Update on Chronic Kidney Disease – Professor Robert Walker

UNIVERSITY of OTAGO

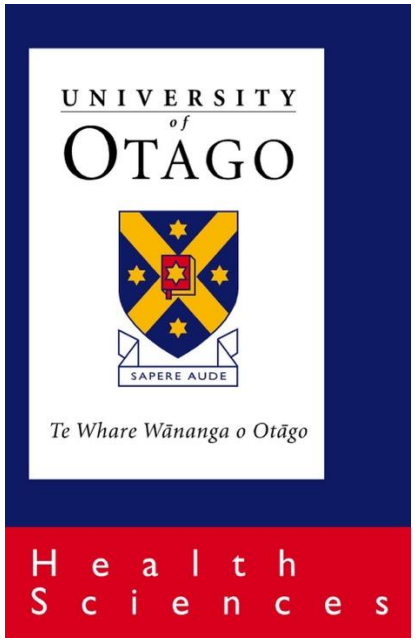
SAPERE AUDE

Te Whare Wānanga o Otāgo

Health Sciences

Conflicts of Interest.

- This workshop is kindly sponsored by Boehringer-Ingleheim.
- The honorarium is paid into my research trust funds.



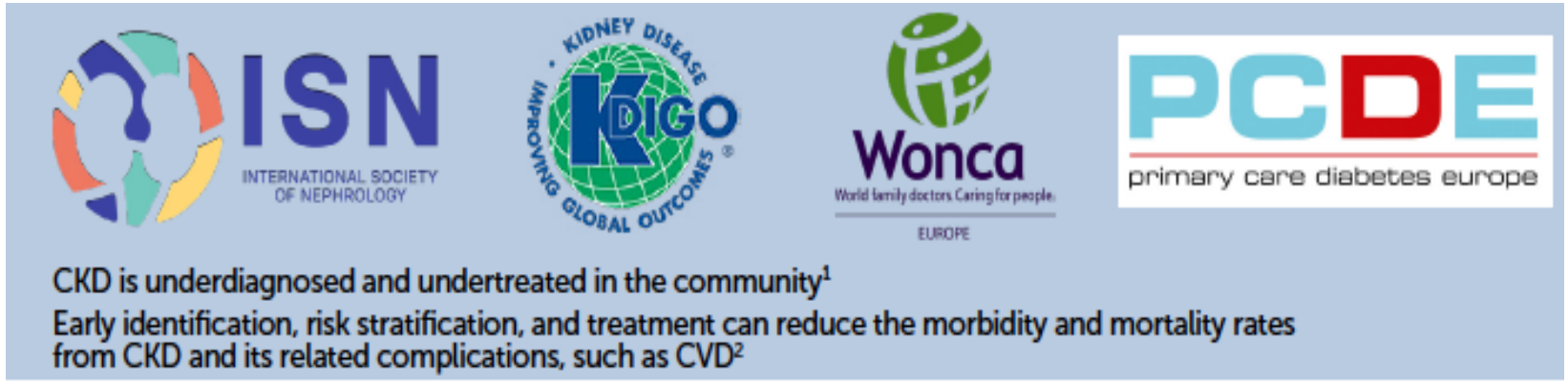
Illustrative Case

- 58 year Māori male with a history of hypertension for 15 years, ischaemic heart disease, T2DM for 5 years and chronic kidney disease.
- BP 156/98, UACR 98mg/mmol, serum creatinine 140umol/l, eGFR 47 ml/min/1.73m^2, HbA1c 63
- 5year Cardiovascular risk 37% ESKD risk 13%

Chronic Kidney Disease and Increased Cardiovascular Risk

Extent of the problem
- identification of CKD
- risk of cardiovascular events

Key management.



Chronic Kidney Disease (CKD)

Early Identification and Intervention in Primary Care

Step 1	Main clinical risk factors for CKD:	Consider other factors:
Identify individuals at risk	• Hypertension • Diabetes • CVD • Family history of CKD	• Systemic disease affecting the kidneys (e.g. SLE) • Obesity • Genetic risk factors (e.g. ADPKD) • Environmental exposures to nephrotoxins • Demographics – older age, race/ethnicity • History of AKI

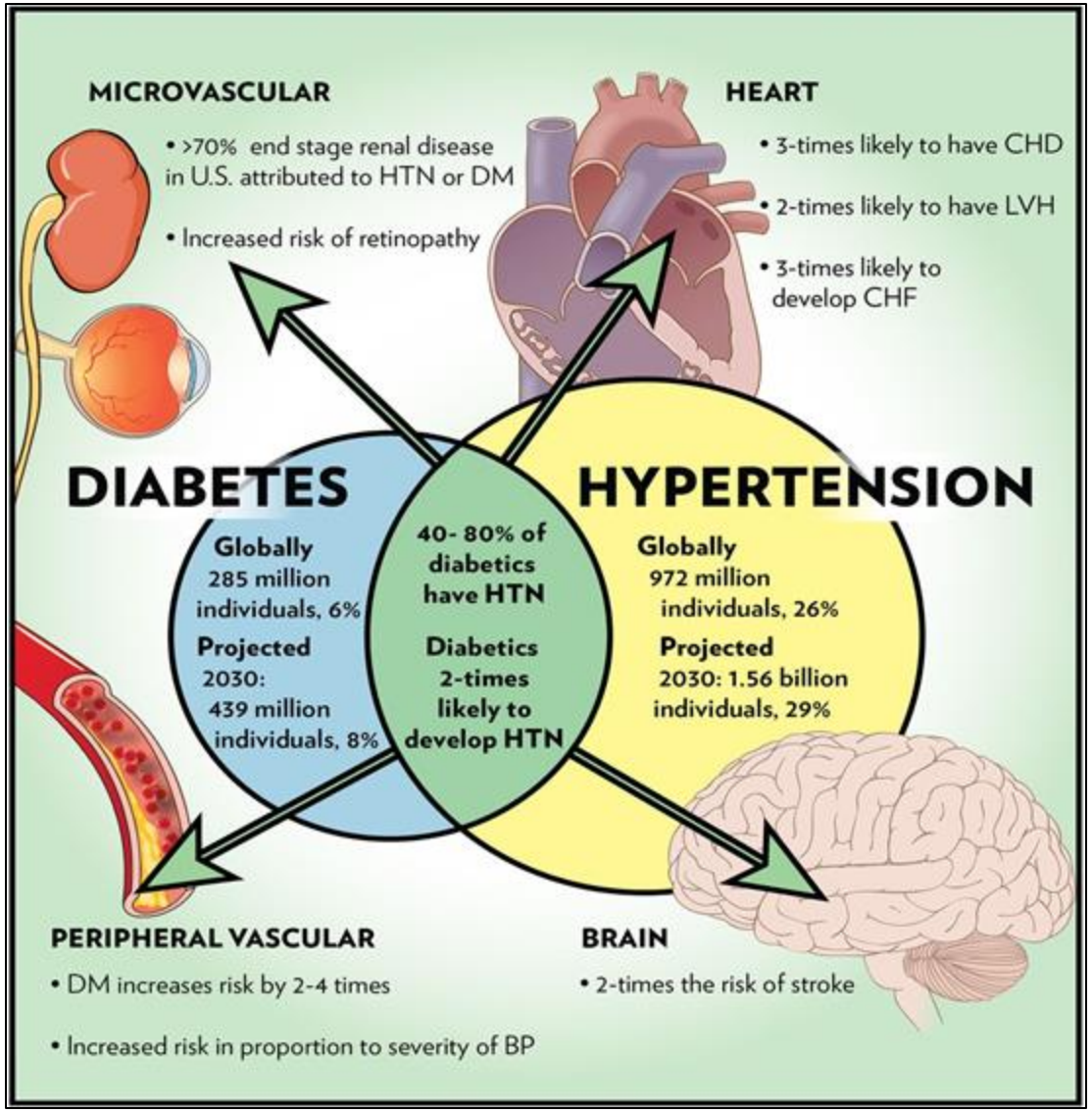
Ethnicity (Māori and Pacific) at significant risk

Screening Strategy

CKD screening should be implemented for groups with these well-accepted CKD risk factors:

- ❑ Hypertension,
- ❑ Diabetes, and/or
- ❑ Cardiovascular disease
- ❑ Māori & Pacific

Screening:
Urinalysis – proteinuria & haematuria
Kidney function - eGFR



https://heart.bmj.com/content/99/8/577

CKD testing characteristics and prevalence of CKD between the different Pacific ethnicities in a population of patients from two Pacific Island health providers in Auckland

Ethnicity	PHO popn (N)	Tested for CKD (n)	CKD	Prevalence of CKD out of total sample	Prevalence of CKD out of those tested
NonMaori_NonPacific	9415 (37.5)	4099 (43.5)	529	5.6 [5.2, 6.1]	12.9 [11.9, 14.0]
Samoan	7451 (29.7)	3554 (47.7)	1187	15.9 [15.1, 16.8]	36.0 [34.4, 37.7]
Tongan	1983 (7.9)	969 (48.9)	310	15.6 [14.1, 17.3]	32.4 [29.4, 35.5]
Cook Island Maori	698 (2.8)	339 (48.6)	101	14.5 [11.9, 17.3]	32.5 [27.3, 38.0]
Fijian	700 (2.8)	361 (51.6)	90	12.9 [10.5, 15.6]	25.1 [20.7, 29.9]
Niuean	522 (2.1)	277 (53.1)	98	18.8 [15.5, 22.4]	36.2 [30.4, 42.2]
Tokelauan	84 (0.3)	37 (44.0)	12	14.3 [7.6, 23.6]	33.3 [18.6, 51.0]
Other Pacific Island	1192 (4.7)	567 (47.6)	214	18.0 [15.8, 20.3]	38.4 [34.3, 42.5]
NZ Maori	3062 (12.2)	1235 (40.3)	276	9.0 [8.0, 10.1]	22.6 [20.3, 25.0]

Tafuna'i et al. Nephrology 2022; 27: 248 - 259

ISN INTERNATIONAL SOCIETY OF NEPHROLOGY

KDIGO KIDNEY DISEASE IMPROVING GLOBAL OUTCOMES

Wonca World family doctors Caring for people. EUROPE

PCDE primary care diabetes europe

CKD is underdiagnosed and undertreated in the community[1]

Early identification, risk stratification, and treatment can reduce the morbidity and mortality rates from CKD and its related complications, such as CVD[2]

Step 2

Test high-risk adults to detect CKD
(not population-wide)

Evaluate kidney function – eGFR
- eGFR calculated based on serum creatinine and/or cystatin C

AND

Evaluate kidney damage – albuminuria
- UACR or dipstick* (if UACR is unavailable)

If UACR ≥30 mg/g (>3 mg/mmol) OR eGFR <60 mL/min/1.73 m²

If UACR <30 mg/g (<3 mg/mmol) AND eGFR >60 mL/min/1.73 m²

Re-test in 3 months

Step 3

Diagnose CKD

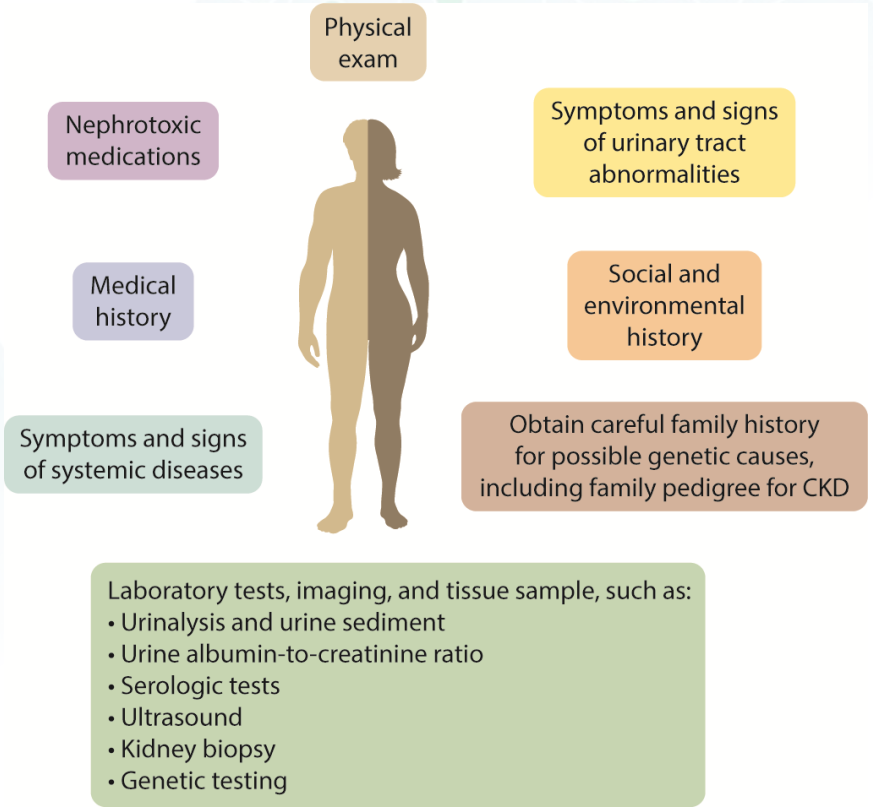
If low eGFR or high UACR are present for ≥3 months, diagnose CKD

Re-test at least once a year†

Do not forget the importance of urinalysis for CKD and CVD
Less than 25% had urinalysis!!

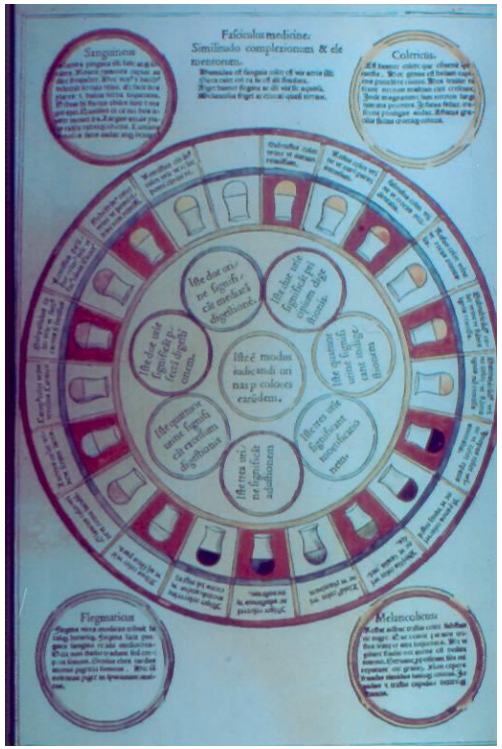
EVALUATION – CKD DEFINITION

CKD is defined as abnormalities of kidney structure or function, present for >3 months, with implications for health. The definition includes many different markers of kidney damage, not just decreased GFR and ACR and the cause of CKD should be actively sought (Figure). CKD is classified according to **C**ause, **G**FR, and **A**CR to establish severity and guide the type and timing of interventions.



Urinalysis – the oldest art of medicine

Physician with the matula



Step 4

Stratify and treat

(also see Table 1)

Risk categories for CKD progression, morbidity, and mortality; monitoring frequency (number of check-ups per year in parentheses); and nephrology consultation[3]

eGFR categories (mL/min/1.73 m²) Description and range	Albuminuria categories A1 <30 mg/g <3 mg/mmol	A2 30–299 mg/g 3–29 mg/mmol	A3 ≥300 mg/g ≥30 mg/mmol
≥90 G1	Monitor (1)	Treat (1)	Treat & consult (3)
60–89 G2	Monitor (1)	Treat (1)	Treat & consult (3)
45–59 G3a	Treat (1)	Treat (2)	Treat & consult (3)
30–44 G3b	Treat (2)	Treat & consult (3)	Treat & consult (3)
15–29 G4	Treat & consult (3)	Treat & consult (3)	Treat & consult (4+)
<15 G5	Treat & consult (4+)	Treat & consult (4+)	Treat & consult (4+)

Adapted from de Boer et al. 2022[5]

Low risk

Stable disease *OR* NO CKD in absence of other markers of kidney damage.[1]
Requires measurements once a year or earlier in case of new symptoms / risk factors.

Moderately increased risk

Requires measurements at least once a year

High risk

Requires measurements at least twice a year

Very high risk

Treat in agreement with a nephrologist

Requires measurements at least three times a year

Requires the closest monitoring at least four times a year (every 1–3 months)

Remember an almost identical risk profile for CVD

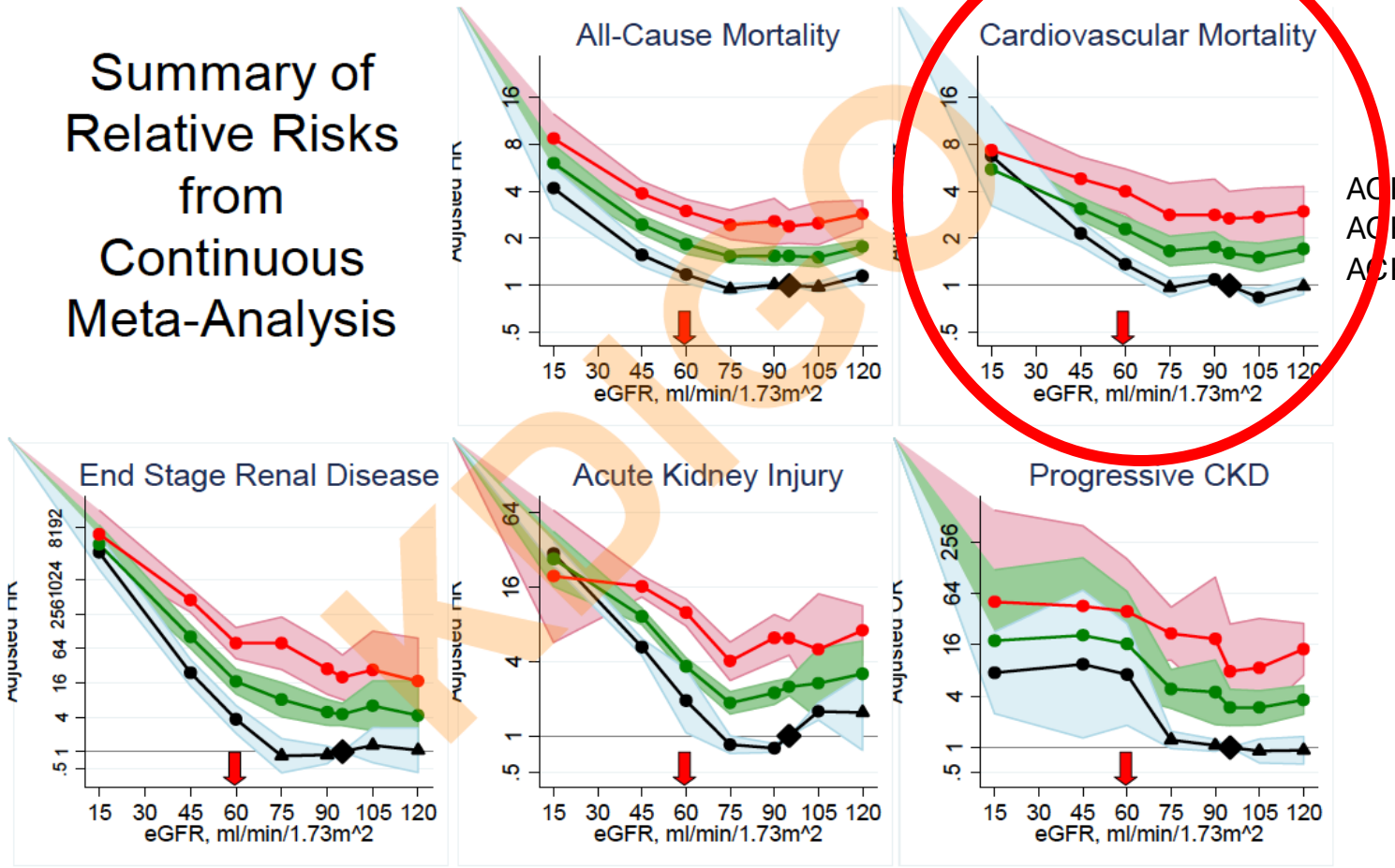
EVALUATION – DIAGNOSIS OF CKD IN OLDER ADULTS

Epidemiological population data support retaining the threshold GFR of 60 ml/min/1.73 m2 for diagnosis of CKD in older adults, even in the absence of significant albuminuria, with consistently elevated and increasing relative risk of adverse outcomes below this threshold.

Age 65+	ACR, mg/g				ACR, mg/g			
eGFRcr-cys	<10	10–29	30–299	300+	<10	10–29	30–299	300+
	All-cause mortality				Myocardial infarction			
105+	1.2	1.4	1.9	3.5	0.97	1.4	2.0	19
90–104	ref	1.2	1.4	2.0	ref	1.2	1.1	1.9
60–89	1.2	1.5	1.8	2.3	1.1	1.4	1.5	1.9
45–59	1.6	2.0	2.4	2.9	1.6	1.9	2.3	3.4
30–44	2.0	2.4	3.2	4.1	2.1	2.6	3.1	3.8
<30	3.4	4.1	5.1	6.5	4.9	3.0	5.1	5.0
	Cardiovascular mortality				Stroke			
105+	1.1	1.5	2.0	12	1.2	1.3	1.5	3.3
90–104	ref	1.4	1.4	3.4	ref	1.3	1.3	2.8
60–89	1.2	1.7	2.2	3.1	1.1	1.4	1.8	2.5
45–59	1.7	2.4	3.0	4.3	1.5	1.7	2.0	2.3
30–44	2.4	3.1	4.5	5.8	1.5	2.0	2.1	2.3
<30	5.7	5.2	5.1	7.8	1.7	2.0	2.4	4.8
	Kidney failure replacement therapy				Heart failure			
105+	2.0	1.0	2.1		0.99	1.5	1.7	7.0
90–104	ref	1.9	4.7	10	ref	1.3	1.5	2.2
60–89	1.4	2.6	6.2	19	1.2	1.5	2.0	3.2
45–59	3.7	7.9	16	42	1.6	2.0	2.9	4.1
30–44	14	14	46	137	2.3	2.9	3.5	6.1
<30	87	364	241	406	4.4	4.1	5.5	7.2
	Acute kidney injury				Atrial fibrillation			
105+	0.91	1.1	1.3	1.9	0.95	1.1	1.0	3.7
90–104	ref	1.3	1.4	3.9	ref	1.2	1.3	2.4
60–89	1.5	2.1	2.7	4.7	1.1	1.2	1.5	2.0
45–59	3.6	4.3	5.1	7.3	1.2	1.4	1.7	1.9
30–44	5.7	5.9	7.2	9.8	1.5	1.8	2.0	2.2
<30	10	11	11	22	1.8	1.8	2.2	3.2
	Hospitalization				Peripheral artery disease			
105+	1.0	1.1	1.2	2.2	1.1	2.3	2.9	4.9
90–104	ref	1.1	1.3	1.4	ref	1.3	2.0	4.8
60–89	1.1	1.2	1.3	1.5	1.3	1.6	2.0	3.2
45–59	1.2	1.2	1.4	1.6	2.0	2.8	3.1	3.1
30–44	1.5	1.4	1.6	2.0	3.5	2.8	3.8	5.9
<30	1.9	1.9	2.0	2.6	8.4	4.1	5.9	10

CV/renal prognosis related to GFR and ACR

Summary of Relative Risks from Continuous Meta-Analysis



ACR > 30mg/mmol
ACR 3– 30 mg/mmol
ACR < 3 mg/mmol

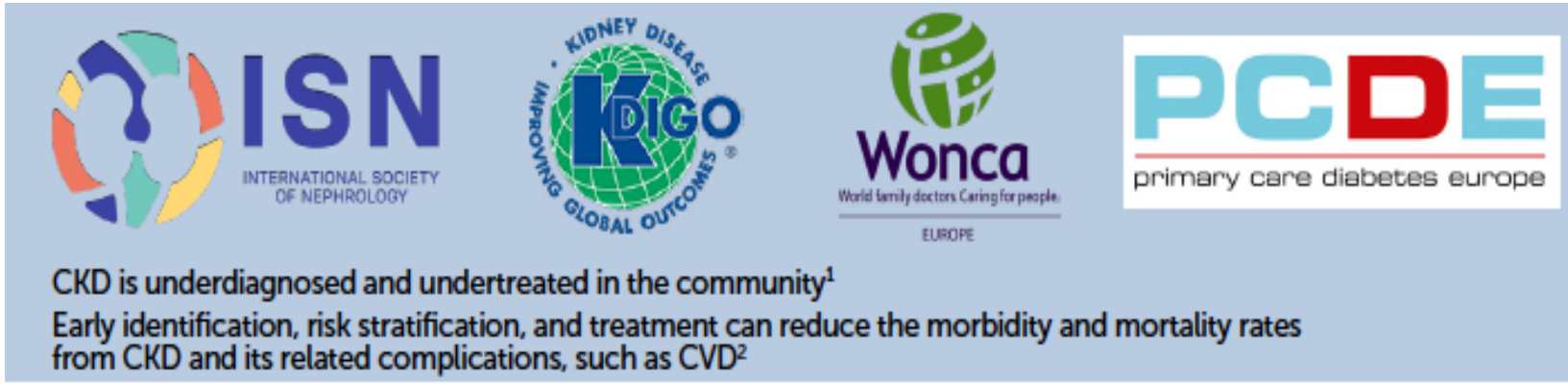
Matsushita K et al, Lancet 2010;375:2073-81 and Gansevoort RT et al Kidney Int 2011;80:93-104

Risk management strategies

Table 1. Treat to slow CKD progression, reduce mortality risk, and manage comorbidities

Lifestyle modification	
Smoking cessation; regular exercise; well-balanced diet (avoid excessive protein intake and processed food, limit sodium intake <2 g/day)	
Medical treatment	
Treat diabetes, hypertension, and CVD: Optimise blood pressure and glycemic control	Ensure guideline-directed medical treatment to slow down CKD progression and reduce CVD risk: maximally tolerated doses of **ACEIs/ARBs, SGLT2 inhibitors, nonsteroidal MRAs** with proven benefits in renal and cardiovascular outcome trials for T2D; also consider **lipid-lowering therapy (statins)** and/or **antiplatelet therapy** (for patients with CKD at risk of atherosclerotic events)
Considerations	
Adjust dosing of medications based on eGFR; exercise caution when prescribing analgesics, antimicrobials, hypoglycemics, chemotherapeutics, or anticoagulants; avoid nephrotoxins (e.g. NSAIDs) and some contrast media	

ACEI/ARB, SGLT2i, MRA are NOW standard of care of proteinuric non-diabetic kidney disease



Step 5

Nephrology consultation

Take action based on the risk categories for CKD progression, morbidity, and mortality, and monitoring frequency (see above).

Primary care practitioners should consult with a nephrologist while initiating treatment; some patients may be under the direct care of a nephrologist if indicated (see Table 3).

Table 3. Additional considerations for nephrology consultation

- Unexplained, progressive decline in eGFR ≥5 mL/min/1.73 m^2 over 12 months or sudden decline in eGFR over days to weeks
- Unexplained significant albuminuria/proteinuria or hematuria
- Persistent hyperkalemia, resistant hypertension (defined as uncontrolled hypertension on three antihypertensive agents, including a diuretic), recurring kidney stones, or hereditary kidney diseases (e.g. ADPKD)
- Other complications identified (anemia, mineral and bone disorders, metabolic acidosis, etc.)

Consultation with a nephrologist can be for identifying other treatable causes or for developing a treatment plan. Although some patients may be maintained further in nephrology care, most will return to primary care.

Key Pathway – CKD (1)

- National referral guidelines:
  - Stage 3+ CKD any aetiology
  - Māori – Pacific with proteinuria and early CKD (not just diabetes) – much higher risk
  - Proteinuria > 50 mg/mmol
  - Haematuria with proteinuria and CKD (any stage)
    - Haematuria alone is UROLOGICAL until proven otherwise both macro and microscopic exception – young adult with synpharyngetic presentation – IgA nephropathy.
      - Red cell casts no longer done by lab
        If urology negative still need to consider GN
  - Rapid decline in renal function (remember fluctuations in eGFR related to loss of autoregulation)*
  - Recurrent kidney stones

Key Pathway – CKD (2)

Maximise management to slow progression.

- Control hypertension: ARB/ACEI first choice – maximise dose especially if proteinuric.

  Add SGLT2 inhibitor
- Cardiac protection – statins and SGLT2inhibitor
- Add Spironolactone for hypertension, CVD & CKD progression.
- Diabetes (T2D) – maximise control – metformin*, SGLT2 inhibitors, GLP1agonists
- Anaemia – iron status (iron infusions in community) EPO
- CKD metabolic bone disease – calcitriol, sodium bicarbonate
- Treat gout
- Review medications:
  Increasing evidence PPIs contribute to CKD. Avoid codeine, phosphate containing enemas

Key Pathway – CKD (3)

- Progressive CKD especially CKD stage 4: Refer to nephrology
- Early referral – education about kidney replacement therapy options
  includes active conservative care.
- Maximise CKD and CVD management

The utility of nurse-led management programmes for CKD: the DEFEND trial

General practice can help support improved outcomes for patients at high risk of progressing to kidney failure through relatively simple complementary nurse-led interventions involving the use of healthcare assistants.

The **DE**lay **F**uture **E**nd-stage **N**ephropathy due to **D**iabetes (DEFEND) trial involved 65 Māori and Pacific patients aged 47 – 75 years with type 2 diabetes, moderate CKD and hypertension, living in Auckland.[36] Patients received either routine medical care and follow-up or nurse-led, community based, monthly assessments and monitoring delivered by healthcare assistants.[36] This study found that community care resulted in clinically significant decreases in systolic blood pressure and proteinuria as well as delayed progression of left ventricular hypertrophy and diastolic dysfunction.[36] The success of the programme was attributed to Māori and Pacific healthcare assistants providing culturally appropriate care, the more frequent follow-up and prompting of patients to take medicines, and reduced costs to patients because of home visits.[36]

After the intervention ended in 11 – 21 months, patients reverted back to routine medical care. In a 2015 follow-up study, the initial short-term improvements in systolic blood pressure and proteinuria for the intervention cohort did not result in long-term reductions in mortality and end-stage kidney disease rates compared with the usual care group.[37] These findings indicate that such community-based interventions may need to be initiated earlier and maintained throughout care to have a more meaningful impact for people with CKD.[37]

Evaluation – Accuracy and Reliability

Understand the variability of GFR and urinary albumin and the value and limitations of the methodology of assessment when determining whether a change is a true change. Implement the requisite laboratory standards of care to ensure accuracy and reliability.

Progressive Nephropathy

- In CKD fluctuations in serum creatinine and eGFR are normal
  - Lose the protective vasodilation or vasoconstriction
  - Kidney senses these highs and lows in perfusion pressure
  - Creatinine does rise and fall as you correct this (hence eGFR)
  - (a linear relationship almost)
  - Pre-glomerular pressure response lost and post-glomerular compensation lost

	26.Jul.2011	06.Oct.2011	06.Mar.2012	11.Jun.2012
	08:50	08:45	08:30	09:14
Sodium	**147**	143	142	142
Potassium	4.5	4.6	4.4	4.4
Creatinine	**223**	**237**	**255**	**260**
Uric Acid				
eGFR	**27**	**25**	**23**	**22**
Urea	**12.6**	**12.7**	**11.7**	**12.6**

The GFR range for a young adult male is 87-167. From ag

65 year old male:

Please review deteriorating kidney function decline in eGFR 5ml/min/year

	28.Nov.2009	27.Feb.2010	12.May.2010	21.Jun.2010	26.Jul.2010	01.Nov.2010	08.Feb.2011	08.Apr.2011	12.Jul.2011	26.Jul.2011	06.Oct.2011	06.Mar.2012	11.Jun.2012	09.Jul.2012
	09:30	10:04	08:15	09:45	08:39	08:25	08:40	08:30	08:35	08:50	08:45	08:30	09:14	08:38
Sodium	143	139	140	135	145	143	143	144	142	**147**	143	142	142	**146**
Potassium	4.5	**3.4**	**3.3**	4.5	4.5	4.7	4.7	4.5	4.6	4.5	4.6	4.4	4.4	4.6
Creatinine	**219**	**229**	**279**	**271**	**217**	**230**	**219**	**217**	**243**	**223**	**237**	**255**	**260**	**233**
Uric Acid			**0.67**											**0.51**
eGFR	**29**	**28**	**22**	**21**	**28**	**26**	**27**	**28**	**24**	**27**	**25**	**23**	**22**	**25**
Urea	**11.6**	**15.7**	**16.1**	**17.6**	**10.6**	**11.8**	**11.4**	**10.7**	**13.1**	**12.6**	**12.7**	**11.7**	**12.6**	

No change in eGFR over 3 years.

Variation around a mean.

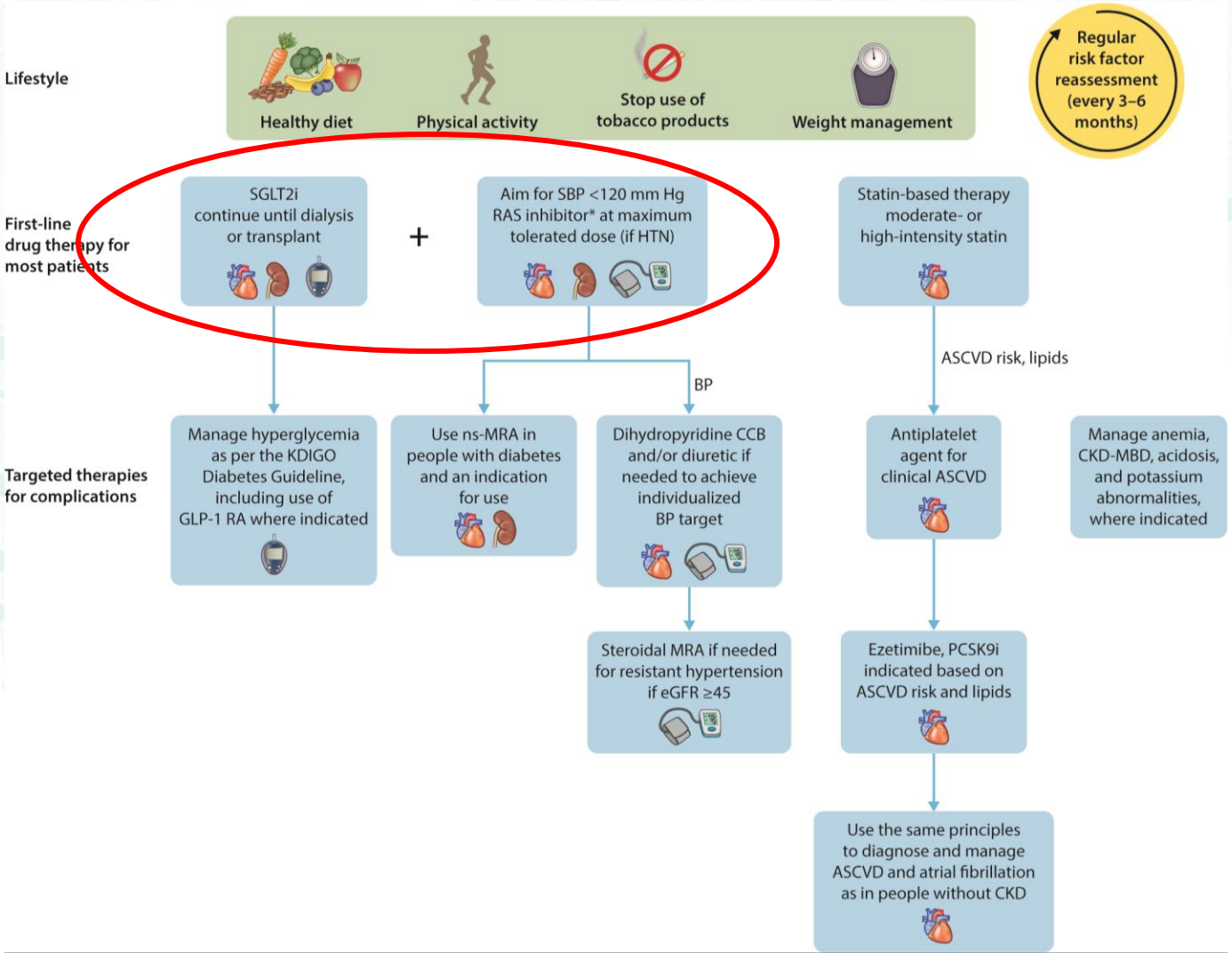
Calculated creatinine clearances 42 – 36 ml/min.

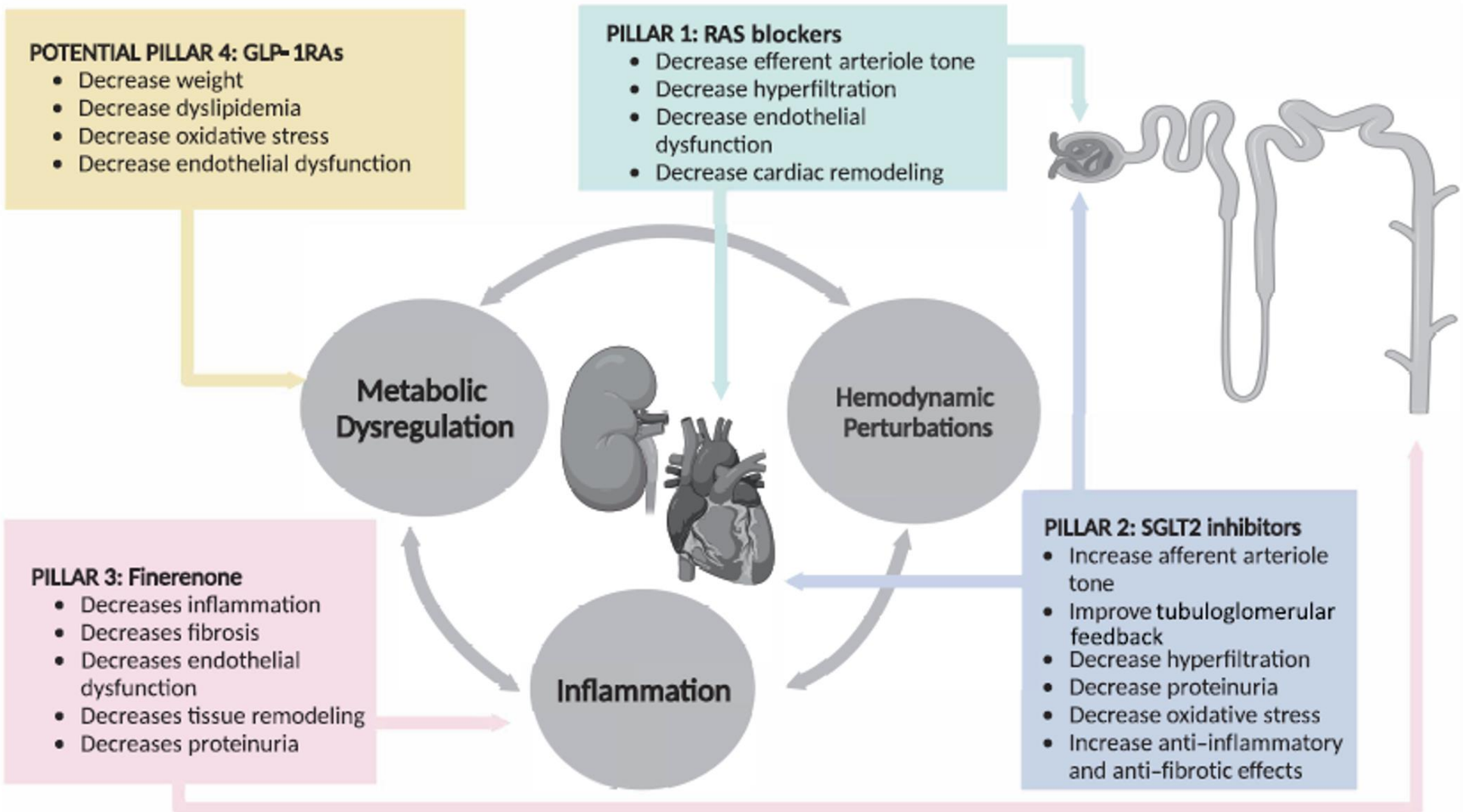
Interpreting eGFR

- Extra creatinine and eGFR values over previous years very useful.
- Important to add other variables
  proteinuria and blood pressure control.
  Medications
  Proportion muscle mass
  Intercurrent illness or excess activity.

MANAGEMENT – RASI AND SGLT2I

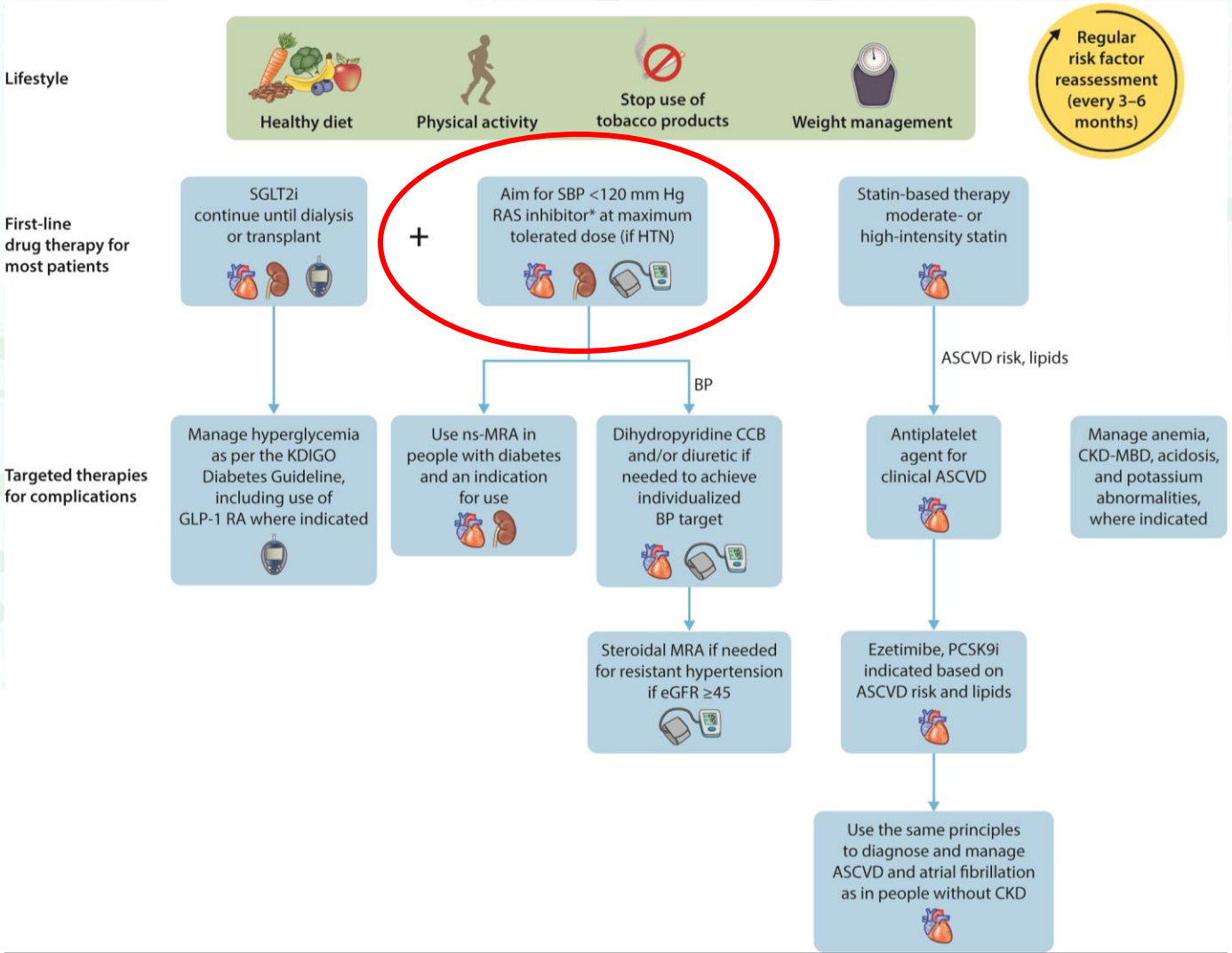
Treatments that delay progression of CKD with a strong evidence base include RASi and SGLT2i. In people with CKD and heart failure, SGLT2i confer benefits irrespective of albuminuria.




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

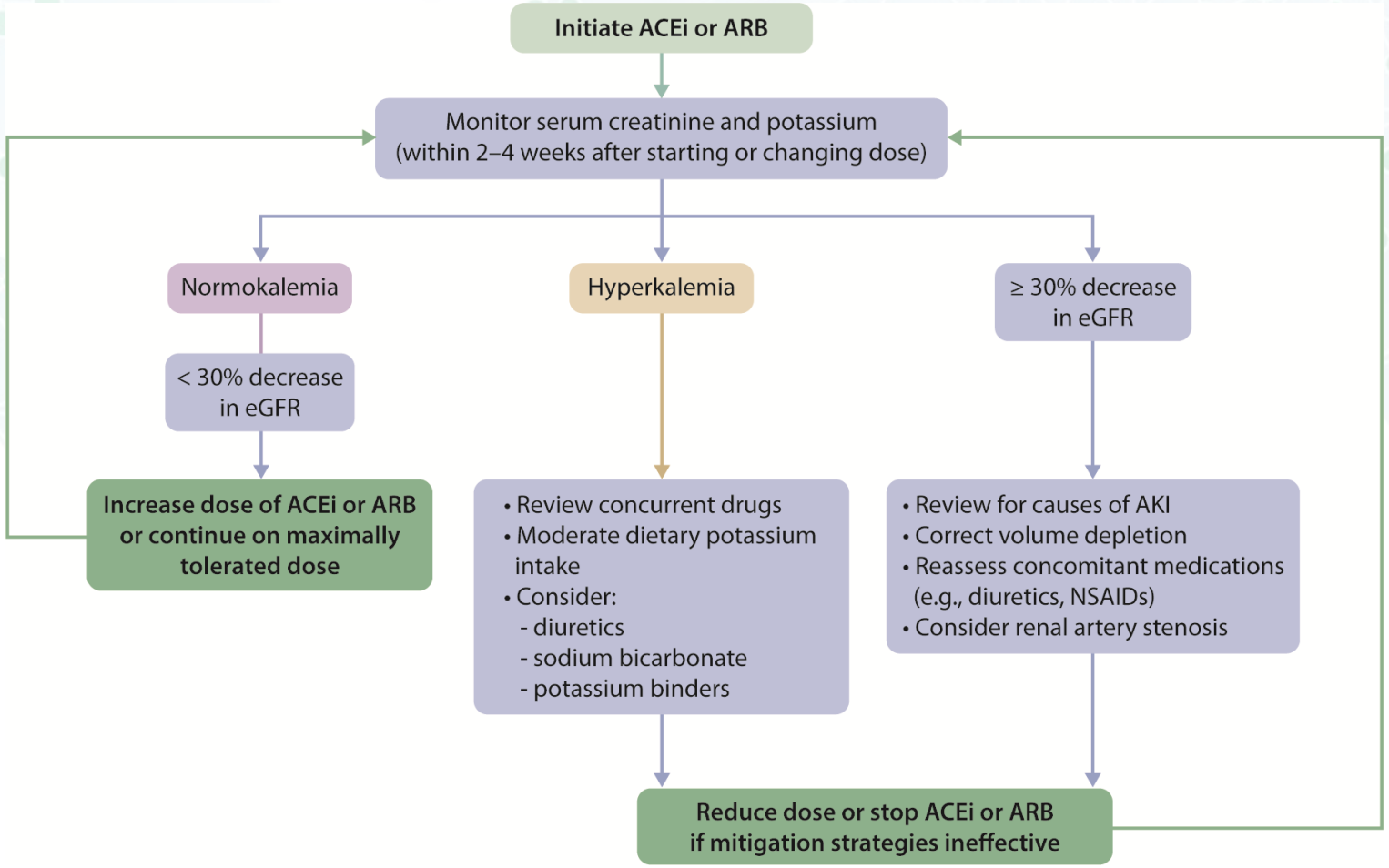
MANAGEMENT – INDIVIDUALIZE BP CONTROL

Individualize BP-lowering therapy and treatment targets in people with frailty, high risk of falls, very limited life expectancy, or symptomatic postural hypotension.



MANAGEMENT – ACUTE CHANGES IN eGFR

Initial dips in eGFR are expected following initiation of hemodynamically active therapies, including both RASI and SGLT2i. GFR reductions of ≥30% from baseline exceed the expected variability and warrant evaluation.



Illustrative Case

- 58 year Māori male with a history of hypertension for 15 years, ischaemic heart disease, T2DM for 5 years and chronic kidney disease.
- BP 156/98, UACR 88mg/mmol, serum creatinine 140umol/l, eGFR 47 ml/min/1.73m^2, HbA1c 63
- 5year Cardiovascular risk 37% ESKD risk 13%
- Commenced on candesartan 8mg daily
  BP 132/86
  eGFR now 41 ml/min/1.73m^2
  Do you stop the ARB?

Impact of Vasoactive Drugs on Renal Function

- Systemic effects vs local renal haemodynamic effects
- Need to remember changes over time
- GFR a continuous variable

Illustrative Case

- 58 year Māori male with a history of hypertension for 15 years, ischaemic heart disease, T2DM for 5 years and chronic kidney disease.
- HbA1c 63
- 5year Cardiovascular risk 37% ESKD risk 13%
- Cardiovascular risk and Diabetic management

Annals of Internal Medicine | Review

Benefits and Harms of Statin Therapy for Persons With Chronic Kidney Disease

A Systematic Review and Meta-analysis

Suetonia C. Palmer, MBChB, PhD; Jonathan C. Craig, MBChB, MM, MPH, PhD; Sankar D. Navaneethan, MD, MPH; Marcello Tonelli, MD, PhD; Fabio Pellegrini, MSc; and Giovanni F.M. Strippoli, MD, MM, MPH, PhD

Ann Intern Med. 2012;157:263-275.

Statins decrease mortality and cardiovascular events in persons with early stages of CKD, have little or no effect in persons receiving dialysis, and have uncertain effects in kidney transplant recipients.

In persons not receiving dialysis

All-cause mortality (relative risk [RR], 0.81 [95% CI, 0.74 to 0.88])

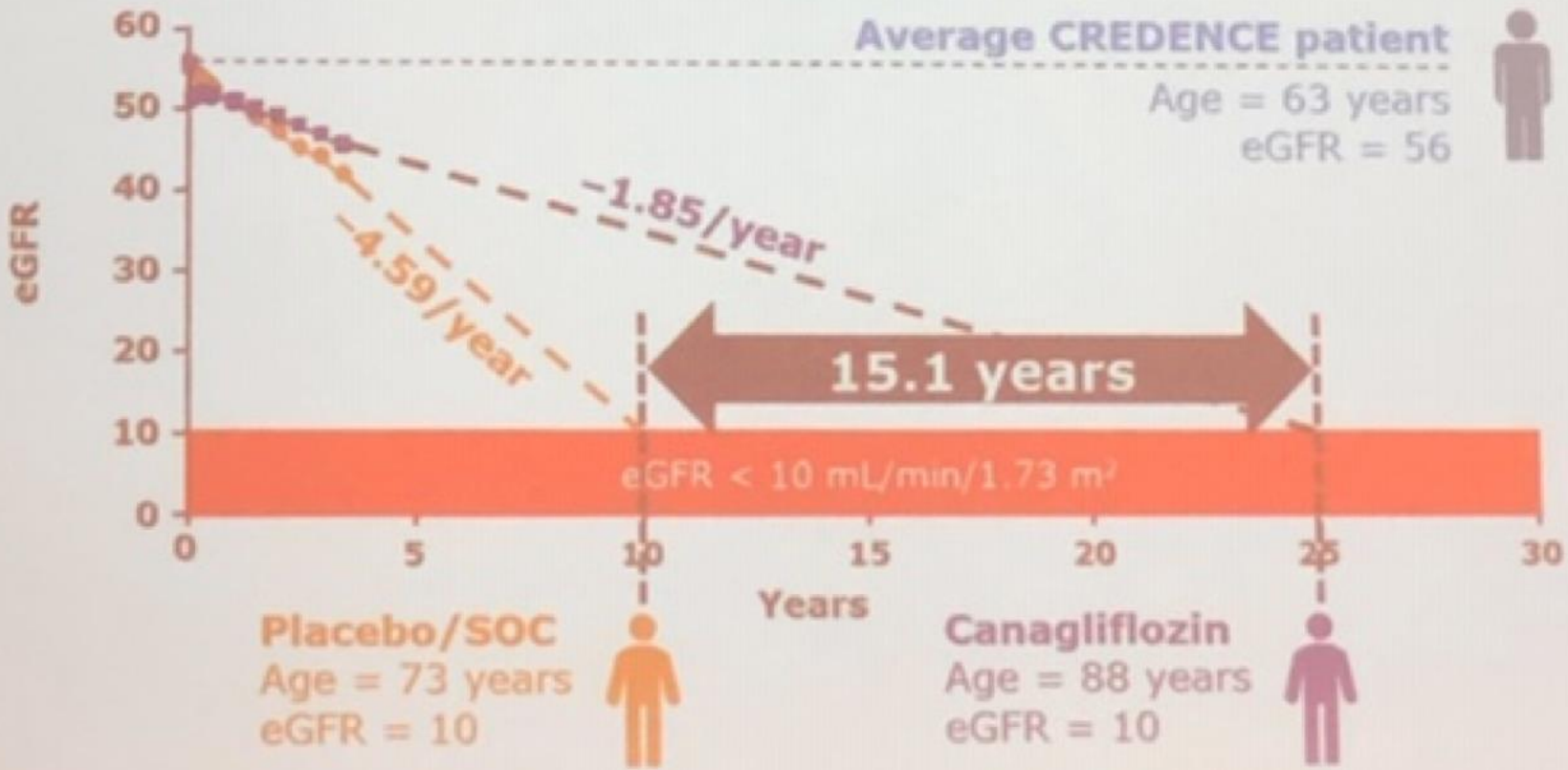
Cardiovascular mortality (RR, 0.78 [CI, 0.68 to 0.89])

Cardiovascular events (RR, 0.76 [CI, 0.73 to 0.80])

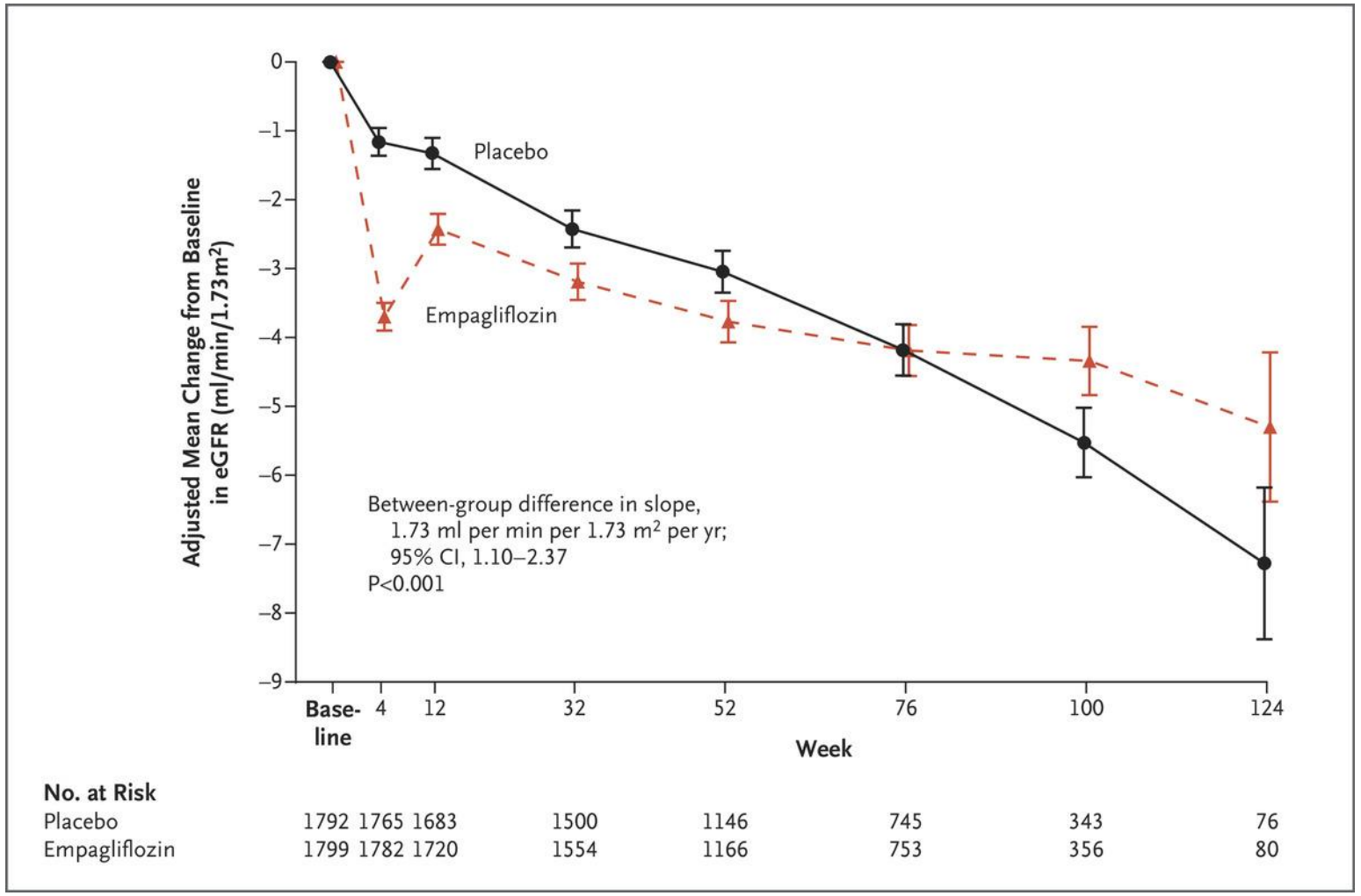
Illustrative Case

- 58 year Māori male with a history of hypertension for 15 years, ischaemic heart disease, T2DM for 5 years and chronic kidney disease.
- BP 156/98, UACR 98mg/mmol, serum creatinine 140umol/l, eGFR 47 ml/min/1.73m^2, HbA1c 63
- 5year Cardiovascular risk 37% ESKD risk 13%
- Candesartan 8mg daily and commenced on Metformin Empagliflozin 500mg/5mg twice a day
  BP 132/86
  eGFR now 33 ml/min/1.73m^2
  UACR now 32mg/mmol

Projected Effects on eGFR



Changes in the Estimated Glomerular Filtration Rate.



Reduction in eGFR up to 30% acceptable

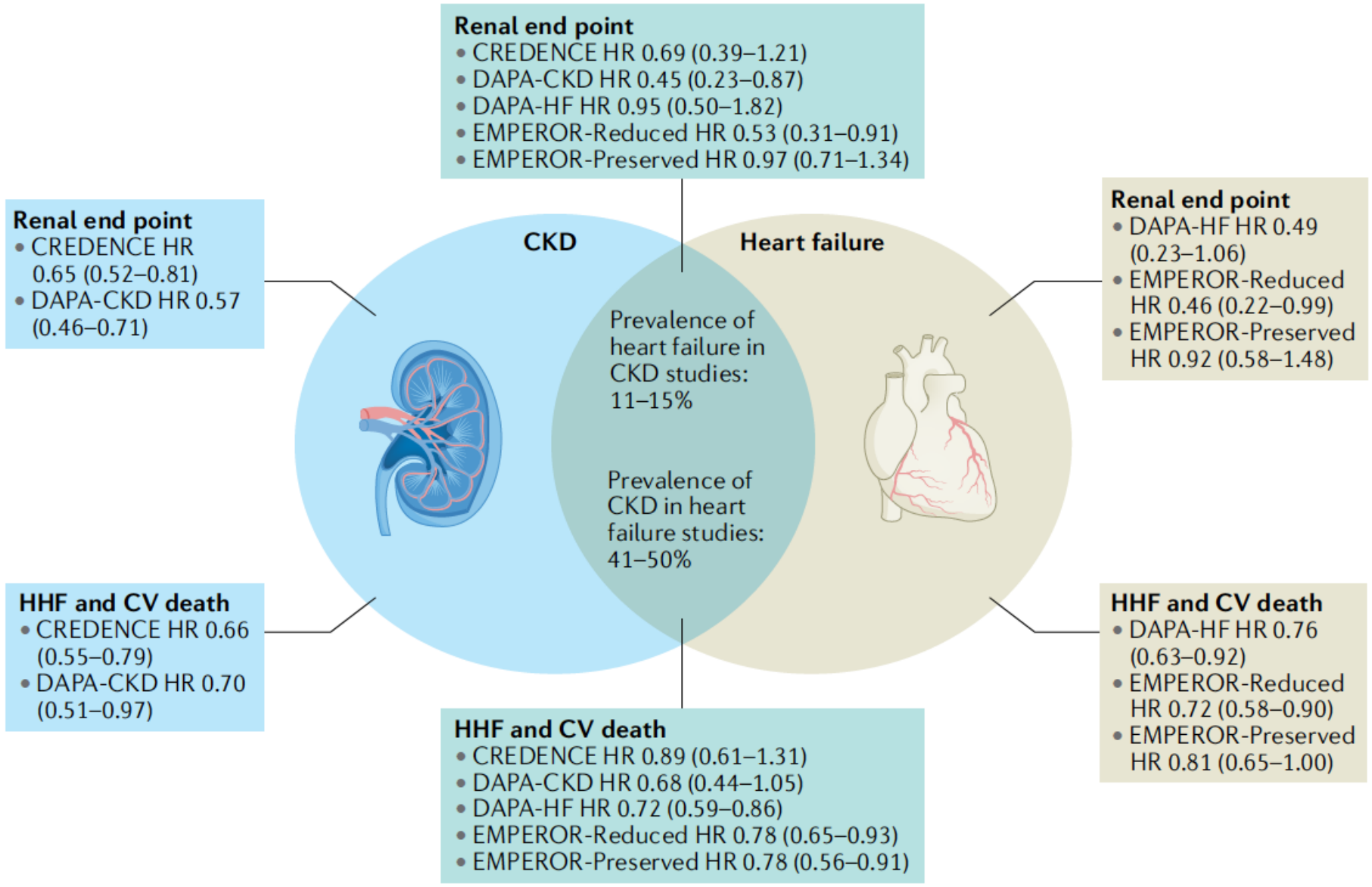
M Packer et al. N Engl J Med 2020;383:1413-1424.

The NEW ENGLAND JOURNAL of MEDICINE

Clinical Impact: CREDENCE
SGLT2 and Diabetic CKD

- Over 4000 participants. T2DM plus eGFR 30 – 90 ml/min
- Primary composite outcome (doubling creatinine, renal or CV death
  NNT= 22 (95%CI 15-32)
- Renal outcome. NNT=28 (95%CI 19-54)
- Greater the proteinuria the greater the effect.
- ESKD. NNT = 43 (95%CI 26-121)
- Cardiovascular endpoints.
  NNT = 40 (95%CI 23-165)
- Other studies similar results.

Impact of FDA requirements of clinical trial reporting.



Kidney and heart failure outcomes with SGLT2 inhibitors.
Nat Rev Nephrol. 2022; 18: 294- 306

Empagliflozin in Patients with Chronic Kidney Disease (EMPA-KIDNEY)

ISN INTERNATIONAL SOCIETY OF NEPHROLOGY

- Multicenter
  - 241 centers
  - 8 countries
- Double-blind
- 6,609 patients
  - eGFR 20 – 45 ml/min/1.73m2
    Regardless of the level of albuminuria
  - eGFR 45 – 90 ml/min/1.73m2
    UACR >200
- Feb 2019 – Apr 2021

Pre-randomization phase 15wks of placebo

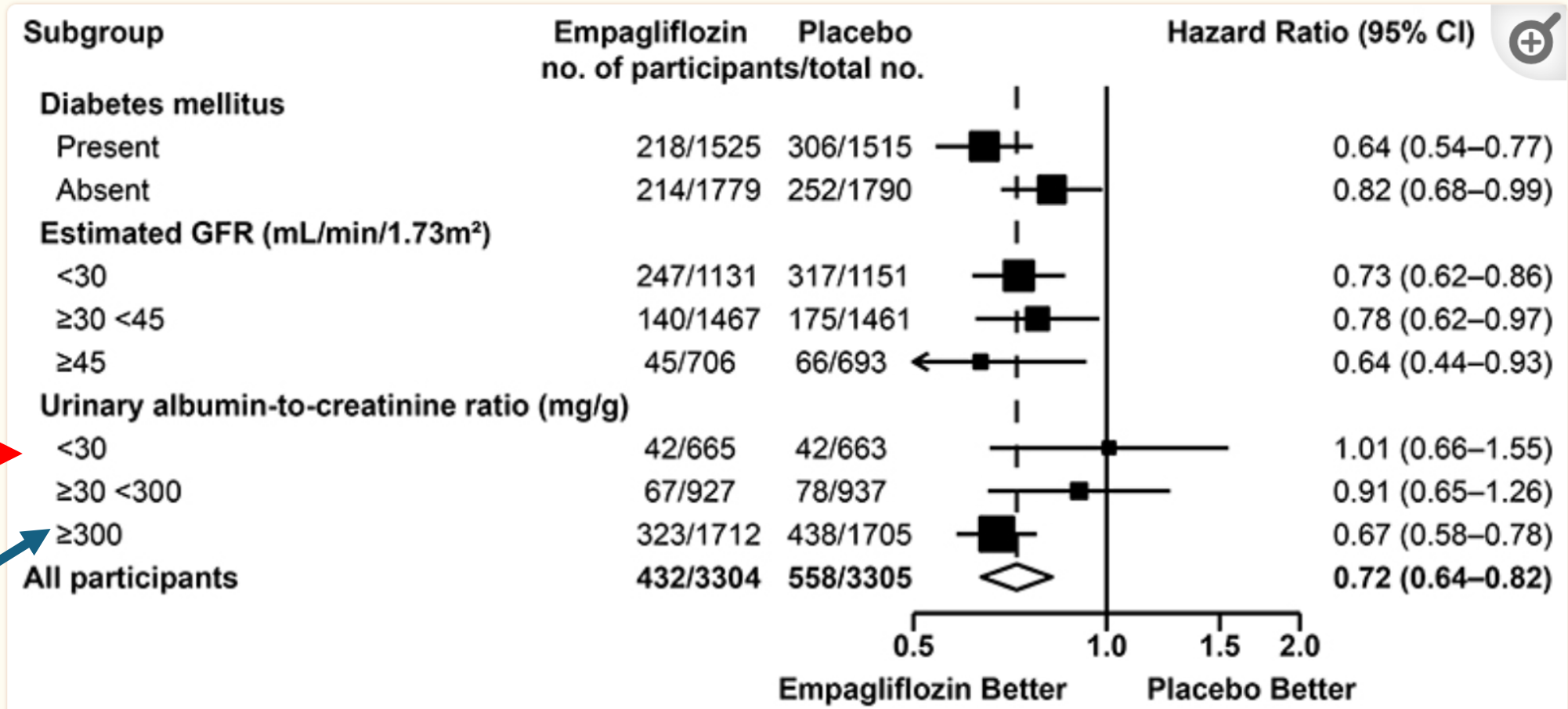
- Empagliflozin 10mg/day
- Placebo

	CKD progression & death from CV causes	Hospitalizations from any cause	Hospitalization for HF or death from CV causes	Death from any cause
Empagliflozin	13.1%	24.8% per 100 patients/year	4.0%	4.5%
	0.72 0.64 – 0.82 P<0.001	0.86 0.78 – 0.95 P = 0.003	0.84 0.67 – 1.07 P = 0.15	0.87 0.70 – 1.08 P = 0.21
Placebo	16.9%	29.2% per 100 patients/year	4.6%	5.1%

Conclusion: among a wide range of patients with chronic kidney disease who were at risk for disease progression, empagliflozin therapy led to a lower risk of progression of kidney disease or death from cardiovascular causes than placebo.

Reference: EMPA-KIDNEY Collaborative Group. (2022). Empagliflozin in Patients with Chronic Kidney Disease. *New England Journal of Medicine*.

VA by Denisse Arellano, MD @deniise_am



UACR <3mg.mmol

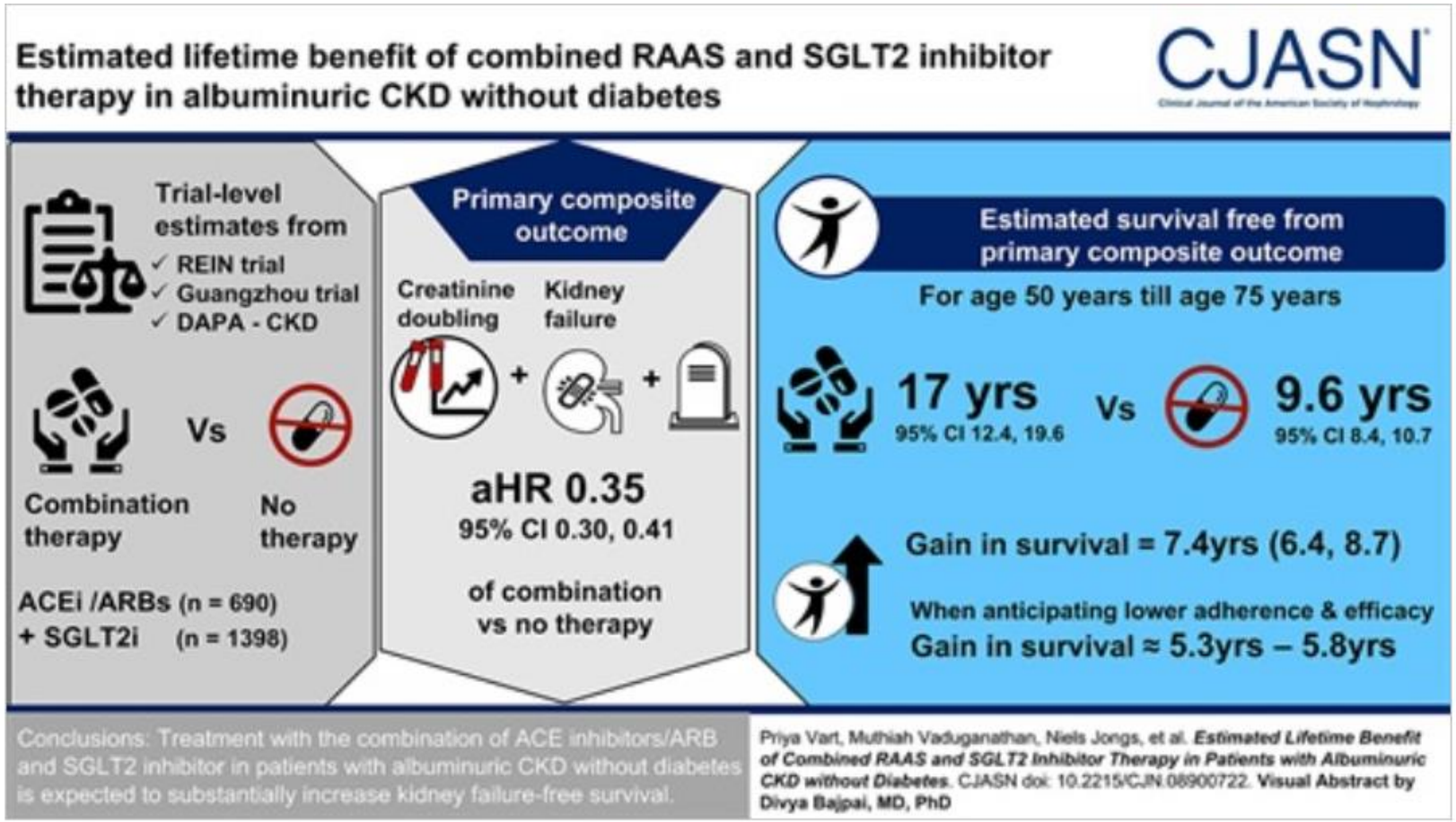
*

UACR > 34mg/mmol

EMPA Kidney NEJM 2022

* Unanswered question

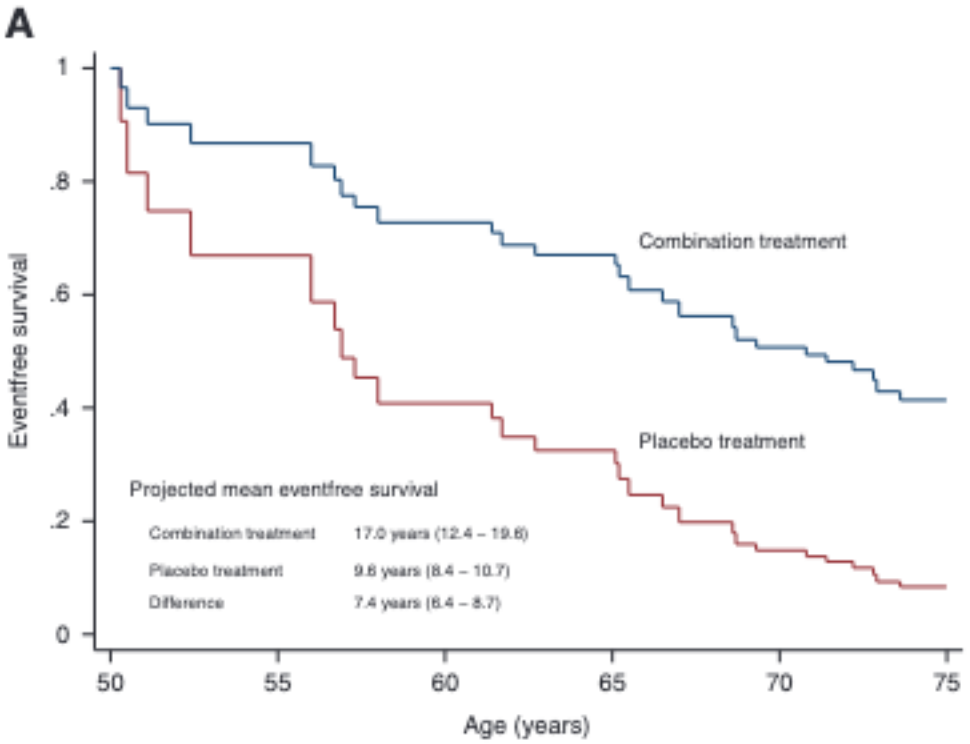
Role of SGLT2 inhibition in non-proteinuric kidney disease



CJASN 17: 1754–1762, 2022. doi: https://doi.org/10.2215/CJN.08900722

Estimated Lifetime Benefit of Combined RAAS and SGLT2 Inhibitor Therapy in Patients with Albuminuric CKD without Diabetes

Priya Vart,[1,2] Muthiah Vaduganathan,[3] Niels Jongs,[1] Giuseppe Remuzzi,[4] David C. Wheeler,[5] Fan Fan Hou,[6] Finnian McCausland,[3] Glenn M. Chertow,[7,8] and Hiddo J.L. Heerspink[1,9]



Results from clinical trials show that an individual aged 50 with albuminuric non-diabetic CKD when treated with combination ARB/ACEI and SGLT2I may experience an additional 7.5 years free of kidney failure and death compared to placebo

CJASN 17: 1754–1762, 2022. doi: https://doi.org/10.2215/CJN.08900722

Management – Discontinuation and Restart of Medications

If medications are discontinued during an acute illness, communicate a clear plan of when to restart the discontinued medications to the affected person and healthcare providers, and ensure documentation in the medical record. Failure to restart these medications may lead to unintentional harm.

Review original indication for the drug.

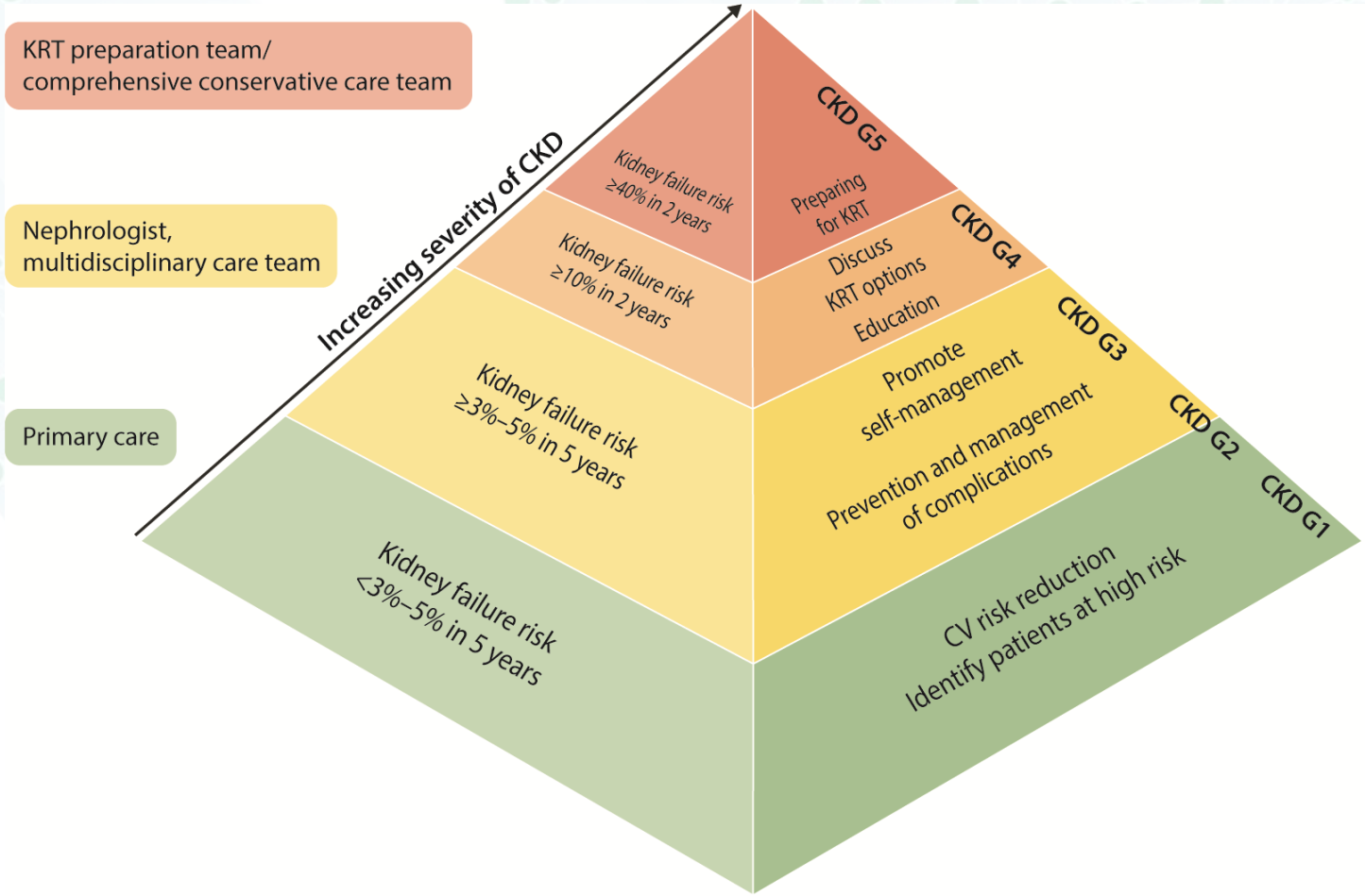
Identify patients with clinical indication for restarting inhibitors ACE-I/ARB (unless there is a new contraindication):

- Heart failure with reduced ejection fraction
- History of myocardial infarction
- Diabetes with albumin:creatinine ratio > 3 mg/mmol
- Hypertension with albumin:creatinine ratio >30 mg/mmol
- Albumin:creatinine ratio > 70 mg/mmol irrespective of hypertension or cardiovascular disease

Cardiovascular risk reduction
Statins – aspirin as appropriate
Diabetes – metformin & SGLT2 inhibitors
Non-diabetes CKD add in SGLT2 inhibitors

MANAGEMENT – ADVANCED CARE PLANNING

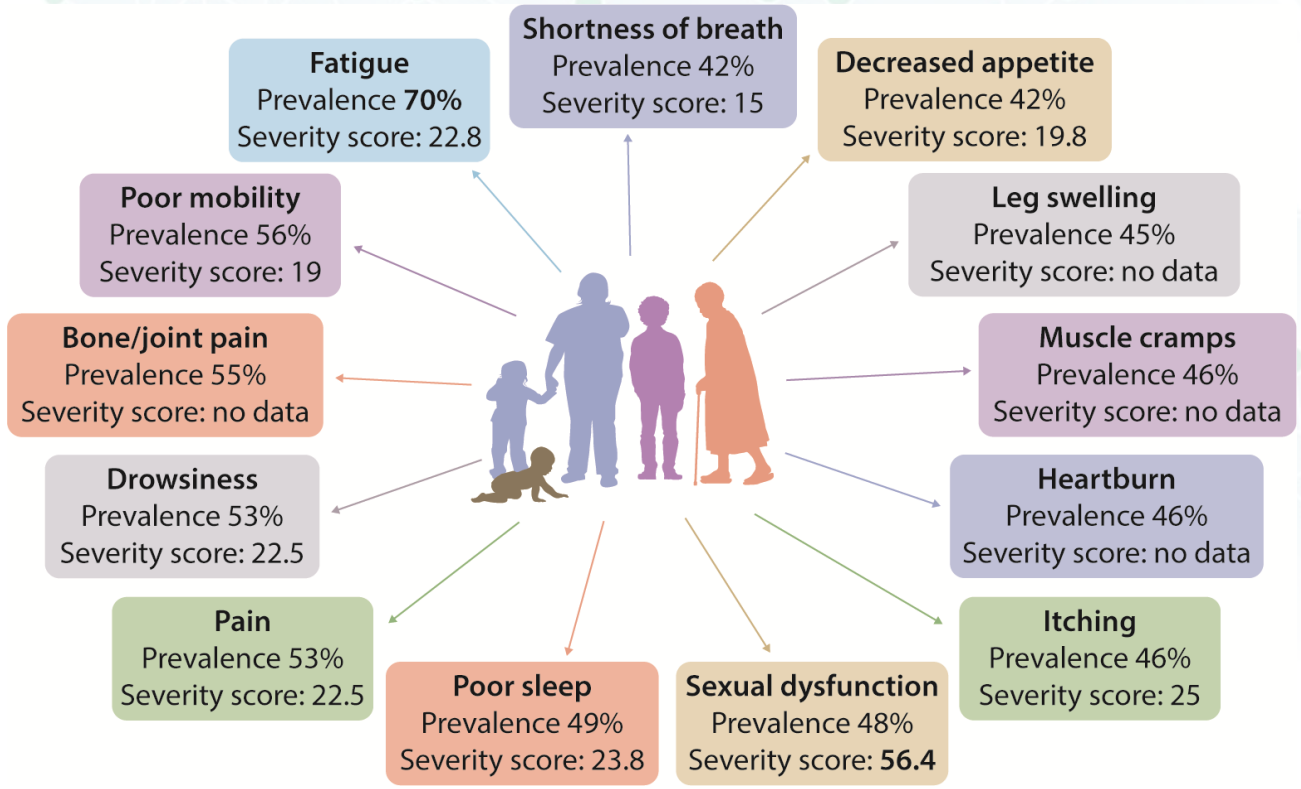
Plans addressing future health care states should be jointly agreed with people with CKD and their families/carers and known to all. Advanced care planning for those choosing supportive care is particularly important.



MANAGEMENT – SYMPTOM CONTROL IN CKD

The identification and assessment of symptoms in people with progressive CKD is important for highlighting changes in clinical management, redirecting treatment toward patient-centered management, and may lead to discussion about appropriate supportive care options. Effective communication and shared decision-making should be key principles between healthcare providers and the people they treat, allowing them to work in partnership to identify symptom burden, possible treatment strategies and person-centered solutions.

CKD grade 4-5



Future monitoring in CKD

- Timely monitoring of kidney function, urinary albumin / creatinine ratio and blood pressure
  Review cardiovascular risk
  Consider referral to nephrology – CKD guidelines
- Patient education – increased risk especially in context of chronic disease – ‘no renal reserve’
  Rapid escalation of treatment in setting of acute illness
  Medication modification as required

Summary:

Given the high cardiovascular risk for individuals with CKD, management should be maximised using current evidence-based practice.

Important roles for ACEI/ARB, SGLT2 inhibitors, statins and Mineralocorticoid receptor antagonists.

Questions

“One must learn by doing the task, for, though you think you know it, you have no certainty until you try.”
Sophocles 5th Century BC.