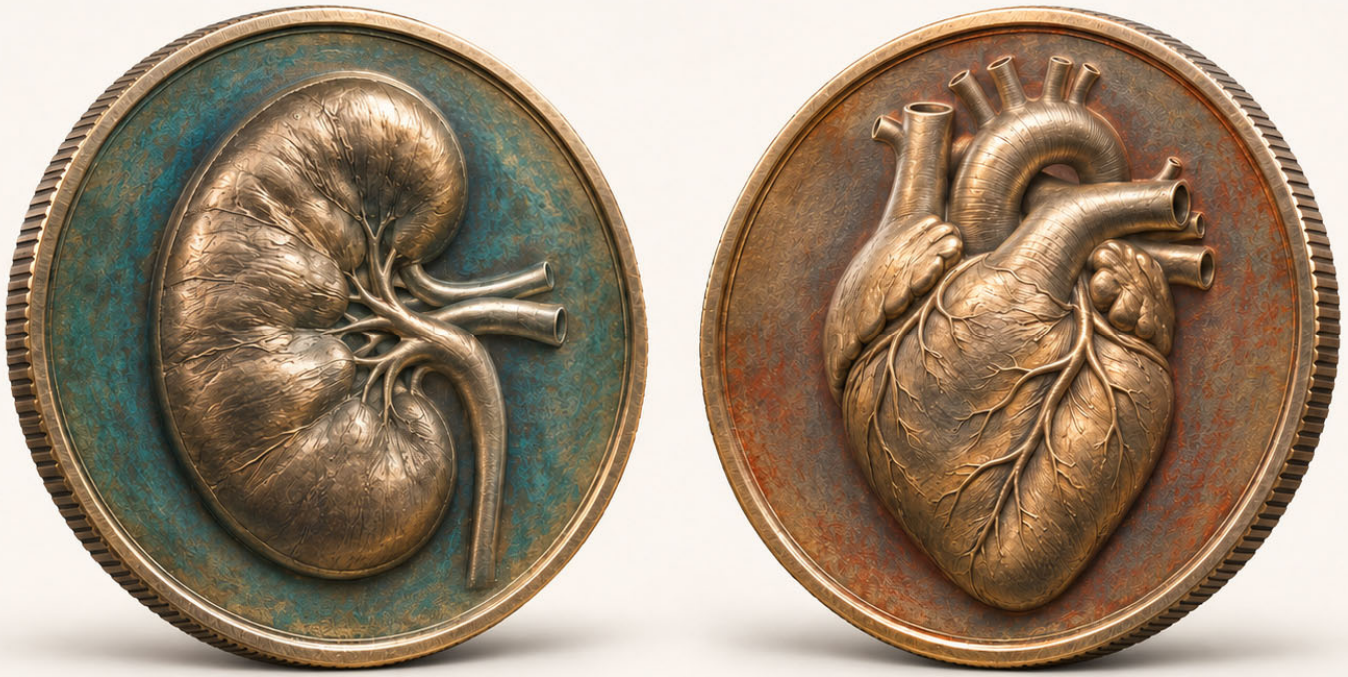
Njurprotektion och kardiovaskulär prevention



Två sidor av samma mynt

Bild - GTP-6.1 Sol

Jonas Spaak
Professor, överläkare kardiologi, ESH hypertension specialist
Hjärtkliniken och HND-centrum Danderyds sjukhus, och Karolinska Institutet
Vice ordförande Svenska Hjärtförbundet

www.hypertoni.org

Jävsdeklaration

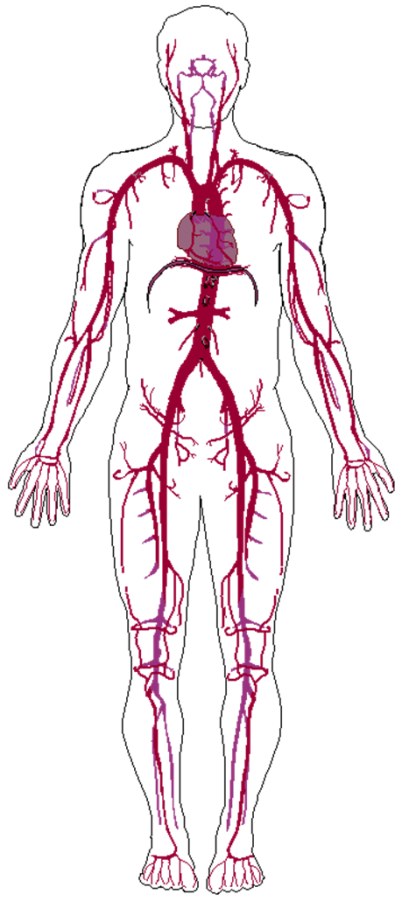
Speaker honoraria:	AstraZeneca, Bayer, NovoNordisk, Novartis, Sanofi Boehringer-Ingelheim, NEKO Health
Advisory:	Beat Vascular Health
PI:	NEKO Health, AstraZeneca, NovoNordisk, Boehringer-Ingelheim
Delägare:	Beat Vascular Health

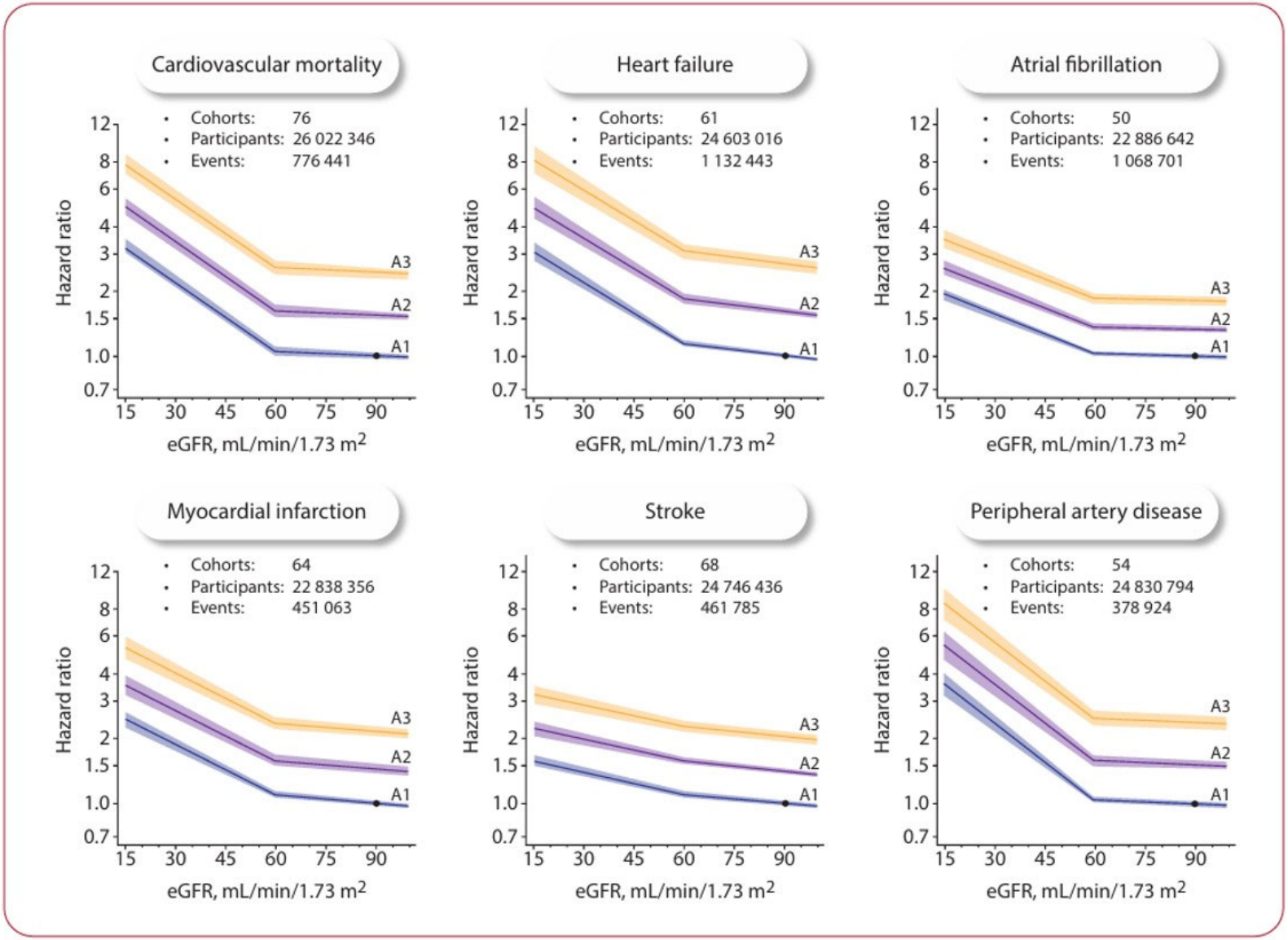
Blodtrycksrelaterade folksjukdomar

- 0.5 miljoner minst måttlig njursjukdom – fler med lindrig
- 0.3-0.5 miljoner har diabetes mellitus
- 0.2-0.3 miljoner har hjärtsvikt
- 0.15 miljoner med demenssjukdom



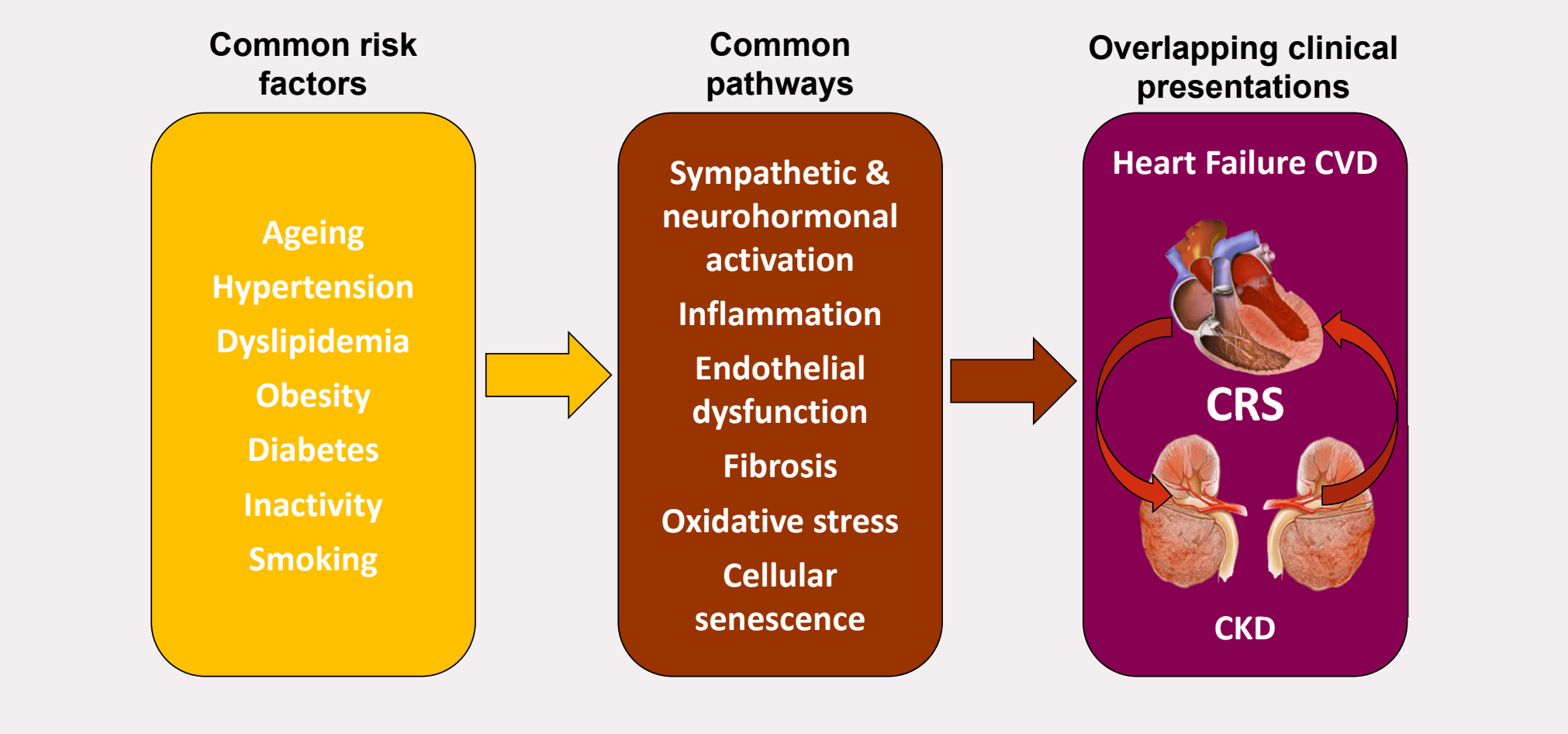
Hjärtsjukdom
Njursjukdom
Diabetes





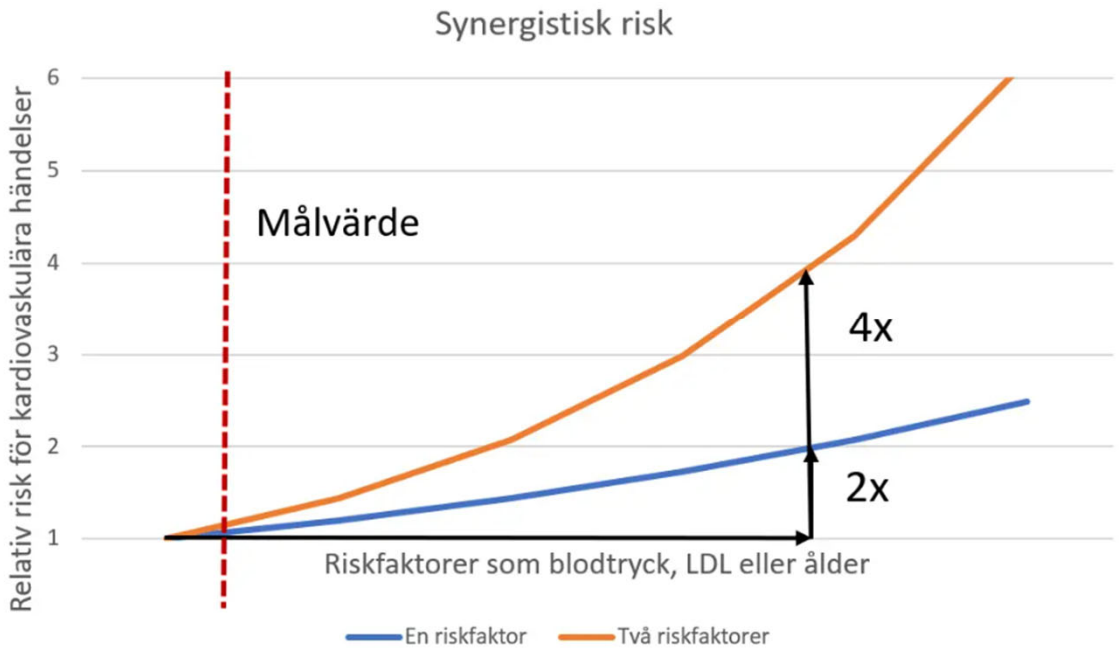
ESC CVD CKD guidelines 2026

Gemensamma riskfaktorer, sjukdomsmekanismer samt klinisk presentation



Zannad and Rossignol, Circulation 138;2018

Janusinfo: Om riskskattning och riskvärdering av kardiovaskulära sjukdomar i primärvården



Figur 2 . Schematisk bild av synergistisk risk. En ökning av LDL med cirka 3 mmol/l eller systoliskt blodtryck 25–30 mmHg ger uppskattningsvis en dubblerad risk för kardiovaskulära händelser.

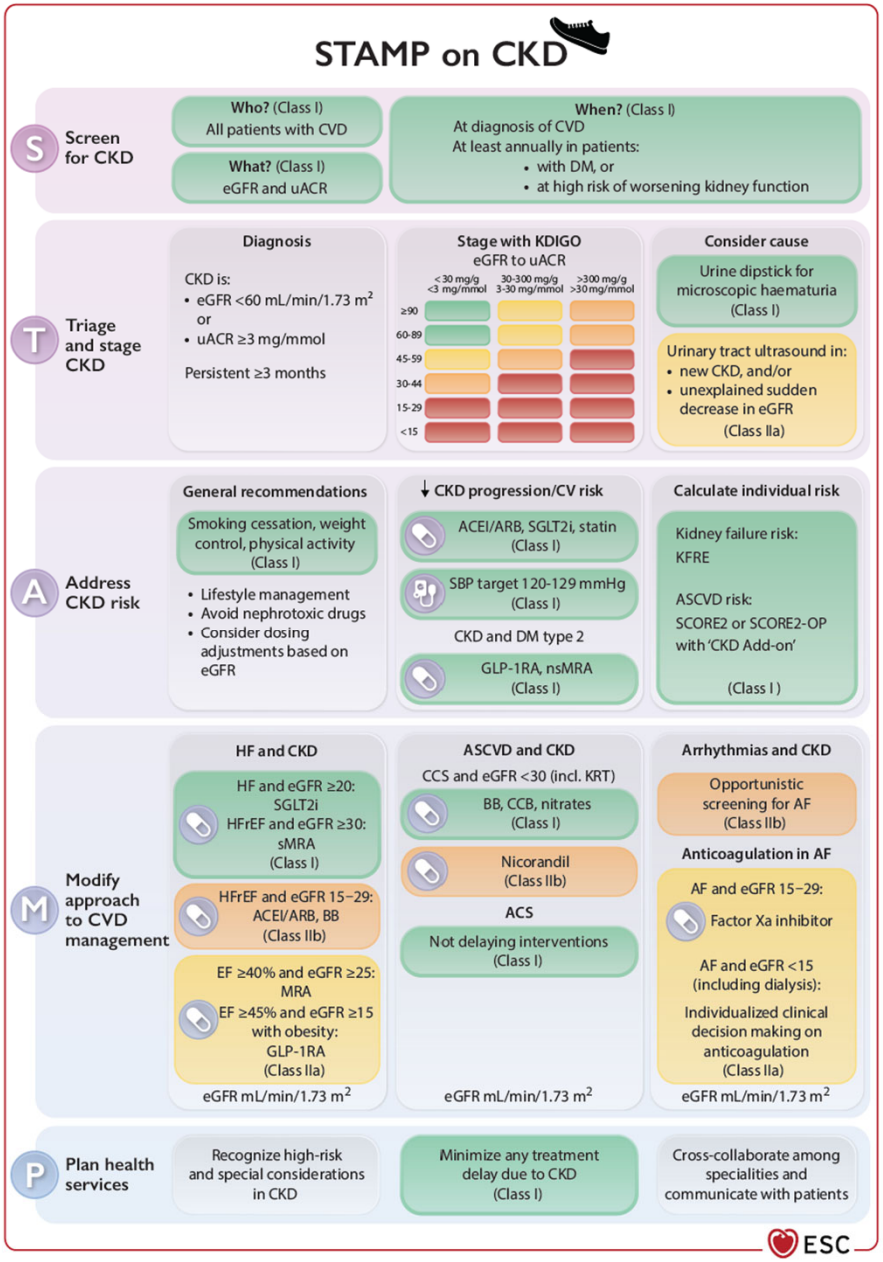
https://janusinfo.se/behandling/expertgruppsutlatanden/hjartochkarlsjukdomar/hjartochkarlsjukdomar/omriskskattningochriskvarderingavkardiovaskularasjukdomaripprimarvarden.5.26a2ee5b17c8f2cf4a6ae348.html

ESC European Society of Cardiology
European Heart Journal (2026) 00, 1–102
https://doi.org/10.1093/eurheartj/ehag098

ESC GUIDELINES

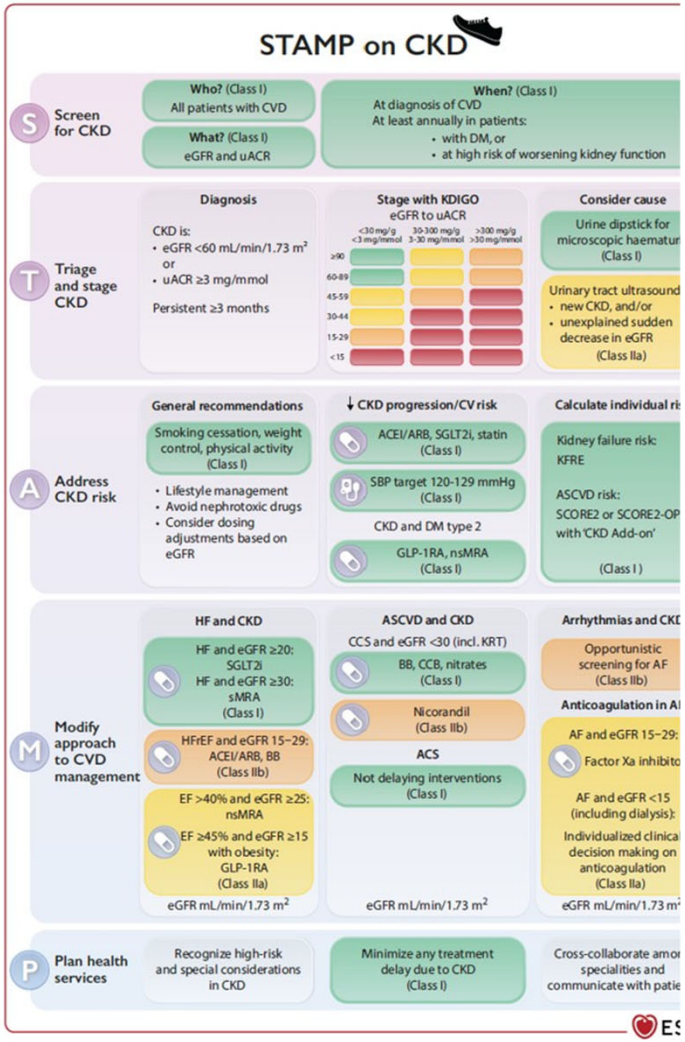
2026 ESC Guidelines for the management of cardiovascular disease and chronic kidney disease, in collaboration with the European Renal Association (ERA)

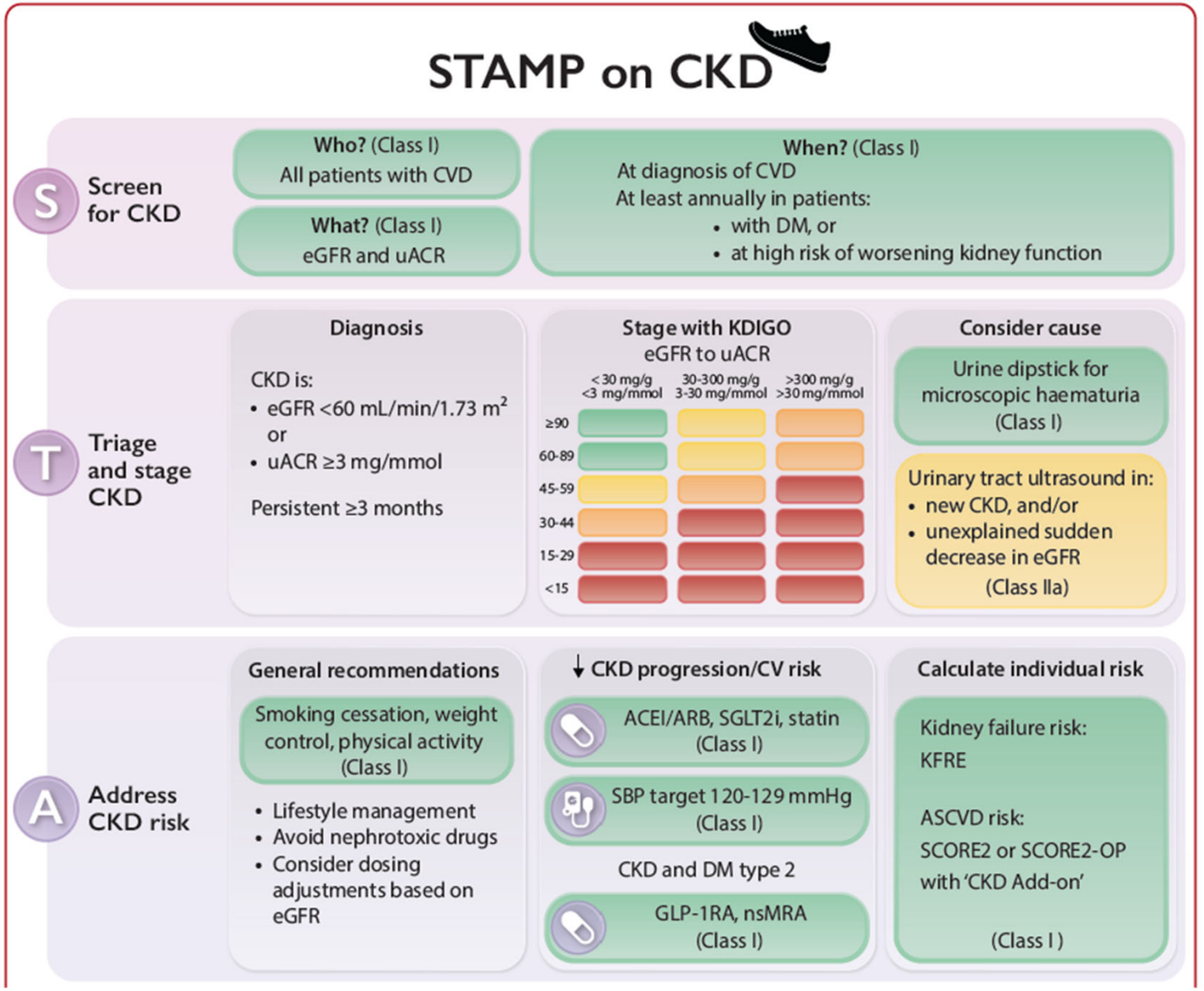
Developed by the task force on the management of cardiovascular disease and chronic kidney disease of the European Society of Cardiology (ESC)



Jonas Spaak

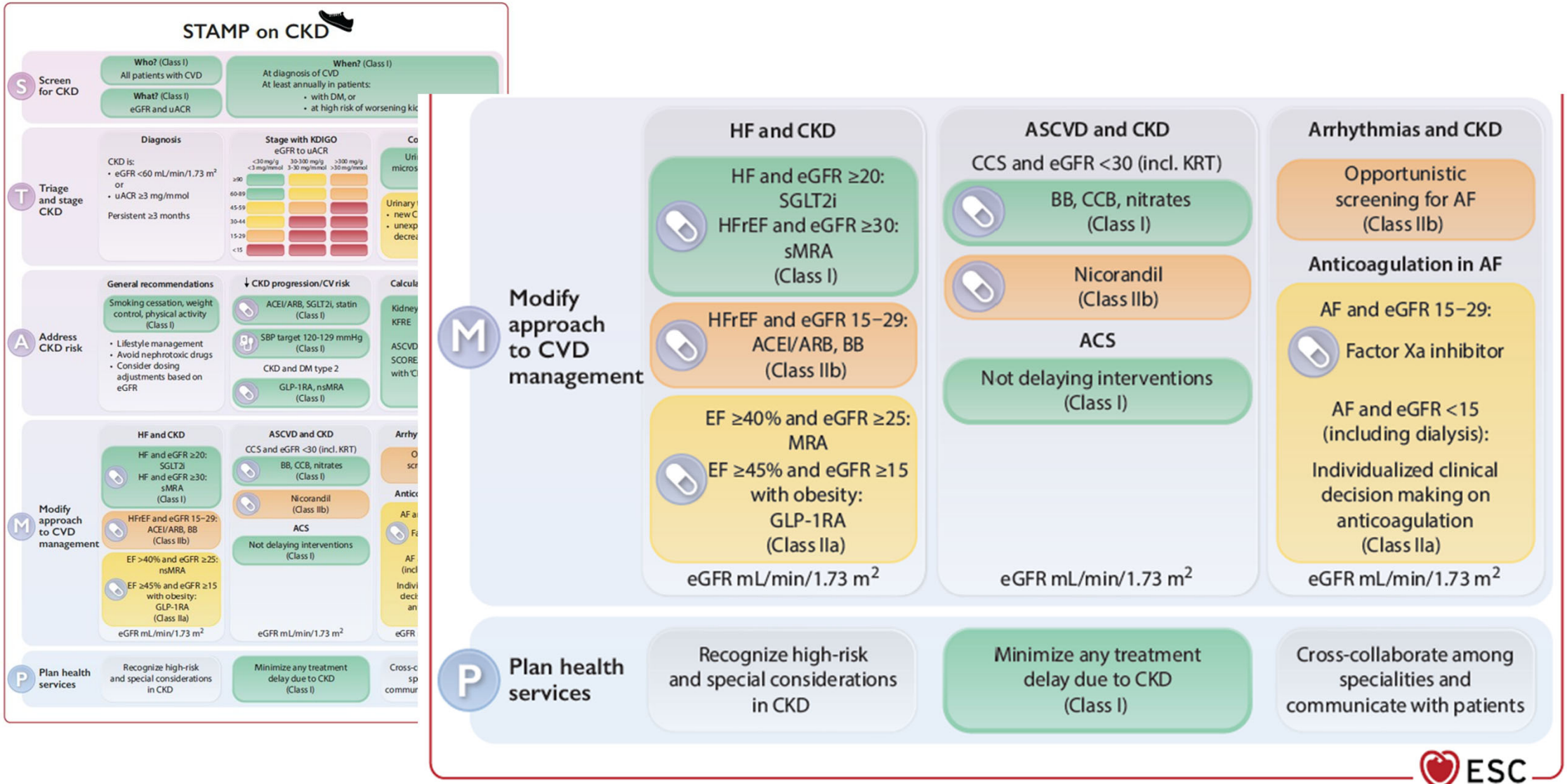
2026 ESC CKD Guidelines





Jonas Spaak

2026 ESC CKD Guidelines



Jonas Spaak

2026 ESC CKD Guidelines

Circulation

AHA PRESIDENTIAL ADVISORY

Cardiovascular-Kidney-Metabolic Health: A Presidential Advisory From the American Heart Association

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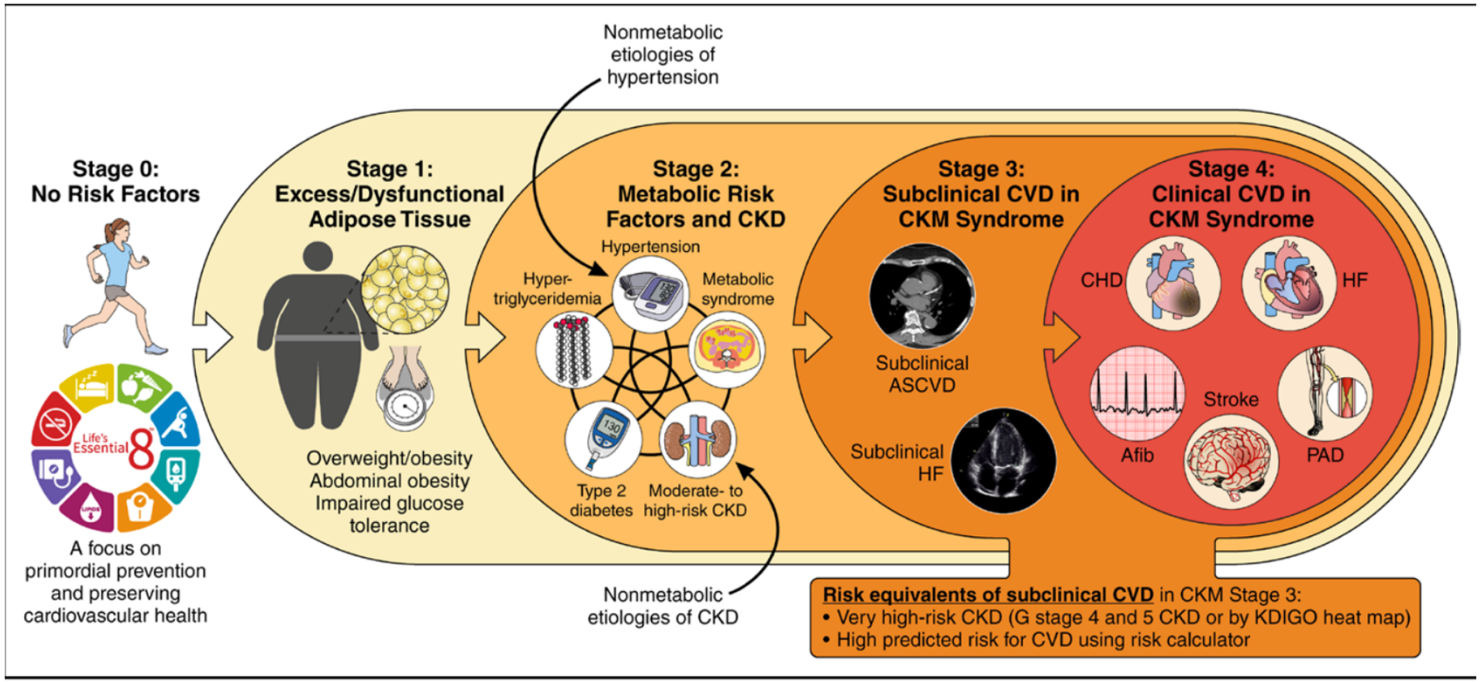
Circulation
Volume 154, Issue 4, 28 July 2026; Pages e50-e158
https://doi.org/10.1161/CIR.0000000000001453

CLINICAL PRACTICE GUIDELINES

2026 AHA/ACC/ADA/ASN Guideline for the Prevention, Detection, Evaluation, and Management of Cardiovascular-Kidney-Metabolic Syndrome: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

Cardiovascular-Kidney-Metabolic Syndrome



Ndumele CE et al. Circulation Nov 2023; 148 (20)
2026 AHA/ACC/ADA/ASN Guideline

Samsjuklighet

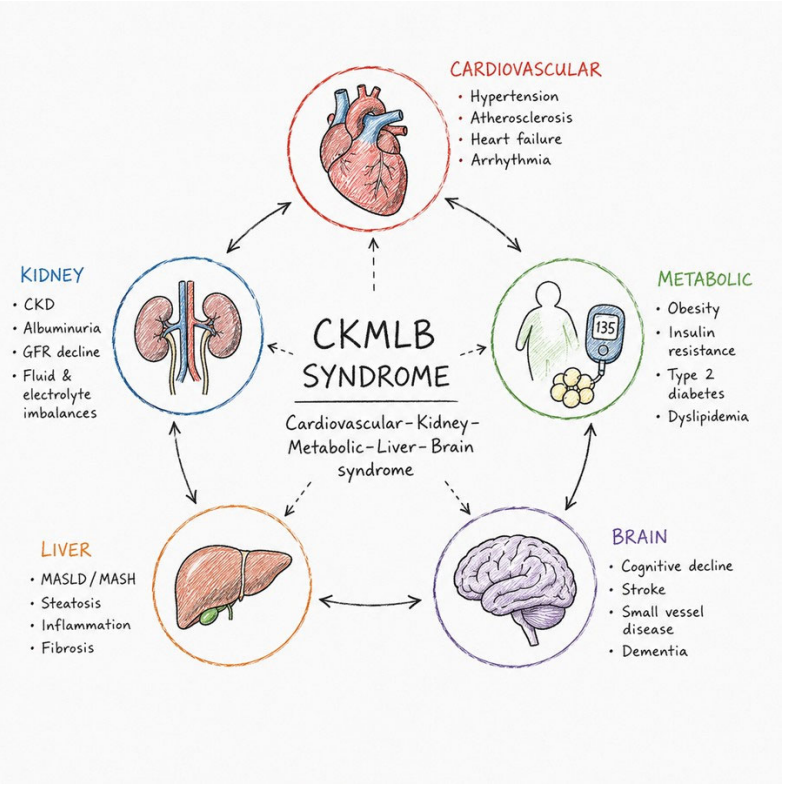
Cardio-Renal syndrome

Cardio-Kidney-Metabolic

Cardio-Vascular-Renal-Metabolic

Cardiovascular-Kidney-Metabolic-Liver

Cardiovascular-Kidney-Metabolic-Liver-Brain



GTP prompt: Make an illustration of Cardiovascular-Kidney-Metabolic-Liver-Brain syndrome, keep it simple and as a sketch

ÄR BLODTRYCKSSÄNKNING EFFEKTIVT ÄVEN VID NJURFUNKTIONSNEDSÄTTNING OCH NJURSVIKT?

Potentiella problem vid njursvikt

- Försämrad njurfunktion → försämrad elimination ger ackumulation

  - Dosering?
  - Försämrad effekt?
  - Risk för biverkningar och njursvikt?
  - Sämre adherens?
  - Sämre evidens?
  - Kan CKD i sig påverka CV fenotyp?

    i.e. är hjärtsvikt vid CKD en annorlunda sjukdom än vid normal njurfunktion?

Pharmacological blood-pressure lowering for the prevention of cardiovascular disease and death across the full spectrum of chronic kidney disease severity: an individual-participant data meta-analysis

*Guyu Zeng, Zeinab Bidel, Qianqian Yang, Dexter Canoy, Mark Woodward, Julia Lewis, Sverre E Kjeldsen, William C Cushman, Jinqing Yuan, Koon Teo, Barry R Davis, John Chalmers, Carl J Pepine, Kazem Rahimi, Milad Nazarzadeh, on behalf of the Blood Pressure Lowering Treatment Trialists' Collaboration**

Lancet 2026; 407: 1626–38

See **Comment** page 1578

*Members listed in the appendix (p 4)

Deep Medicine Group, Nuffield Department of Women's and Reproductive Health, Medical Sciences Division, University of Oxford, Oxford, UK (G Zeng MD, Z Bidel MSc, Q Yang MSc, Prof M Woodward PhD, Prof K Rahimi FRCP, M Nazarzadeh DPhil); **Department of Cardiology, Fuwai Hospital, National Center for Cardiovascular Disease, Peking Union Medical College, Chinese Academy of Medical Sciences, Beijing, China** (G Zeng, Prof J Yuan PhD); **Population Health Sciences Institute, Newcastle University, Newcastle, UK** (D Canoy PhD); **The George Institute for Global Health, School of Public Health,**

Summary

Background Individuals with chronic kidney disease (CKD), particularly those at more advanced stages, have been systematically under-represented in randomised controlled trials (RCTs) of blood-pressure-lowering treatment due to safety concerns, leading to a persistent paucity of evidence for cardiovascular risk management in this high-risk group. We investigated the effect of blood-pressure-lowering treatment on the risk of major cardiovascular disease and death across the full spectrum of CKD stages and by key clinical subgroups.

Methods We conducted a one-stage meta-analysis of individual-participant data from RCTs in which participants were randomly assigned to a blood-pressure-lowering therapy versus a comparator. We used RCTs collated in the Blood Pressure Lowering Treatment Trialists' Collaboration dataset, published at any time in any language, which were eligible for inclusion if they had at least 1000 person-years of follow-up per arm, baseline blood-pressure and creatinine measurements, and time-to-event outcomes; those with unclear randomisation procedures or restricted to heart failure or acute care settings were excluded. Participants with a documented history of heart failure or extreme creatinine values were excluded. No age criteria were applied. The primary outcome was major cardiovascular events, defined as a composite of fatal or non-fatal stroke, ischaemic heart disease, or hospitalisation for, or death from, heart failure. Relative treatment effects were estimated with a stratified Cox proportional hazards model. Heterogeneity of treatment effects was evaluated across prespecified subgroups defined by CKD status, CKD stage (1–5), diabetes, proteinuria, and baseline blood pressure. A stratified network meta-analysis was performed to examine whether treatment effects differed by defined subgroups within each of five principal antihypertensive drug classes. The systematic review was registered in PROSPERO (CRD42018099283).

Findings From 52 RCTs (363 684 participants), a total of 285 124 participants from 46 randomised trials met the

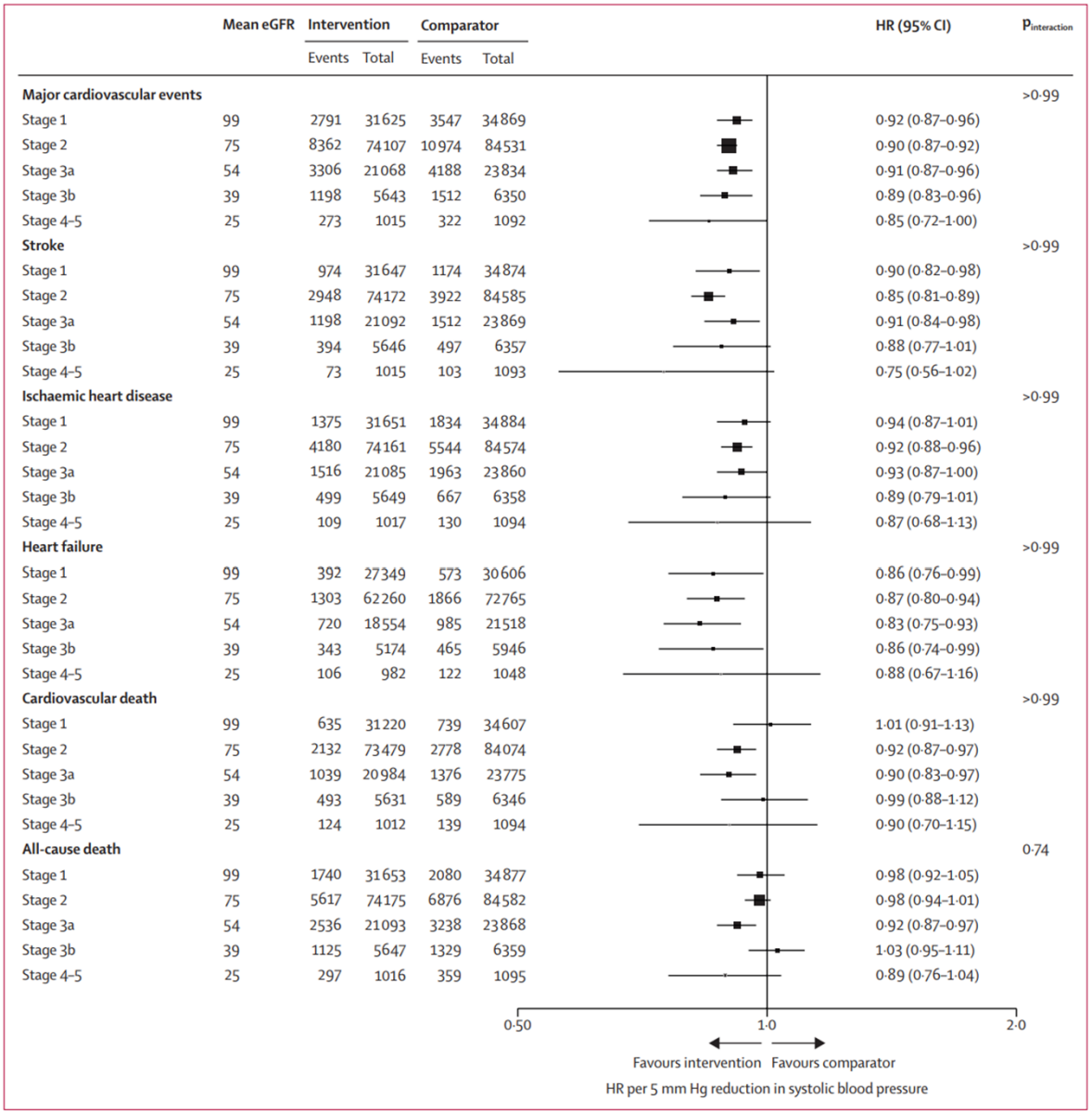


Figure 2: **Effects of blood-pressure-lowering treatment on primary and secondary outcomes by CKD stage**

Ingen interaktion mellan olika utfall och CKD stadium

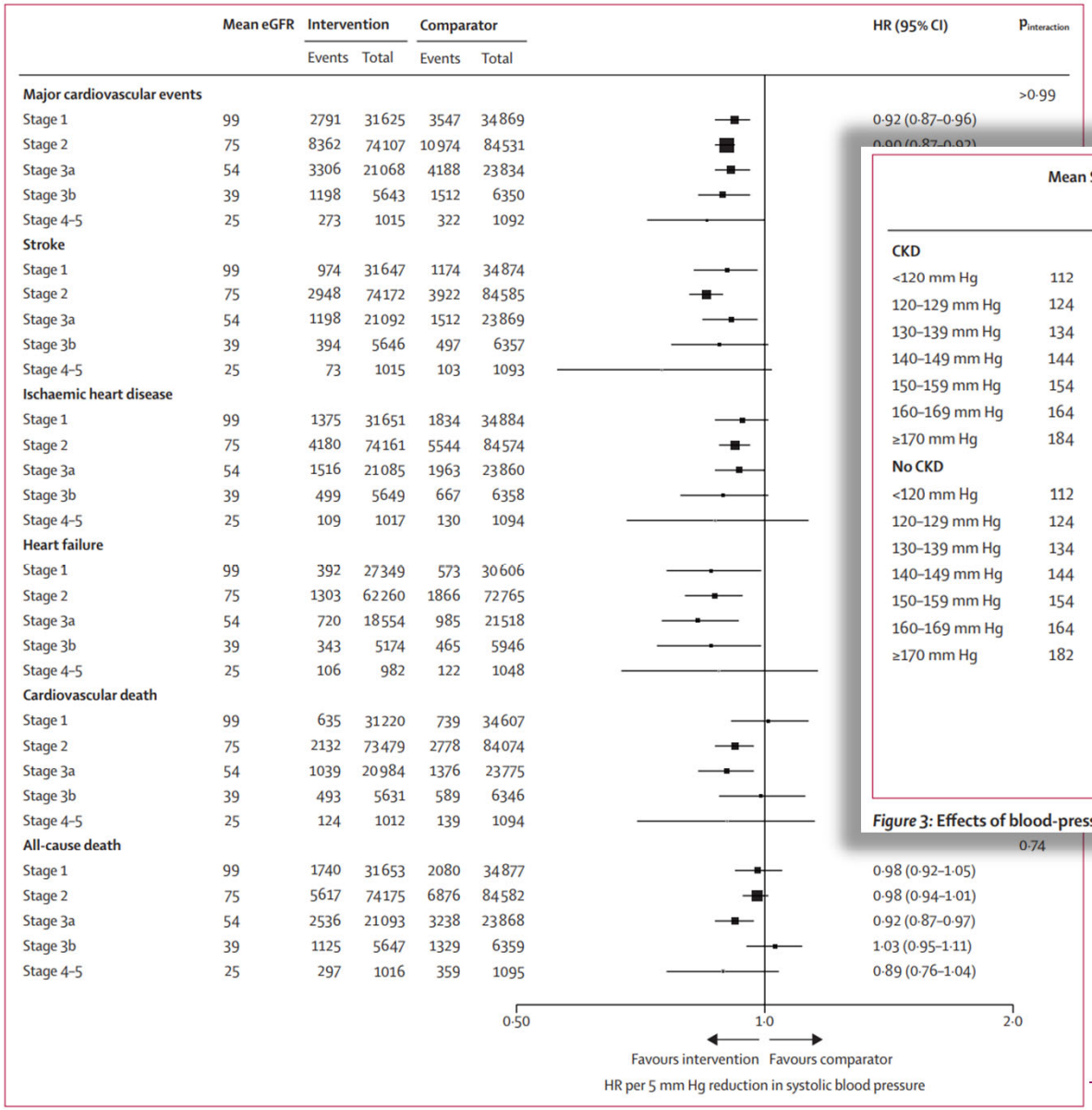


Figure 2: **Effects of blood-pressure-lowering treatment on primary and secondary outcomes by CKD stage**

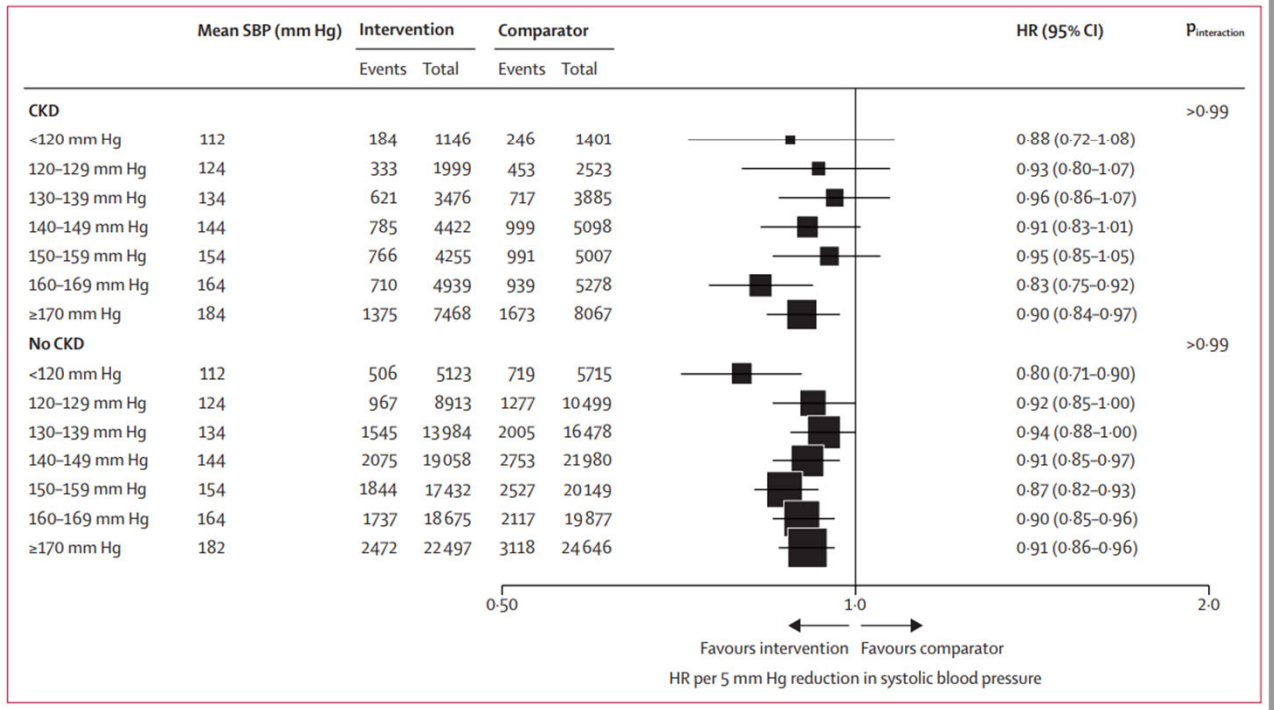
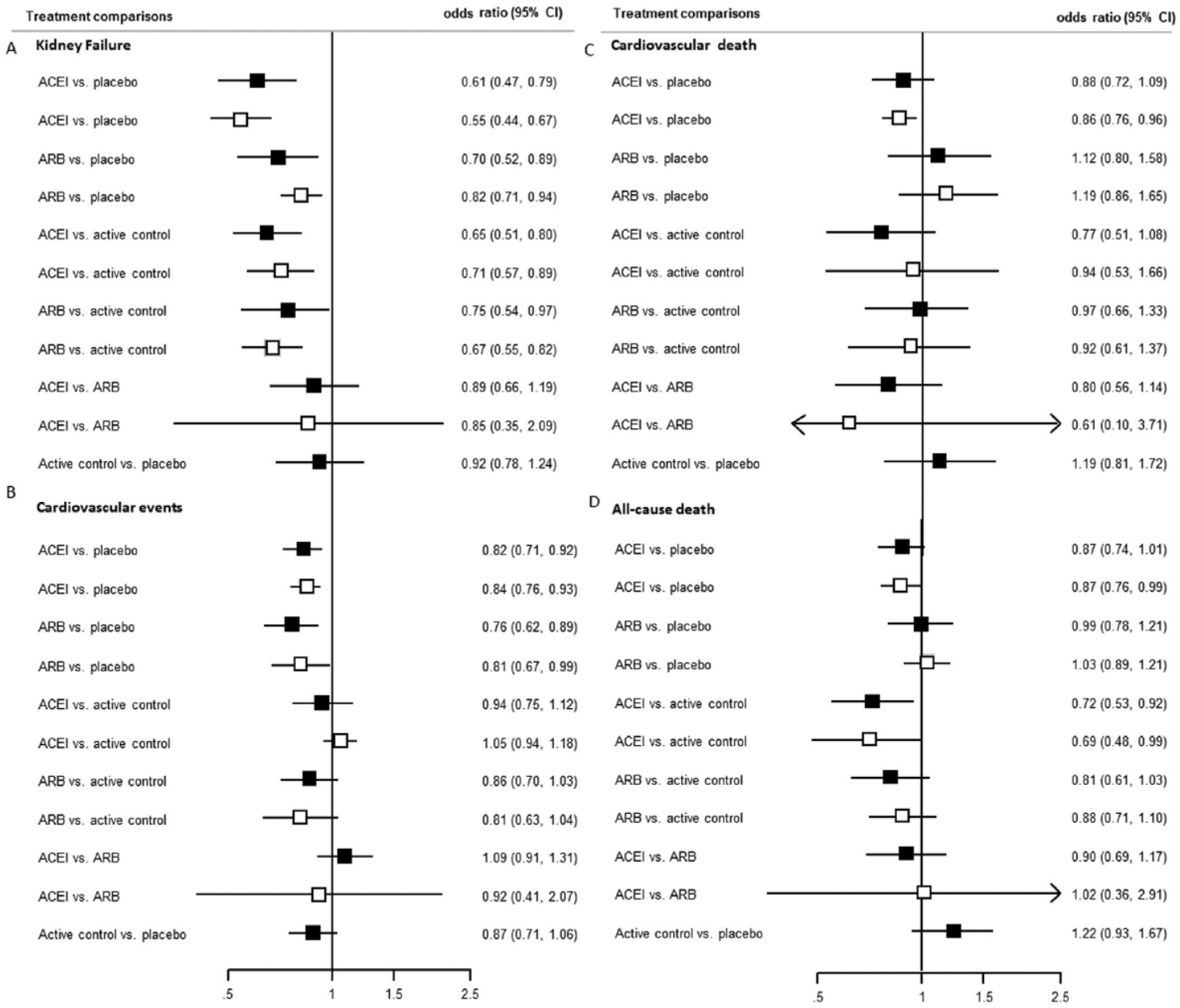


Figure 3: **Effects of blood-pressure-lowering treatment on major cardiovascular events by baseline CKD status and SBP**

Ingen interaktion mellan olika målblodtryck och CKD

ÄR ACE/ARB BRA PÅ ATT SÄNKA BLODTRYCK OCH FÖREBYGGA MACE VID CKD?

Jonas Spaak



Karolinska Institutet

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Metaanalys 119 RCT (n=64,768)

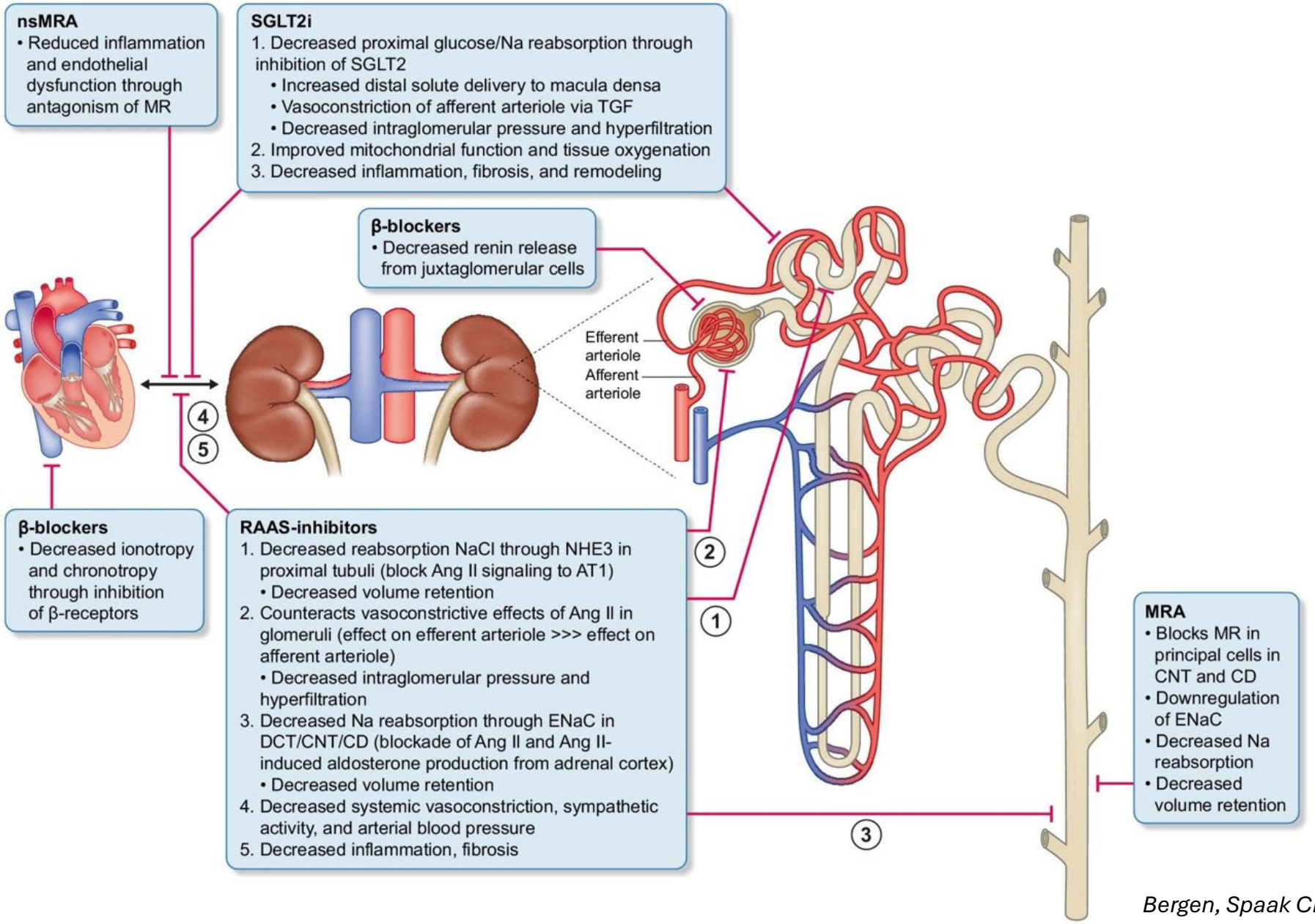
Bayesian network meta-analysis (solid squares; CI, credible interval) and traditional meta-analysis (open squares; CI, confidence interval)

Xie etal AJKD 67(5) 2016

Recommendations	Class[a]	Level[b]
Treatment with maximally tolerated ACEI or ARB is recommended in patients with CKD[c] to achieve target blood pressure, reduce risk of progression of CKD and risk of cardiovascular events.[210,211,213,215,219]	I	A

Recommendations	Class[a]	Level[b]
Treatment with maximally tolerated ACEI or ARB is recommended in patients with CKD[c] to achieve target blood pressure, reduce risk of progression of CKD and risk of cardiovascular events.[210,211,213,215,219]	I	A

ÄR ACE/ARB *BÄTTRE* ÄN ANDRA BLODTRYCKSSÄNKANDE LÄKEMEDEL?



Jonas Spaak

Bergen, Spaak CKJ 19 (1) 2026

VALUE trial

- **Findings:** Blood pressure was reduced by both treatments, but the effects of the amlodipine-based regimen were more pronounced…
- **Interpretation:** The main outcome of cardiac disease did not differ between the treatment groups…

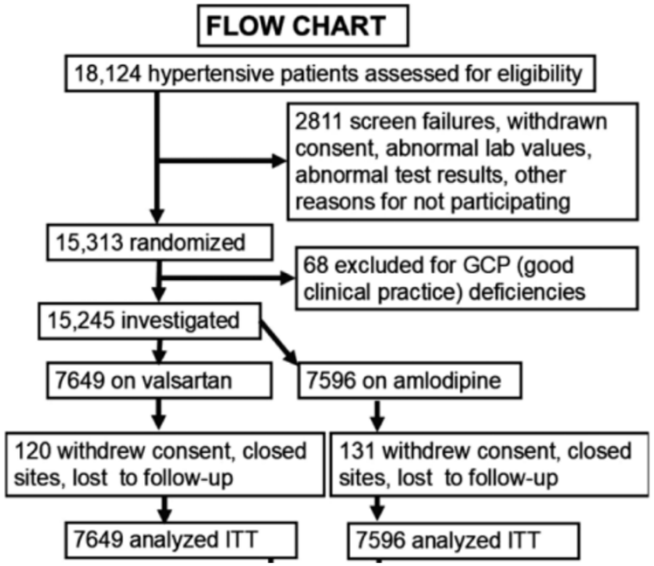
- "In VALUE, the BP treatment target was <140/90 mmHg, achieved by 56 % and 62 % in the valsartan and amlodipine arms, respectively"
- After the initial 6 months, patients with at least three study visits were included, and BP was registered as achieved average BP during the follow-up period.

THE LANCET

ARTICLES

Outcomes in hypertensive patients at high cardiovascular risk treated with regimens based on valsartan or amlodipine: the VALUE randomised trial

*Stevo Julius, Sverre E Kjeldsen, Michael Weber, Hans R Brunner, Steffan Ekman, Lennart Hansson, Tsushung Hua, John Laragh, Gordon T McInnes, Lada Mitchell, Francis Plat, Anthony Schork, Beverly Smith, Alberto Zanchetti, for the VALUE trial group**



PLOS ONE

RESEARCH ARTICLE

Effects of calcium channel blockers comparing to angiotensin-converting enzyme inhibitors and angiotensin receptor blockers in patients with hypertension and chronic kidney disease stage 3 to 5 and dialysis: A systematic review and meta-analysis

Yen-Chung Lin[1,2,3,4], Jheng-Wei Lin[1]*, Mai-Szu Wu[2], Kuan-Chou Chen[2], Chiung-Chi Peng[2], Yi-No Kang[1]*

1 Center for Evidence-based Medicine, Department of Education, Taipei Medical University Hospital, Taipei, Taiwan, R.O.C, **2** Graduate Institute of Clinical Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan, R.O.C, **3** Division of Nephrology, Department of Internal Medicine, Taipei Medical University Hospital, Taipei, Taiwan, R.O.C, **4** Department of Internal Medicine, School of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan, R.O.C

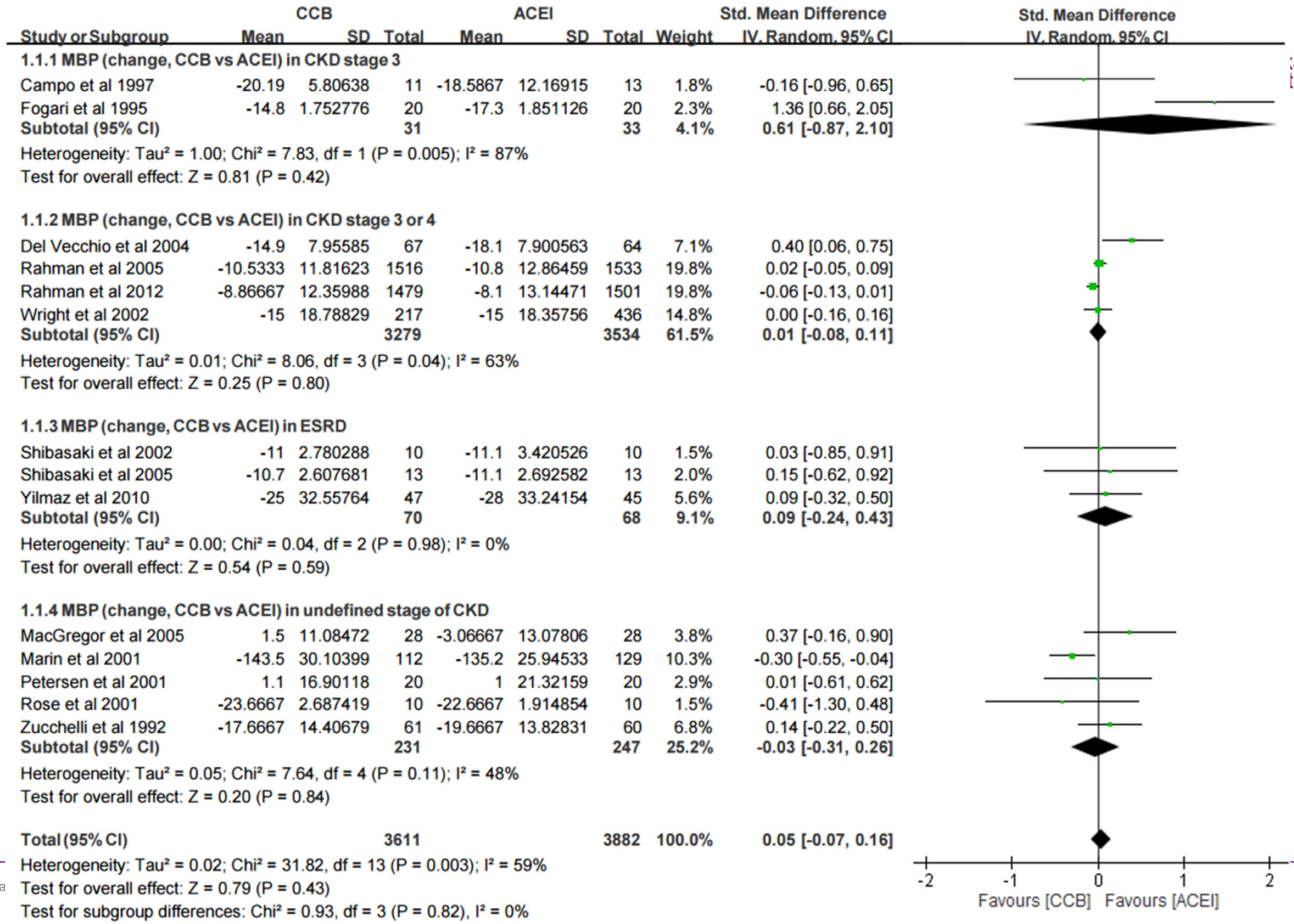
Check for updates

Karolinska Institutet

Danderyds Sjukhus

Metaanalys från 2017

21 RCT (n=9,492) med CKD 3-5



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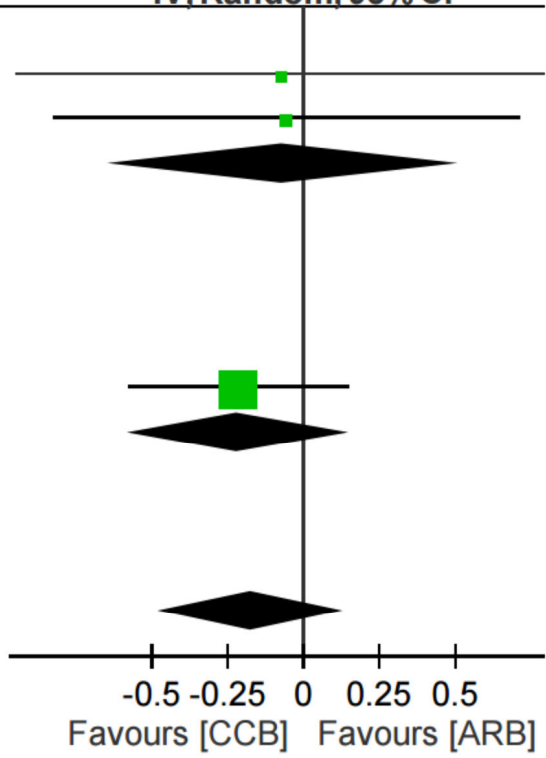
Study or Subgroup	CCB Mean	CCB SD	CCB Total	ARB Mean	ARB SD	ARB Total	Weight	Std. Mean Difference IV, Random, 95% CI
1.2.1 MBP(change, CCB vs ARB) in ESRD								
Shibasaki et al 2002	-11	2.780288	10	-10.7	4.716991	10	12.3%	-0.07 [-0.95, 0.80]
Shibasaki et al 2005	-10.7	2.607681	13	-10.5	3.996248	13	16.0%	-0.06 [-0.83, 0.71]
Subtotal (95% CI)			**23**			**23**	**28.3%**	**-0.06 [-0.64, 0.51]**
Heterogeneity: Tau² = 0.00; Chi² = 0.00, df = 1 (P = 0.98); I² = 0%								
Test for overall effect: Z = 0.22 (P = 0.83)								
1.2.2 MBP(change, CCB vs ARB) in undefined stage of CKD								
Iino et al 2004	-16.2333	10.84097	59	-13	18.28004	58	71.7%	-0.21 [-0.58, 0.15]
Subtotal (95% CI)			**59**			**58**	**71.7%**	**-0.21 [-0.58, 0.15]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 1.15 (P = 0.25)								
Total (95% CI)			**82**			**81**	**100.0%**	**-0.17 [-0.48, 0.14]**
Heterogeneity: Tau² = 0.00; Chi² = 0.18, df = 2 (P = 0.91); I² = 0%								
Test for overall effect: Z = 1.09 (P = 0.27)								
Test for subgroup differences: Chi² = 0.18, df = 1 (P = 0.67), I² = 0%								



Original Investigation AJKD

Comparative Effectiveness of Renin-Angiotensin System Inhibitors and Calcium Channel Blockers in Individuals With Advanced CKD: A Nationwide Observational Cohort Study

Edouard L. Fu, Catherine M. Clase, Marie Evans, Bengt Lindholm, Joris I. Rotmans, Friedo W. Dekker, Merel van Diepen, and Juan-Jesus Carrero

Rationale & Objective: It is unknown whether initiating renin-angiotensin system (RAS) inhibitor therapy in patients with advanced chronic kidney disease (CKD) is superior to alternative antihypertensive agents such as calcium channel blockers (CCBs). We compared the risks for kidney replacement therapy (KRT), mortality, and major adverse cardiovascular events (MACE) in patients with advanced CKD in routine nephrology practice who were initiating either RAS inhibitor or CCB therapy.

Study Design: Observational study in the Swedish Renal Registry, 2007 to 2017.

Settings & Participants: 2,458 new users of RAS inhibitors and 2,345 CCB users with estimated glomerular filtration rates < 30 mL/min/1.73 m^2 (CKD G4-G5 without KRT) who were being followed up by a nephrologist. As a positive control cohort, new users of the same drugs with CKD G3 (estimated glomerular filtration rate, 30-60 mL/min/1.73 m^2) were evaluated.

Exposures: RAS inhibitor versus CCB therapy initiation.

Outcome: Initiation of KRT (maintenance dialysis or transplantation), all-cause mortality, and MACE (composite of cardiovascular death, myocardial infarction, or stroke).

Analytical Approach: HRs with 95% CIs were estimated using propensity score–weighted Cox proportional hazards regression adjusting for demographic, clinical, and laboratory covariates.

Results: Median age was 74 years, 38% were women, and median follow-up was 4.1 years. After propensity score weighting, there was significantly lower risk for KRT after new use of RAS inhibitors compared with new use of CCBs (adjusted HR, 0.79 [95% CI, 0.69-0.89]) but similar risks for mortality (adjusted HR, 0.97 [95% CI, 0.88-1.07]) and MACE (adjusted HR, 1.00 [95% CI, 0.88-1.15]). Results were consistent across subgroups and in as-treated analyses. The positive control cohort of patients with CKD G3 showed similar KRT risk reduction (adjusted HR, 0.67 [95% CI, 0.56-0.80]) with RAS inhibitor therapy compared with CCBs.

Limitations: Potential confounding by indication.

Conclusions: Our findings provide evidence from real-world clinical practice that initiation of RAS inhibitor therapy compared with CCBs may confer kidney benefits among patients with advanced CKD, with similar cardiovascular protection.

Visual Abstract online

Complete author and article information provided before references.

Correspondence to E.L. Fu (e.l.fu@lumc.nl)

Am J Kidney Dis. 77(5):719-729. Published online November 24, 2020.

doi: 10.1053/j.ajkd.2020.10.006

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Svenska njurregistret
2007-17
2458 nya RAAS
2345 nya CCB

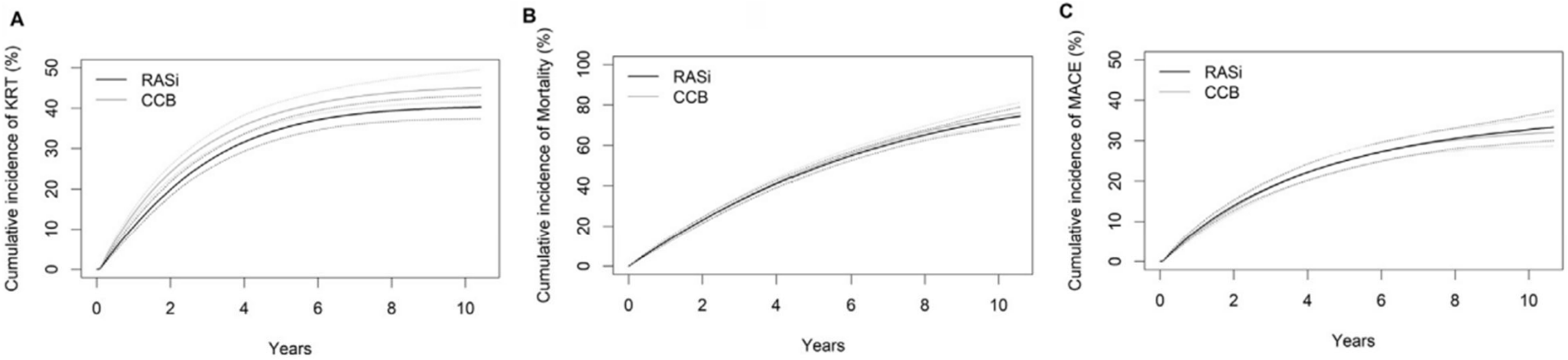


Figure 1. Weighted standardized survival curves for (A) kidney replacement therapy (KRT), (B) mortality, and (C) major adverse cardiovascular events (MACE) stratified by renin-angiotensin system inhibitor (RASi) or calcium channel blocker (CCB) use.

Recommendations	Class[a]	Level[b]
Treatment with maximally tolerated ACEI or ARB is recommended in patients with CKD[c] to achieve target blood pressure, reduce risk of progression of CKD and risk of cardiovascular events.[210,211,213,215,219]	I	A

ÄR ACE/ARB *BÄTTRE* ÄN ANDRA BLODTRYCKSSÄNKANDE LÄKEMEDEL?

INTE BEVISAT

Drugs (2026) 86:737–752
https://doi.org/10.1007/s40265-026-02302-4

SYSTEMATIC REVIEW

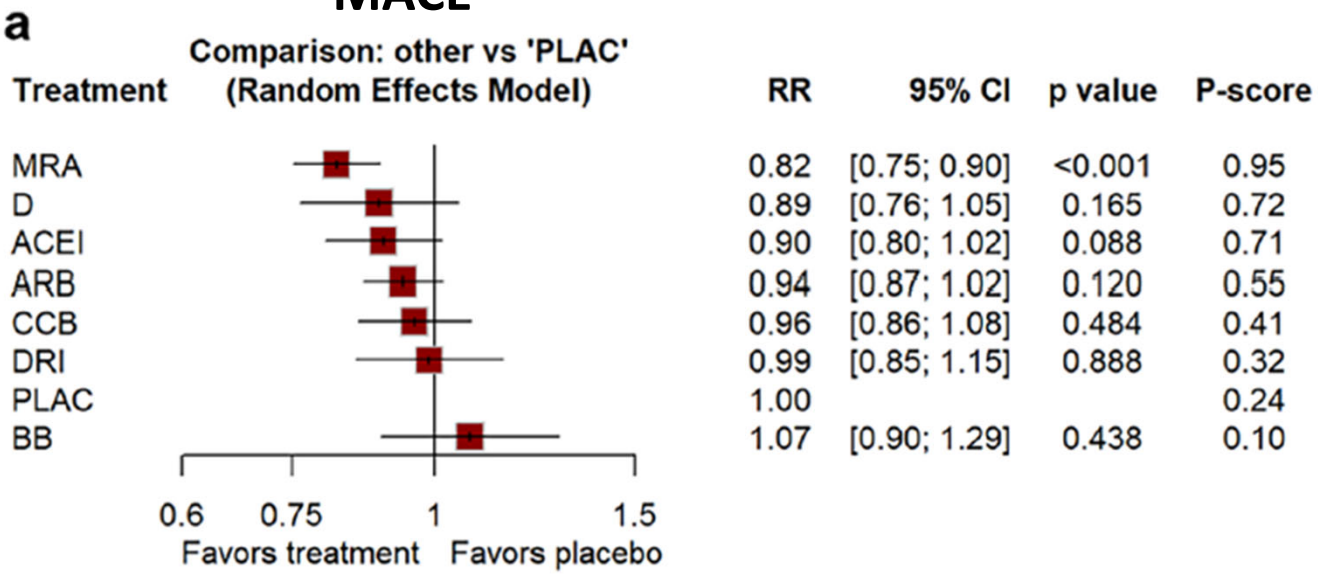
Efficacy of Renin Angiotensin Aldosterone System Inhibitors on Cardiovascular Outcomes in Hypertensive Population: A Network Meta-analysis of Randomized Controlled Trials

Yueming Jiang[1] · Jinbo Hu[1] · Xiaoyan Yi[1,2] · Qifu Li[1] · Shumin Yang[1]

Received: 26 October 2025 / Accepted: 10 February 2026 / Published online: 17 March 2026
© The Author(s), under exclusive licence to Springer Nature Switzerland AG 2026

Metaanalys från 2026
43 RCT (n=160,698)
– inte begränsad till CKD!

MACE



PLAC:placebo, D:Diuretics, DRI:direct renin inhibitors,

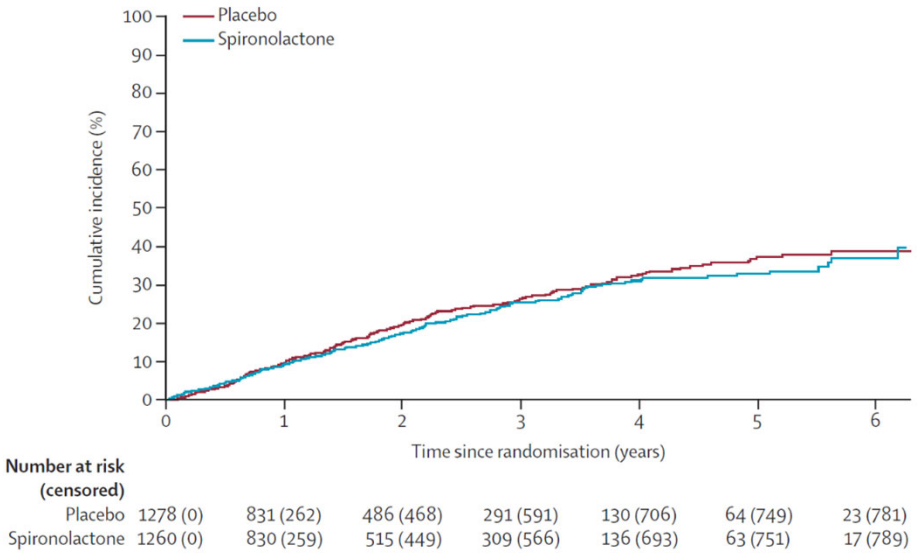
MRA

Steroidal MRA vid avancerad CKD

- Kontraindicerat vid eGFR < 30 enligt FASS
- Används ofta off-label baserat på posthoc analyser av RALES, EMPHASIS-HF.

Vid dialys:

- ACHIEVE (n=2538 HD pat; ca 10-12% känd hjärtsvikt) → ingen effect kardiovaskulär mortalitet, hospitalisering för hjärtsvikt. Hög risk hyperkalemi. Avbröts efter 78% recruitment och 1,8 år för futility.
- ALCHEMIST (n = 644 HD pat; ca 11-12% hjärtsvikt). Ingen skillnad kardiovaskulär komposit. Numeriskt lägre antal HHF i behandlingsgrupp men ej signifikant.



Walsh et al. Lancet 2025
Rossignol et al. Lancet 2025

nsMRA

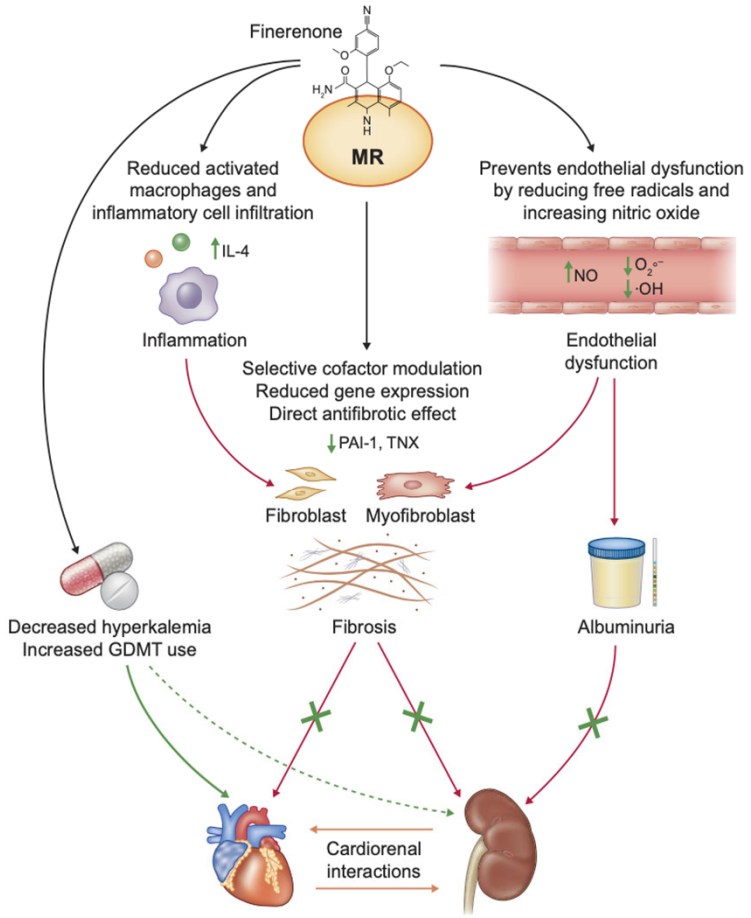
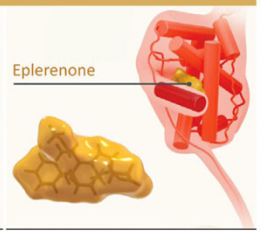


Figure 2: Summary of the cardiorenal mechanisms involved with non-steroidal MRAs.

	Steroidal MRAs		Finerenone
			
Structural properties	Flat (steroidal)	Flat (steroidal)	Bulky (nonsteroidal)
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	+++	++	+

Lo et al. NDT 2023;38:845-854

Epstein et al. AJKD 2022; 80(5):658-666

FIDELITY

ESC European Society of Cardiology

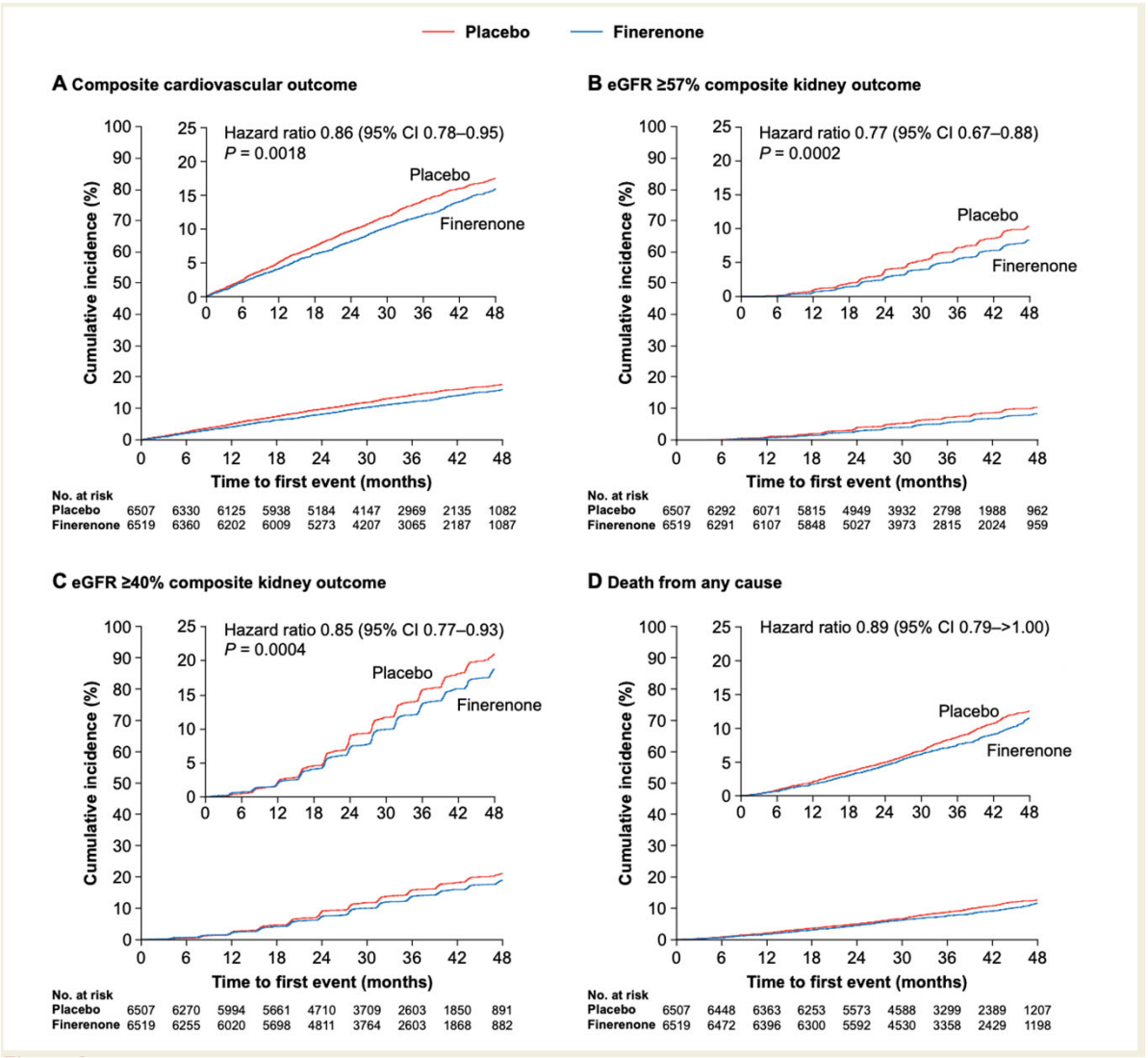
European Heart Journal (2022) **43**, 474–484
https://doi.org/10.1093/eurheartj/ehab777

FASTTRACK CLINICAL RESEARCH
Diabetes and metabolic disorders

Cardiovascular and kidney outcomes with finerenone in patients with type 2 diabetes and chronic kidney disease: the FIDELITY pooled analysis

Rajiv Agarwal [1*†], Gerasimos Filippatos[2*†], Bertram Pitt [3], Stefan D. Anker[4], Peter Rossing [5,6], Amer Joseph[7], Peter Kolkhof [8], Christina Nowack[9], Martin Gebel [10], Luis M. Ruilope [11,12,13], and George L. Bakris [14]; on behalf of the FIDELIO-DKD and FIGARO-DKD investigators[‡]

FIDELITY studien – Finerenon vid typ 2 diabetes



Ca 13 000 patienter med typ 2 diabetes och CKD

Medel eGFR 57,
median UACR ca 50 mg/mmol

>99% RAAS-blockad

6,7% SGLT2-hämmare

7,2% GLP1-analog

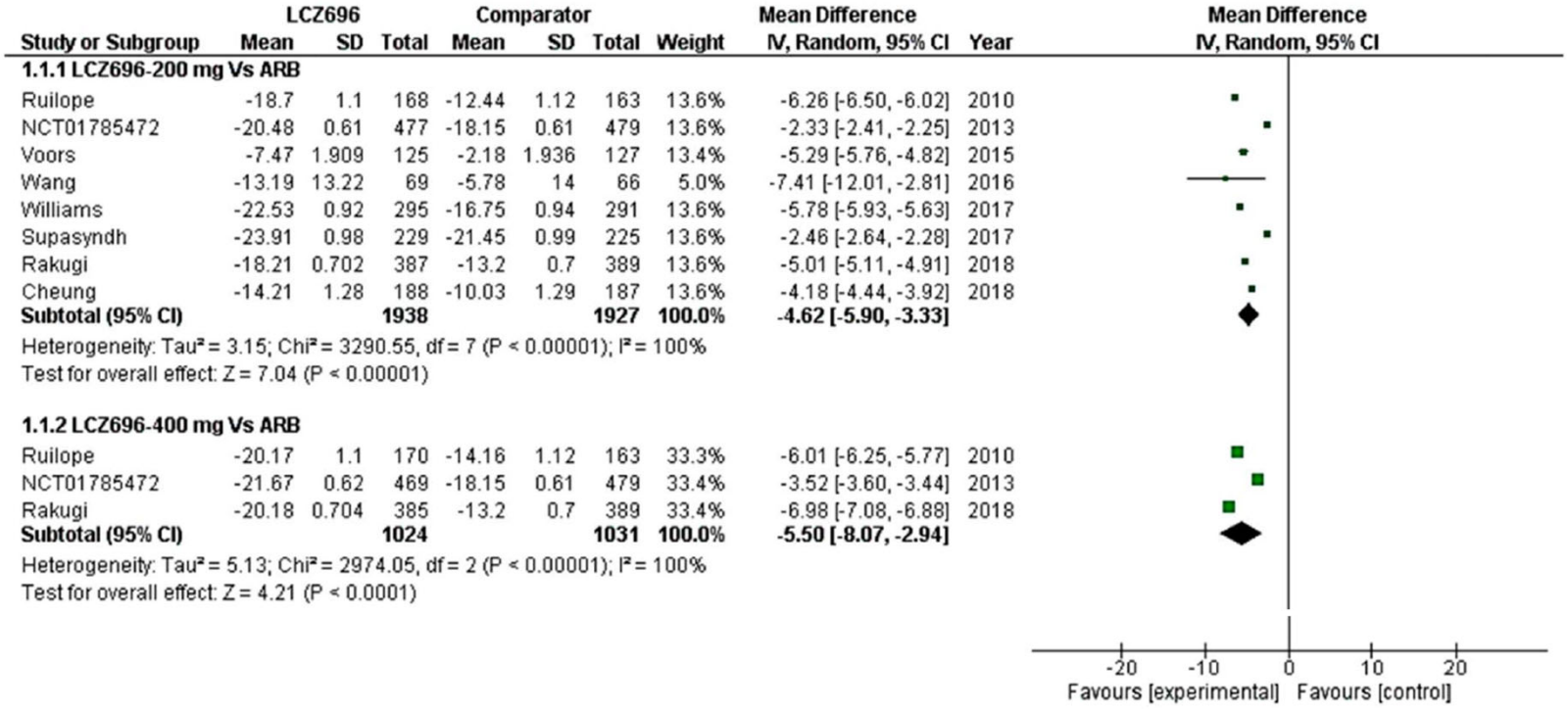
Agarwal et al.
European Heart Journal
*(2022) **43**, 474–484*

SAMSJUKLIGHET
BLODTRYCKSEFFEKTER AV ANDRA BEHANDLINGAR

Jonas Spaak

ARNI - Saccubitril Valsartan (Entresto®)

- Initiala hypertonistudier – uppnått signifikant lägre bltr än valsartan
- Har indikation hypertoni i Kina och Japan



Study or Subgroup	LCZ696 Mean	SD	Total	Comparator Mean	SD	Total	Weight	Mean Difference IV, Random, 95% CI	Year
1.1.1 LCZ696-200 mg Vs ARB									
Ruilope	-18.7	1.1	168	-12.44	1.12	163	13.6%	-6.26 [-6.50, -6.02]	2010
NCT01785472	-20.48	0.61	477	-18.15	0.61	479	13.6%	-2.33 [-2.41, -2.25]	2013
Voors	-7.47	1.909	125	-2.18	1.936	127	13.4%	-5.29 [-5.76, -4.82]	2015
Wang	-13.19	13.22	69	-5.78	14	66	5.0%	-7.41 [-12.01, -2.81]	2016
Williams	-22.53	0.92	295	-16.75	0.94	291	13.6%	-5.78 [-5.93, -5.63]	2017
Supasyndh	-23.91	0.98	229	-21.45	0.99	225	13.6%	-2.46 [-2.64, -2.28]	2017
Rakugi	-18.21	0.702	387	-13.2	0.7	389	13.6%	-5.01 [-5.11, -4.91]	2018
Cheung	-14.21	1.28	188	-10.03	1.29	187	13.6%	-4.18 [-4.44, -3.92]	2018
Subtotal (95% CI)			**1938**			**1927**	**100.0%**	**-4.62 [-5.90, -3.33]**	
Heterogeneity: Tau² = 3.15; Chi² = 3290.55, df = 7 (P < 0.00001); I² = 100%									
Test for overall effect: Z = 7.04 (P < 0.00001)									
1.1.2 LCZ696-400 mg Vs ARB									
Ruilope	-20.17	1.1	170	-14.16	1.12	163	33.3%	-6.01 [-6.25, -5.77]	2010
NCT01785472	-21.67	0.62	469	-18.15	0.61	479	33.4%	-3.52 [-3.60, -3.44]	2013
Rakugi	-20.18	0.704	385	-13.2	0.7	389	33.4%	-6.98 [-7.08, -6.88]	2018
Subtotal (95% CI)			**1024**			**1031**	**100.0%**	**-5.50 [-8.07, -2.94]**	
Heterogeneity: Tau² = 5.13; Chi² = 2974.05, df = 2 (P < 0.00001); I² = 100%									
Test for overall effect: Z = 4.21 (P < 0.0001)									

Malik etal American Journal of Therapeutics 29, e322–e333 (2022)

SGLT2-hämmare i diabetesstudier och office SBP

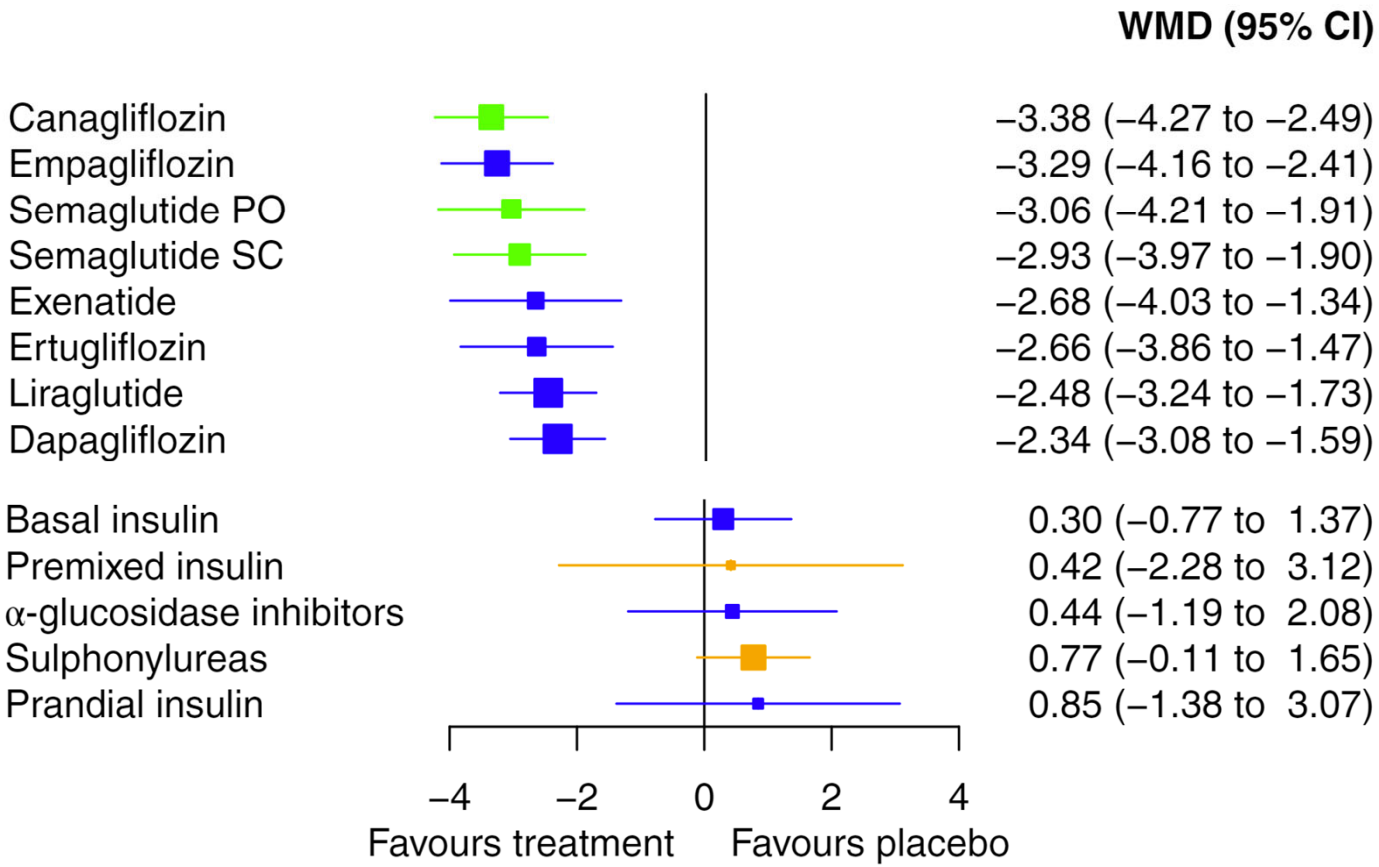


FIGURE 3 Network meta-analysis results for change in systolic blood pressure (mm Hg) compared with placebo. Treatments are presented according to their effect estimate compared with placebo. Effect sizes are presented as weighted mean differences (WMDs) with 95% confidence intervals (CIs). Colours indicate the confidence in the effect estimates according to the CINeMA

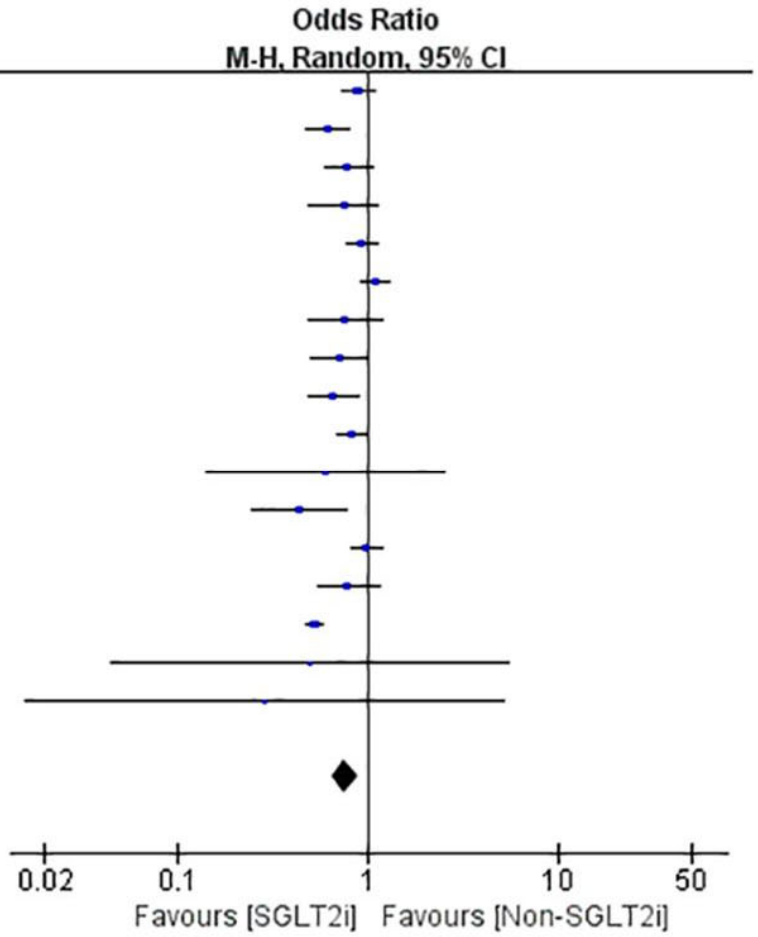
Tsapas etal Diabetes Obes Metab.2021;23:2116–2124

SGLT2i and cardiovascular mortality

Study or Subgroup	SGLT2i Events	SGLT2i Total	Non-SGLT2i Events	Non-SGLT2i Total	Weight	Odds Ratio M-H, Random, 95% CI
Antonio Gonzalez Perez 2023	127	11811	481	39744	8.0%	0.89 [0.73, 1.08]
Björn Pasternak 2019	100	20983	163	20983	7.4%	0.61 [0.48, 0.79]
Caroline H. Nørgaard 2022	70	5275	150	8913	7.0%	0.79 [0.59, 1.05]
Chang Hee Kwon 2022	27	2958	130	10691	5.6%	0.75 [0.49, 1.14]
Christopher P Cannon 2020	341	5499	184	2747	8.1%	0.92 [0.77, 1.11]
Donna Shu-Han Lin 2022	814	81152	187	20288	8.3%	1.09 [0.93, 1.28]
Edouard L Fu 2022	31	5489	51	6886	5.3%	0.76 [0.49, 1.19]
Frederik Persson 2018	38	10227	160	30681	6.3%	0.71 [0.50, 1.01]
Hsin-Fu Lee 2020	69	11431	104	11431	6.8%	0.66 [0.49, 0.90]
Iskandar Idris 2021	213	28720	259	28720	8.1%	0.82 [0.68, 0.98]
Jayoung Lim 2022	3	1842	5	1842	1.1%	0.60 [0.14, 2.51]
Kohei Kaku 2017	22	1006	25	511	4.1%	0.43 [0.24, 0.78]
Stephen D Wiviot 2019	245	8582	249	8578	8.1%	0.98 [0.82, 1.18]
Tadarro L Richardson Jr 2023	48	21170	61	21170	6.0%	0.79 [0.54, 1.15]
Wei-Syun Hu 2023	646	17588	1189	17588	8.8%	0.53 [0.48, 0.58]
Ying-Ying Liu 2023	1	112	2	112	0.4%	0.50 [0.04, 5.54]
Young Sang Lyu 2023	0	186	5	593	0.3%	0.29 [0.02, 5.21]
Total (95% CI)		**234031**		**231478**	**100.0%**	**0.76 [0.64, 0.89]**
Total events	2795		3405			

Heterogeneity: Tau² = 0.07; Chi² = 95.37, df = 16 (P < 0.00001); I² = 83%

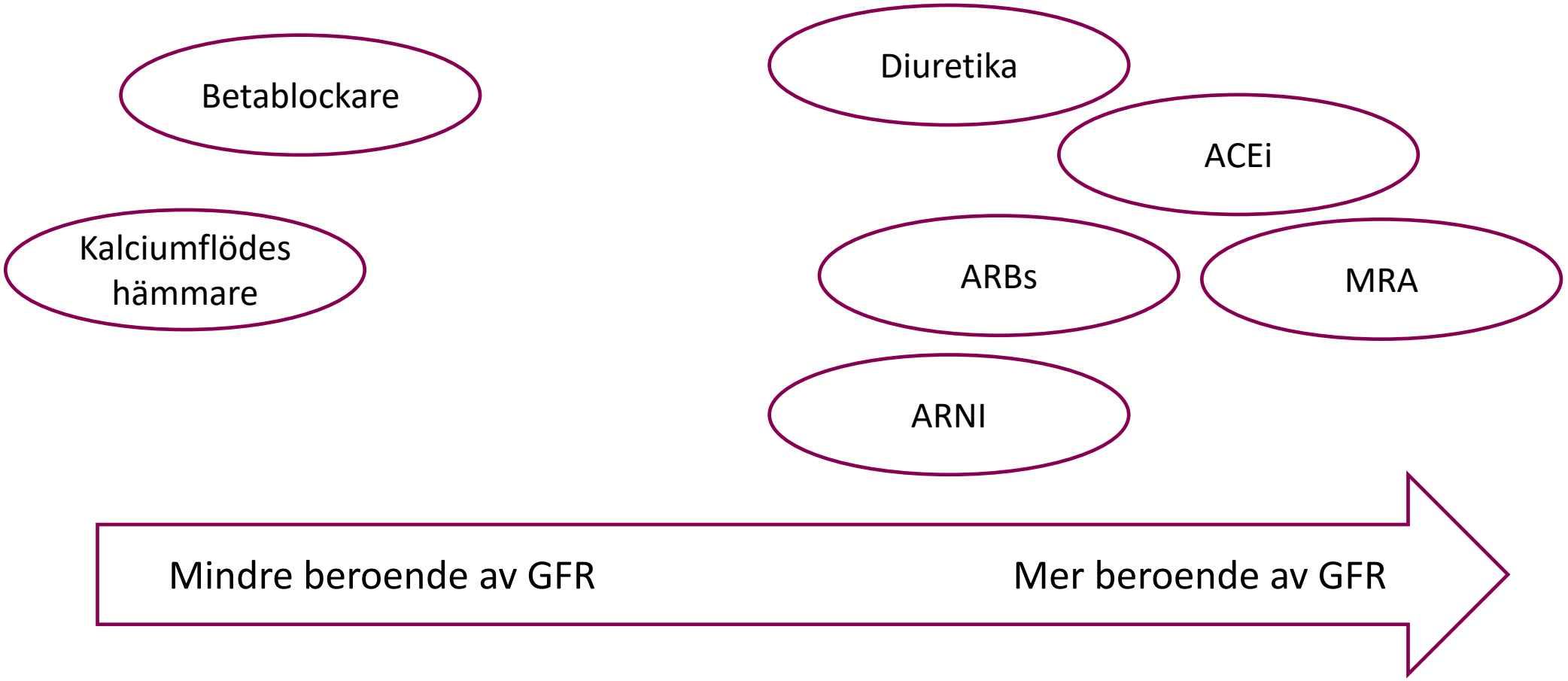
Test for overall effect: Z = 3.39 (P = 0.0007)



Wang F etal Frontiers Endocrin 2024:15

ALL DENNA EVIDENS
– MEN HUR GÖR MAN?

Njursvikt - doseringsfråga…



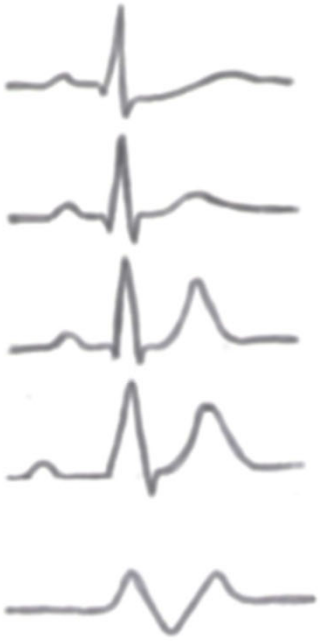
”Lathund” för ACEi/ARB/MRA/ARNi/SGLT2i

1. Sätt ut NSAID
2. Undvik överbehandling med diuretika
3. Vid njursvikt – standardbic/koldioxid!
4. ”Sjukskriv medicinerna”
   – paus vid annan sjukdom med risk för dehydrering
5. Kontrollera blodtryck, kreatinin och kalium 10-14d efter doshöjning, sedan var (3-6)-12 månad. *Gärna med hjälp av sköterskemott.*
6. Njurskyddande trots kreastegring.
   Om >30(-50)% - sätt inte ut, halvera dosen (gäller ej SGLT2i)!



Bergen& Spaak CKJ 2026:19:1

K^+ 5.0 to 5.5	K^+ 5.5 to 6.0	K^+ 6.0 to 6.4	$K^+ \geq 6.5$
Maintain dose If dose increase is planned, re-test within 1-2 weeks	**MILD** If clinically well, repeat K+ within 7-14 days. Consider K^+ lowering maintenance treatment and if persisting, dose reduction of MRA or ACE/ARB/ARNI	**MODERATE** Refer to hospital if acutely developed hyper-K^+ or clinically unwell / AKI, urgent EKG. If clinically well, consider K^+ lowering maintenance treatment, discontinue MRA and potentially ACE/ARB/ARNI. Repeat K^+ within one working day	**SEVERE** Refer to hospital for immediate assessment and treatment. EKG changes → urgent K^+ correction No EKG changes → initiate K^+ lowering treatment, discontinue MRA/ACE/ARB/ARNI until K^+ is under control



Hypokalemia: ST-depression, flat T, U wave, long Q-T

Normokalemia: normal EKG

Mild hyperkalemia: peaked T-waves

Moderate: also widened QRS, prolonged P-R

Severe: no P-wave, sinusoidal bradycardia

Assess potential sampling error – but do not delay
Assess kidney function and potential acidosis
Review diet and drugs
Schedule follow up

Bergen, Spaak CKJ 19 (1) 2026

Varför har patientens kreatinin stigit?



Hemodynamiskt betingad kreastegring förväntat vid insättning eller upptitrering av SGLT2i, RAAS-block eller MRA. Accepterar upp till 30% (50%?)

Variabilitet mellan provtagningar.

Prerenal – postrenal?

Dehydrering

Dekompensation?

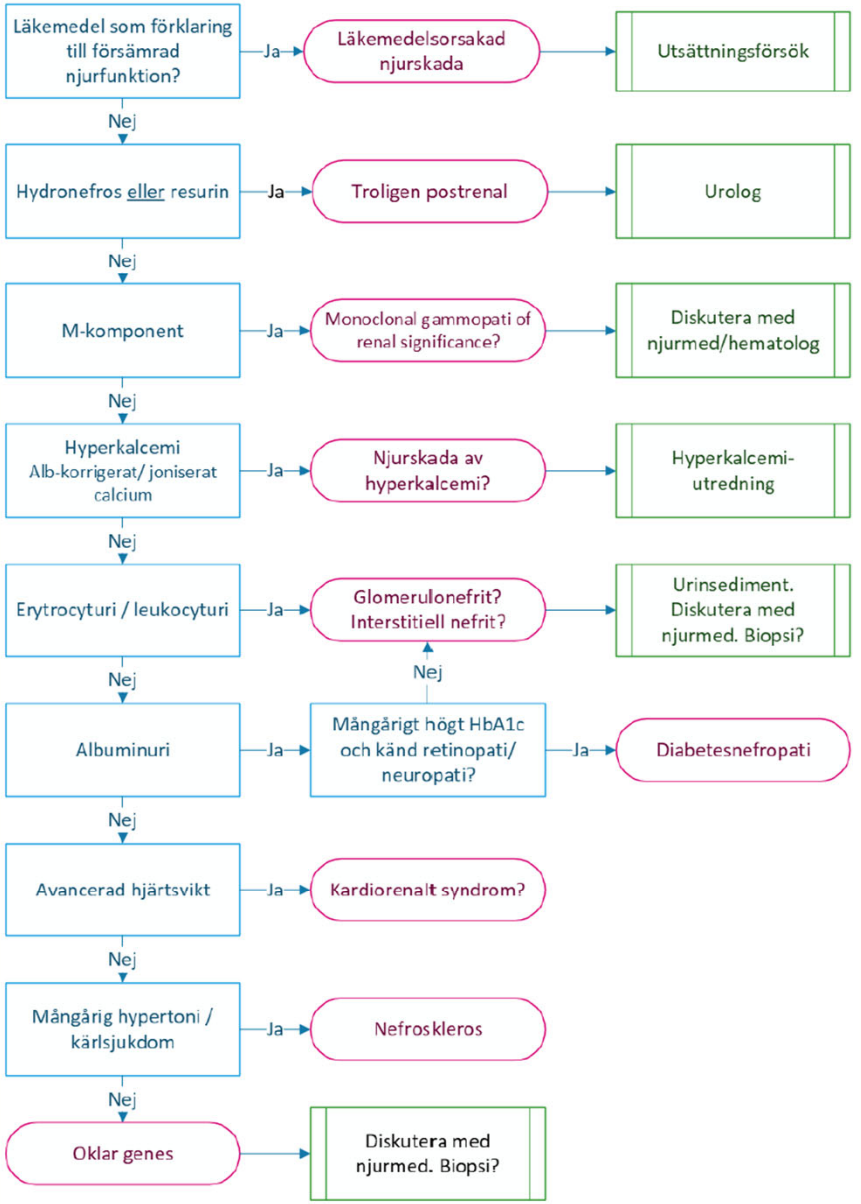
Infektion?

Nya läkemedel?

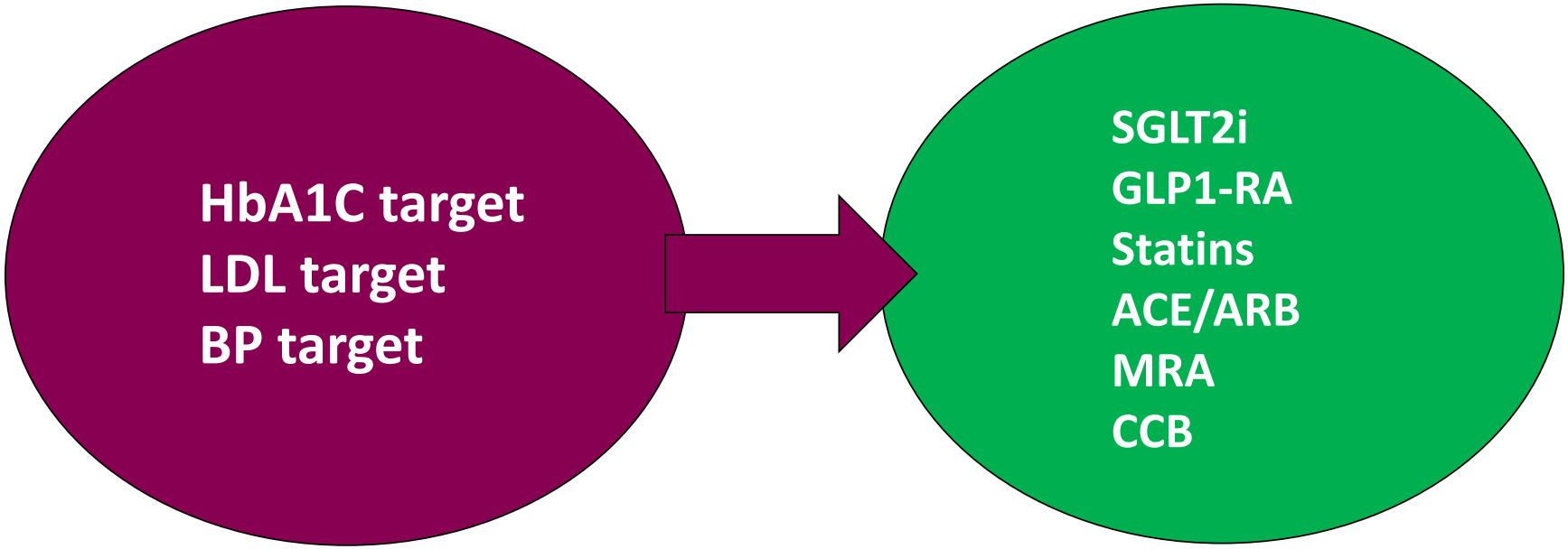
Basutredning av kronisk njursjukdom

- Kreatinin, Cystatin C vid ett tillfälle.
- CRP, albumin, kalcium, HbA1c.
- P-proteinfraktioner. S-FLC alt. U-proteinfraktioner.
- U-sticka, U-alb/krea.
- Ultraljud njurar eller DT buk.

Förenklat flödesschema för tolkning av utredningen



From focus on treatment target to focus on multimodal risk reduction



Sammanfattande punkter

- Blodtryckssänkning minskar CVD likvärdigt över hela CKD spektret
  - Studier med olika målblodtryck vid CKD saknas dock
  - RAASi inte säkert bättre än CCB
- Lägre blodtrycksmål minskar CVD händelser
  - men till priset av GFR försämring och något ökat antal som hamnar i dialys
  - osäkrare evidens vid samtidig DM2
  - Riktlinjerna börjar konvergera mot <130/<80 även vid CKD
    KDIGO 2021/2024->SBP <120 nu public review draft <130; ESH 2023 advises against <120; ESC 2026-> 120–129; AHA/ACC <130/80
- För njurprotektion viktigare med flera läkemedelsklasser
  - RAASi, nsMRA, SGLT2i
  - i njursviktsstudierna drivs den positiva effekten av dessa av grad av proteinuri