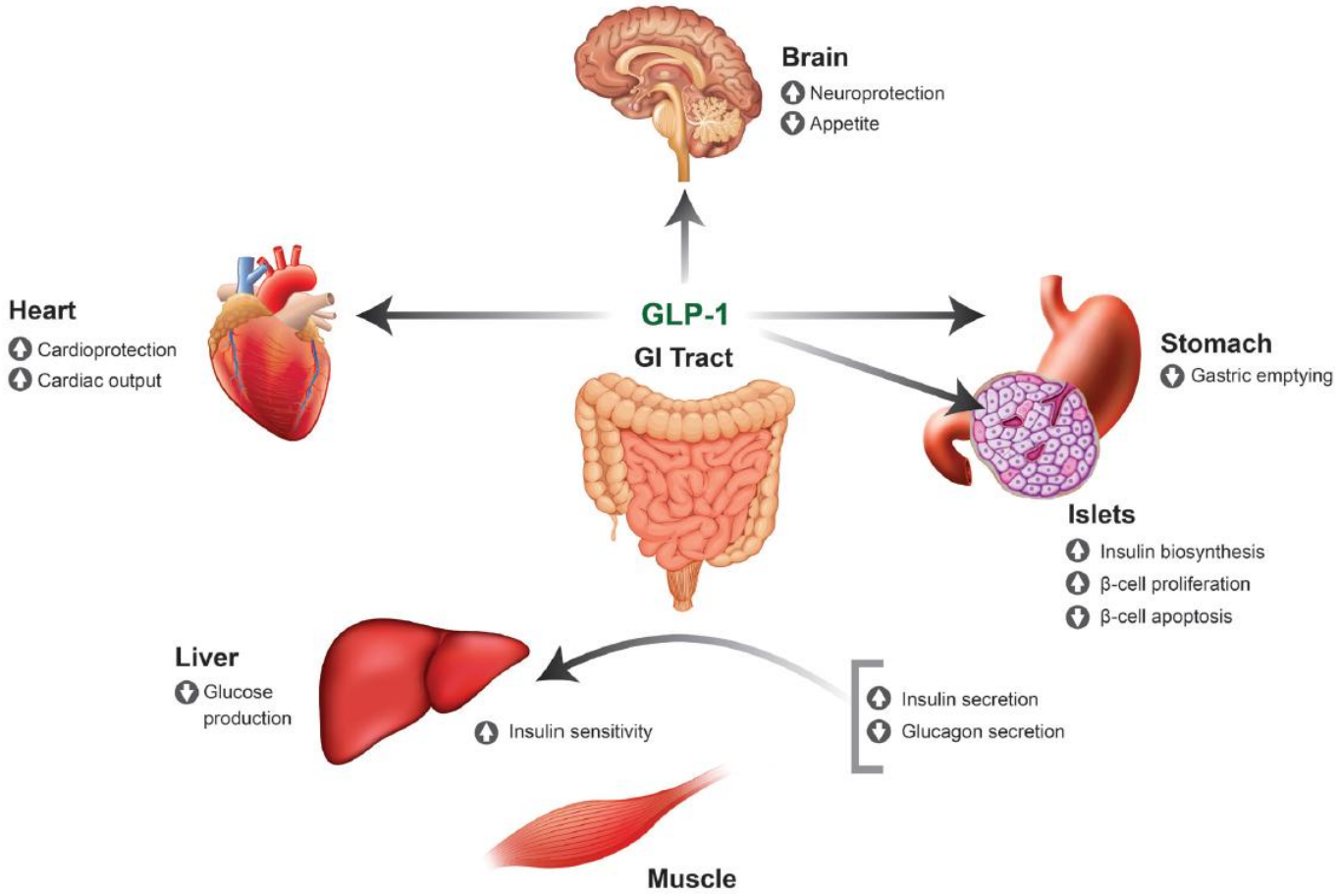
Placebo
−4.23 mL/min/1.73 m² per year

Between-group absolute difference,
0.73 mL/min/1.73 m² per year
(95% CI, 0.22 to 1.24 mL/min/1.73 m² per year)

Finerenone treatment reduced the risk of kidney failure or sustained ≥40% decline in eGFR:
Hazard ratio, 0.74 (95% CI, 0.57 to 0.97)

Neuen BL, Perkovic V, Agarwal R; FIND-CKD Investigators. Finerenone in patients with chronic kidney disease due to glomerular diseases: a randomized clinical trial. *JAMA*. Online June 5, 2026. doi: 10.1001/jama.2026.9923

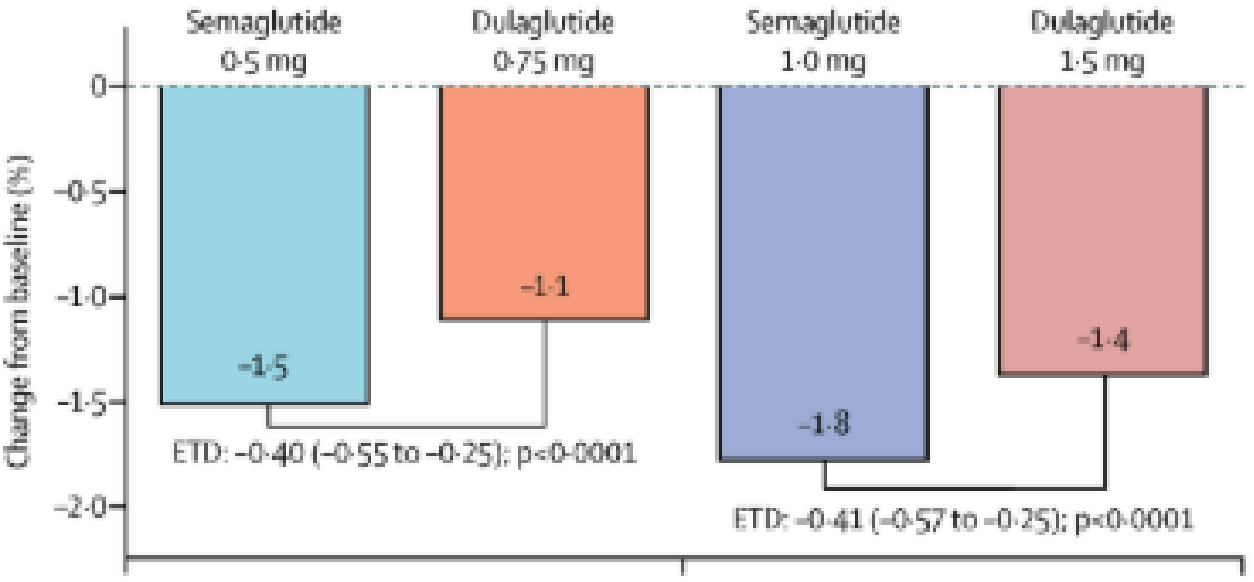
Actions of GLP- 1 inhibitors

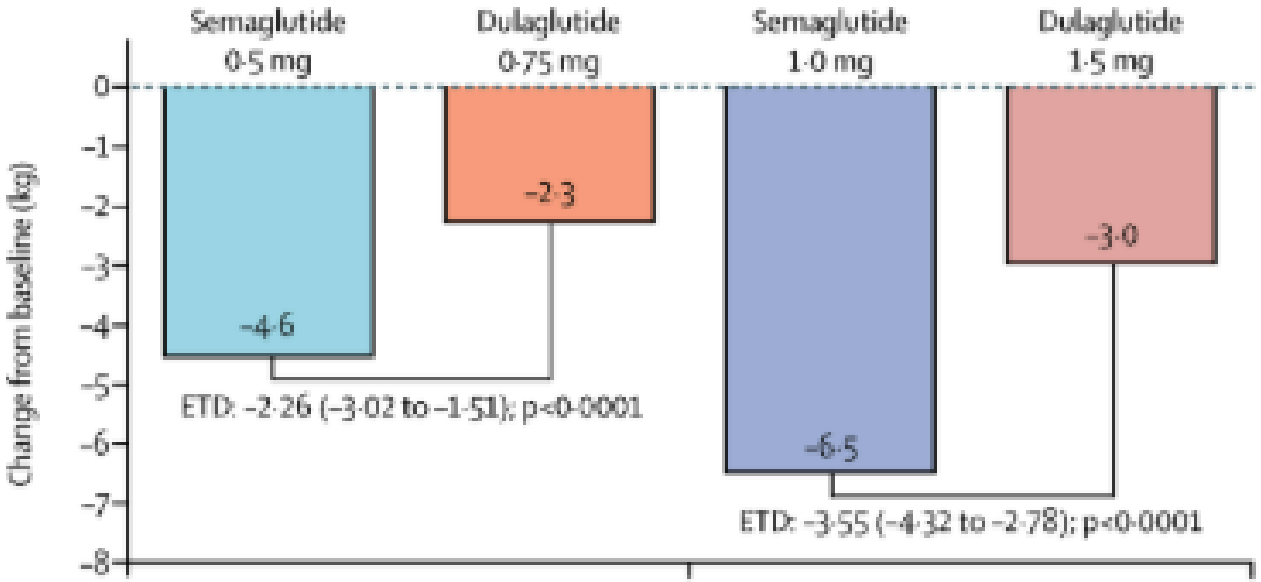


Hinnen D. Glucagon-Like Peptide 1 Receptor Agonists for Type 2 Diabetes. *Diabetes Spectrum*. 2017;30(3):202-210. doi:10.2337/ds16-0026

GLP1RA - SUSTAIN 7

Change from baseline in percentage HbA1C and change in body weight





Semaglutide 1mg mean HbA1c reduction 1.8%

Semaglutide 1mg mean weight reduction 6.5kg

Semaglutide versus dulaglutide once weekly in patients with type 2 diabetes (SUSTAIN 7): a randomised, open-label, phase 3b trial
Pratley, Richard E et al. The Lancet Diabetes & Endocrinology, Volume 6, Issue 4, 275 - 286

RAPID initiation of GDMT initiation is being promoted/debated

- Many CKD and CV events occur early in disease progression
- New approach being explored is initiate foundations therapies early then titrate and individualise
- Approach not new to Cardiologists but is to nephrology practice

Concerns re rapid initiation of multiple treatments was

- AKI
- Hyperkalaemia
- Hypotension
- Excessive eGFR decline

Trial Data demonstrated

- Acceptable safety with monitoring
- Initial drop in eGFR stabilizes over time
- Greater improvement in long term preservation of kidney function

Limitations in rapid commence of GDMT

- Frailty
- Recurrent aKI
- Orthostatic hypotension
- Advanced CKD with hyperkalaemia
- Poly pharmacy
- Elderly patients

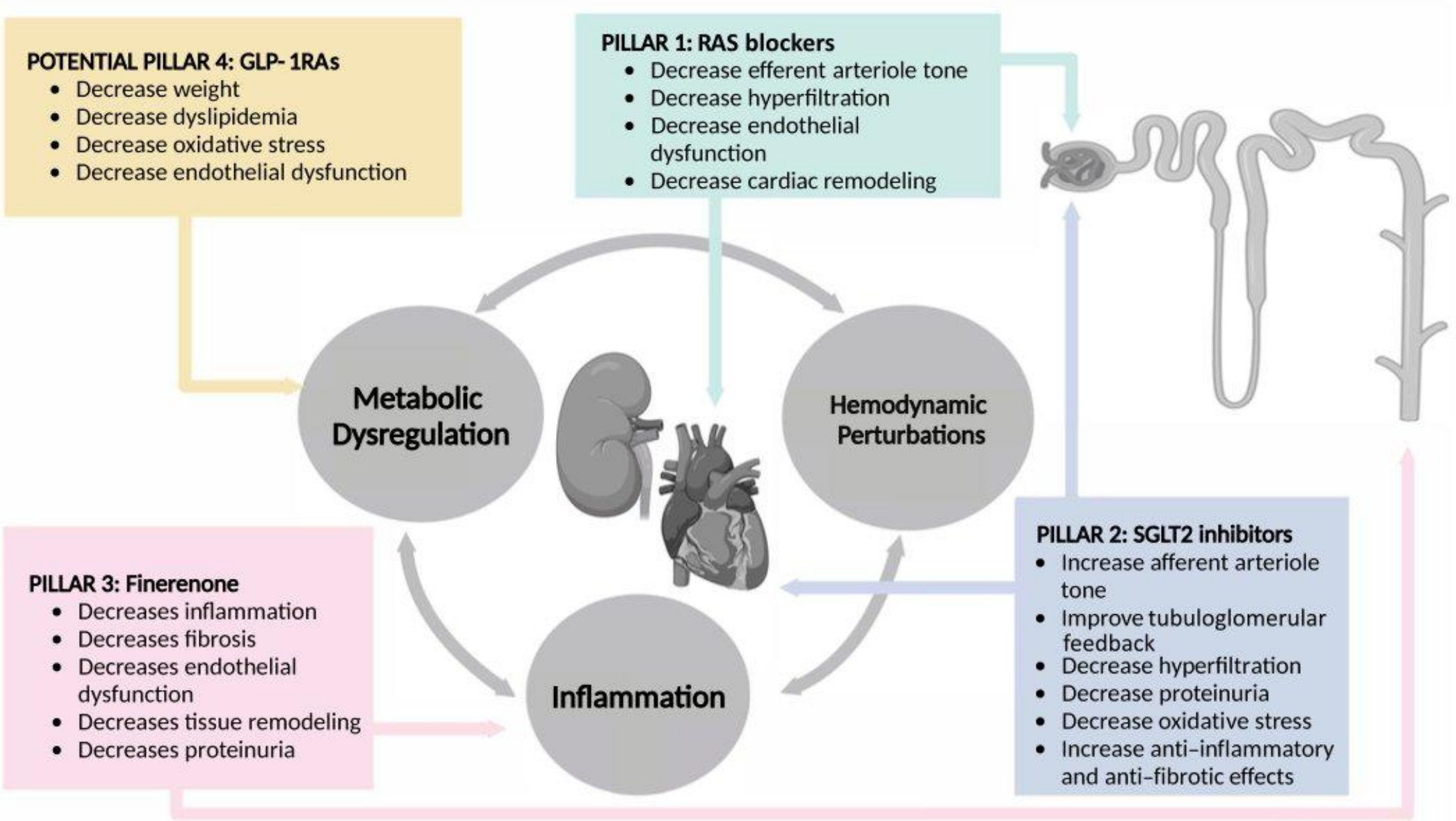
Generally

ARB/ACE & statin

SGLT2 plus nsMRA

GLP1 before if hyperglycaemia

But optimal sequencing yet to be researched in detail


POTENTIAL PILLAR 4: GLP- 1RAs
Decrease weight
Decrease dyslipidemia
Decrease oxidative stress
Decrease endothelial dysfunction
PILLAR 1: RAS blockers
Decrease efferent arteriole tone
Decrease hyperfiltration
Decrease endothelial dysfunction
Decrease cardiac remodeling
Metabolic Dysregulation
Hemodynamic Perturbations
Inflammation
PILLAR 3: Finerenone
Decreases inflammation
Decreases fibrosis
Decreases endothelial dysfunction
Decreases tissue remodeling
Decreases proteinuria
PILLAR 2: SGLT2 inhibitors
Increase afferent arteriole tone
Improve tubuloglomerular feedback
Decrease hyperfiltration
Decrease proteinuria
Decrease oxidative stress
Increase anti–inflammatory and anti–fibrotic effects

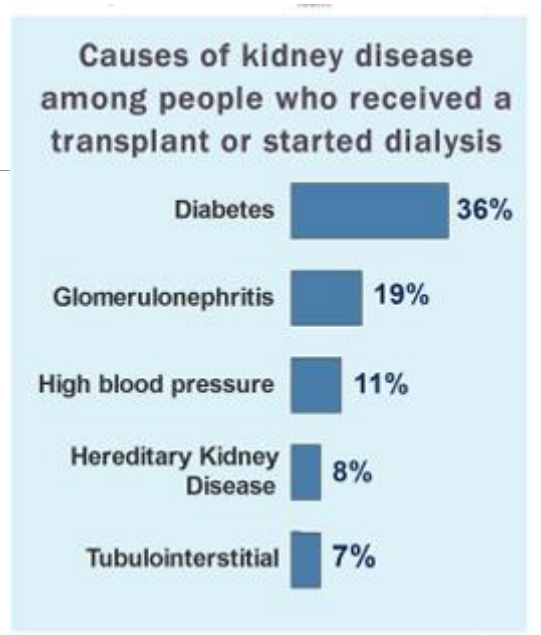
Revolution in Ig A Treatment- New Therapies

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Ig A Nephropathy

- Chronic immune – mediated kidney disease – abnormal IgA antibiodies deposit in glomeruli – causing inflammation and progressive kidney damage
- Most common cause of GN worldwide
- Mechanism – body produces abnormal IgA1 Abs – thought to occur in the gut and saliva– immune system forms Ab against these and complexes deposit in glomeruli –inflammation and scarring
- Disorder of B-cell origin – at least 50% progress to kidney failure or death
- Mediated by B- cell activating factor (BAFF) and proliferation-inducing ligand(APRIL).

Dialysis and Kidney Transplantation in Australia 2024 Summary



Source: ANZDATA. The Australia and New Zealand Dialysis and Transplant Registry (ANZDATA) collects information about people receiving dialysis or kidney transplant for end-stage kidney disease in Australia and New Zealand.

Latest and Emerging Treatments

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Standard Treatment

- ACE/ARB – manages hypertension and proteinuria
- SGLT2 inhibitors – reduce proteinuria

- Corticosteriods in heavy proteinuria

Remission is not achieved with these treatments – slows progression

Referred to as supportive therapies

1. Targeted release Budensonide – First disease specific approved therapies

Goal to reduce abnormal Ig A production upstream

- release budesonide in the distal ileum
- Supress mucosal IgA production
- Trails – NefigArd trial
- Compared with usual steroid treatment – lower systemic steroid exposure , fewer systemic adverse effects

2. Sparsentan – showed greater proteinuria reduction compared with ARB
    - Dual action enothelian receptor blockade
    - ARB blockage
    - Reduced proteinuria predicts better outcomes
    - Multipathway antiproteinuric strategy
    - Monitor LFTs

Three Major Trials targeting Mechanism of Ig A Nephropathy

Renal Society of Australasia

- Revolutionary RCTs – VISIONARY , ORIGIN , APPALAUSE
- Act on APRIL inhibitor – Sibeprenlimab
- BAFF and April inhibitor - Atacicept , Telitacicept
- Complement inhibition - Iptacopan
- Interim analysis of Phase 3 trails found significant effects on proteinuria – longer time frame needed to observe reduction in decline of eGFR
- Initial analysis promising for significantly reducing eGFR decline (less than 1 year in most studies)
- Good safety profile for all treatments

Three Major Trials targeting Mechanism of Ig A Nephropathy

The NEW ENGLAND JOURNAL of MEDICINE

Telitacicept for IgA Nephropathy — Interim Analysis of a Phase 3 Trial

A Research Summary based on Lv J et al. | 10.1056/NEJMoa2514415 | Published on May 14, 2026

TRIAL DESIGN

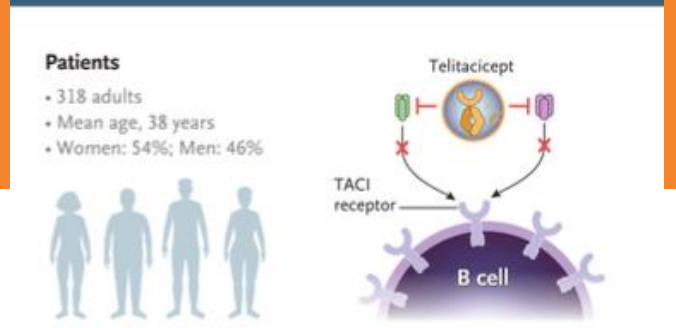
- Phase 3
- Double-blind
- Randomized
- Placebo-controlled
- Location: 72 sites in China

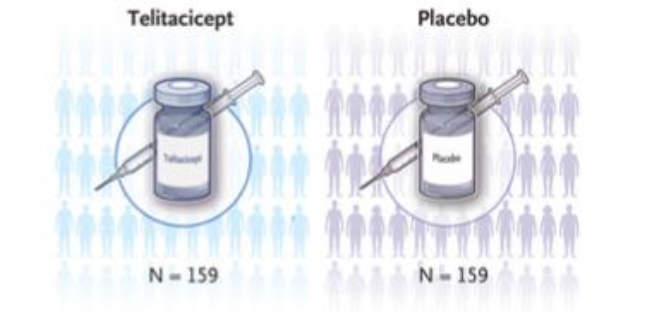
RESULTS

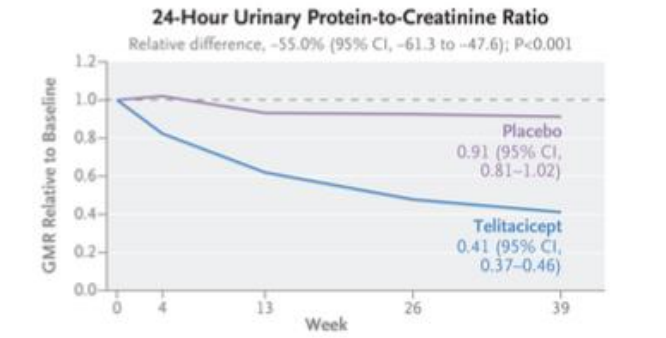
At week 39, the adjusted geometric mean ratio of the 24-hour urinary protein-to-creatinine ratio relative to baseline was 0.41 in the telitacicept group and 0.91 in the placebo group (representing decreases of 58.9% and 8.8%, respectively). The eGFR appeared stable in the telitacicept group and declined in the placebo group. Adverse events occurred more frequently in the telitacicept group than in the placebo group, although serious adverse events were more common with placebo.

LIMITATIONS AND REMAINING QUESTIONS

- These data constitute interim 39-week results; ongoing analyses up to week 104 may enable evaluation of eGFR stability or decline over a longer period.
- The trial population is exclusively Asian, which may limit the generalizability of the results.

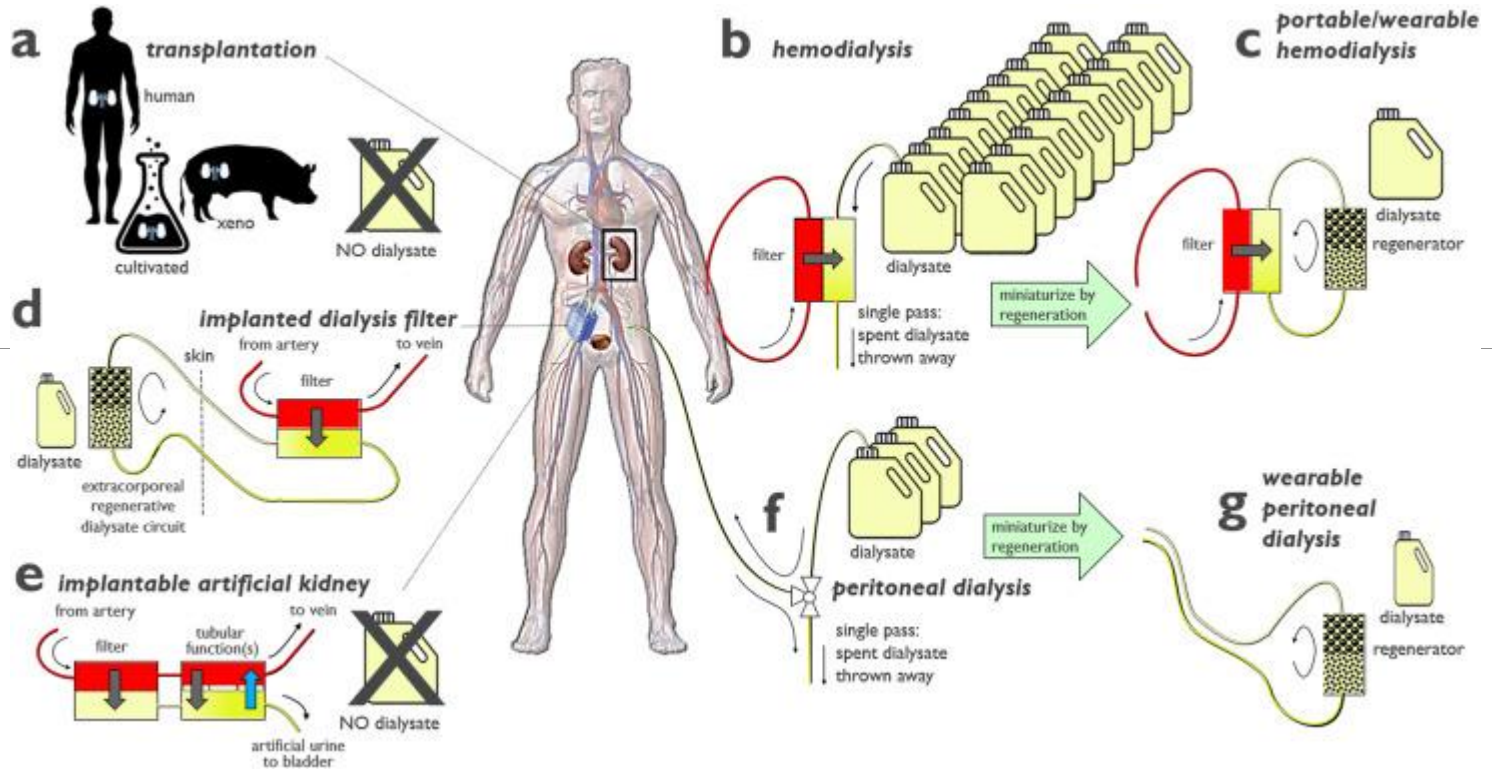








Novel therapies



Wieringa, F. P., et al. (2025). "The Future of Technology-Based Kidney Replacement Therapies: An Update on Portable, Wearable, and Implantable Artificial Kidneys." American Journal of Kidney Diseases **85(6): 787-796.**

Pig kidneys and future of Organ Transplatation

Renal Society of Australasia

- 2025 – Female had Xeno transplant for 130 days
- 2025 – Male had Xeno transplant for 6 months
- Last xeno transplant was 1964 – chimpanzee kidney lasting 9 months
- Two large companies –exploring thymus plus kidney of the pig to re train the immune system

---

- Phase 1 – 2 studies
- Cost is big issue – will it be cheaper than dialysis ??
- Kidneys are grown in sterile facility within pigs which is thought to be resources intensive - ? Zoonotic infections ?
- Plus expensive immune suppressants after – Ecolizumab (approx. $500,000/year)
- Equity of access becomes part of the debate

Reality and Anaemia Management

Renal Society of Australasia

KDIGO Management of Anaemia in CKD Guidelines – 2026. Kidney International (2026) 109 (Suppl 1S), S1–S99

Recommendation 1 – Diagnosis of Anaemia

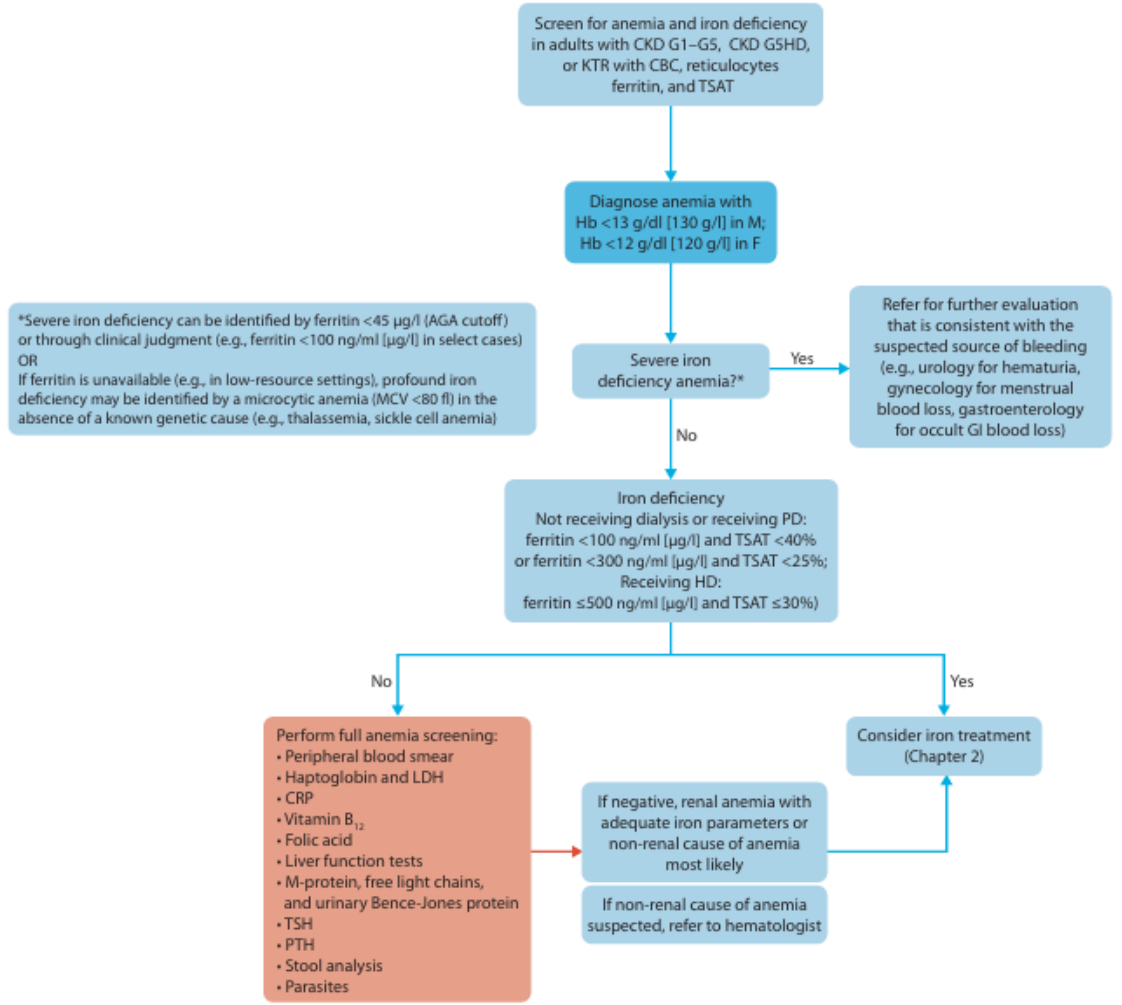


Figure 6 | Flowchart of the different steps to follow when people with chronic kidney disease (CKD) have anemia. AGA, American Gastroenterological Association; CBC, complete blood count; CKD G1–G5, chronic kidney disease not receiving dialysis; CKD G5HD, chronic kidney disease G5 receiving hemodialysis; CRP, C-reactive protein; F, female; GI, gastrointestinal; Hb, hemoglobin; HD, hemodialysis; KTR, kidney transplant recipient; LDH, lactate dehydrogenase; M, male; MCV, mean corpuscular volume; PD, peritoneal dialysis; PTH, parathyroid hormone; TSAT, transferrin saturation; TSH, thyroid-stimulating hormone.

Hb targets

High and low Hb levels[1]

Four international pivotal trials examined high and low haemoglobin concentrations:

1. Normal haematocrit trial[2]
2. CHOIR[3]
3. CREATE[4]
4. TREAT[5]

All demonstrated no clinical benefit to targeting high or low Hb levels

---

CHOIR[3] identified Hb targets >135 g/L were associated with an increased risk of several adverse outcomes including cardiovascular complications and hospitalisations

These studies caused a treatment shift from a specific Hb target for all patients, to an individualised patient approach, considering patients disease state, comorbidities and avoiding targets above 130 g/L[1*]

*Aranesp (darbepoetin) Product Information recommends an Hb target < 120 g/L[6]

1. de Francisco ALM. *NDT Plus* 2010;3:519–26. 2. Besarab A et al. N Engl J Med 1998;339:584–90. 3. Singh AK et al. *New Engl J Med* 2006;255:2085–98. 4. Drueke TB et al. *New Engl J Med* 2006;355:2071–86. 5. Pfeffer MA et al. *New Engl J Med* 2009;361:2019–32. 6. Aranesp Product Information www.amgen.com.au/Aranesp.PI

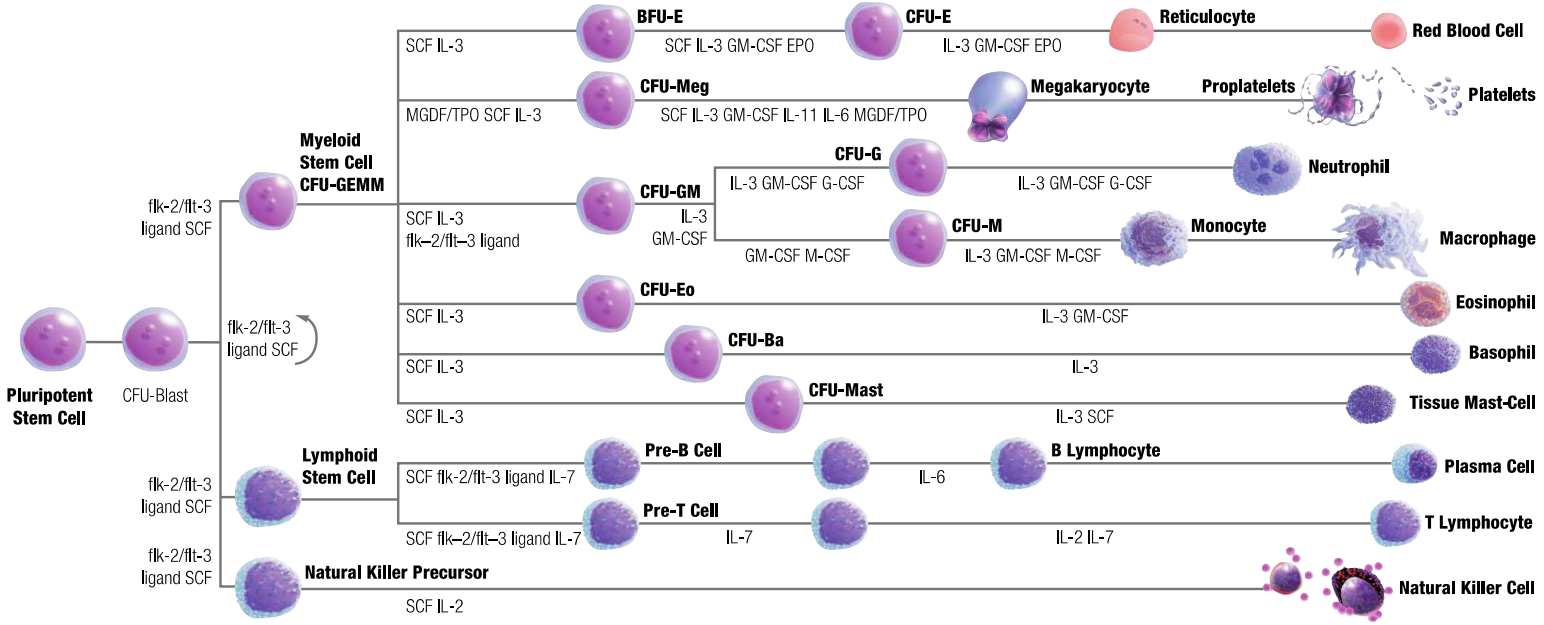
Haematopoiesis

A process of blood cell development and maturation[1,2]

Pluripotent stem cells are present in the bone marrow and give rise to all blood cells

Early in development two cell lineages appear (myeloid and lymphoid)

Red blood cells (RBC) are derived from the myeloid lineage and appear in the blood stream as mature cells



1. Mescher AL. *Junquiera Basic Histology*. Text and Atlas, 12 ed. 2010. 2. Prchal L. Production of erythrocytes. In Prchal JT *et al. Williams Haematology*, 8th ed. 2010.

Erythropoiesis

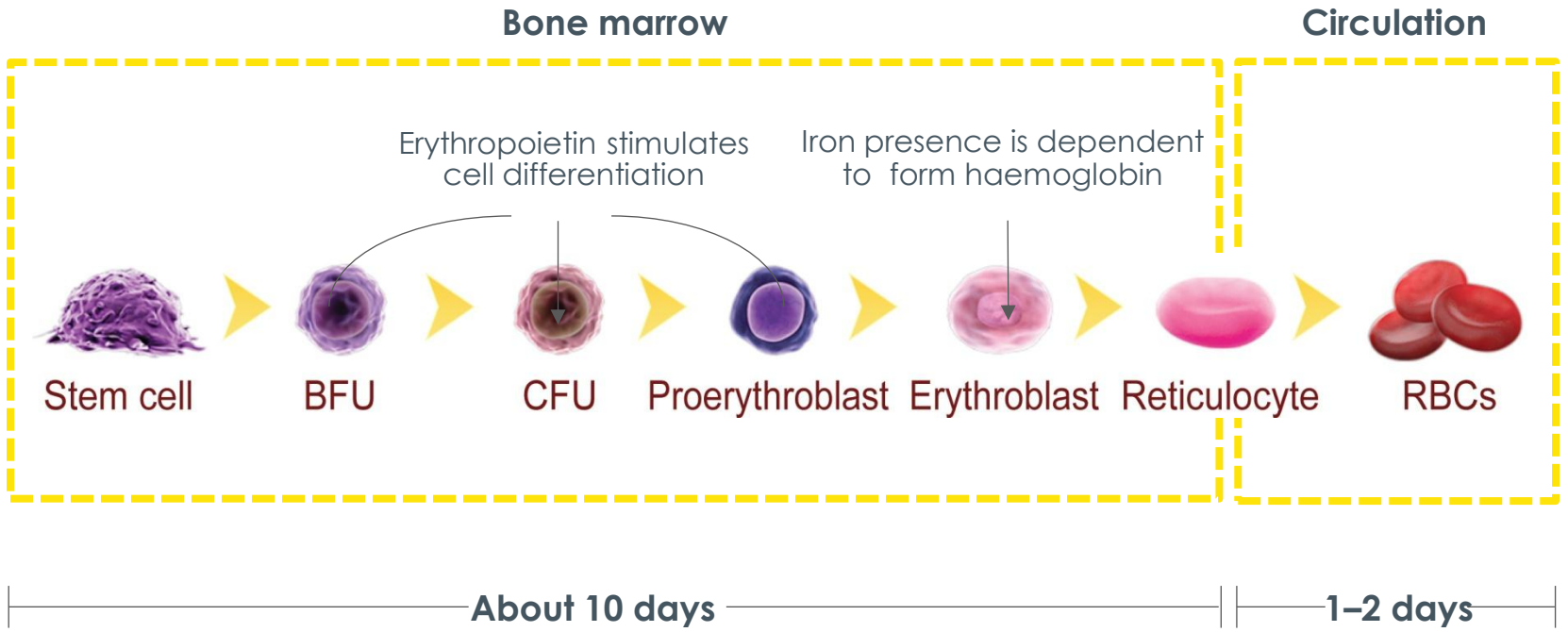
Red blood cells[1,2]

- Densely filled with haemoglobin
- Primary function is to transport oxygen to the body
- Production is regulated by erythropoietin ensuring the correct number of RBC are present
  - 1% of RBC in the circulation need to be replaced daily by young RBC (reticulocytes) to keep the numbers stable
  - Circulating reticulocytes assume the appearance of mature RBC
- Live for approximately 120 days
  - Dialysis patients RBC have a reduced life span ~70 days

1. Mescher AL. *Junquiera Basic Histology*. Text and Atlas, 12 ed. 2010. 2. Prchal L. Production of erythrocytes. In Prchal JT *et al. Williams Haematology*, 8th ed. 2010.

Regulation of RBC production[1,2]

- Erythropoietin modulates the growth and differentiation of RBC
- Iron is required for further RBC maturation including completion



BFU = burst forming unit; CFU = colony forming unit

1. Marieb EN et al. Human anatomy and physiology, 8th ed. 2010. 2. Guyton and Hall. Textbook of Medical Physiology, 12th ed, 2011.

Mechanisms responsible for anaemia of CKD

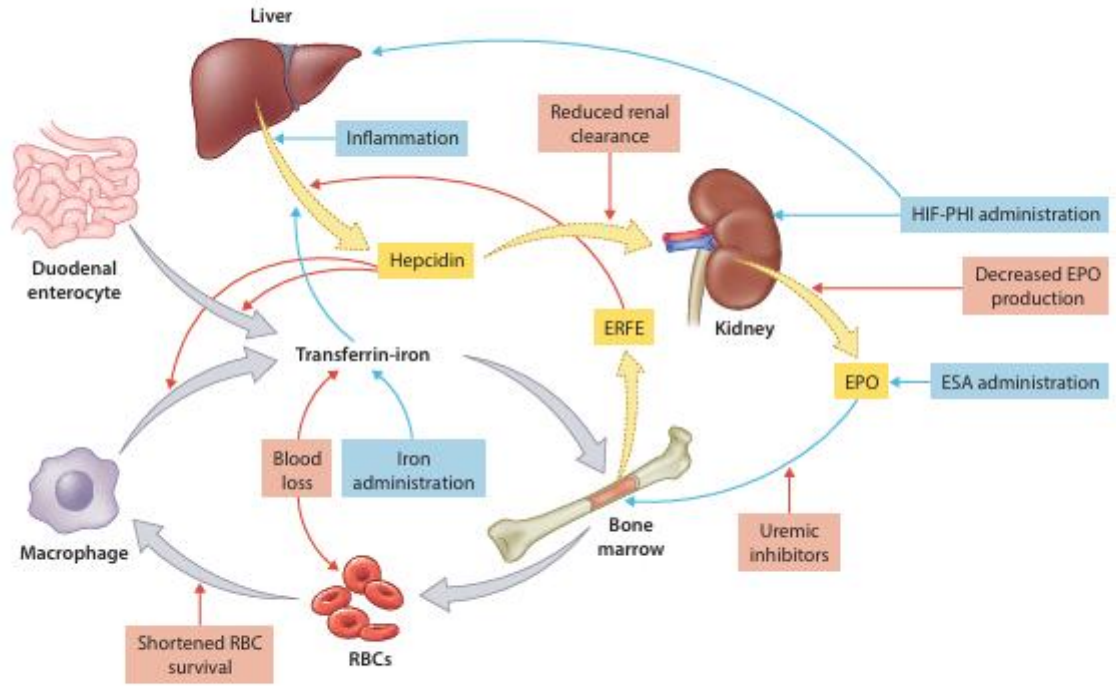


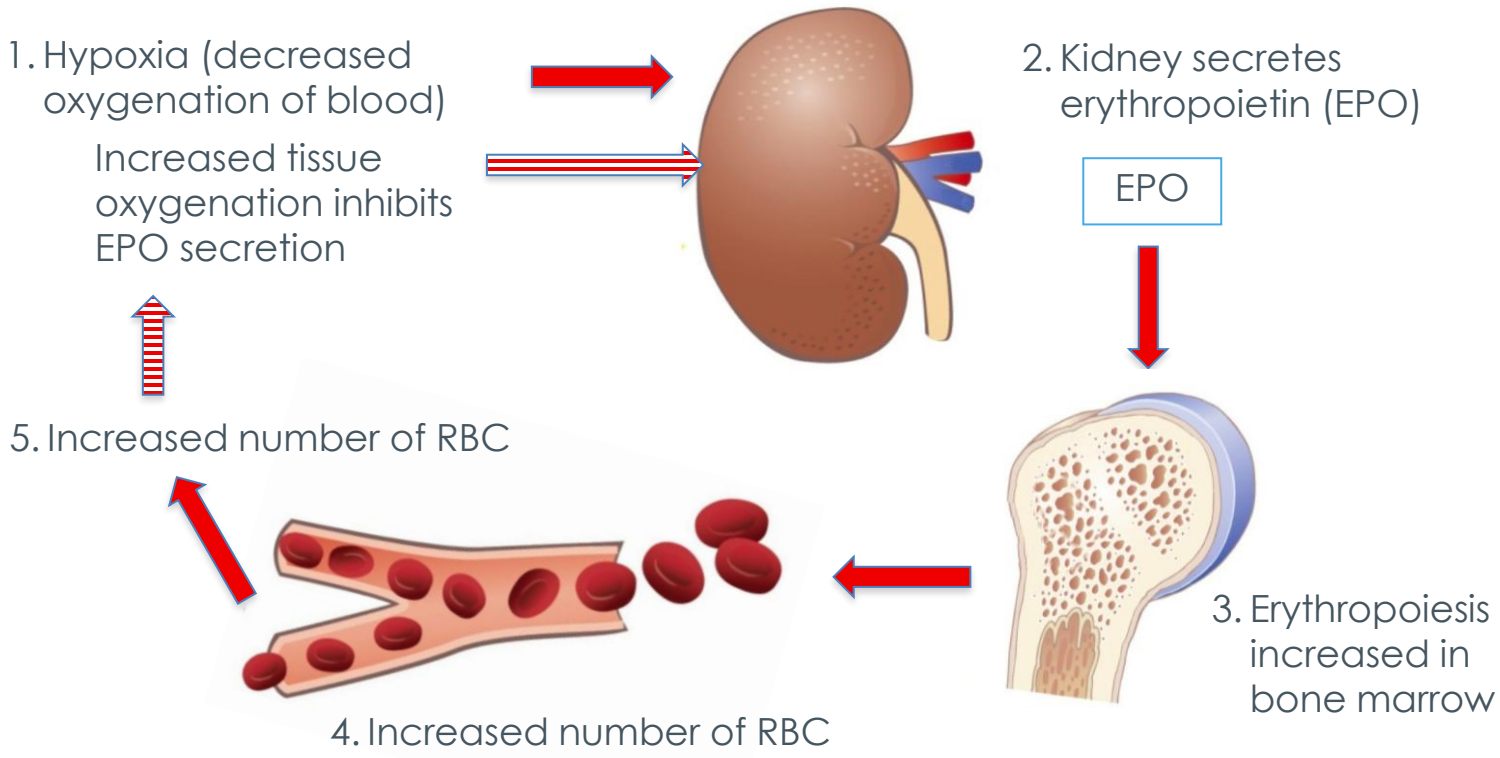
Figure 2 | Mechanisms underlying anemia of chronic kidney disease. The red color arrows indicate inhibitory effects, and the blue arrows indicate stimulatory effects. EPO, erythropoietin; ERFE, erythroferrone; ESA, erythropoiesis-stimulating agent; HIF-PHI, hypoxia-inducible factor–prolyl hydroxylase inhibitor; RBC, red blood cell. Modified from Babitt and Lin.[29]

Kidney International (2026) 109 (Suppl 1S), S1–S99

Regulation of RBC production

Erythropoietin stimulates RBC production[1]

Erythropoietin is the primary regulator of RBC production. It is primarily produced in the kidneys.



1. Marieb EN, *et al. Human anatomy & physiology*, 8th ed. 2010.

Iron management summary

Renal Society of Australasia

In people with anaemia and CKD G5 on HD – suggest initiating iron therapy if ferritin < 500 Ug/l and TSAT , 30% (2D) – use IV iron

In people not on HD or receiving PD – Ferritin < 100ug/l and TSAT < 40% or Ferritin > 100ug/l and < 300ub/l and TSAT , 25%

In people with anaemia and CKD not receiving HD in whom iron is initiated , suggest using oral iron or IV iron based on person's values, preference and degree of anaemia and iron deficiency and relative efficacy, tolerability, availability and cost of each

When do you check iron ?

In people with CKD treated with iron, it is reasonable to test haemoglobin (Hb), ferritin, and TSAT every 3 months for those with CKD not receiving dialysis or CKD G5PD and every 1–3 months for those with CKD G5HD

Events that may warrant more frequent iron testing

- Initiation of or increase of ESAs
- Episode of known blood loss
- Recent hospitalisation

Consider not administering iron in setting of systemic infection

Use of ESAs, hypoxia-inducible factor and other agents

Renal Society of Australasia

Practice Point 3.1.2: In people with anemia and CKD, address all correctable causes of anemia, including iron deficiency, prior to the initiation of treatment with an ESA or a HIF-PHI (Figure 8).

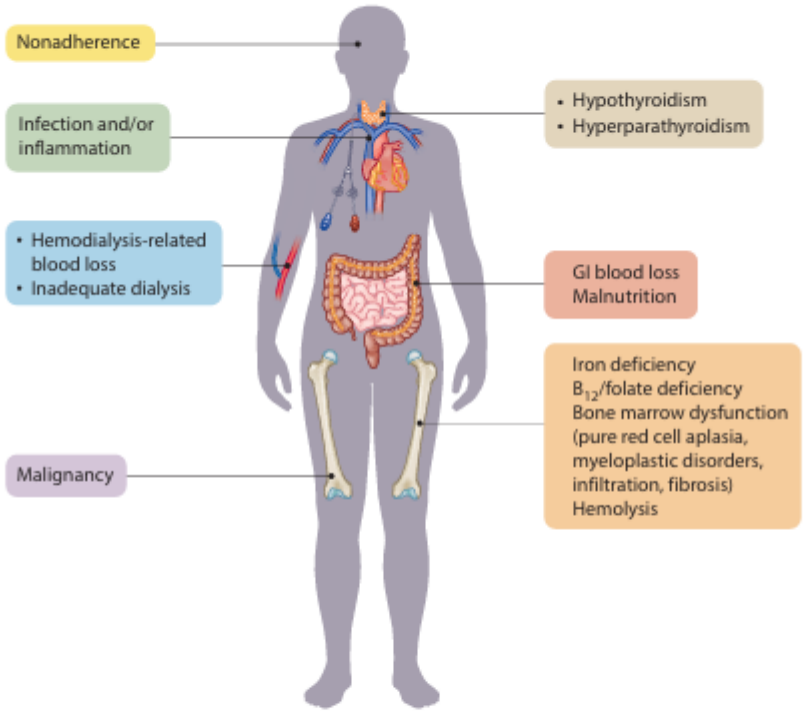


Figure 8 | Potentially reversible causes of anemia in chronic kidney disease in addition to decreased erythropoietin production. GI, gastrointestinal.

Recommendation 3.1.1: In people with anemia and CKD in whom correctable causes of anemia have been addressed, we suggest using an ESA rather than a HIF-PHI as first-line treatment of anemia (*2D*).

Theoretical risk or experimental evidence

- Active cancer or history of cancer not in complete remission – (2-5 yrs)
- ADPKD
- Proliferative retinal disease
- Pulmonary arterial hypertension

Kidney International (2026) 109 (Suppl 1S), S1–S99

ESA initiation

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CKD Grade 5 D – HD or PD start ESA when Hb < 90 – 100 g/l

CKD not on Dx including transplant recipients –

- **Hb of when this is commenced is based on the symptoms and in Australia the PBS criteria – recommendation is target Hb < 115 g/l (1D)**

Recent research into frequency of testing for HD patients

Renal Society of Australasia

Reducing Surveillance Bloodwork in Haemodialysis: Safety, Cost, and Sustainability

NEPHROLOGY

Aim: / Background:

Limited evidence on optimal frequency of surveillance bloodwork for haemodialysis patients.



Aim: To assess the safety, sustainability, and cost of 6-weekly vs. monthly bloodwork.

Methods:

Design: Retrospective cohort study comparing the 12 months before and after practice change in Sept 2023

Outcomes:

- Blood test frequency
- Hospitalisations
- Hyperkalaemia
- % Hb in target
- Cost & environmental impact
- Staff acceptability

Outcomes: / Results:

6-weekly vs. monthly bloods

Blood draws ↓ by 380
Blood tubes ↓ by 870

No ↑ in hospitalisations, hyperkalaemia, or ESA use

For every 100 patients each year

Pathology tests ↓ by 1340
>AUD $20,000 saved

↓ Hb levels between 100-115g/L
Slight ↑ in unplanned Hb tests
Well-accepted by dialysis staff

↓ 0.1 tonnes carbon emissions

CONCLUSION: 6-weekly bloodwork was safe, reduced costs and waste, and had positive environmental impacts. No increase in hospitalisations or clinically significant hyperkalaemia.

Daniel Hirsch, Donia George, Debbie Pugh, *et al.*, 2026

APSN

NPs can prescribe ESA's in Australia !!!!

Renal Society

DARBEPOETIN ALFA

Source S100 HSD Public

Body System BLOOD AND BLOOD FORMING ORGANS > ANTIANEMIC PREPARATIONS > OTHER ANTIANEMIC PREPARATIONS

⚠ Authority Required (STREAMLINED)

6294

Anaemia associated with intrinsic renal disease

Clinical criteria:

- Patient must require transfusion,

AND

- Patient must have a haemoglobin level of less than 100 g per L,

AND

- Patient must have intrinsic renal disease, as assessed by a nephrologist.

Code & Prescriber	Medicinal Product Pack (Name, form & strength and pack size)	Max qty packs	Max qty units	No. of repeats	DPMQ	Max Safety Net	General Patient Charge
5646E (MP) (NP)	DARBEPOETIN ALFA darbepoetin alfa 40 microgram/0.4 mL injection, 0.4 mL pen device (PI, CMI) **Available brands** Aranesp SureClick	8	8	5	$701.60	$25.00	$25.00

Anaemia management with ESA therapy

Iron status should be evaluated before and during ESA treatment as without adequate iron stores ESAs will not be effective[1,2]

Individualisation of ESA treatment is fundamental to anaemia management[3-5]

Hb concentration should be gradually increased to approach a target of ≤115 g/L[2]

- rate of Hb increase should not exceed 10 g/L in any 2-week period[2–4]
- dose adjustments should not be made more often than every 4 weeks[2]

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1. O'Mara NB, *Diabetes Spectrum* 2008;21:12–19. 2. Aranesp Product Information. www.amgen.com.au/Aranesp.PI. 3. Locatelli F, *et al. Nephrol Dial Transplant*. 2013;28(6):1346–59. 4. KDIGO Clinical Practice Guidelines *Kidney Int*. Suppl 2012;2:279–335. 5. McMahon LP et al. *Nephrology* 2012;17:17–9.
6. Kidney Health Australia. CKD Management in General Practice, 3rd ed 2015. www.kidney.org.au accessed November 2017.

How to prescribe ESA's

Renal Society Australasia

Table 7 | Dosing of ESAs

ESA	Initial dose	Dose adjustment[a]
Epoetin alfa and beta	CKD not receiving dialysis: ~50 U/kg once or twice weekly (some use up to 100 U/kg once every 2 wk) (may also round to a convenient dose in units, such as 4000 or 10,000 U, using the lower dose range once or twice weekly and a higher dose range every 2 wk)	CKD not receiving dialysis: Increase or decrease the dose and/or dosing frequency as needed (generally not given more than once weekly)
	CKD G5D: 50–100 U/kg 3 times weekly (may round to a convenient dose in units)	CKD G5D: Increase the dose by 25 U/kg/dose if Hb rise is <1.0 g/dl (<10 g/l) after 4 wk. Decrease the dose by 10–25 U/kg/dose if Hb rise is >2 g/dl (>20 g/l) in 4 wk
Erythropoietin biosimilars	*Product names and doses vary by region (refer to individual product information)*	
Darbepoetin	CKD not receiving dialysis: 0.45 μg/kg weekly or 40–100 μg every 2–4 wk	CKD not receiving dialysis: Increase or decrease the dose and/or dosing frequency as needed (generally not given more than once weekly)
	CKD G5D: 0.45 μg/kg weekly or 0.75 μg/kg every 2 wk (may round to convenient dose: 25, 40, 60, 100, 150, or 200 μg; 300 and 500 μg also available)	CKD G5D: Increase the dose by 25% if Hb rise is <1.0 g/dl (<10 g/l) after 4 wk. Decrease the dose by 25% if Hb rise is >2 g/dl (>20 g/l) in 4 wk
Methyl polyethylene glycol–epoetin beta	CKD not receiving dialysis: 0.6 μg/kg or 50–120 μg every 2 wk, or 1.5 mg/kg or 120–200 μg/kg every month	CKD not receiving dialysis: Increase or decrease the dose and/or dosing frequency as needed (generally not given more than once every 2 wk)
	CKD G5D: 0.6 μg/kg every 2 wk (may round to a convenient dose)	CKD G5D: Increase the dose by 30–50 μg/dose if Hb rise is <1.0 g/dl (<10 g/l) in 4 wk. Decrease the dose by 30–50 μg/dose if Hb rise is >2 g/dl (>20 g/l) in 4 wk

CKD, chronic kidney disease; CKD G5D, CKD G5 receiving dialysis; ESA, erythropoiesis-stimulating agent; Hb, hemoglobin.
[a]Refer to product labeling or dialysis facility protocols for other details of dosing and conversion from an ESA. In general, weight-based dosing is used for children.

ESA response to therapy

Hyporesponsiveness is defined as no increase in Hb concentration from baseline after 1 month of appropriate weight-based ESA therapy[1]

A poor response to an ESA has been shown to be a marker of poor health status[2]

Potential factors that may contribute to hyporesponsiveness include:[1,3]

Easily correctable	Potentially correctable	Unable to correct
Absolute iron deficiency	Infection/inflammation	Haemoglobinopathies
Vitamin B_{12}/folate deficiency	Inadequate dialysis	Bone marrow disorders
Hypothyroidism	Haemolysis	
ACEi/ARB medication	Bleeding	
Non-adherence	Hyperparathyroidism	
	PRCA	
	Malignancy	
	Malnutrition	

ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin-receptor blocker; PRCA = pure red cell aplasia
Table adapted from KDIGO guidelines[1]

1. KDIGO Clinical Practice Guidelines *Kidney Int* Suppl 2012;2: 279-335. 2. McMahon LP *et al. BMC Nephrology* 2012;13:40.
3.de Francisco ALM, *et al. NDT Plus* 2009; 2[Suppl 1]:i18-26.

Conclusion

- GDMT – will change landscape of CKD management
- Promising outcomes for future of people newly diagnosed with IgA
- Novel therapies still long way off – are they really the answer
- Anamia management updated with path testing more environmentally friendly
- Research in relation to testing frequency environmentally friendly and cost effective

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- Wholistic approach should be adopted by all

Thanks for Listening

