Management of Diabetes and CKD –circa 2022

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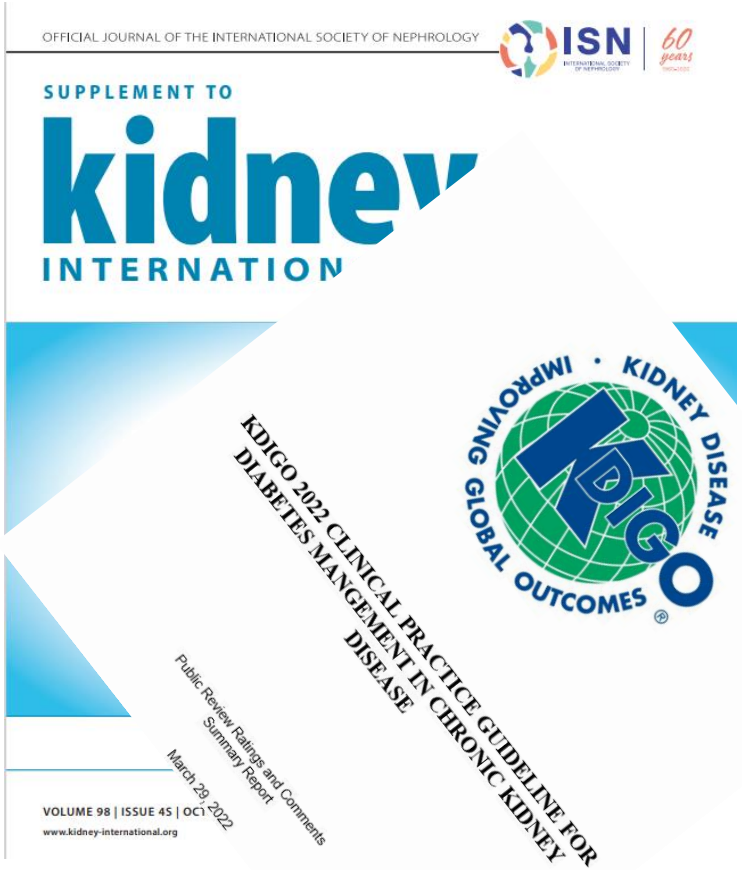
The 6th Annual
HEART IN DIABETES
CME Conference - June 24-26, 2022
Loews Philadelphia Hotel - 1200 Market St, Philadelphia PA 19107
Also Interactive Online Streaming and On-Demand are Available

Disclosures

- Professor Rossing has received the following:
  - Consultancy and/or speaking fees (to his institution) from Astellas, AstraZeneca, Bayer, Boehringer Ingelheim, Eli Lilly, Gilead, MSD, Mundipharma, Novo Nordisk, Vifor, and Sanofi Aventis
  - Research grants from AstraZeneca and Novo Nordisk

Living Guidelines



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KIDNEY DISEASE · IMPROVING GLOBAL OUTCOMES

Chronic Kidney Disease Testing Among Primary Care Patients With Type 2 Diabetes Across 24 U.S. Health Care Organizations

Nikita Stempniewicz,[1] Joseph A. Vassalotti,[2,3] John K. Cuddeback,[1] Elizabeth Ciemins,[1] Amy Storfer-Isser,[4] Yingying Sang,[5] Kunihiro Matsushita,[5] Shoshana H. Ballew,[5] Alex R. Chang,[6] Andrew S. Levey,[7] Robert A. Bailey,[8] Jesse Fishman,[8] and Josef Coresh[5]

Diabetes Care 2021;44:2000–2009 | https://doi.org/10.2337/dc20-2715

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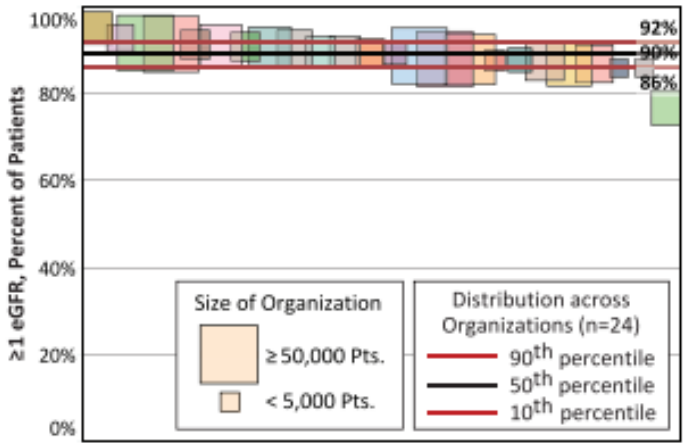

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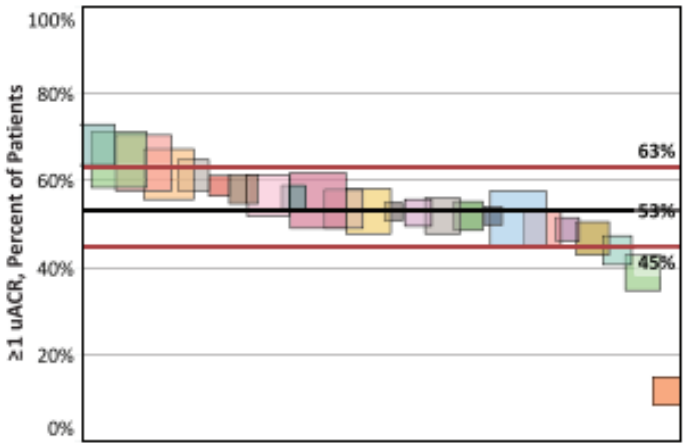
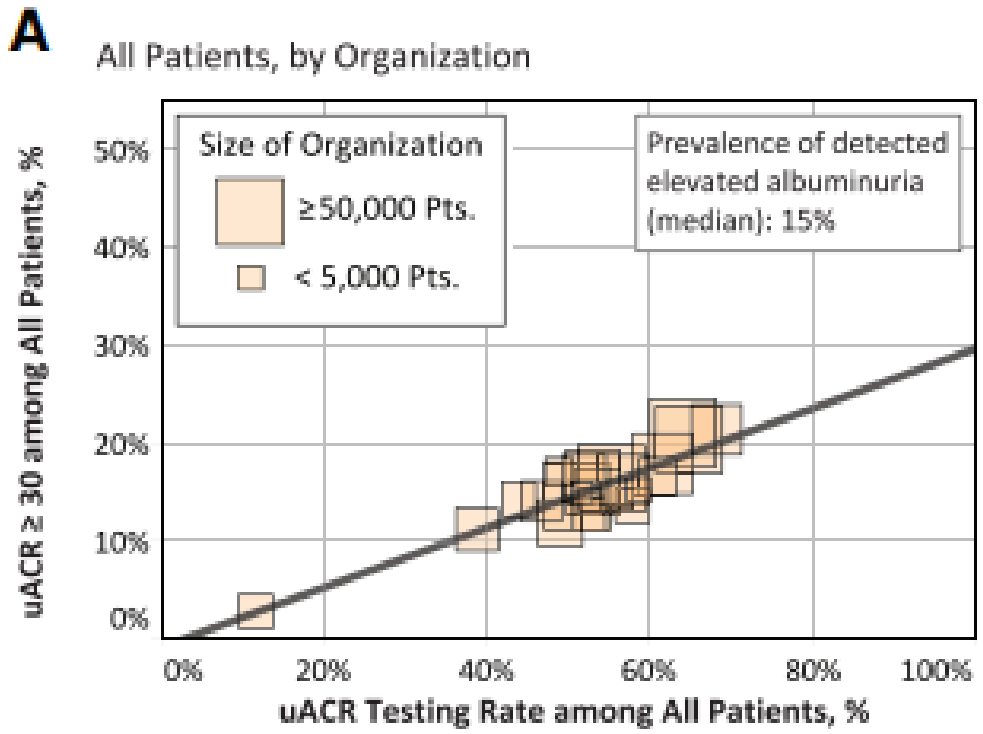
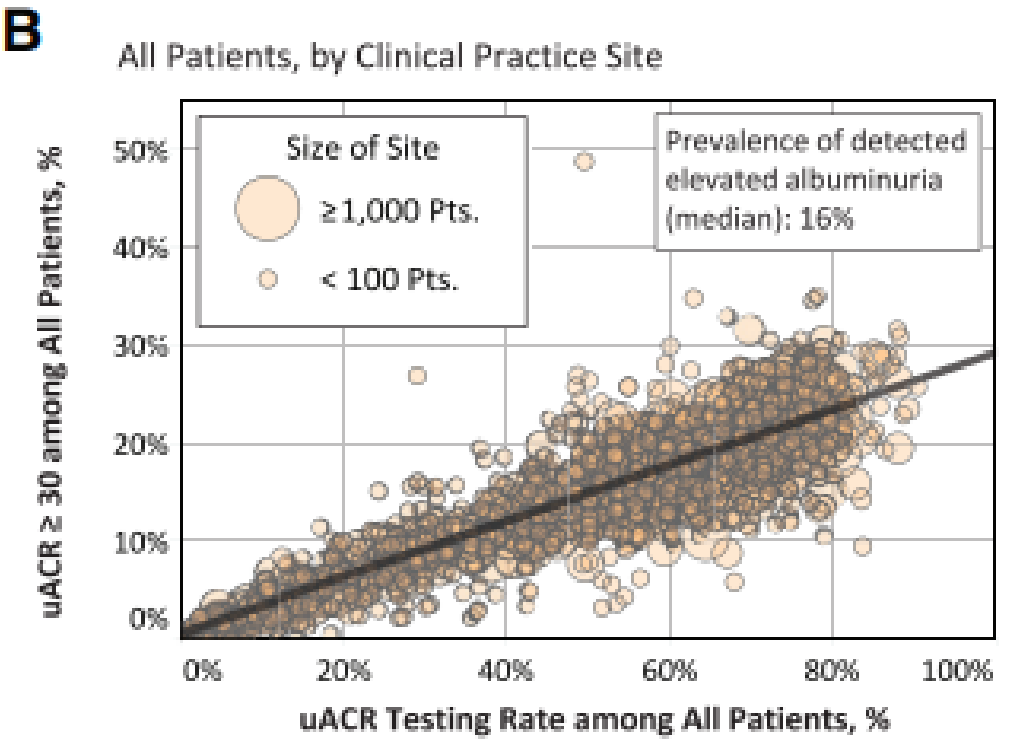


Only 50% are tested within 1 year for UACR and eGFR

You only find what you are looking for (and you don't treat what you don't find)





Screening for CKD in people living with diabetes

Who and when to screen?

T1D Yearly starting 5 years after diagnosis

T2D Yearly starting at diagnosis

How to screen?

Spot urine albumin–creatinine ratio (ACR)

and

Estimated glomerular filtration rate (eGFR)

What to do with a positive result?

Repeat and confirm:

- Evaluate possible temporary or spurious causes
- Consider using cystatin C and creatinine to more precisely estimate GFR
- Only persistent abnormalities define CKD

Initiate evidence-based treatments

What defines CKD diagnosis?

Persistent urine ACR ≥30 mg/g

Persistent eGFR <60 mL/min/1.73 m^2

Other evidence of kidney damage

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Risk of CKD progression, frequency of visits, and referral to nephrology according to GFR and albuminuria

CKD is classified based on: • Cause (C) • GFR (G) • Albuminuria (A)				Albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				< 30 mg/g < 3 mg/mmol	30–299 mg/g 3–29 mg/mmol	≥ 300 mg/g ≥ 30 mg/mmol
GFR categories (ml/min/1.73 m²) Description and range	G1	Normal or high	≥ 90	Screen 1	Treat 1	Treat and refer 3
	G2	Mildly decreased	60–89	Screen 1	Treat 1	Treat and refer 3
	G3a	Mildly to moderately decreased	45–59	Treat 1	Treat 2	Treat and refer 3
	G3b	Moderately to severely decreased	30–44	Treat 2	Treat and refer 3	Treat and refer 3
	G4	Severely decreased	15–29	Treat and refer* 3	Treat and refer* 3	Treat and refer 4+
	G5	Kidney failure	< 15	Treat and refer 4+	Treat and refer 4+	Treat and refer 4+

Low risk (if no other markers of kidney disease, no CKD)

/ increased risk

High risk

Very high risk

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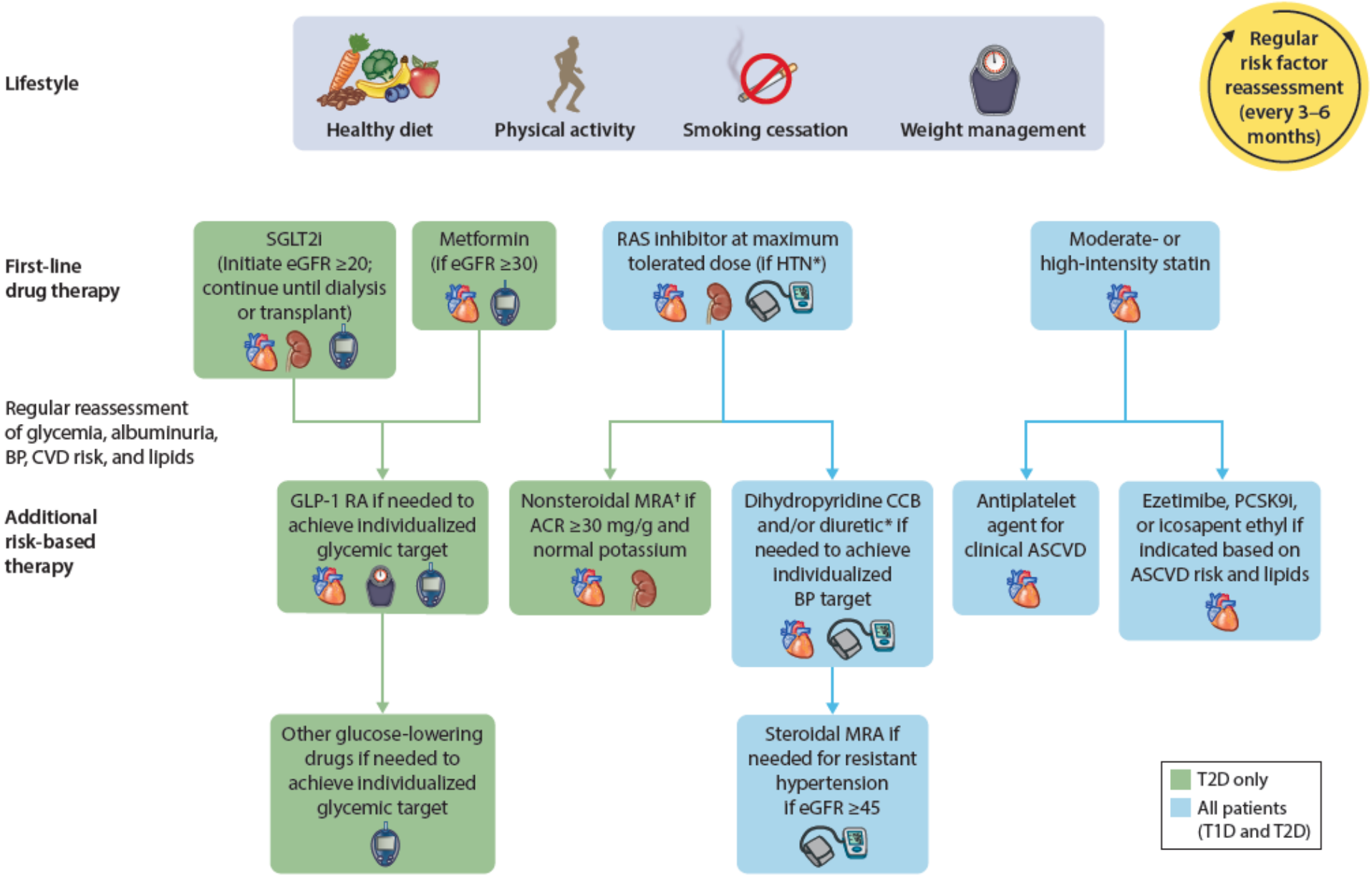
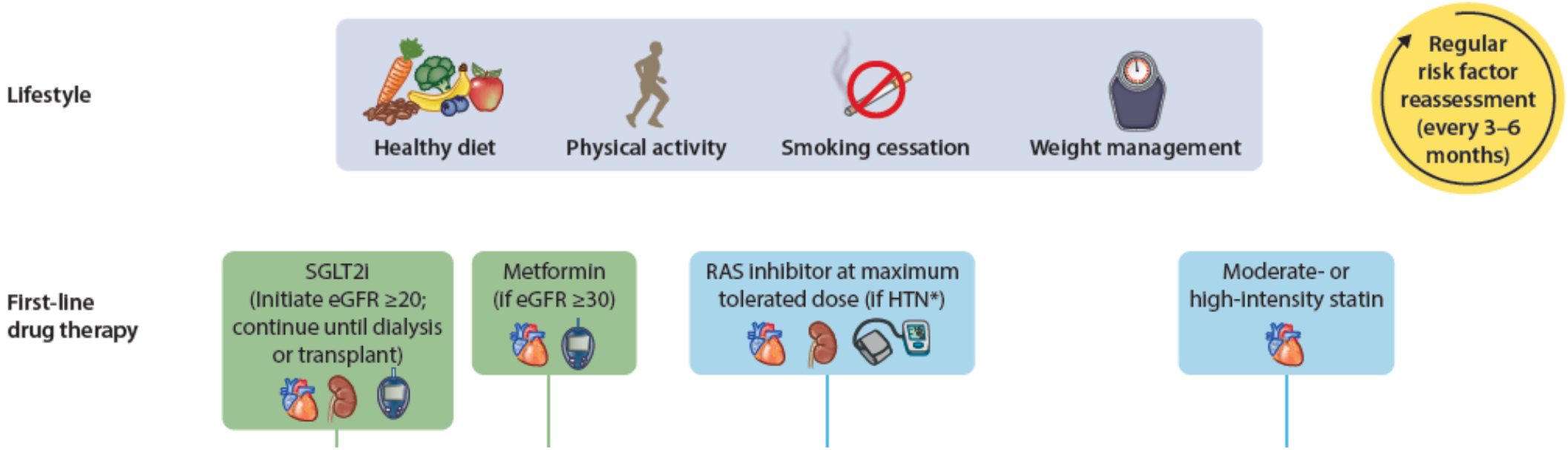
KIDNEY DISEASE · IMPROVING GLOBAL OUTCOMES · KDIGO

Holistic approach for improving outcomes in patients with diabetes and CKD





- **Consensus statement:**
- An SGLT2 inhibitor with proven kidney or cardiovascular benefit is recommended for patients with T2D, CKD, and an eGFR ≥20 mL/min/1.73 m^2.
- Once initiated, the SGLT2 inhibitor can be continued at lower levels of eGFR.

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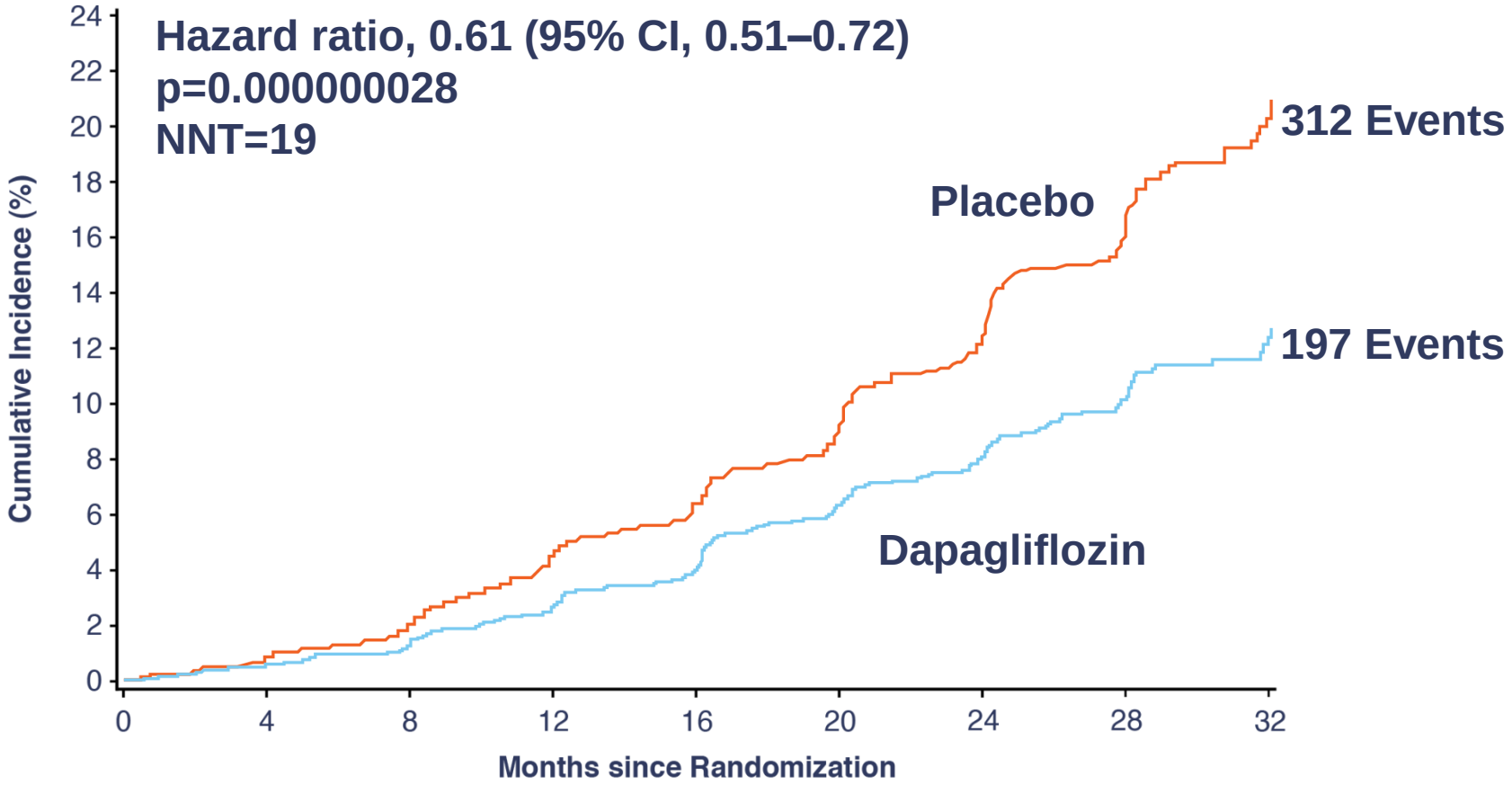
KDIGO KIDNEY DISEASE · IMPROVING GLOBAL OUTCOMES

Primary outcome:

Sustained ≥50% eGFR decline, ESKD, renal or cardiovascular death



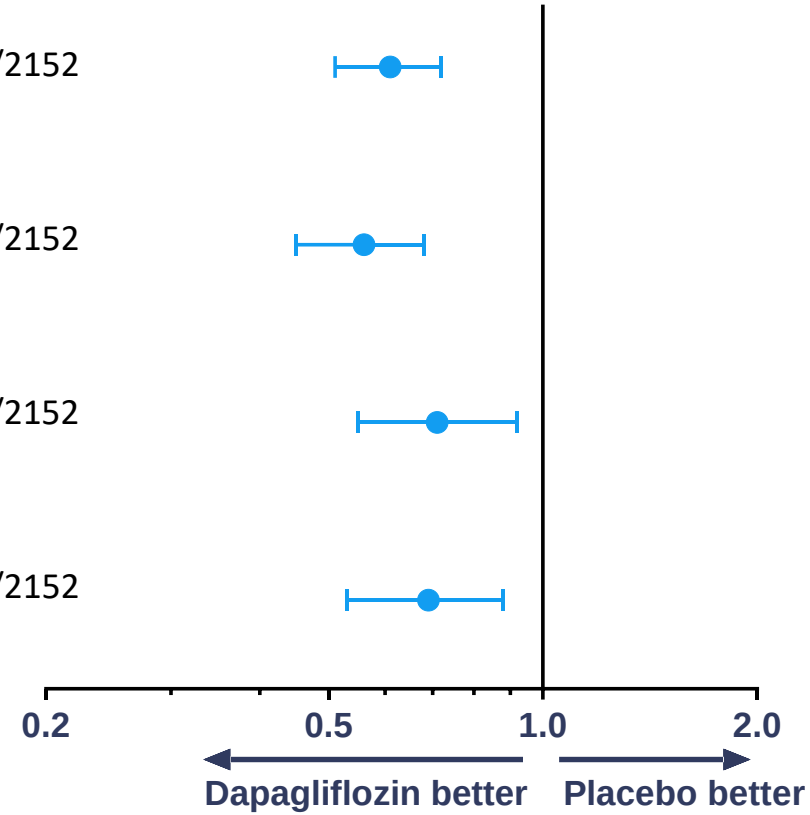
No. at Risk									
Dapagliflozin	2152	2001	1955	1898	1841	1701	1288	831	309
Placebo	2152	1993	1936	1858	1791	1664	1232	774	270

DAPACKD

Heerspink HJL. et.al. NEJM 2020.

Primary and secondary outcomes[1]

	Dapagliflozin	Placebo	Hazard Ratio (95% CI)
	No. of participants/total no.		
Primary outcome	197/2152	312/2152	0.61 (0.51, 0.72)
Kidney-specific outcome	142/2152	243/2152	0.56 (0.45, 0.68)
Cardiovascular death or heart failure hospitalization	100/2152	138/2152	0.71 (0.55, 0.92)
All-cause mortality	101/2152	146/2152	0.69 (0.53, 0.88)

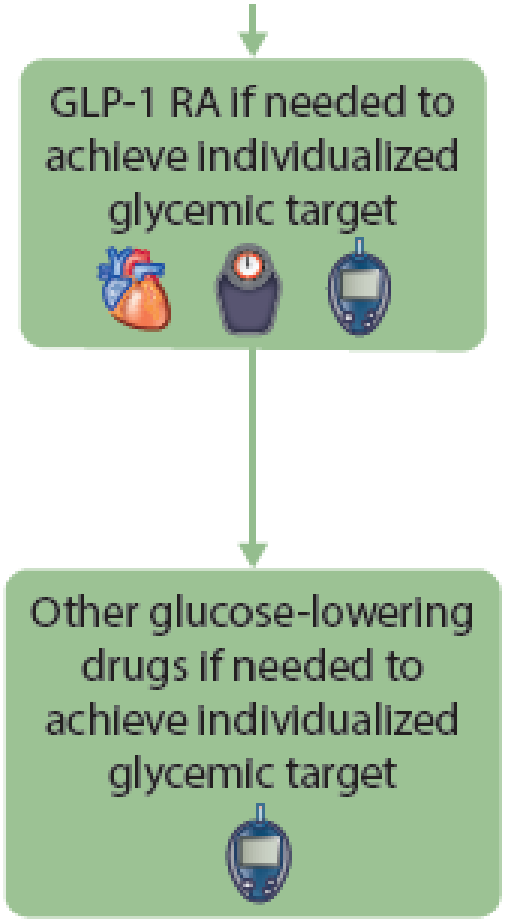


Primary outcome: sustained ≥50% eGFR decline, ESKD, renal or cardiovascular death; Kidney-specific outcome: sustained ≥50% eGFR decline, ESKD, renal death

eGFR, estimated glomerular filtration rate; ESKD, end-stage kidney disease

1: Heerspink HJL. et.al. *N Engl J Med* 2020;383:1436-1446.

GLP-1 RA when glucose control is not at target



Consensus statement:

A GLP-1 receptor agonist with proven cardiovascular benefit is recommended for patients with T2D and CKD who do not meet their individualized glycemic target with metformin and/or an SGLT2 inhibitor or because they are unable to use these drugs.

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KDIGO KIDNEY DISEASE · IMPROVING GLOBAL OUTCOMES

Importance of Glycemic Control

Averting symptomatic hyperglycemia

Substantial and enduring reduction in microvascular complications

- 50-76% reduction DCCT with A1c 7% vs 9%
- 25% reduction UKPDS with A1C 7% vs 7.9%
- Greatest benefit with reduction from higher levels of A1C

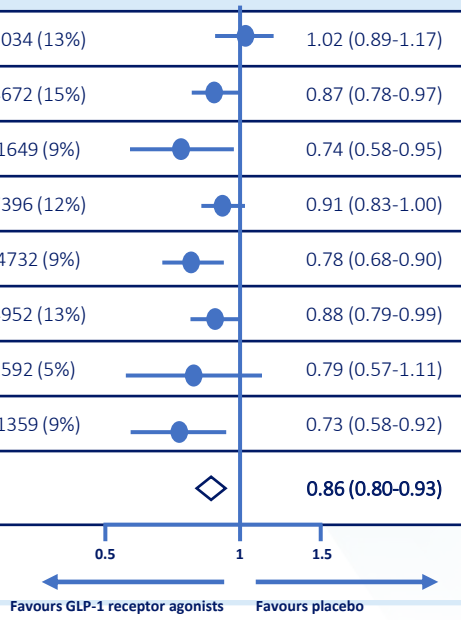
Uncertainty regarding macrovascular benefit of BG control in T2D

Benefits emerge slowly while harms of glucose control medications can be more immediate

GLP-1 Receptor Agonists Cardiovascular Outcomes Trials in Type 2 Diabetes

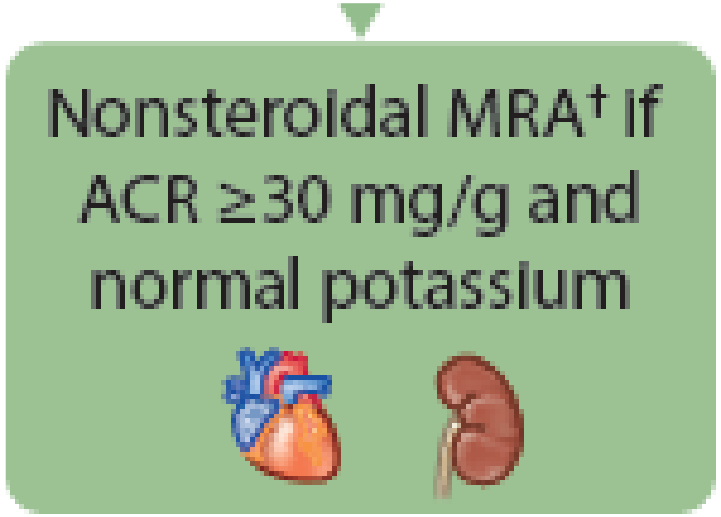
- Reduce risk of major adverse CVD events.
  - Atherosclerotic events
  - CVD death
  - Decrease macroalbuminuria.
- Reduce eGFR decline from early- to late-stage CKD.
- CVD and CKD benefits and safety have been demonstrated in patients with pre-existing CKD.

	GLP-1 Receptor agonist, n/N (%)	Placebo n/N (%)	Hazard ratio (95% CI)	NNT (95% CI)	*p* value
Three-point MACE					
ELIXA	400/3034 (13%)	392/3034 (13%)	1.02 (0.89-1.17)		0.78
LEADER	608 (4668 (13%)	694/4672 (15%)	0.87 (0.78-0.97)		0.01
SUSTAIN-6	108 (1648 (7%)	146/1649 (9%)	0.74 (0.58-0.95)		0.016
EXSCEL	839/7356 (11%)	905/7396 (12%)	0.91 (0.83-1.00)		0.061
Harmony Outcomes	338/4731 (7%)	428/4732 (9%)	0.78 (0.68-0.90)		0.0006
REWIND	594/4949 (12%)	663/4952 (13%)	0.88 (0.79-0.99)		0.026
PIONEER 6	61/1591 (4%)	76/1592 (5%)	0.79 (0.57-1.11)		0.17
AMPLITUDE-O	189/2717 (7%)	125/1359 (9%)	0.73 (0.58-0.92)		0.0069
Subtotal (I^2=44.5%, p=0.082)			0.86 (0.80-0.93)	65 (45-130)	<0.0001



Sattar N et al. Lancet Diabetes Endocrinol. 2021; S2213-8587(21)00203-5

Nonsteroidal MRA after RASi in T2D with residual kidney and CV risk



Consensus statement

A nonsteroidal mineralocorticoid receptor antagonist with proven kidney and cardiovascular benefit is recommended for patients with T2D, an eGFR ≥25 mL/min/1.73 m^2, normal serum potassium concentration, and albuminuria (ACR ≥30 mg/g) despite maximum tolerated dose of RAS inhibitor.

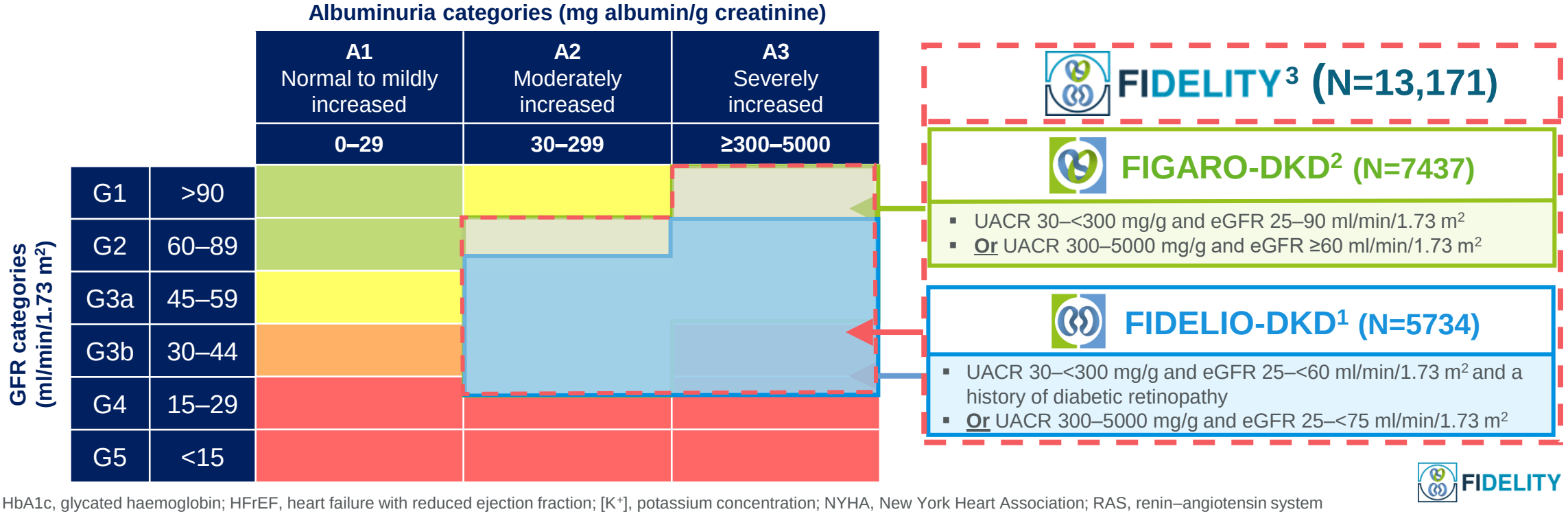
The FIDELITY prespecified pooled analysis combined data from FIDELIO-DKD and FIGARO-DKD, including patients across a broad spectrum of eGFR and CKD categories

Key inclusion criteria

- Aged ≥18 years with T2D
- On maximum tolerated dose of RAS inhibitor for ≥4 weeks
- Diabetic retinopathy for patients with A2 albuminuria (FIDELIO-DKD only)
- Moderately/severely increased albuminuria
- Serum [K^+] ≤4.8 mmol/l

Key exclusion criteria

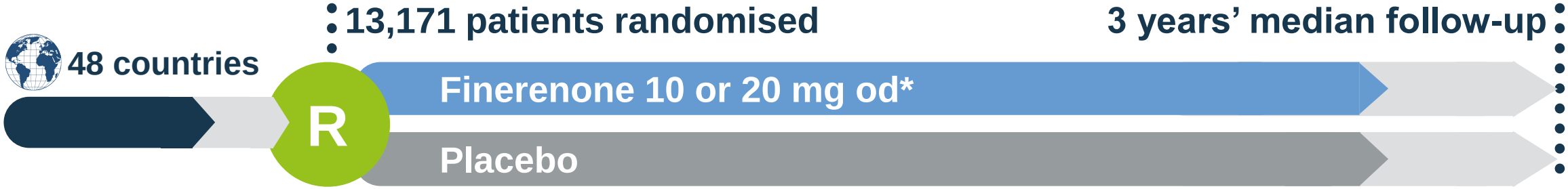
- HFrEF with NYHA Class II–IV
- HbA1c >12%
- Uncontrolled arterial hypertension
- Other kidney disease



HbA1c, glycated haemoglobin; HFrEF, heart failure with reduced ejection fraction; [K^+], potassium concentration; NYHA, New York Heart Association; RAS, renin–angiotensin system

1. Bakris G, *et al. N Engl J Med* 2020;383:2219–2229; 2. Ruilope LM, *et al. Am J Nephrol* 2019;50:345–356; 3. Filippatos G, *et al. ESC* 2021; abstract 7161

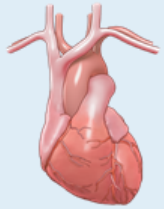
FIDELITY is a prespecified pooled analysis of FIDELIO-DKD and FIGARO-DKD



Key outcomes

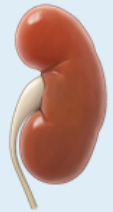
CV composite

Time to CV death, non-fatal MI, non-fatal stroke, or HHF



57% eGFR kidney composite

Time to kidney failure,[#] sustained ≥57% decrease in eGFR from baseline, or renal death



Other outcomes

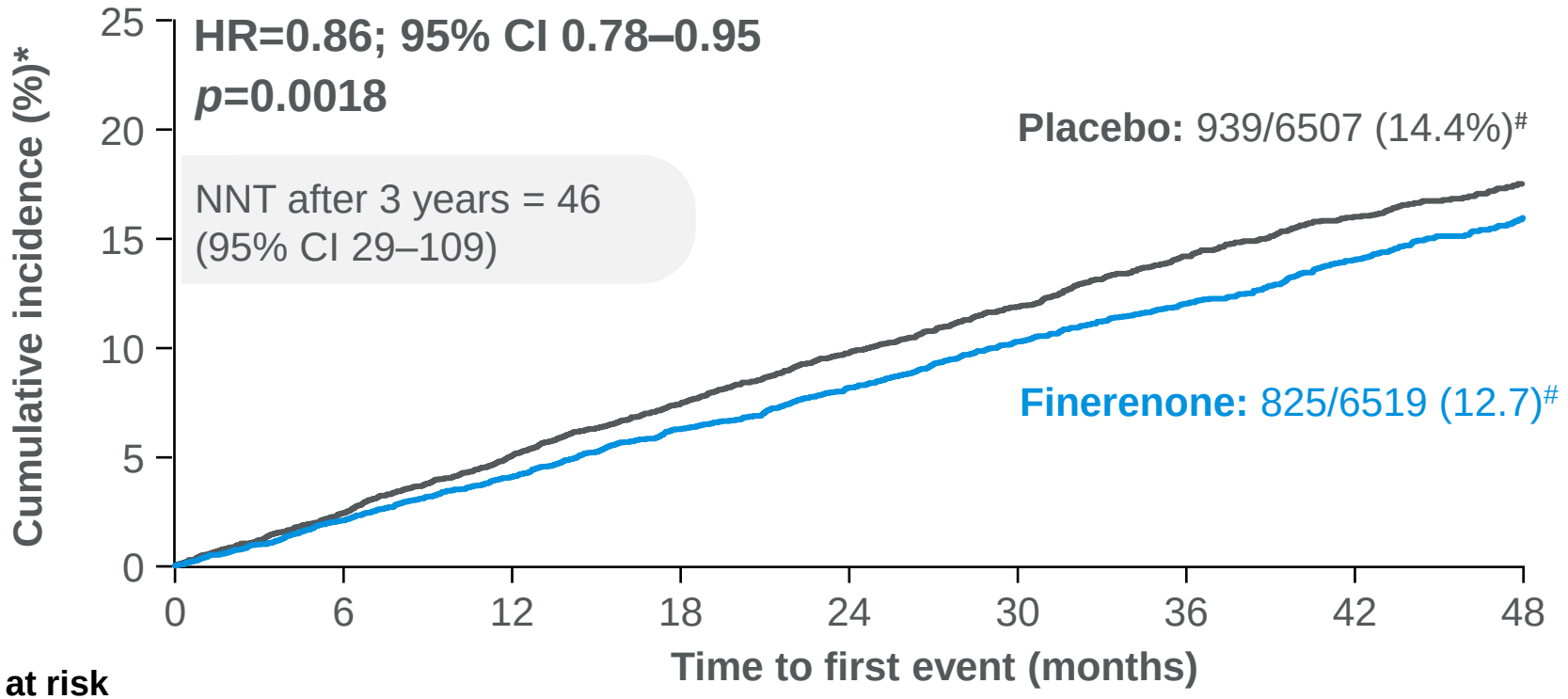
- Time to first occurrence of onset of kidney failure, a sustained ≥40% decrease of eGFR from baseline,* or renal death[#]
- Time to death from any cause
- Time to hospitalisation for any cause
- Change in UACR from baseline to month 4

*10 mg if screening eGFR 25–<60 ml/min/1.73 m^2; 20 mg if ≥60 ml/min/1.73 m^2, up-titration encouraged from month 1 if serum potassium ≤4.8 mEq/l and eGFR stable; [#]kidney failure defined as either ESKD (initiation of chronic dialysis for ≥90 days or kidney transplant) or sustained decrease in eGFR <15 ml/min/1.73 m^2
CV, cardiovascular; HHF, hospitalisation for heart failure; MI, myocardial infarction; od, once daily

R Agarwal et al Eur Heart J. 2022 Feb 10;43(6):474-484

On top of optimised RAS blockade, finerenone significantly reduced the risk of the composite CV outcome by 14%

Time to CV death, non-fatal MI, non-fatal stroke, or hospitalisation for HF

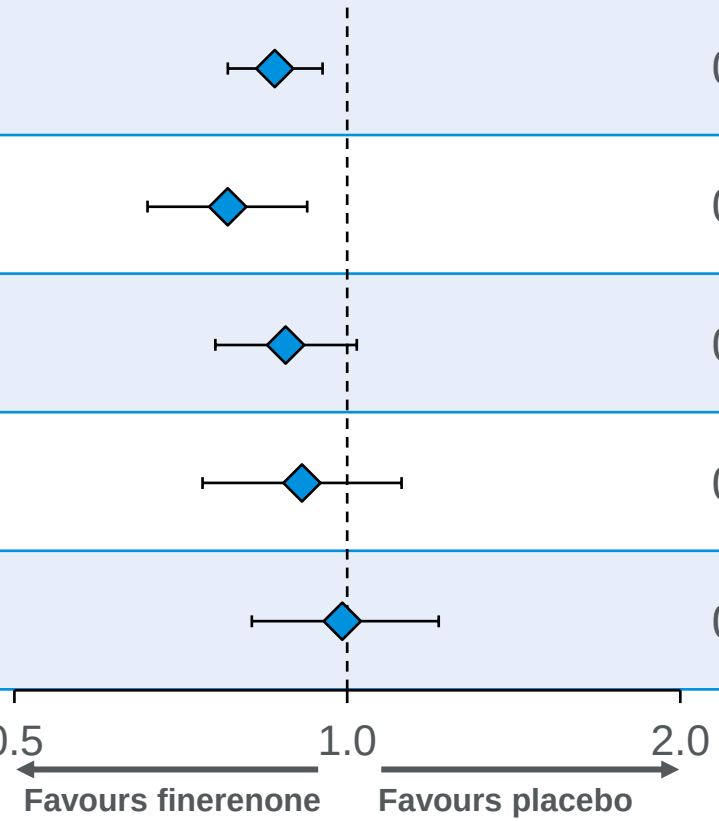


No. at risk									
Finerenone	6519	6360	6202	6009	5273	4207	3065	2187	1087
Placebo	6507	6330	6125	5938	5184	4147	2969	2135	1082

*Cumulative incidence calculated by Aalen–Johansen estimator using deaths due to other causes as competing risk; #number of patients with an event over a median of 3.0 years of follow-up
CI, confidence interval; CV, cardiovascular; HF, heart failure; HR, hazard ratio; MI, myocardial infarction; NNT, number needed to treat; RAS, renin–angiotensin system
Filippatos G. Abstract 7161 presented at the European Society of Cardiology 2021 (ESC 2021)

The CV benefits of finerenone were primarily driven by reduction in HHF, and also CV death

Outcome	Finerenone (n=6519) n (%)	Placebo (n=6507) n (%)	HR (95% CI)	*p*-value
Composite CV outcome	825 (12.7)	939 (14.4)	0.86 (0.76–0.95)	0.0018
HHF	256 (3.9)	325 (5.0)	0.78 (0.66–0.92)	0.0030
CV death	322 (4.9)	364 (5.6)	0.88 (0.76–1.02)	0.092
Non-fatal MI	173 (2.7)	189 (2.8)	0.91 (0.74–1.12)	0.36
Non-fatal stroke	198 (3.0)	198 (3.0)	0.99 (0.82–1.21)	0.95

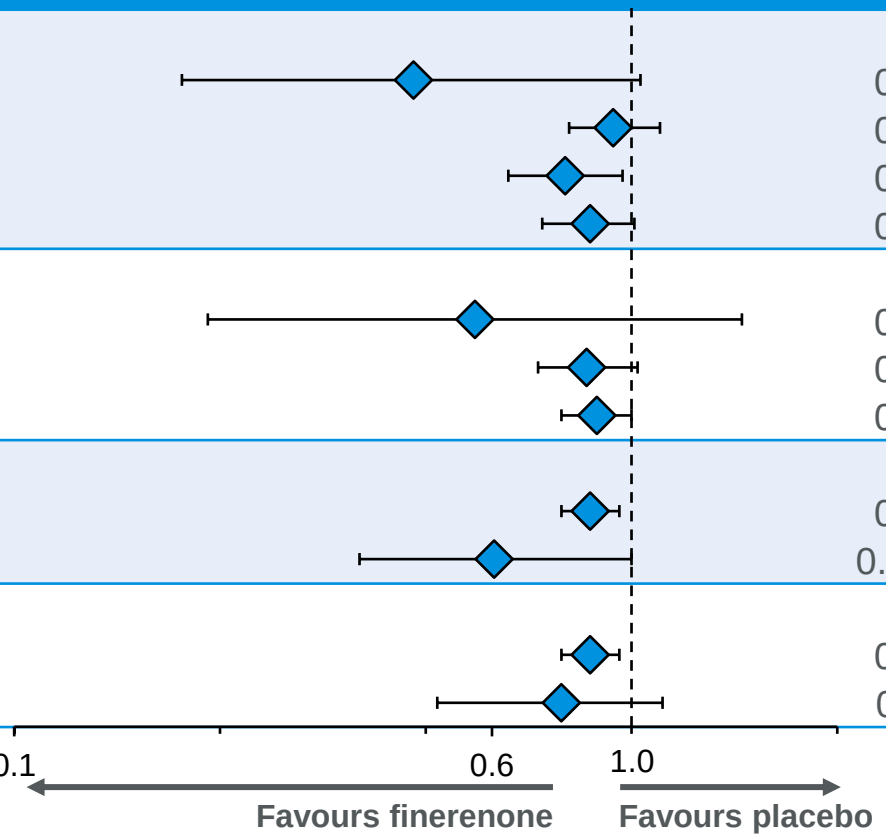


CI, confidence interval; CV, cardiovascular; HHF, hospitalisation for heart failure; HR, hazard ratio; MI, myocardial infarction

Filippatos G. Abstract 7161 presented at the European Society of Cardiology 2021 (ESC 2021)

The CV benefits of finerenone were consistent regardless of baseline eGFR or UACR, and use of SGLT-2is or GLP-1RAs

Category	Finerenone (n=6519) n/N (n/100PY)	Placebo (n=6507) n/N (n/100PY)	HR (95% CI)	$p_{interaction}$
Baseline eGFR, ml/min/1.73 m^2				0.14
<25	11/81 (5.2)	23/81 (12.2)	0.48 (0.22–1.03)	
25–<45	321/2117 (5.7)	331/2115 (5.8)	0.94 (0.81–1.10)	
45–<60	197/1717 (4.0)	247/1717 (5.1)	0.80 (0.66–0.97)	
≥60	295/2603 (3.6)	337/2592 (4.2)	0.87 (0.74–1.01)	
Baseline UACR, mg/g				0.41
<30	10/120 (2.4)	15/110 (4.3)	0.59 (0.24–1.45)	
30–<300	260/1076 (3.8)	292/2023 (4.4)	0.86 (0.73–1.02)	
≥300	554/4321 (4.7)	631/4371 (5.4)	0.89 (0.79–1.00)	
Baseline SGLT-2i				0.41
No	786/6081 (4.4)	887/6068 (5.1)	0.87 (0.79–0.96)	
Yes	39/438 (3.0)	52/439 (4.1)	0.63 (0.40–<1.00*)	
Baseline GLP-1RA				0.63
No	767/6022 (4.4)	875/6060 (5.0)	0.87 (0.79–0.96)	
Yes	58/497 (3.8)	64/447 (4.9)	0.76 (0.52–1.11)	



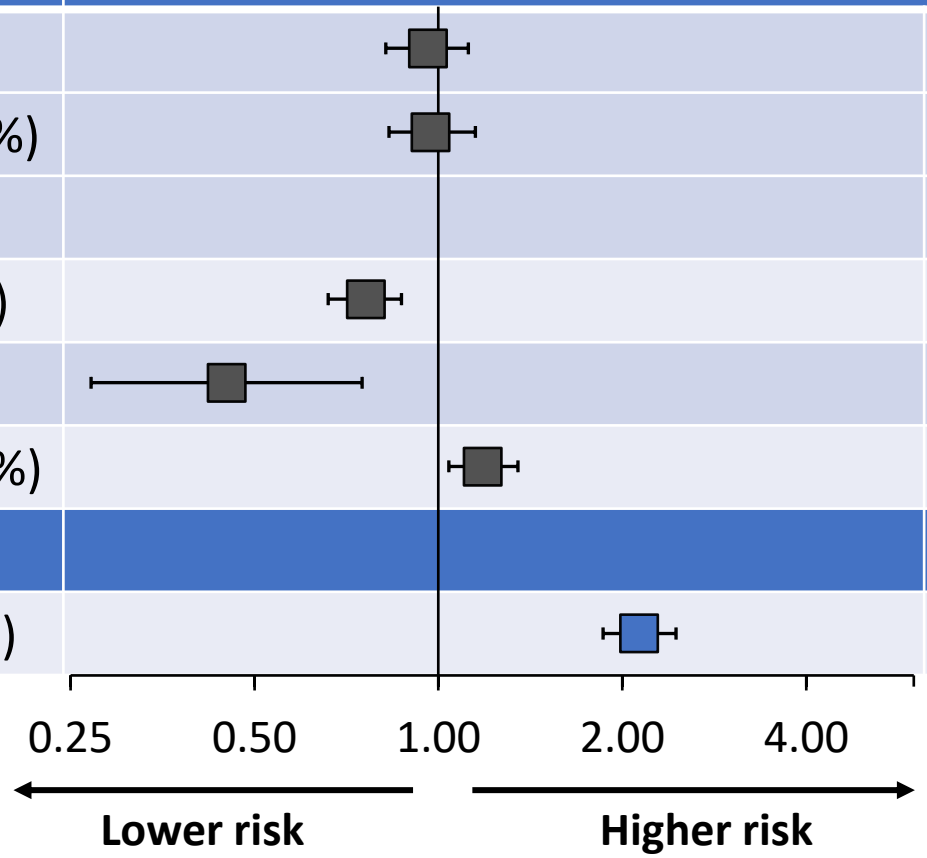
*Upper CI=0.996

CI, confidence interval; CV, cardiovascular; eGFR, estimated glomerular filtration rate; GLP-1RA, glucagon-like peptide-1 receptor agonist; HR, hazard ratio; PY, patient-years; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; ; UACR, urine albumin-to-creatinine ratio

Filippatos G. Abstract 7161 presented at the European Society of Cardiology 2021 (ESC 2021)

SGLT2i and diuretic use at baseline, but not RASi dosing was associated with lower risk of hyperkalemia

Variable, %*		HR (95% CI)	*p*-value
ACEi or ARB dosing#	≤min dose (30%)	0.96 (0.82–1.12)	0.8508
	>min but <max dose (25%)	0.97 (0.83–1.15)	
	≥max dose (45%)	Reference	
Diuretic use, yes (57%) vs no (43%)		0.76 (0.66–0.87)	<0.0001
SGLT-2i use, yes (5%) vs no (95%)		0.45 (0.27–0.75)	0.0023
β-blocker use, yes (52%) vs no (48%)		1.18 (1.04–1.35)	0.0133
Treatment assignment			
Finerenone (50%) vs placebo (50%)		2.13 (1.86–2.45)	<0.0001

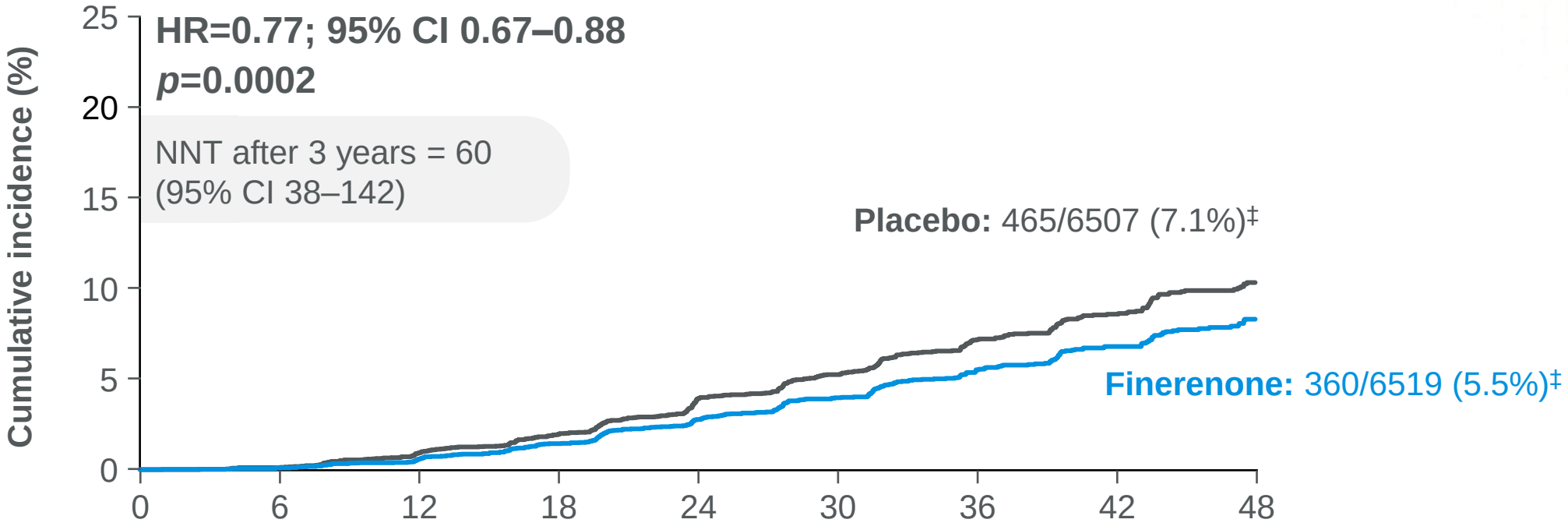


*Proportion of patients per group in safety population
#According to dose recommended by the product label; dosing information was missing for 0.3% of patients
ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CI, confidence interval; HR, hazard ratio; RASi, renin-angiotensin system inhibitor; SGLT-2i, sodium-glucose co-transporter-2 inhibitor

R Agarwal et al J Am Soc Nephrol. 2022 Jan;33(1):225-237

Finerenone significantly reduced the risk of the ≥57% eGFR kidney composite outcome by 23%

Time to kidney failure,* sustained ≥57% decrease in eGFR from baseline, or renal death#



No. at risk									
Finerenone	6519	6291	6107	5848	5027	3973	2815	2024	959
Placebo	6507	6292	6071	5815	4949	3932	2798	1988	962

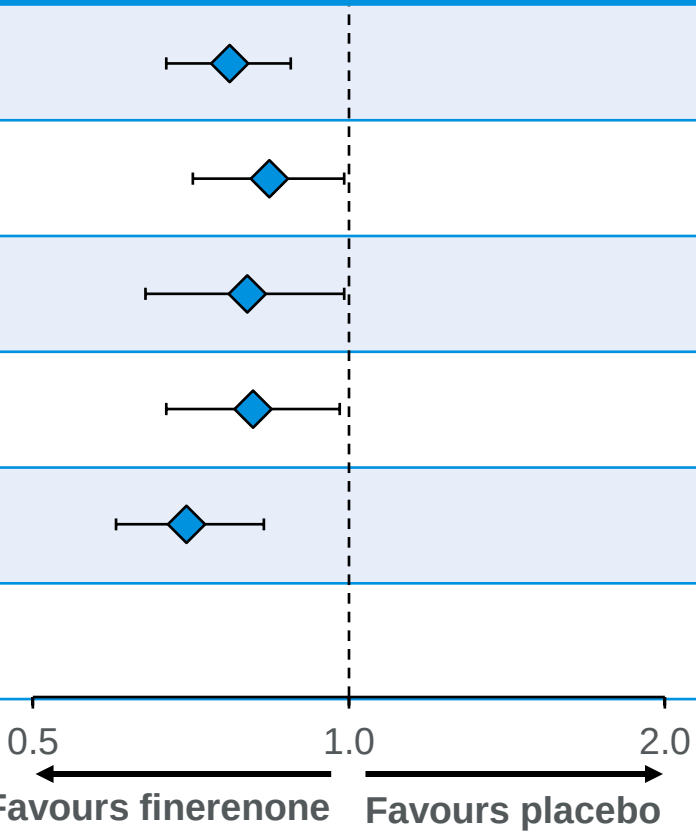
*ESKD or an eGFR <15 ml/min/1.73 m^2; #events were classified as renal death if: (1) the patient died; (2) kidney replacement therapy had not been initiated despite being clinically indicated; and (3) there was no other likely cause of death; ‡cumulative incidence calculated by Aalen–Johansen estimator using deaths due to other causes as competing risk; ¶number of patients with an event over a median of 3.0 years of follow-up

CI, confidence interval; eGFR, estimated glomerular filtration rate; ESKD, end-stage kidney disease; HR, hazard ratio; NNT, number needed to treat

Filippatos G. Abstract 7161 presented at the European Society of Cardiology 2021 (ESC 2021)

Finerenone significantly reduced the incidences of all components of the kidney composite outcome including ESKD (except renal death*)

Outcome	Finerenone (n=6519) n (%)	Placebo (n=6507) n (%)	HR (95% CI)	*p*-value
eGFR 57% composite kidney outcome	360 (5.5)	465 (7.1)	0.77 (0.67–0.88)	0.0002
Kidney failure	254 (3.9)	297 (4.6)	0.84 (0.71–0.99)	0.039
ESKD[#]	151 (2.3)	188 (2.9)	0.80 (0.64–0.99)	0.040[‡]
eGFR <15 ml/min/1.73 m^2[¶]	195 (3.0)	237 (3.6)	0.81 (0.67–0.98)	0.026[‡]
≥57% decrease in eGFR from baseline[¶]	257 (3.9)	361 (5.5)	0.70 (0.60–0.83)	<0.0001
Renal death	2 (<0.1)	4 (<0.1)	0.53 (0.10–2.91)	–



≥57% decrease in eGFR is equivalent to doubling of serum creatinine

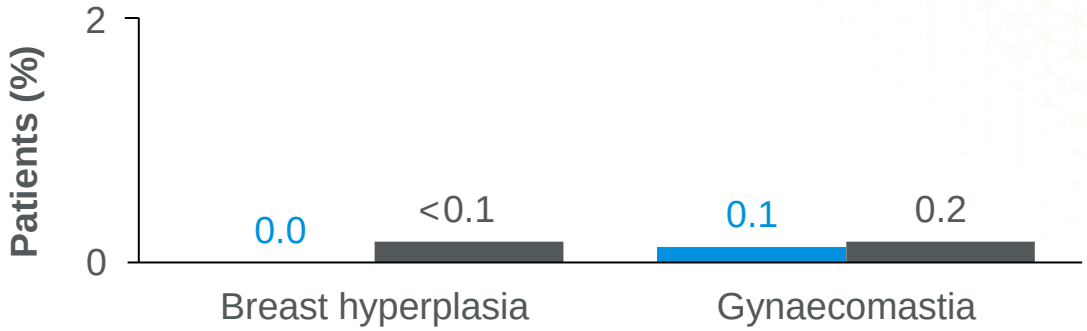
*Only 6 patients experienced renal death; [#]initiation of chronic dialysis for ≥90 days or kidney transplant; [‡]analysis for *p*-values not prespecified; [¶]confirmed by two eGFR measurements ≥4 weeks apart
CI, confidence interval; eGFR, estimated glomerular filtration rate; ESKD, end-stage kidney disease; HR, hazard ratio
Filippatos G. Abstract 7161 presented at the European Society of Cardiology 2021 (ESC 2021)

Finerenone showed modest effects on SBP and no sexual side effects. Hyperkalaemia was increased but clinical impact was low

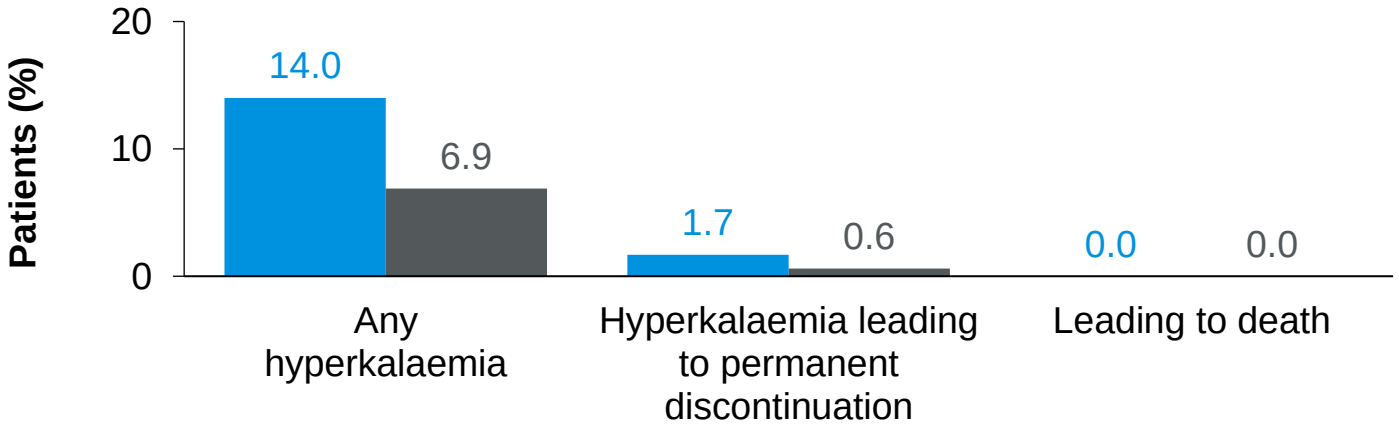
Modest effect on systolic blood pressure

Placebo-corrected change in mean SBP of **–3.7 mmHg** at 4 months

No sexual side-effects



Increased hyperkalaemia with minimal impact



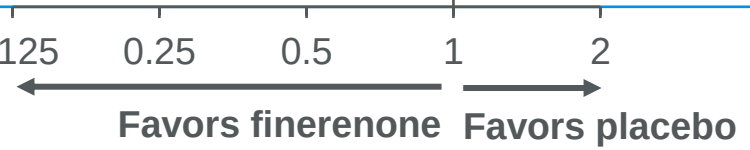
Finerenone (n=6510)
Placebo (n=6489)

SBP, systolic blood pressure

Bakris GL, *et al. N Engl J Med* 2020;383:2219–2229; Pitt B, *et al*. *N Engl J Med* 2021;doi:10.1056/NEJMoa2110956

Finerenone reduced the risk of the kidney composite endpoint outcomes irrespective of SGLT-2i use

Endpoint	Finerenone n/N (n/100 PY)	Placebo n/N (n/100 PY)	HR (95% CI)	Adjusted HR (95% CI)*	*p*-value for interaction
eGFR 57% kidney composite#					
No SGLT-2i	351/6081 (2.1)	448/6068 (2.6)		0.80 (0.69–0.92)	0.29
SGLT-2i	9/438 (0.7)	17/439 (1.4)		0.42 (0.16–1.08)	
eGFR 40% kidney composite‡					
No SGLT-2i	818/6081 (5.0)	961/6068 (5.9)		0.84 (0.76–0.92)	0.59
SGLT-2i	36/438 (2.9)	34/439 (2.8)		0.70 (0.41–1.21)	



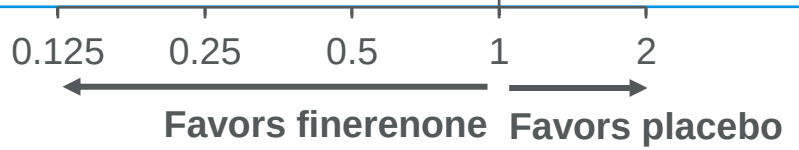
*Adjusted HR for HbA1c, SBP, UACR at baseline (log-transformed), eGFR at baseline; #eGFR 57% kidney composite outcome defined as kidney failure (ESKD or eGFR <15 ml/min/1.73 m^2), a sustained ≥57% decrease in eGFR from baseline (equivalent to a doubling of serum creatinine) for ≥4 weeks, or renal death; ‡ eGFR 40% kidney composite outcome defined as kidney failure (ESKD or eGFR <15 ml/min/1.73 m^2), a sustained ≥40% decrease in eGFR from baseline maintained for ≥4 weeks, or renal death

CI, confidence interval; eGFR, estimated glomerular filtration rate; ESKD, end-stage kidney disease; HbA1c, glycated hemoglobin; HR, hazard ratio; PY, patient-years; SBP, systolic blood pressure; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urine albumin-to-creatinine ratio

FIDELITY

The CV benefit of finerenone was consistent irrespective of SGLT-2i use

Endpoint	Finerenone n/N (n/100 PY)	Placebo n/N (n/100 PY)	HR (95% CI)	Adjusted HR (95% CI)*	*p*-value for interaction
CV composite#					
No SGLT-2i	786/6081 (4.4)	887/6068 (5.1)		0.87 (0.79–0.96)	0.41
SGLT-2i	39/438 (3.0)	52/439 (4.1)		0.63 (0.40–1.00)	
HHF					
No SGLT-2i	246/6081 (1.4)	303/6068 (1.7)		0.80 (0.68–0.95)	0.16
SGLT-2i	10/438 (0.7)	22/439 (1.7)		0.45 (0.21–0.99)	



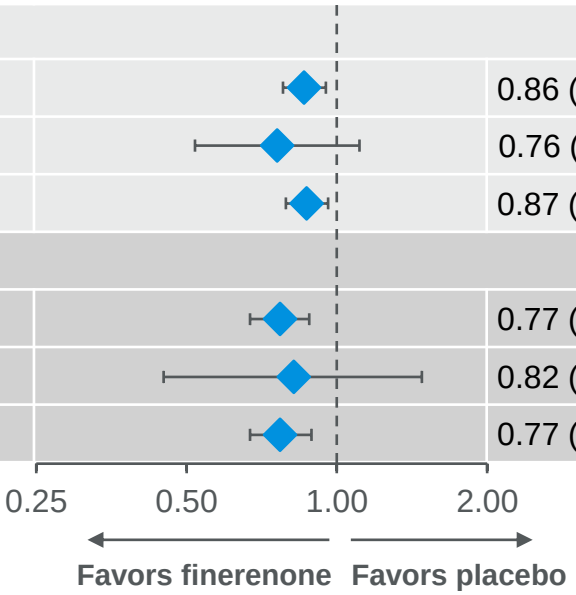
*Adjusted HR for HbA1c, SBP, UACR at baseline (log-transformed), eGFR at baseline; #CV composite defined as composite of CV death, non-fatal MI, non-fatal stroke, or HHF
CI, confidence interval; CV, cardiovascular; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; HHF, hospitalization for heart failure; HR, hazard ratio; MI, myocardial infarction; PY, patient-years; SBP, systolic blood pressure; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; UACR, urine albumin-to-creatinine ratio

FIDELITY

Finerenone reduced the risk of the CV and kidney composite outcomes compared with placebo, irrespective of GLP-1RA use

Composite efficacy outcomes by GLP-1RA use at baseline

Endpoint	n/N (%)		n events per 100 PY		Hazard ratio (95% CI)	$P_{interaction}$
	Finerenone	Placebo	Finerenone	Placebo		
Composite CV outcome*						
Overall	825/6519 (12.6)	939/6507 (14.4)	4.34	5.01	0.86 (0.78–0.95)	0.63
GLP-1RA use at baseline	58/497 (11.7)	64/447 (14.3)	3.79	4.90	0.76 (0.52–1.11)	
No GLP-1RA use at baseline	767/6022 (12.7)	875/6060 (14.4)	4.38	5.02	0.87 (0.79–0.96)	
Kidney composite outcome#						
Overall	360/6519 (5.5)	465/6507 (7.1)	1.96	2.55	0.77 (0.67–0.88)	0.79
GLP-1RA use at baseline	22/497 (4.4)	27/447 (6.0)	1.47	2.10	0.82 (0.45–1.48)	
No GLP-1RA use at baseline	338/6022 (5.6)	438/6060 (7.2)	2.01	2.59	0.77 (0.67–0.89)	



Time-varying analyses showed that finerenone reduced the risk of the composite CV outcome and kidney composite outcome vs. placebo regardless of GLP-1RA use at any time during the study and not just at baseline ($P_{interaction}$=0.40 and $P_{interaction}$=0.33, respectively)

*Included time to CV death, non-fatal MI, non-fatal stroke or HHF; #Included time to kidney failure, sustained ≥57% eGFR decline from baseline or renal death
PY, patient-years

Chronic Kidney Disease—Treatment (continued)

11.3c In patients with chronic kidney disease who are at increased risk for cardiovascular events or chronic kidney disease progression or are unable to use a sodium–glucose cotransporter 2 inhibitor, a nonsteroidal mineralocorticoid receptor antagonist (finerenone) is recommended to reduce chronic kidney disease progression and cardiovascular events (Table 9.2).A

APPROVED BY FDA AND EMA

RECOMMENDED IN GUIDELINES!!

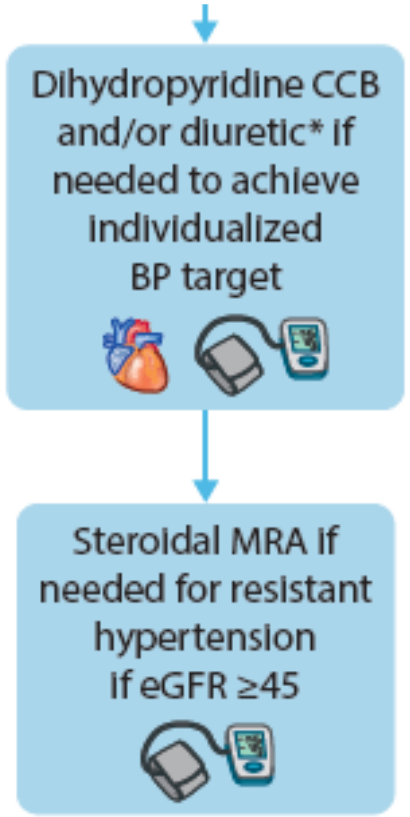
Summary for finerenone

- Overall, AEs were balanced between finerenone and placebo[1,2]
- Modest reduction in SBP with finerenone[1,2]
- Finerenone has no effects on HbA1c[1,2]
- Sexual side effects were balanced between groups[1–3]
- Routine serum [K^+] monitoring minimised the clinical impact of hyperkalaemia with low rates of finerenone discontinuation due to hyperkalaemia[1–4]

Finerenone shows long-term kidney and CV benefits in patients with CKD and T2D[1–3]
Elevations in serum [K^+] are predictable and manageable through routine monitoring[5]

1. Bakris GL, *et al*. *N Engl J Med* 2020;383:2219–2229; 2. Pitt B, *et al. N Engl J Med* 2021; doi: 10.1056/NEJMoa2110956; 3. Filippatos G and Agarwal R, presented at the ESC Congress presented at ESC Congress 2021 Hot Line session 28 August 2021 available at: https://esc365.escardio.org/presentation/238815; 4. Agarwal R. *WCN* 2021; abstract WCN21-0607

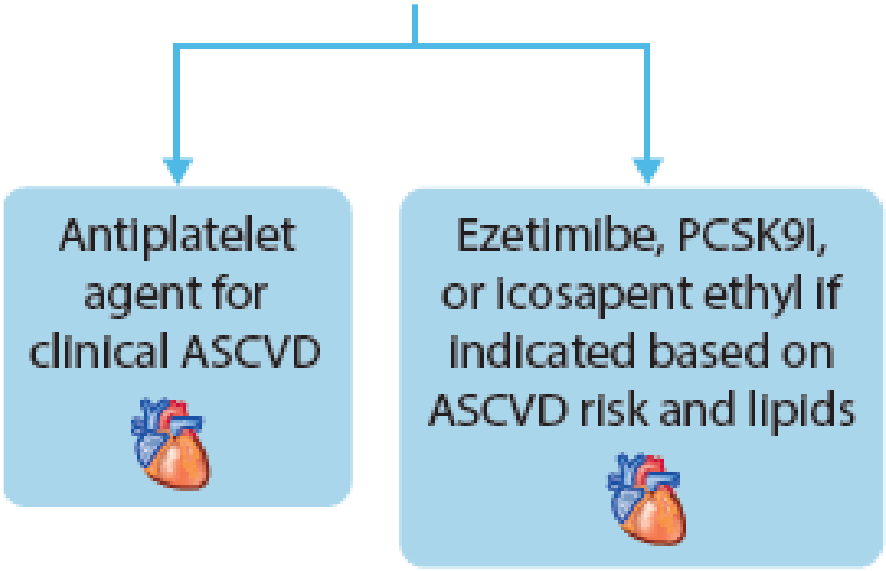
Control of BP after RASi



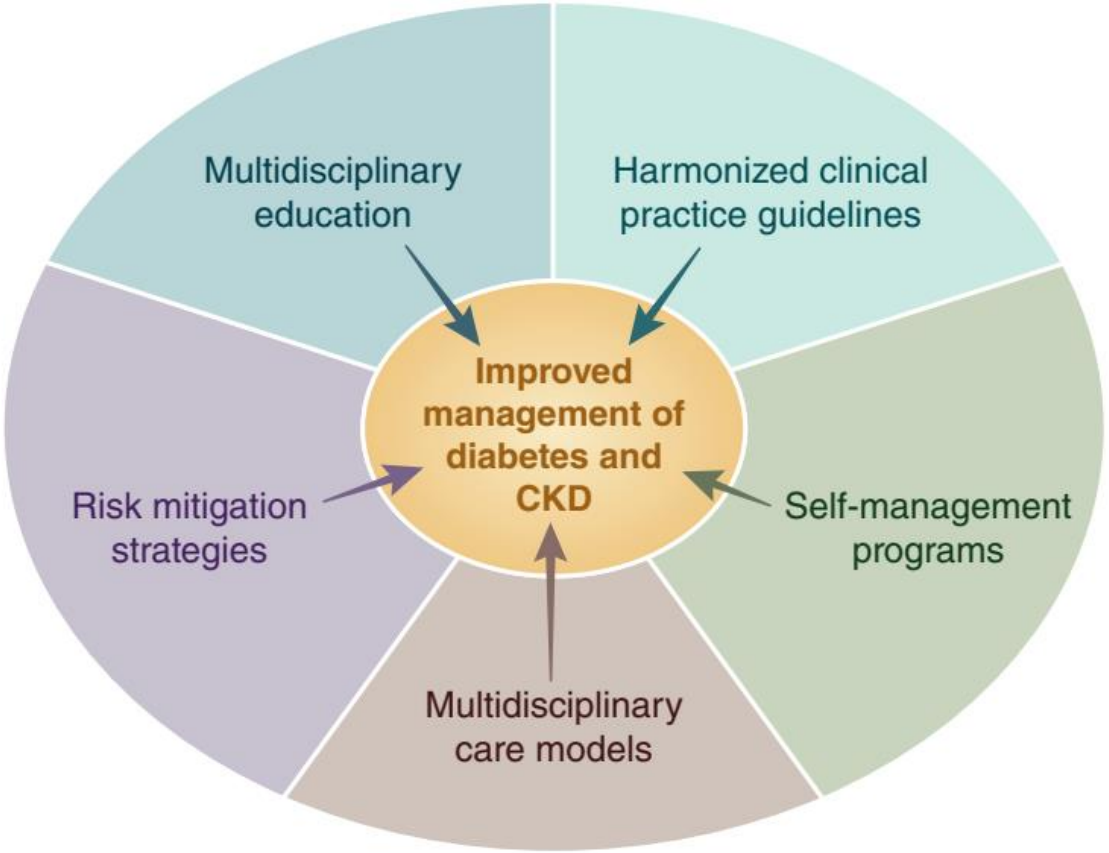
American Diabetes Association.

KDIGO KIDNEY DISEASE · IMPROVING GLOBAL OUTCOMES

Optimize lipid and ASCVD prevention

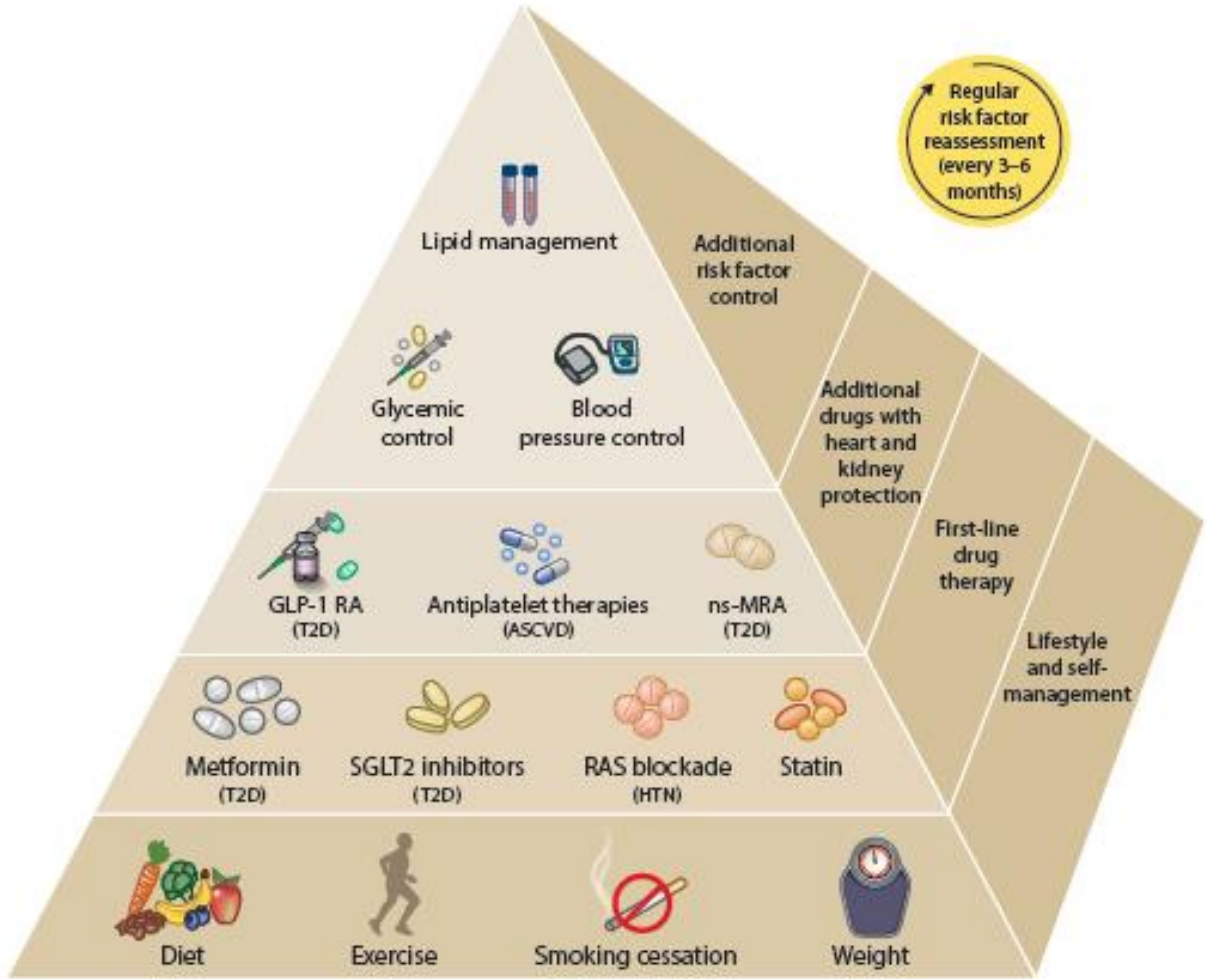


. Overcoming barriers to management of CKD in patients with diabetes



American Diabetes Association.

KIDNEY DISEASE · IMPROVING GLOBAL OUTCOMES · KDIGO



Diabetes with CKD

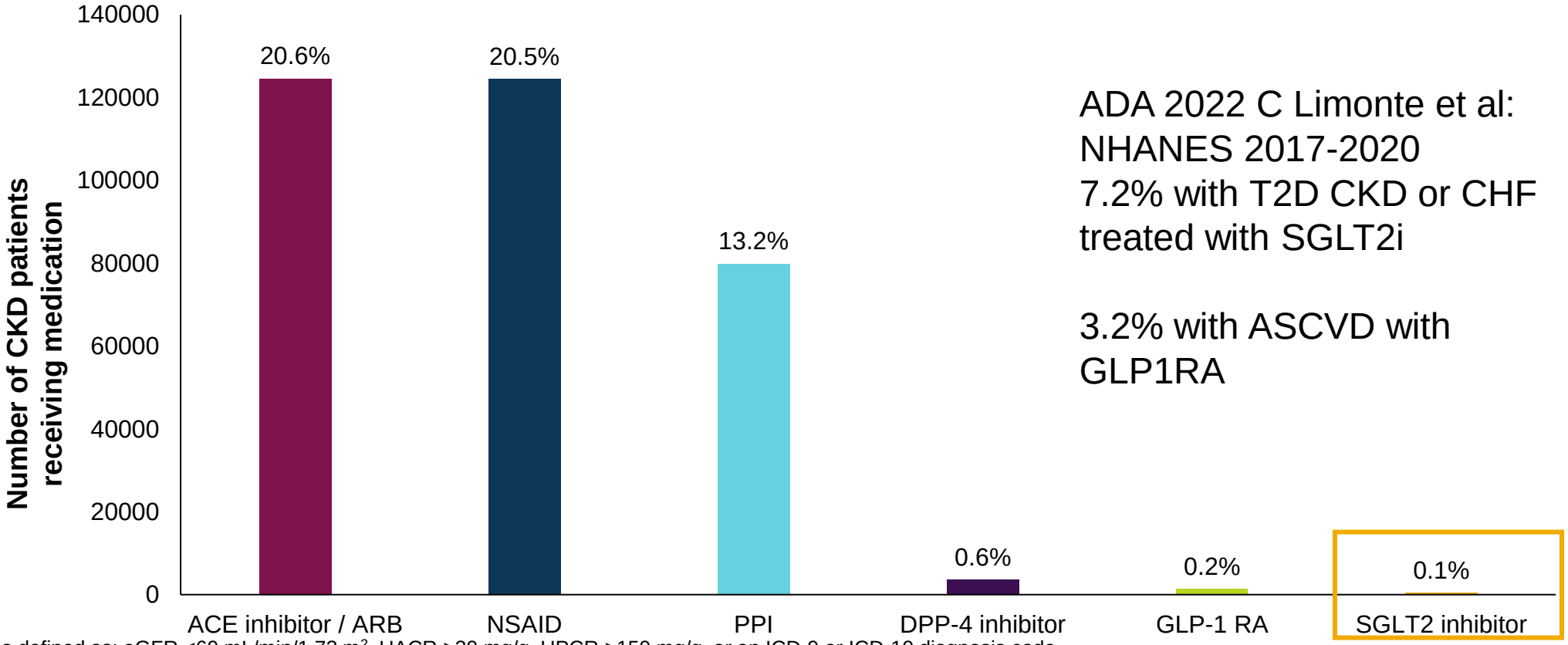
American Diabetes Association.

KDIGO KIDNEY DISEASE · IMPROVING GLOBAL OUTCOMES

The uptake of SGLT2 inhibitors in CKD is lower compared with other glycemic and nonglycemic agents

- The CURE-CKD registry investigated prescription patterns of 606,064 adult US CKD patients

Prevalence of prescription medication use in CKD patients[1,a]



ADA 2022 C Limonte et al:
NHANES 2017-2020
7.2% with T2D CKD or CHF treated with SGLT2i

3.2% with ASCVD with GLP1RA

[a]CKD was defined as: eGFR <60 mL/min/1.73 m^2, UACR >30 mg/g, UPCR >150 mg/g, or an ICD-9 or ICD-10 diagnosis code

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; CKD, chronic kidney disease; DPP-4, dipeptidyl peptidase-4; eGFR, estimated glomerular filtration rate; GLP-1 RA, glucagon-like peptide-1 receptor agonist; ICD, International Classification of Diseases; NSAID, nonsteroidal anti-inflammatory drug; PPI, proton pump inhibitor; SGLT2, sodium–glucose co-transporter 2; UACR, urine albumin:creatinine ratio; UPCR, urine protein:creatinine ratio

1. Tuttle KR, et al. *JAMA Netw Open* 2019;2:e1918169;

Key take away points

- Remember to screen for CKD with eGFR and UACR
- Healthy lifestyle and education foundation
- First line therapy includes SGLT2i, RASi, metformin and statins
- CKD and CVD protection with SGLT2i and nsMRA, and CVD protection with GLP1-RA
- Organisation of care is key to overcoming barriers to implementation and treatment inertia

Thank you

Fot. Peter Rossing