PLACE 2026
PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

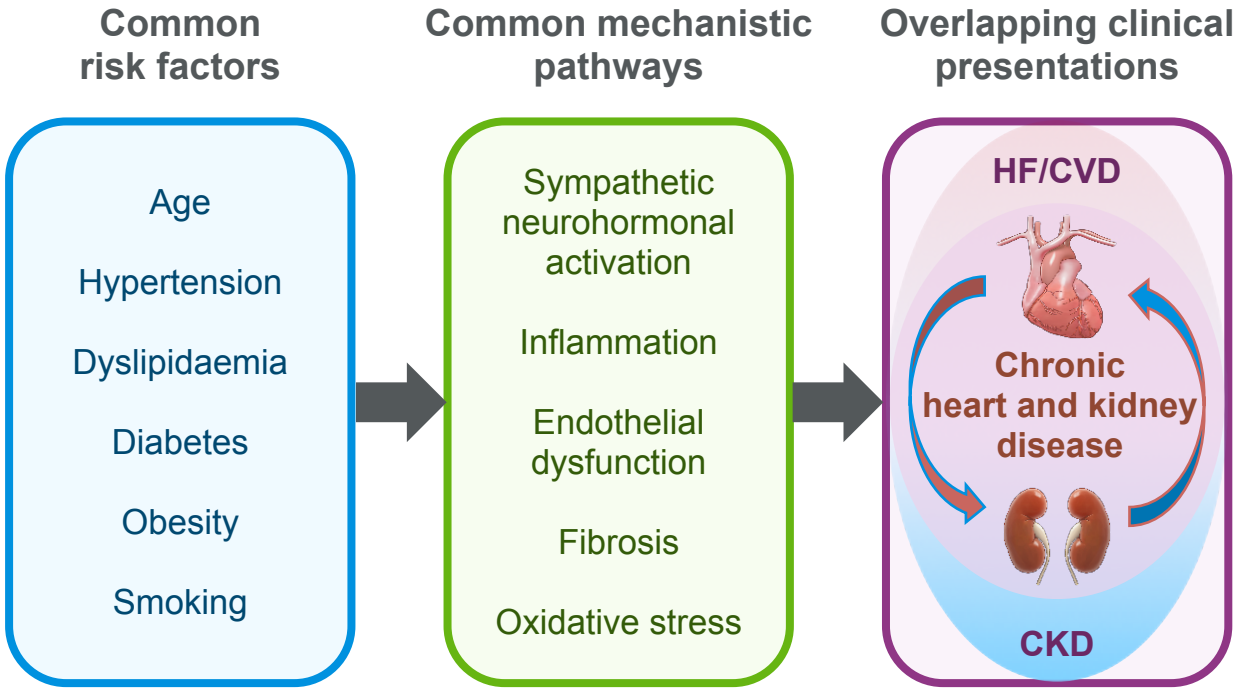
CONGRESSO
ROMA, 25 - 27 Giugno 2026

INSUFFICIENZA CARDIACA CRONICA: LE COMPLESSITA'

Cardio nefro-protezione nel paziente scompensato: cosa fare e cosa evitare

Dott. Saporito Pasquale

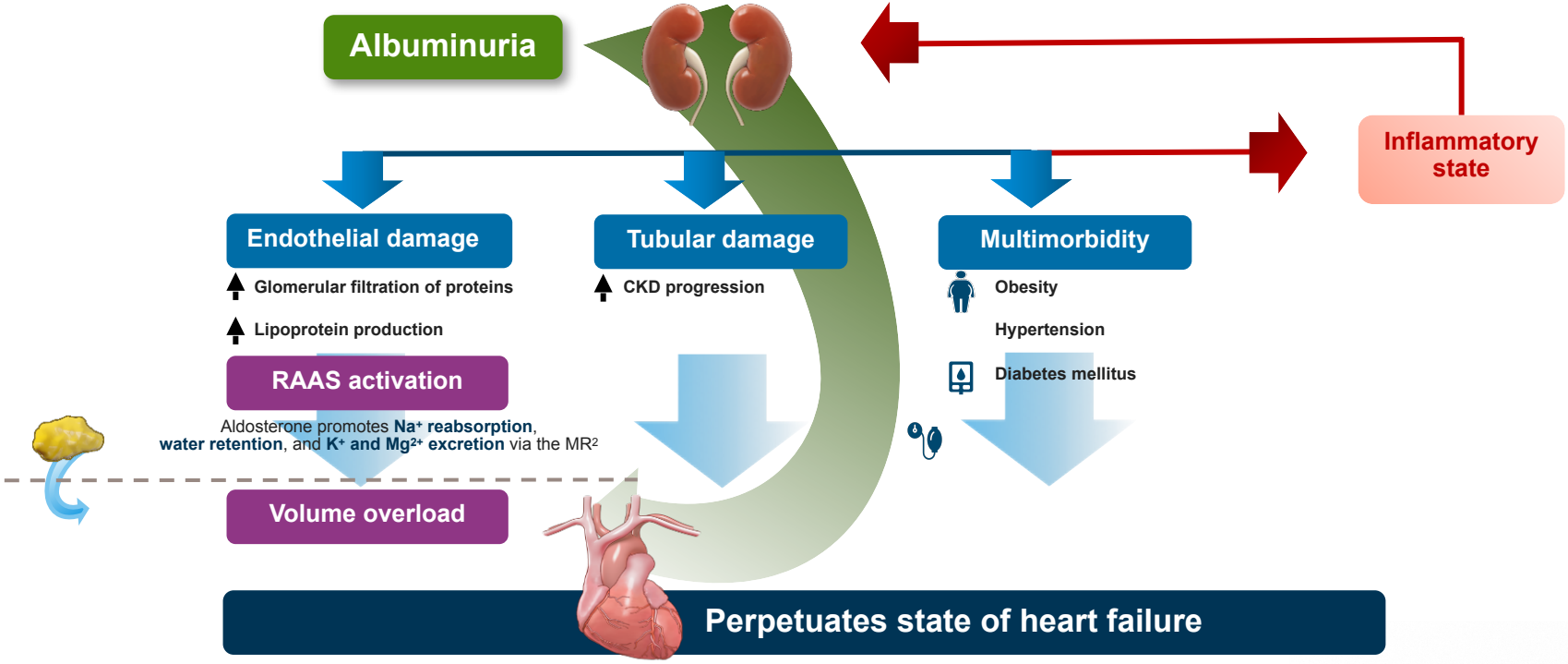
Chronic heart and kidney disease: CKD and heart dysfunction, such as HF, share common risk factors and pathogenic mechanisms[1,2]



CKD, chronic kidney disease; CVD, cardiovascular disease; HF, heart failure; MRAs, mineralocorticoid receptor antagonists; RASi, renin–angiotensin system inhibitors; SGLT-2is, sodium-glucose co-transporter-2 inhibitors

1. Zannad F & Rossignol P. *Circulation* 2018;138:929–944; 2. McCullough PA, *et al. J Am Heart Assoc* 2022;11:e024139

Chronic heart and kidney disease: CKD and heart dysfunction, such as HF, share common risk factors and pathogenic mechanisms[1,2]



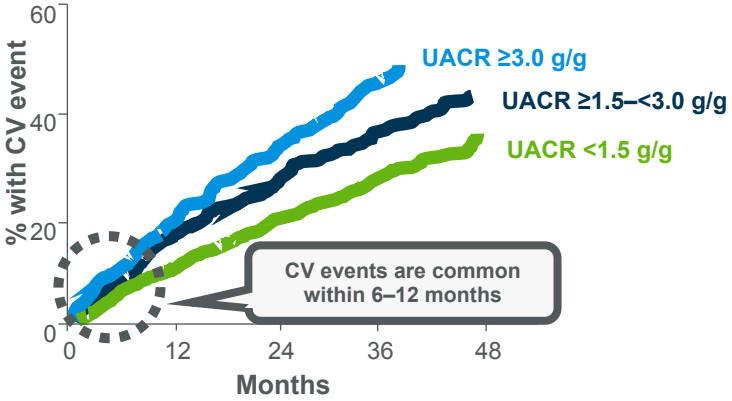
K^+, potassium; Mg^{2+}, magnesium; MR, mineralocorticoid receptor; Na^+, sodium; RAAS, renin-angiotensin-aldosterone system; UACR, urine albumin-to-creatinine ratio

1. Khan MS, *et al. JACC Rev* 2023;81:270–282; 2. Kolkhof P, *et al. Int J Mol Sci* 2022;23:9243

As albuminuria is an early predictor of heart and kidney disease outcomes, routine UACR screening is recommended in international cardiology guidelines to mitigate CV risk [1]

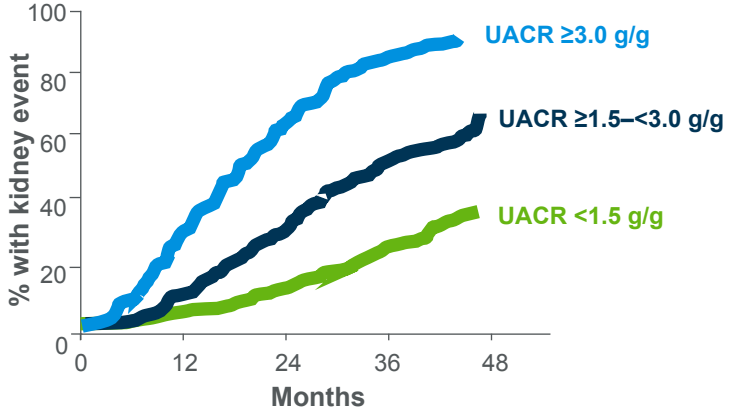
CV composite outcome

Composite of MI, stroke, first HHF or unstable angina, coronary or peripheral revascularisation, or CV death[3]



Kidney composite outcome

Composite of the time to first doubling of serum creatinine, ESKD or death[2]



ESC European Society of Cardiology

NEW

Recommendation for patients with chronic kidney disease and diabetes[4]

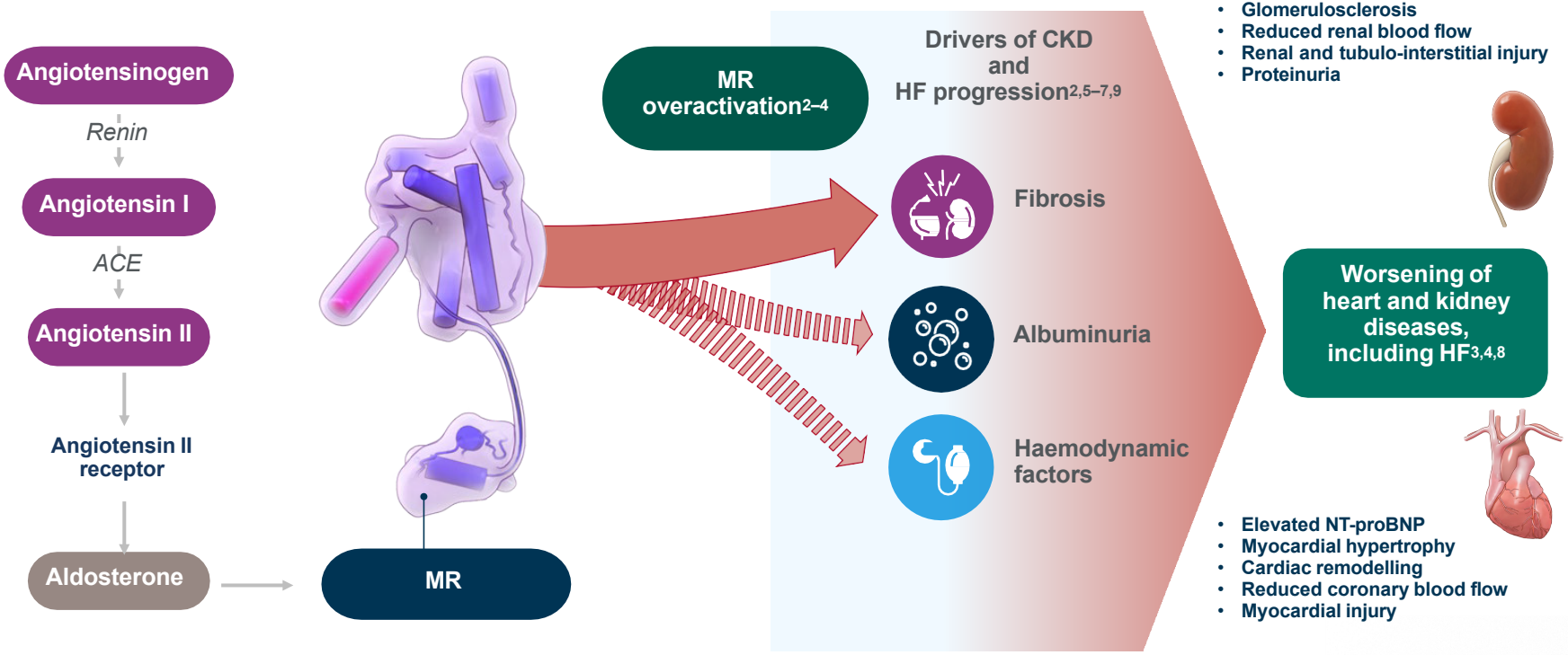
Evidence level 1B

It is recommended that patients with diabetes are **routinely screened for kidney disease** by **assessing eGFR** defined by CKD-EPI and **UACR**

HHF, hospitalisation for heart failure; MI, myocardial infarction

1. de Jong PE. *J Nephrol* 2007;20:375–380; 2. de Zeeuw D, *et al. Kidney Int* 2004;65:2309–2320; 3. de Zeeuw D, *et al. Circulation.* 2004;110:921–927;
4. Marx N *et al. J Hypertens* 2023; doi.org/10.1093/eurheartj/ehad192

Drivers of CKD progression include fibrosis, albuminuria and haemodynamic factors, all associated with MR overactivation[1–9]

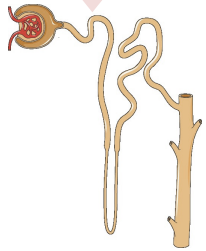


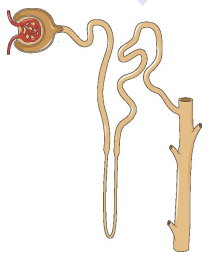
1. Fountain JH, *et al*. Physiology, renin angiotensin system. In: StatPearls [Internet]. Treasure Island, FL: StatPearls Publishing; 2023;
2. Bauersachs J, *et al*. *Hypertension* 2015;65:257–263; 3. Kolkhof P, *et al*. *Handb Exp Pharmacol* 2017;243:271–305; 4. Barrera-Chimal J, *et al*. *Kidney Int* 2019;96:302–319;
5. Alicic RZ, *et al*. *Clin J Am Soc Nephrol* 2017;12:2032–2045; 6. Mora-Fernández C, *et al*. *J Physiol* 2014;592:3997–4012; 7. Hahn VS, *et al*. *JACC Heart Fail* 2020;8:712–724;
8. Buonafine M, *et al*. *Am J Hypertens* 2018;31:1165–1174; 9. Khan MS, *et al*. *JACC Rev* 2023; 81:270–282

The complex pathophysiology underpinning CV and kidney disease in people with T2D calls for a holistic approach to treatment1,2

RAASi
Decrease efferent arteriole tone, hyperfiltration, endothelial dysfunction and cardiac remodelling

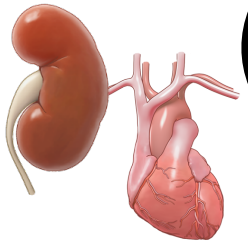
Finerenone
Decreases inflammation, fibrosis, endothelial dysfunction, tissue remodelling and proteinuria





Haemodynamic perturbations

Inflammation and fibrosis



Metabolic dysregulation

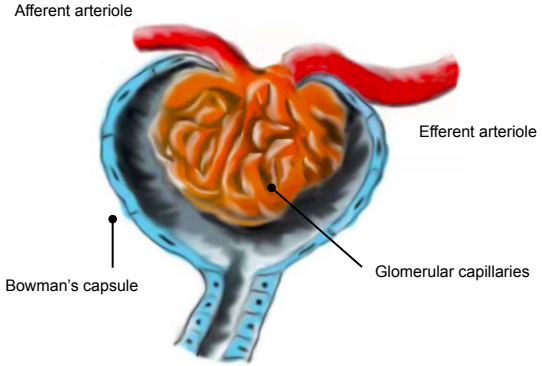
SGLT-2is
Increase afferent arteriole tone, tubuloglomerular feedback and anti-inflammatory and anti-fibrotic effects. Decrease hyperfiltration, proteinuria and oxidative stress

Potentially GLP-1RAs for metabolic control
Decrease weight, dyslipidaemia, oxidative stress and endothelial dysfunction

RAASi & SGLT2i interplay

RAAS inhibitors

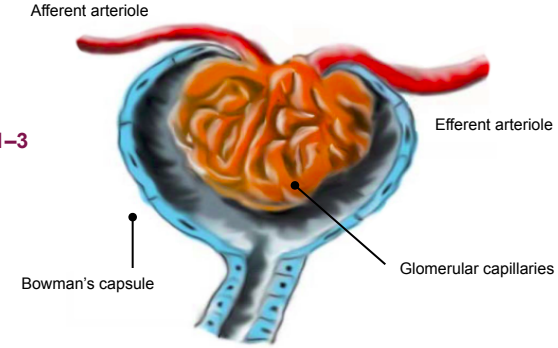
Efferent vasodilation[1]



SGLT2 inhibitors

Afferent vasoconstriction[1–3]
Due to increased Na^+ delivery to the macula densa[1-5]

Efferent vasodilation[5]
Prostaglandins, AT (1-7)



CLINICAL IMPLICATIONS

- **Decreased glomerular pressure[1,3]**
- **Reduction in albuminuria[1,2]**

- **Decreased glomerular pressure[1,3]**
- **Reduction in albuminuria[4]**

RAAS, renin–angiotensin–aldosterone system; SGLT2, sodium–glucose co-transporter 2

1. Van Bommel EJ, et al. *Clin J Am Soc Nephrol* 2017;12:700–710; 2. Seidu S, et al. *Prim Care Diabetes* 2018;12:265–283; 3. Cherney DZ, et al. *Circulation* 2014;129:587–597; 4. Heerspink HJL, et al. *Diabetes Care* 2011;34(Suppl. 2):S325–S329; Van Bommel EJ et al Kidney international 2020, 97(1), 202-212

SGLT2-I: not only anti-hyperfiltration effect

SGLT-2 INHIBITION

Reduce Glomerular Pressure[1]
↑Afferent vasoconstriction
↓Glomerular hyperfiltration

Neurohormonal Improvement
↓Intrarenal RAAS activity[3]
↓SNS activity[4]

Inflammation/Fibrosis Reductions[1,2]
↓Inflammatory markers
↓Fibrotic markers

Decreased Renal Workload and Hypoxia[1,2]
↓Solute transport
↓Oxygen demand
↑ Hematopoiesis

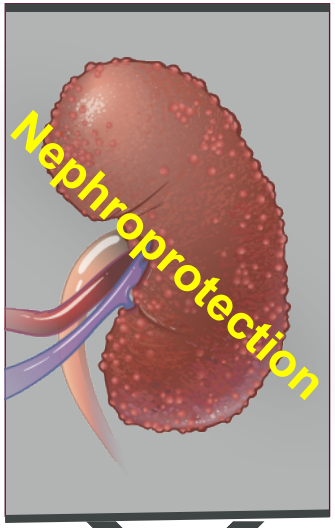
CLINICAL EFFECTS

Stabilization of eGFR[2]

↓Blood Pressure[1]

↓Tubular/Glomerular Injury[1,2]
↓Albuminuria

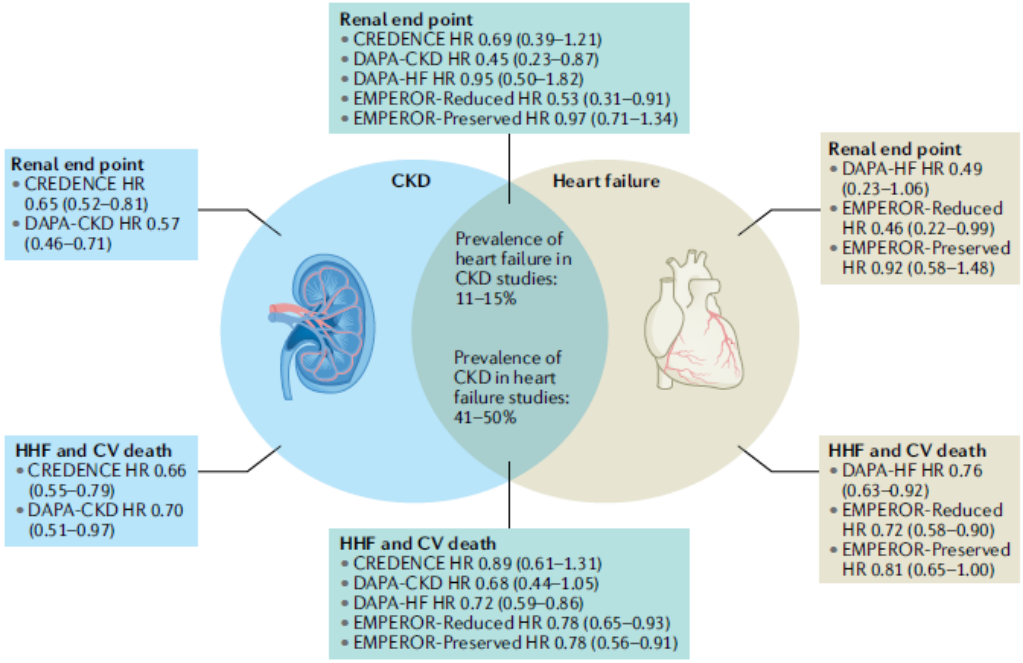
↓Renal Ischemic Injury[1]
↑Hb/hematocrit[2]



Cardiovascular Protection

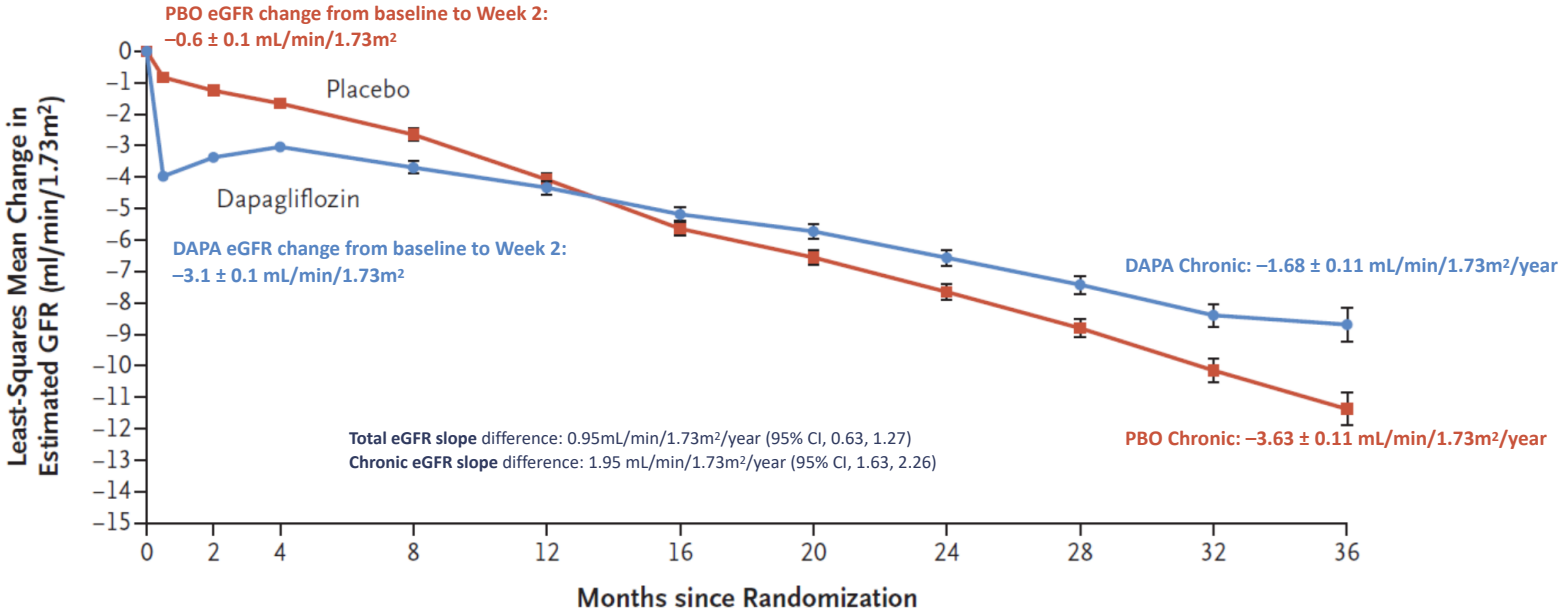
1. Heerspink HJL KI 2018; 2. Tamargo J. Eur Cardiol. 2019

Kidney and Heart Failure outcomes associated with SGLT2i use



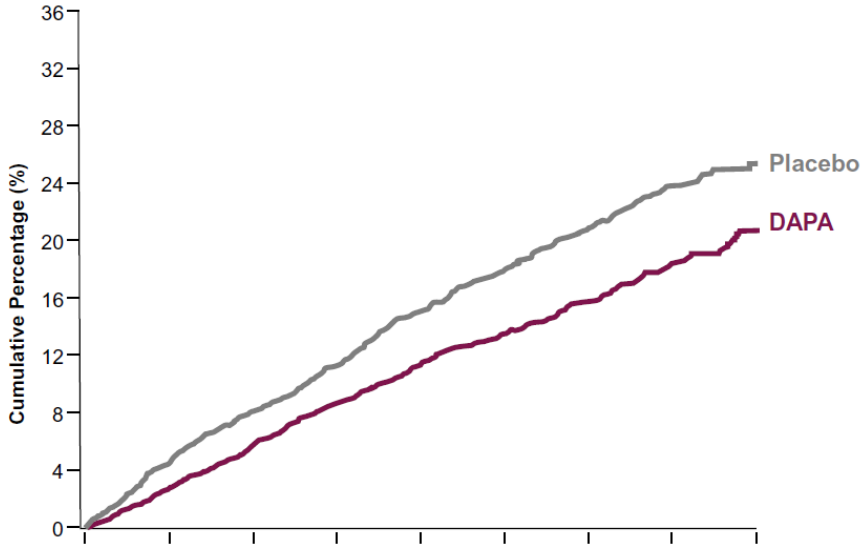
Association between CKD and heart failure.
Chronic kidney disease (CKD) and heart failure share traditional risk factors including age, hypertension and diabetes. In addition, biological changes that occur as kidney function deteriorates (for example, inflammation and haemodynamic changes) increase the onset and progression of heart failure and vice versa. Consequently, CKD and heart failure often coexist, and adequate treatment may positively affect the prognosis of both conditions. Hazard ratios for composite kidney outcomes (defined as kidney failure, a sustained doubling of serum creatinine level or death due to kidney conditions in the CREDENCE trial; as estimated glomerular filtration rate decline ≥50%, kidney failure or death due to kidney conditions in the DAPA- CKD and DAPA- HF trials; and as kidney failure or sustained estimated glomerular filtration rate decline ≥40% in the EMPEROR- Reduced and EMPEROR- Preserved trials) and for the composite of cardiovascular (CV) death and hospitalization for heart failure (HHF) in the major sodium–glucose co- transporter 2 (SGLT2) inhibitor CKD and heart failure trials, across subgroups defined by baseline CKD and heart failure status, demonstrate beneficial effects of SGLT2 inhibitors across levels of kidney function and in patients with and without heart failure

Dapagliflozin in patients with chronic kidney disease (DAPA-CKD trial)



N Engl J Med. 2020; 383:1436-1446;

Primary Endpoint: CV Death or hHF or an Urgent HF Visit



No. at Risk									
DAPA	2373	2305	2221	2147	2002	1560	1146	612	210
Placebo	2371	2258	2163	2075	1917	1478	1096	593	210

26% RRR

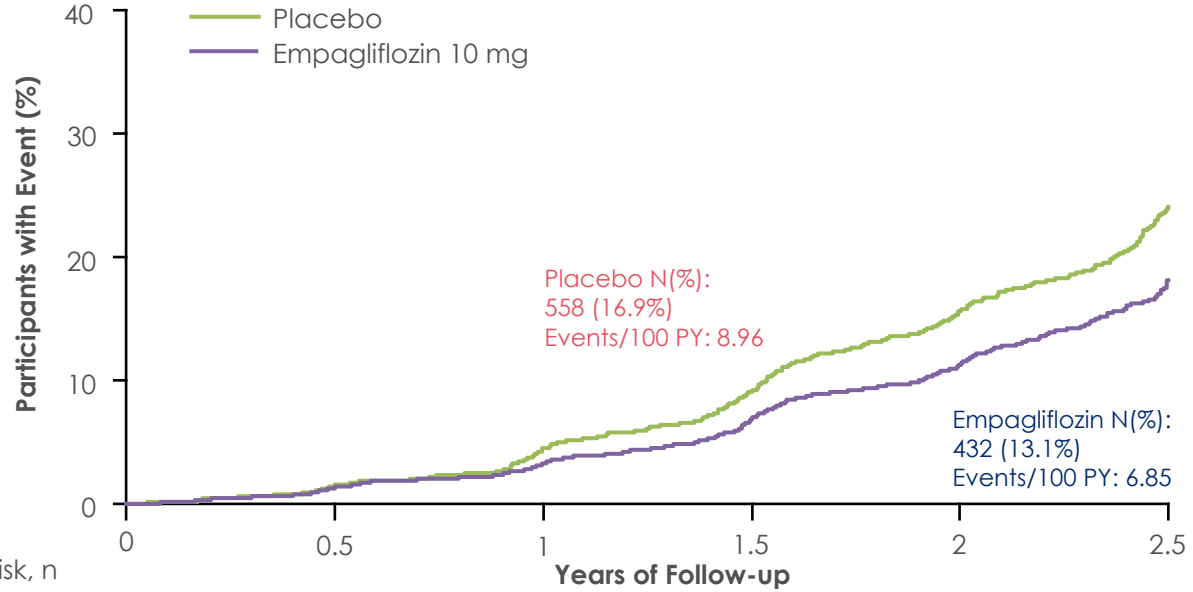
HR 0.74 (0.65, 0.85)
p=0.00001

NNT = 21

Absolute Risk Reduction [ARR]=4%
Event rate/100 patient years:
11.6 vs 15.6; p=0.00001

DAPA = dapagliflozin; HF = heart failure; hHF = hospitalisation for heart failure; HR = hazard ratio; NNT = number needed to treat.

Primary composite outcome:
Kidney disease progression or CV death1



Patients at risk, n

Placebo	3305	3250	3129	2243	1496	592
Empagliflozin	3304	3252	3163	2275	1538	624

HR 0.72
(95% CI 0.64, 0.82)
P < 0.001

RRR 28%

NNT=28*

GLP1-RA and cardiorenal outcomes

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journal homepage: www.elsevier.com/locate/jcte

Anti-inflammatory benefits of semaglutide: State of the art

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[a] Research Center of Physiology, Semnan University of Medical Sciences, Semnan, Iran
[b] Urology and Nephrology Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[c] Medical Toxicology Research Center, Mashhad University of Medical Sciences, Mashhad, Iran
[d] Pharmaceutical Research Center, Pharmaceutical Technology Institute, Mashhad University of Medical Sciences, Mashhad, Iran
[e] Applied Biomedical Research Center, Mashhad University of Medical Sciences, Mashhad, Iran
[f] Biotechnology Research Center, Pharmaceutical Technology Institute, Mashhad University of Medical Sciences, Mashhad, Iran

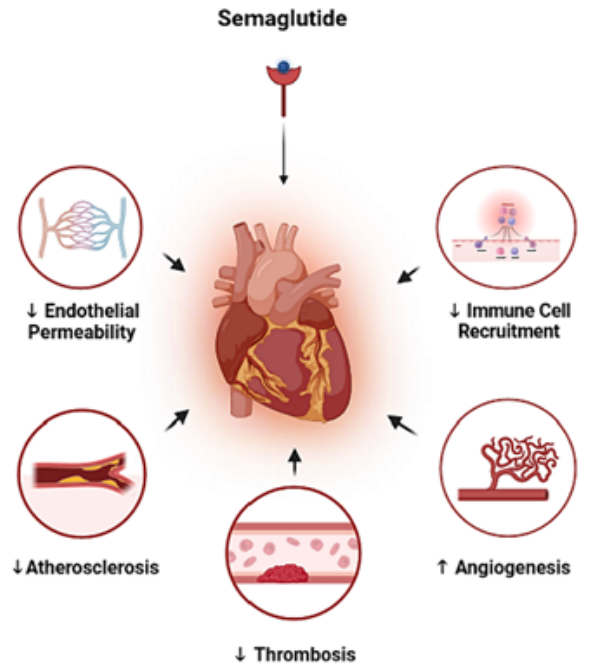
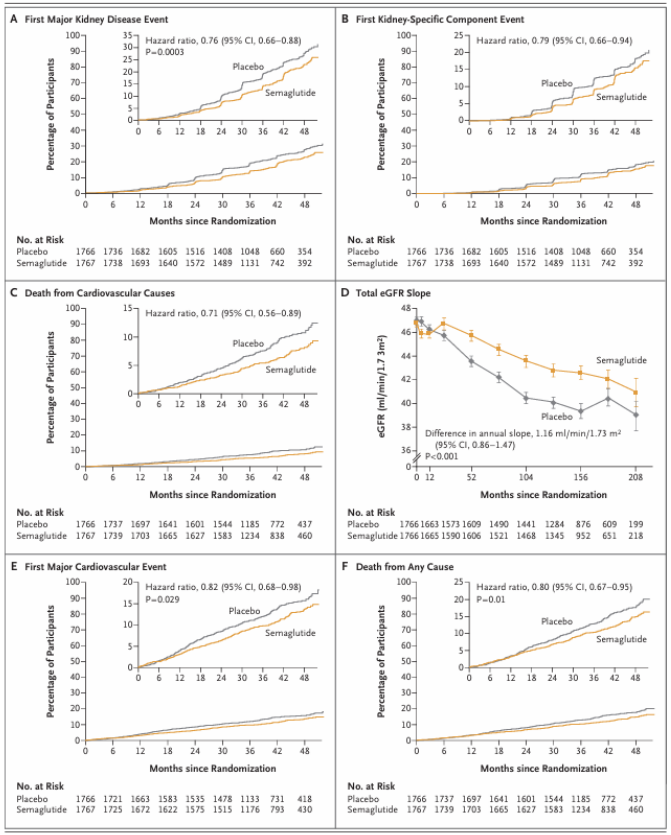


Fig. 2. Semaglutide improves cardiovascular function by its anti-inflammatory benefits thru several mechanisms. It can preserves endothelial permeability, reduce immune cells recruitment into heart tissues, decrease atherosclerotic and thrombotic processes and induce angiogenesis in myocardium.

GLP1-RA and cardiorenal outcomes



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Effects of Semaglutide on Chronic Kidney Disease in Patients with Type 2 Diabetes

Vlado Perkovic, M.B., B.S., Ph.D., Katherine R. Tuttle, M.D.,
Peter Rossing, M.D., D.M.Sc., Kenneth W. Mahaffey, M.D.,
Johannes F.E. Mann, M.D., George Bakris, M.D., Florian M.M. Baeres, M.D.,
Thomas Idorn, M.D., Ph.D., Heidrun Bosch-Traberg, M.D.,
Nanna Leonora Lausvig, M.Sc., and Richard Pratley, M.D.,
for the FLOW Trial Committees and Investigators*

Drivers of CKD progression

ACEis and ARBs[4,5]
Thiazide-like diuretics and dihydropyridine Ca^{2+} channel blockers[4,5]
SGLT-2is[6–9]

SGLT-2is[9,10]
GLP-1RAs[10]
Metformin[10]
Other anti-hyperglycaemic agents[10]

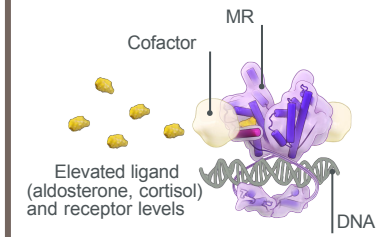
Haemodynamic[1,2]
(elevated BP and/or intraglomerular pressure)

Metabolic[1,2]
(poor glycaemic control)

Inflammation and fibrosis[1–3]

Primary MoA of therapies shown

MR overactivation



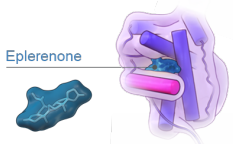
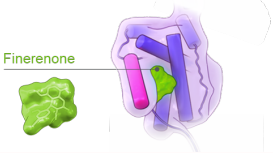
In T2D, MR overactivation may contribute to CKD progression[11,12]

Finerenone

BP, blood pressure; GLP-1RA, glucagon-like peptide-1 receptor agonist; MoA, mechanism of action

1. Alicic RZ, *et al. Clin J Am Soc Nephrol* 2017;12:2032–2045; 2. Mora-Fernández C, *et al. J Physiol* 2014;18:3997; 3. Bauersachs J, *et al. Hypertension* 2015;65:257–263;
4. American Diabetes Association. *Diabetes Care* 2022;45(Suppl 1):S175–S184; 5. American Diabetes Association. *Diabetes Care* 2022;45(Suppl 1):S144–S174;
6. Kidokoro K, *et al. Circulation* 2019:140;303–315; 7. Zelniker TA & Braunwald E. *J Am Coll Cardiol* 2018;72:1845–1855; 8. Heerspink HJ, *et al. Circulation* 2016;134:752–772; 9. Zelniker TA & Braunwald E. J *Am Coll Cardiol* 2020;75:422–434; 10. American Diabetes Association. *Diabetes Care* 2022;45(Suppl 1):S125–S143;
11. Agarwal R, *et al. Eur Heart J* 2021;42:152–162; 12. Agarwal R, *et al. Nephrol Dial Transplant* 2022;37:1014–1023

Differential MR binding of steroidal MRAs vs finerenone results in distinct effects on gene expression

	Aldosterone antagonists		Finerenone
			
Structural properties	Flat (steroidal)	Flat (steroidal)	Bulky (nonsteroidal)[1,5]
Potency to MR	High[4,10]	Moderate[1,4,10]	High[1,2,10]
Selectivity to MR	Low[4,10]	Moderate[4,10]	High[1,2,10]
Half-life	>20 hours*	4–6 hours*	2–3 hours#
Active metabolites	++	–	–
CNS penetration	Yes	Yes	No based on preclinical data[3]
Gynecomastia	Yes[4]	Less than spironolactone[4]	No signal in phase II studies[7-9]
Indication (SmPC)	Congestive HF[2]	HF and LVEF ≤40% or ≤30%[3]	CKD with albuminuria associated with T2D[4]
Tissue distribution	Kidney > heart (at least 6-fold)[6,10]	Kidney > heart (~3-fold)[6,10]	Balanced kidney : heart (1:1)[6,10]

Based on preclinical data and ARTS phase II programme

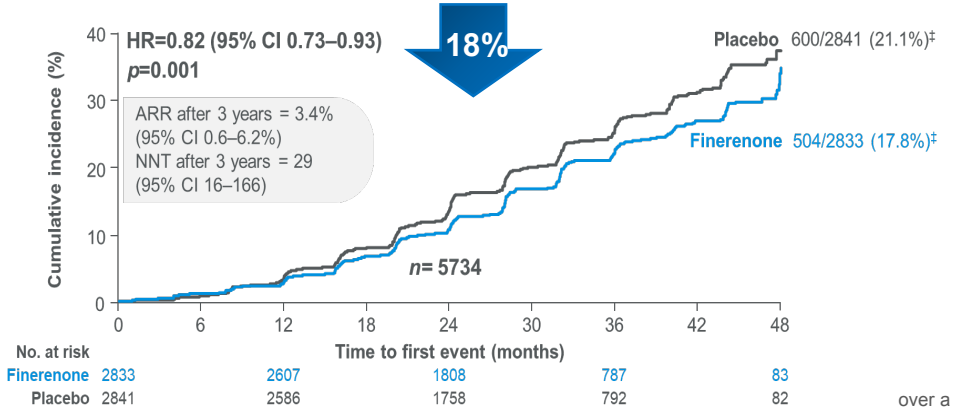
MR, miner
1. Bärfack
2015;290:21876–21889; 6. Kolkhof P, *et al. Curr Opin Nephrol Hypertens* 2015;24:417–424; 7. Pitt B, *et al. Eur Heart J* 2013;34:2453–2463; 8. Bakris GL, *et al. JAMA* 2015;314:884–894; 9. Filippatos G, *et al. Eur Heart J* 2016;37:2435–2443;
kolkhof P, *et al. Handb Exp Pharmacol.* 2017;243:271-305

Evidence for finerenone is being documented by two phase III trials with complementary kidney and CV endpoints

	FIDELIO-DKD [1]	FIGARO-DKD [2]
Clinical efficacy primary endpoint	**Composite endpoint**: time to onset of kidney failure*, sustained decrease of eGFR ≥40% from baseline, or death due to kidney disease	**Composite endpoint**: time to CV death, non-fatal MI, non-fatal stroke or hospitalisation for HF
Key secondary endpoints	Same as primary endpoint in **FIGARO-DKD**	Same as primary endpoint in **FIDELIO-DKD**
Other endpoints	All-cause mortality; All-cause hospitalisation; Change in UACR	Composite: time of first occurrence of kidney failure*, a sustained decrease in eGFR ≥57% from baseline over ≥4 weeks or death due to kidney disease

1. Bakris GL, *et al*. *Am J Nephrol* 2019;50:333–344; 2. Ruilope LM, *et al*. *Am J Nephrol* 2019;50:345–356

Finerenone significantly reduced the risk of the primary composite kidney endpoint vs placebo

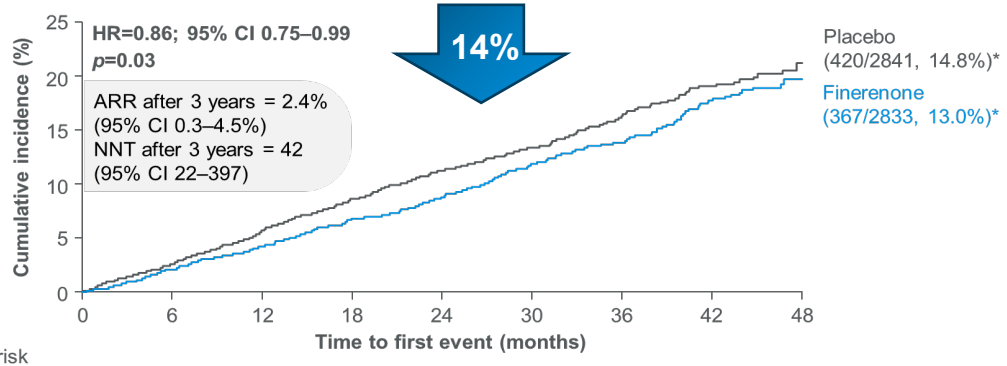


over a median follow-up of 2.6 years

Outcome	Finerenone (n=2833) n (%)	Finerenone (n=2833) n per 100 PY	Placebo (n=2841) n (%)	Placebo (n=2841) n per 100 PY	HR (95% CI)	p-value
Primary composite kidney outcome	**504 (17.8)**	**7.59**	**600 (21.1)**	**9.08**	**0.82 (0.73–0.93)**	**0.001**
Kidney failure*	208 (7.3)	2.99	235 (8.3)	3.39	0.87 (0.72–1.05)	–
End-stage kidney disease	119 (4.2)	1.60	139 (4.9)	1.87	0.86 (0.67–1.10)	–
Sustained# decrease in eGFR to <15 ml/min/1.73 m²	167 (5.9)	2.40	199 (7.0)	2.87	0.82 (0.67–1.01)	–
Sustained# ≥40% decrease in eGFR from baseline	479 (16.9)	7.21	577 (20.3)	8.73	0.81 (0.72–0.92)	–
Renal death	2 (<0.1)	–	2 (<0.1)	–	–	–

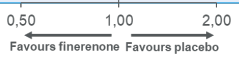
Bakris et al., N Engl J Med 2020; doi: 10.1056/NEJMoa2025845

Finerenone significantly reduced the risk of the key secondary composite cardiovascular endpoint vs placebo



over a median follow-up of 2.6 years

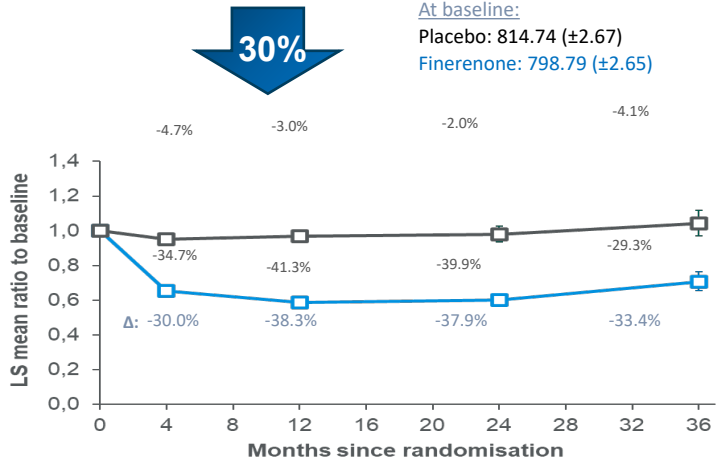
Endpoint	Finerenone (n=2833) n (%)	Finerenone (n=2833) n per 100 PY	Placebo (n=2841) n (%)	Placebo (n=2841) n per 100 PY	Hazard ratio (95% CI)	p-value
Key secondary cardiovascular composite endpoint	367 (13.0)	5.11	420 (14.8)	5.92	0.86 (0.75–0.99)	0.03
Cardiovascular death	128 (4.5)	1.69	150 (5.3)	1.99	0.86 (0.68–1.08)	–
Non-fatal MI	70 (2.5)	0.94	87 (3.1)	1.17	0.80 (0.58–1.09)	–
Non-fatal stroke	90 (3.2)	1.21	87 (3.1)	1.18	1.03 (0.76–1.38)	–
Hospitalisation for HF	139 (4.9)	1.89	162 (5.7)	2.21	0.86 (0.68–1.08)	–



Bakris et al., N Engl J Med 2020; doi: 10.1056/NEJMoa2025845

Finerenone reduces UACR starting from 4th months and worsening of eGFR overtime

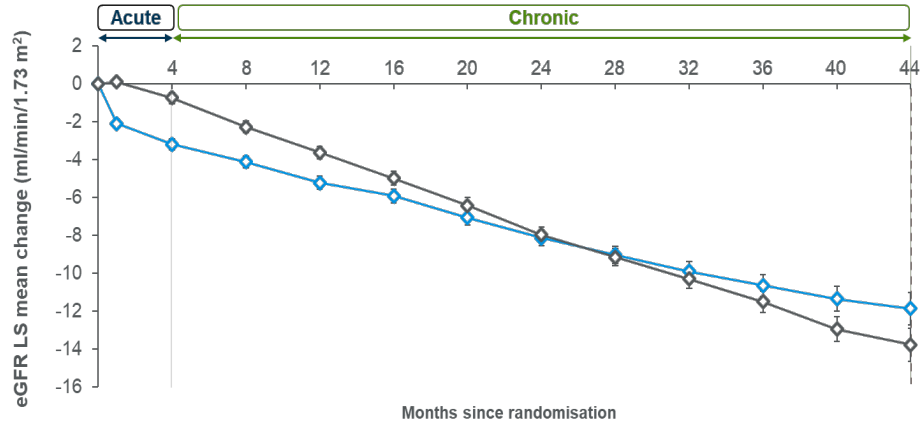
UACR



No. of patients				
Finerenone	2831	2582	1841	856
Placebo	2840	2598	1825	834

Ratio of least-squares mean change from baseline [Finerenone vs placebo]= **0.69; 95% CI 0.66–0.71**

eGFR



Finerenone	2799	2722	2613	1870	867	336
Placebo	2800	2720	2611	1846	844	339

At baseline:
Placebo: 44.3 (±12.6)
Finerenone: 44.4 (± 12.6)

→

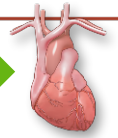
Acute eGRF change:
Placebo: -0.73 (-1.03 to -0.44)
Finerenone: -3.18 (-3.44 to -2.91)

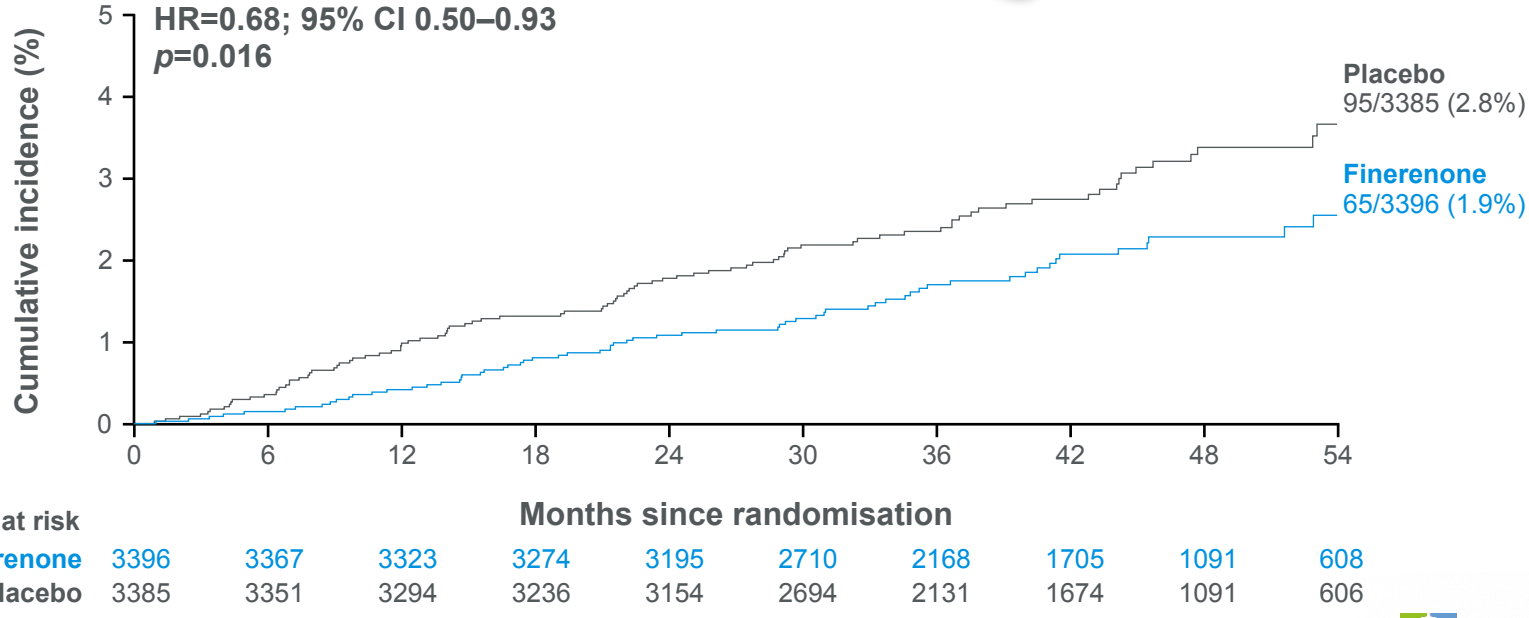
Chronic annulised eGRF change:
Placebo: -3.97 (-4.27 to -3.66)
Finerenone: -2.66 (-2.96 to -2.36)

Bakris et al., N Engl J Med 2020; doi: 10.1056/NEJMoa2025845

FIGARO-DKD: Finerenone significantly reduced the risk of new-onset HF by 32%

New-onset HF in patients without a history of HF at baseline





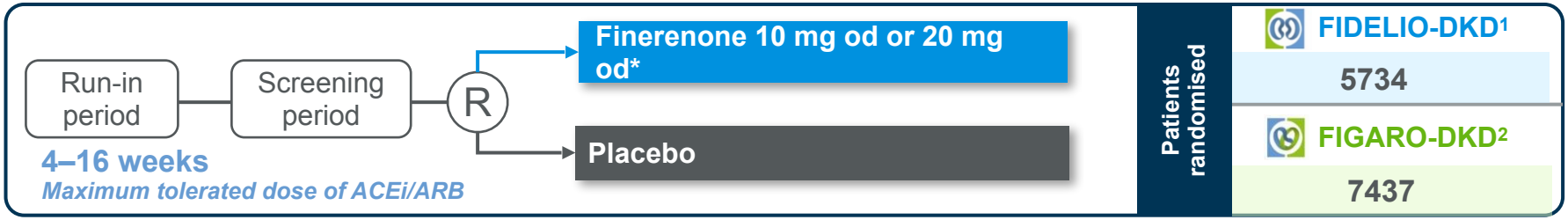
No. at risk										
Finerenone	3396	3367	3323	3274	3195	2710	2168	1705	1091	608
Placebo	3385	3351	3294	3236	3154	2694	2131	1674	1091	606

Finerenone is indicated for the treatment of chronic kidney disease (with albuminuria) associated with type 2 diabetes in adults.[2] For prescribing information please refer to the SmPC of the product applicable in your country.

1. Filippatos G, *et al. Circulation* 2022;145:437–447; 2. Bayer AG. KERENDIA® (finerenone) Summary of Product Characteristics. 2023. https://www.ema.europa.eu/documents/product-information/kerendia-epar-product-information_en.pdf [accessed 03 July 2023]

FIGARO-DKD

The phase III FIDELIO and FIGARO trials investigated the effects of finerenone on kidney and CV outcomes in >13,000 patients with CKD and T2D1,2



	FIDELIO-DKD[1]	FIGARO-DKD[2]
Clinical efficacy primary endpoint	**Composite endpoint:** Time to kidney failure,# sustained ≥40% eGFR decline, or kidney-related death	**Composite endpoint:** Time to CV death, non-fatal MI, non-fatal stroke, or hospitalisation for HF
Key secondary endpoint	Same as primary endpoint in **FIGARO-DKD**	Same as primary endpoint in **FIDELIO-DKD**

FIDELITY[3]
Prespecified pooled analysis

Key outcomes

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

57% kidney composite:
Time to kidney failure,# sustained ≥57% eGFR decline, or kidney-related death

*Patients received an initial dose of finerenone of 10 mg od or 20 mg od based on an eGFR at the screening visit of 25–<60 or ≥60 ml/min/1.73 m², respectively.[1,2] Up-titration to finerenone 20 mg od was permitted at any time after visit 2 (month 1); down-titration to finerenone 10 mg od was permitted at any time after start of treatment. Dose titrations were initiated in response to changes in potassium and eGFR[1,2]; #kidney failure defined as initiation of chronic dialysis for ≥90 days, kidney transplantation or sustained eGFR <15 ml/min/1.73 m²[2,3]
ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CKD, chronic kidney disease; CV, cardiovascular; eGFR, estimated glomerular filtration rate; HF, heart failure; MI, myocardial infarction; od, once daily; R, randomisation; T2D, type 2 diabetes

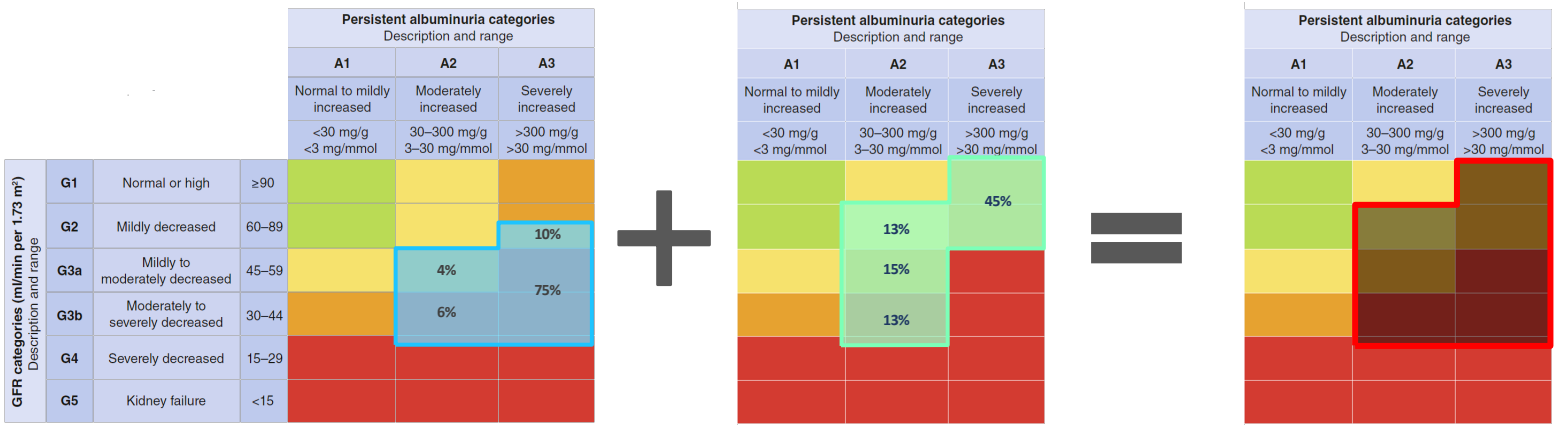
1. Bakris GL, *et al. Am J Nephrol* 2019;50:333–344; 2. Ruilope LM, *et al. Am J Nephrol* 2019;50:345–356; 3. Agarwal R, *et al. Eur J Heart* 2022;43:474–484

FIDELITY

FIDELITY Pooled Analysis on 13.171 DKD patients

FIDELIO-DKD + FIGARO-DKD

FIDELITY
(FIDELIO-DKD + FIGARO-DKD pooled)



PRIMARY CV ENDPOINT

Time to CV death, non-fatal MI, non- fatal stroke or hospitalisation for HF RRR 14%

KEY SECONDARY KIDNEY ENDPOINT

Time to kidney failure (initiation of chronic dialysis for ≥90 days, kidney transplantation or sustained eGFR <15 ml/min/1.73 m²), **sustained ≥57% decrease in eGFR from baseline, or renal death RRR 23%**

30≤UACR<300 mg/g and
25≤eGFR<90 mL/min/1.73m²

or

300≤UACR≤5000 mg/g and
eGFR≥25 mL/min/1.73m²

*Please see slide notes for recruitment caps and eGFR/UACR cut-off details

1. KDIGO. *Kidney Int* 2020;98:S1–S115; 2. Ruilope LM, *et al. Am J Nephrol* 2019;50:345–356; 3. Bakris GL, *et al. Am J Nephrol* 2019;50:333–344 4. Agarwal R et al. *Eur Heart J* 2021. DOI: 10.1093/eurheartj/ehab777/6433104,

Finerenone has demonstrated significant risk reductions in CV and kidney outcomes in two phase III clinical trials

FIDELITY

Prespecified pooled analysis of data from the **FIDELIO-DKD and FIGARO-DKD** trials

>13,000 patients **across the disease continuum of CKD and T2D** (CKD stage 1–4) with moderate-to-severely elevated albuminuria (UACR ≥30 mg/g)

14% reduced **risk of CV morbidity and mortality** (HR=0.86; 95% CI 0.78–0.95; *p*=0.002)[1]

22% reduced **risk of first HHF*** (HR=0.78; 95% CI 0.66–0.92; *p*=0.003)[1]

23% reduced **risk of CKD progression#** (HR=0.77; 95% CI 0.67–0.88; *p*=0.0002)[1]

20% reduced **risk of ESKD** (HR=0.80; 95% CI 0.64–0.99; *p*=0.040)[1,‡]

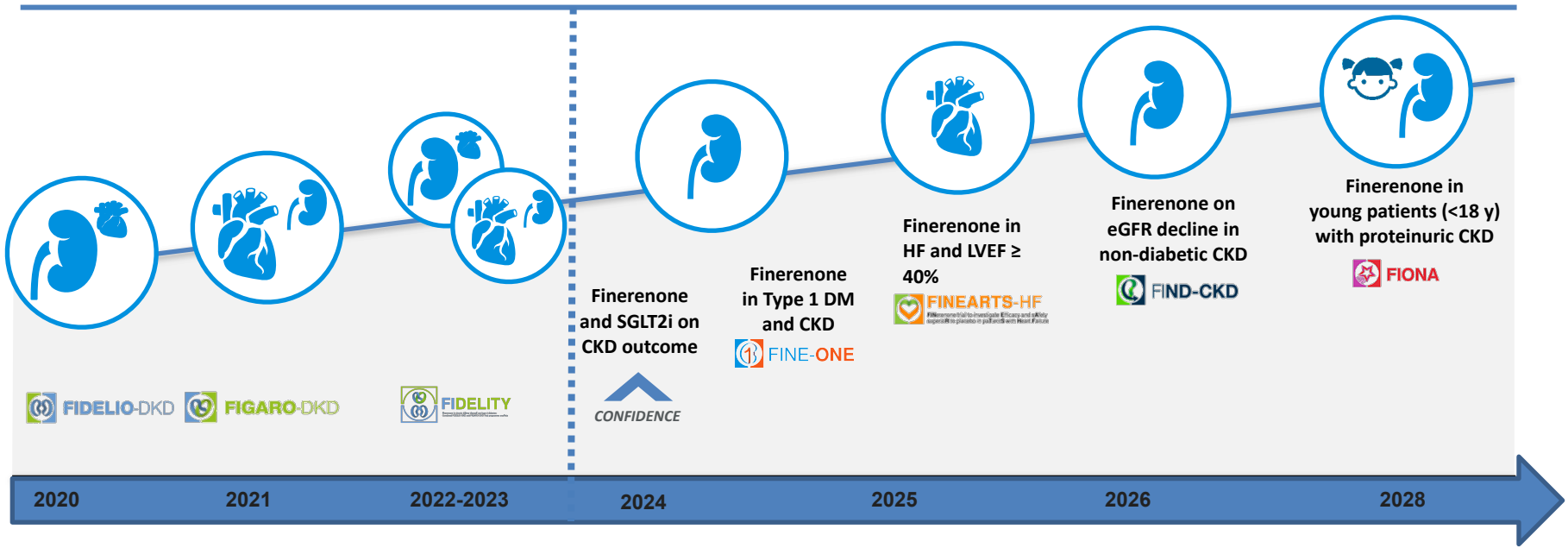
32% reduction in **UACR** (ratio of LS mean change from baseline 0.68; 95% CI 0.66–0.70)[1]

Finerenone is indicated for the treatment of CKD (with albuminuria) associated with T2D in adults[2]

*First HHF defined as first event after randomisation; #ESKD or an eGFR <15 ml/min/1.73 m^2; events were classified as renal death if: (1) the patient died; (2) KRT had not been initiated despite being clinically indicated; and (3) there was no other likely cause of death; ‡analysis for *p*-value not prespecified.
HHF, hospitalisation for heart failure; KRT, kidney replacement therapy; LS, least-squares

1. Agarwal R, *et al. Eur Heart J* 2022;43:474–484; 2. Bayer AG. KERENDIA® (finerenone) Summary of Product Characteristics. 2023. https://www.ema.europa.eu/documents/product-information/kerendia-epar-product-information_en.pdf [accessed 05 Jun 2023]

Clinical development of Finerenone

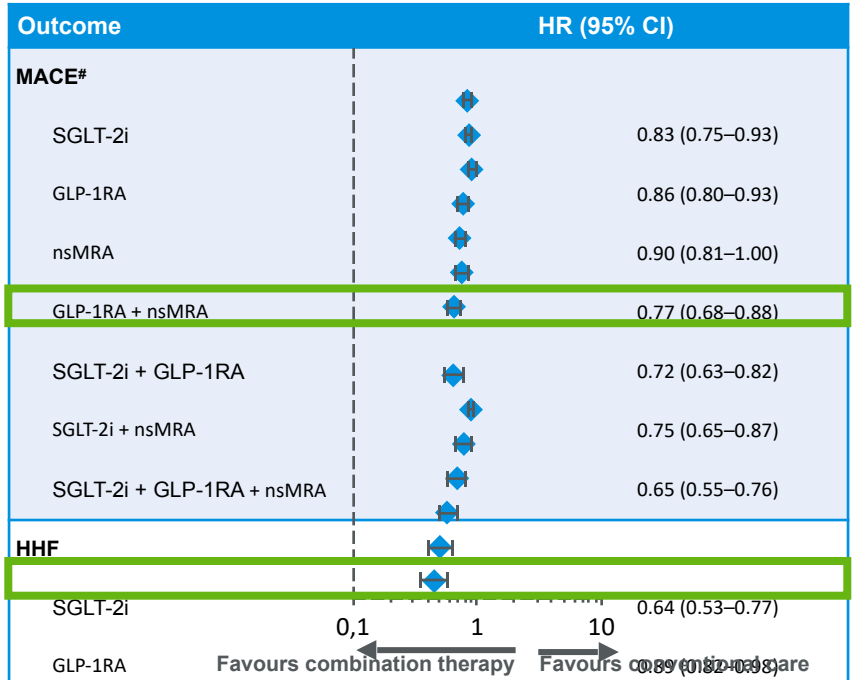


1. Bakris GL, et al. N Engl J Med 2020;383:2219–2229; 2. Pitt B, et al. N Engl J Med 2021;2021:385-2252; 3. Agarwal R, et al. Eur Heart J 2022;43:474–484; 4. Bayer. https://clinicaltrials.gov/ct2/show/NCT05254002; 5. Green JB, et al. Nephrol Dial Transpl 2022; doi: https://doi.org/10.1093/ndt/gfac198; 6. Heerspink HJL et al. Diabetes Research and Clinical Practice 2023. https://doi.org/10.1016/j.diabres.2023.110908; 7. Bayer. https://clinicaltrials.gov/ct2/show/NCT04435626; 8. Bayer. https://clinicaltrials.gov/ct2/show/NCT05047263 9. Bayer. https://clinicaltrials.gov/ct2/show/NCT05196035

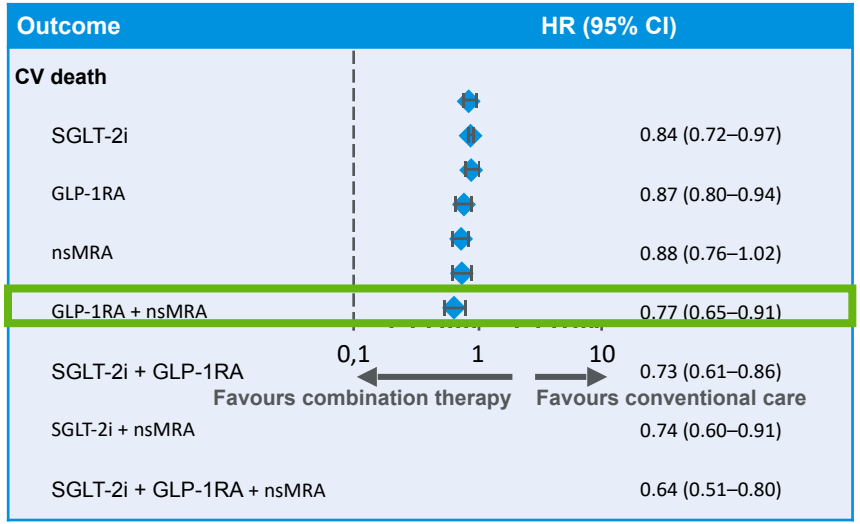
PLACE 2026
PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

Results from a cross-trial analysis suggest potential CV benefits with SGLT-2i, GLP-1 RA and nsMRA combination treatment* in patients with T2D and albuminuria

MACE* and HHF



CV death



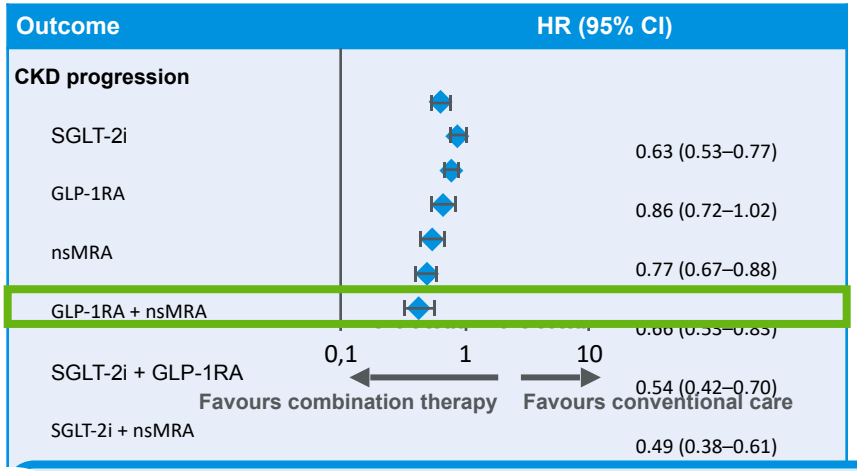
Finerenone is indicated in the EU for the treatment of CKD (with albuminuria) associated with T2D in adults. CV prevention is not an approved indication for finerenone in the EU

MACE, major adverse cardiovascular events; RAS, renin–angiotensin system

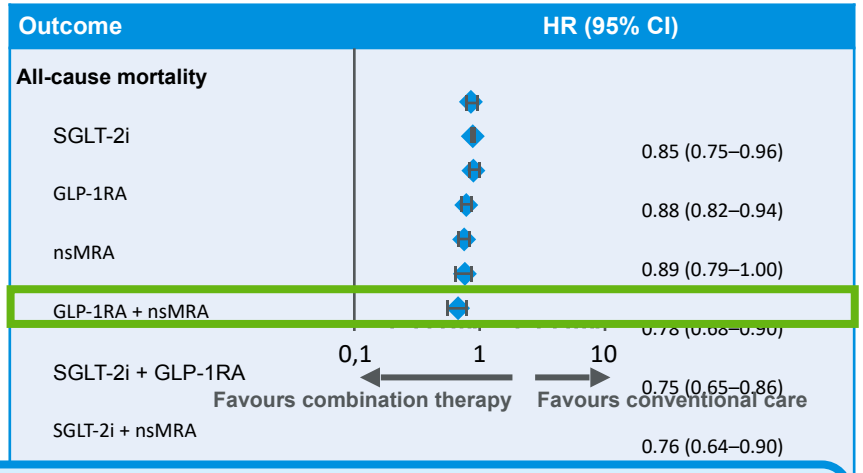
Neuen BL, *et al. Circulation* 2024;149:450–462

Combination treatment* with an SGLT-2i, GLP-1 RA and nsMRA may also reduce the risk of CKD progression and all-cause mortality

CKD progression



All-cause mortality



A **'pillar' approach** combining therapy with an **SGLT2i, GLP-1 RA and nsMRA**, in addition to **RAS blockade,** may be **beneficial for patients with T2D and albuminuria** as these drugs **target different complementary pathophysiological pathways** involved in CV and kidney diseases

Finerenone is indicated in the EU for the treatment of CKD (with albuminuria) associated with T2D in adults. CV prevention is not an approved indication for finerenone in the EU
*When added to RAS blockade
Neuen BL, *et al. Circulation* 2024;149:450–462

Finerenone is a key treatment pillar for the management of CKD and T2D1–6

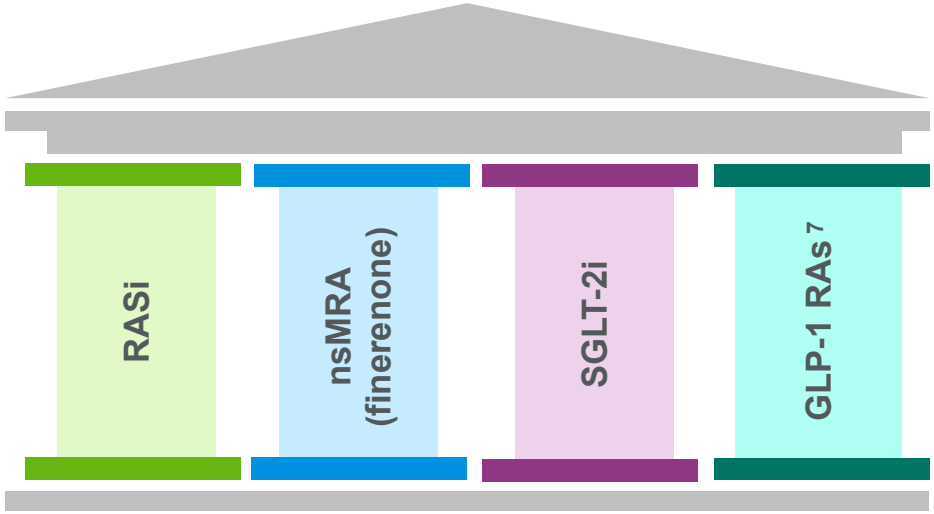


Figure adapted from Blazek O & Bakris GL. 2022[6]

CKD, chronic kidney disease; ESC, European Society of Cardiology; ESH, European Society of Hypertension; ERBP, European Renal Best Practice; GLP-1 RA, glucagon-like peptide-1 receptor agonist; nsMRA, non-steroidal mineralocorticoid receptor antagonist; RASi, renin–angiotensin system inhibitor; SGLT-2i; sodium-glucose co-transporter-2 inhibitor; T2D, type 2 diabetes.

1. Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney Int.* 2022;102:S1-S128; 2. American Diabetes Association. *Diabetes Care*. 2023;46:S191–S202; 3. de Boer I, et al. *Diabetes Care*. 2022;45:3075–3090; 4. Sarafidis P, et al. *Clin Kidney J*. 2023;16:1885–1907; 5. Mancia G, et al. *J Hypertens*. 2023;41:1874–2071; 6. Blazek O & Bakris GL. *Am Heart J Plus*. 2022;19:100187; 7. Perkovic V, et al. *N Engl J Med*. 2024;391:109–121.

COSA EVITARE

- Sospensione ingiustificata: non sospendere terapia con RAASi per lievi rialzi creatininemia (<30%) o per iperkaliemia moderata
- Inerzia Terapeutica: non ritardare l'impiego della terapia massimale in base alle caratteristiche del paziente

- Trattare Iperkaliemia: nuovi chelanti sicuri ed efficaci a disposizione
- Monitoraggio frequente del paziente anche in ambulatori multispecialistici


Grazie!
Gracias
Merci
谢谢
謝謝
Dziękuję
Gracias
Obrigado
Спасибо
감사합니다
شكرا لك
متشكرم