

LiNS 2026

Studienübersicht 2025/26

Ralph Wendt



Wir bauen Zukunft. Für Ihre Gesundheit.

Disclosure

lecture fees / advisory board honaria:

Fresenius Medical Care, Vifor Fresenius Pharma,
Amgen, Shire, Novartis, Alexion, Otsuka, Cellpharm,
Ablynx, Daiichi Sankyo, Sanofi, AstraZeneca,
Boehringer Ingelheim / Lilly, Travere, Bayer, Stadapharm,
Roche, GSK

research grants

BMBF, BMG, ERA-PerMed



1976



1995



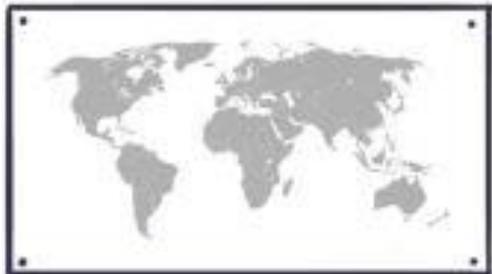
2005



2011



2026



697.294

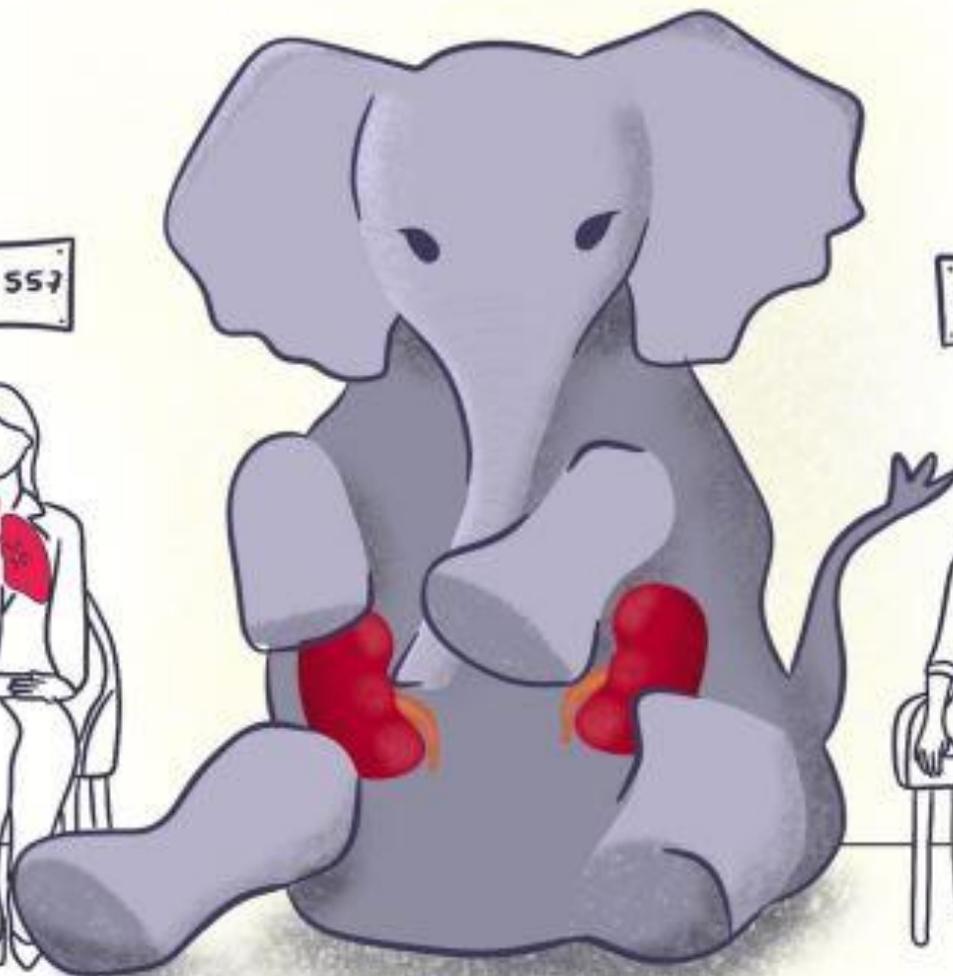
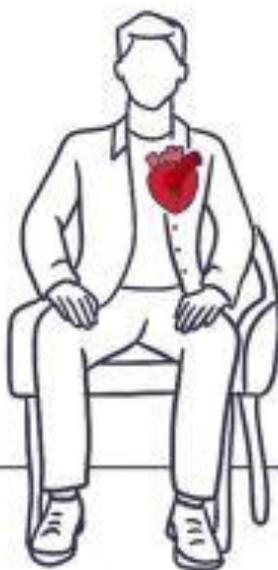
421.725

101.474

454.557

459.875

485.403



Extremely common

843,6 Million
in 2017

Approximately **1 in 10**



Increasing death rate

+41.5% 1990 to 2017

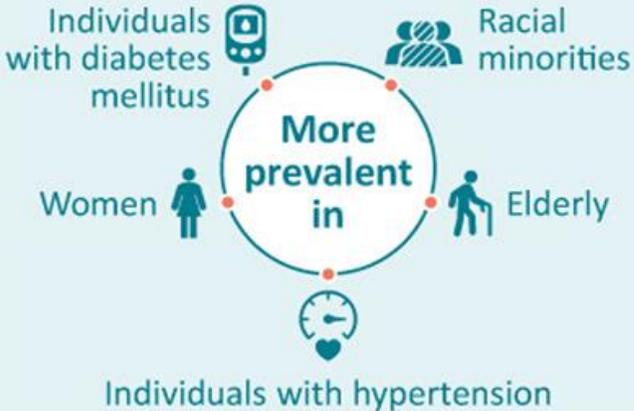


Rank in cause of death

Large burden in low- and middle-income countries



Among the **top 10 causes** of death
in Singapore, Greece, and Israel



CONCLUSION

Chronic kidney disease (CKD) occurs frequently and has devastating consequences. This should prompt major efforts to develop preventative and therapeutic measures that are effective. The aim of these measures should be lowering the incidence of CKD and slowing its progression.



RASi

SGLT2i

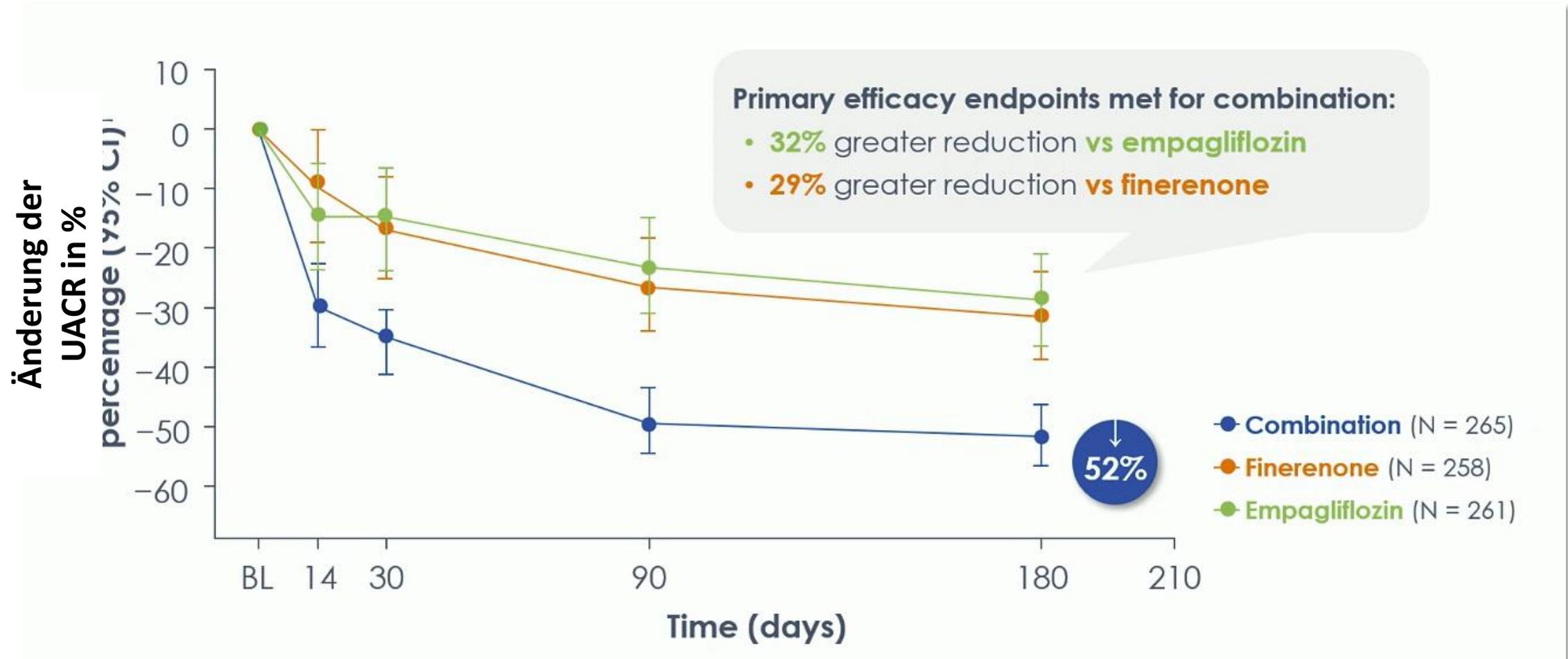
nsMRA

GLP1-RA

Die nephrologischen „fantastic Four“ in CKD (mit T2DM)

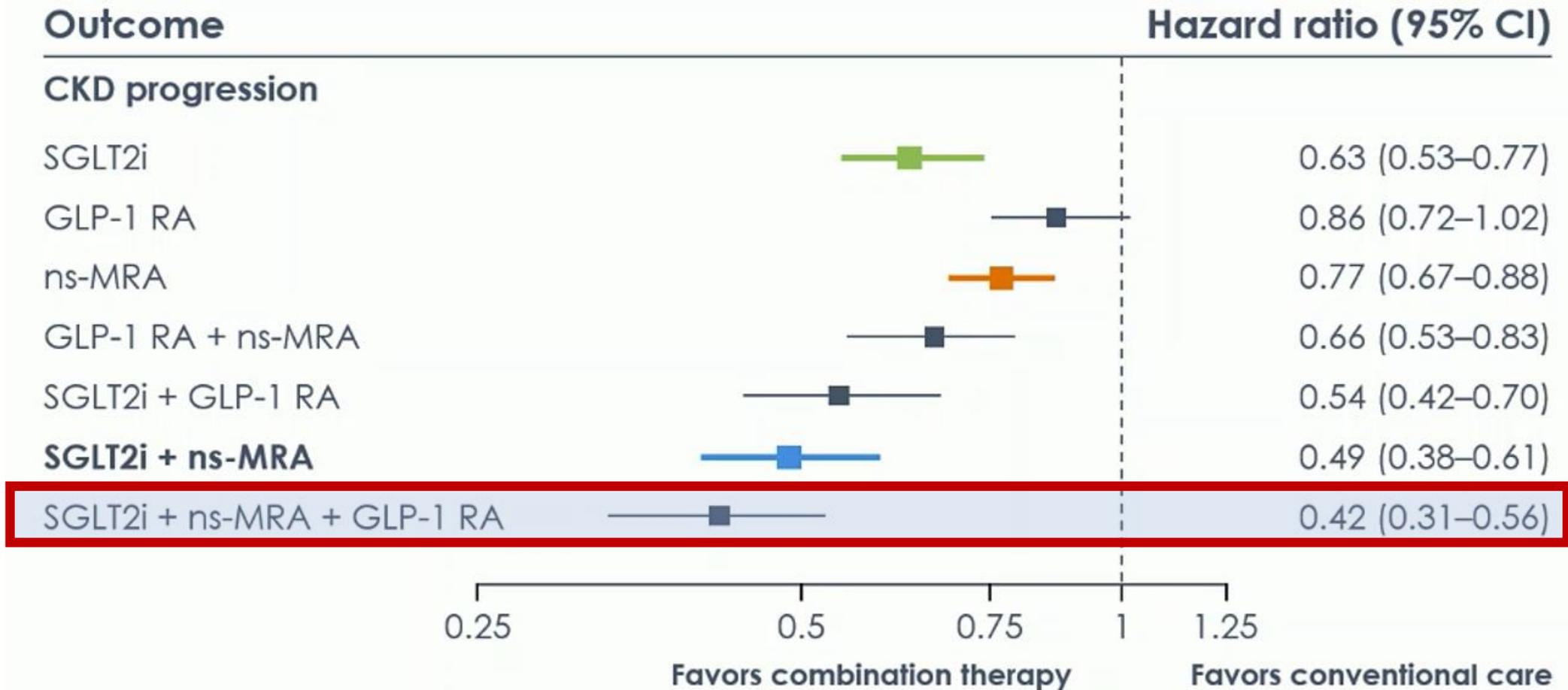
CONFIDENCE: Senken Finerenon und SGLT2-I additiv die Albuminurie?

n=800: T2D, eGFR 30-90, UACR \geq 100 mg/g – <5000 mg/g; max. tolerierter RAS-I



Klinikum **St.GEORG**

CKD-Progressionshemmung durch Kombinationstherapie



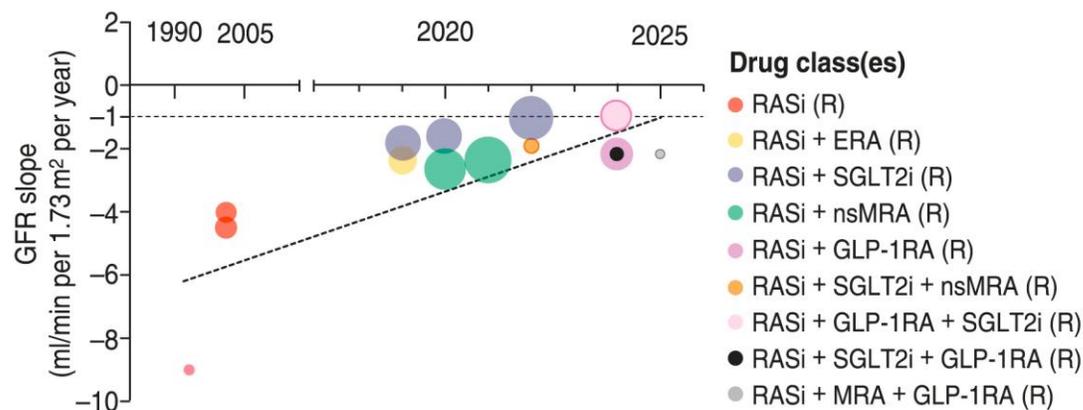
From progression to **remission**: a new paradigm for success in CKD

a

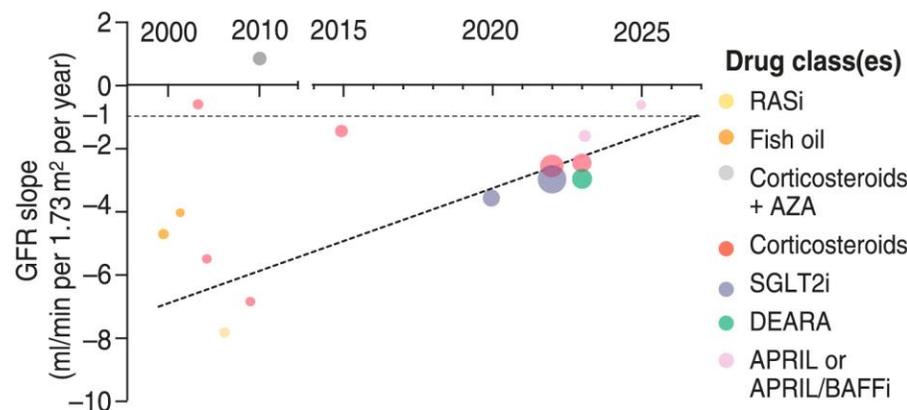
	Historical paradigm: slow CKD progression	New paradigm: aim to achieve CKD remission
Goal	<ul style="list-style-type: none"> Delay the inevitable loss of kidney function 	<ul style="list-style-type: none"> Halt decline in kidney function to normal healthy aging (<1 ml/min per 1.73 m² per year) OR achieve normalization of GFR and albuminuria
Therapeutic context	<ul style="list-style-type: none"> Few effective therapies to prevent loss of kidney function 	<ul style="list-style-type: none"> Combination therapy with highly effective and safe agents (RASi, SGLT2i, ns-MRA, GLP-1RA, disease-specific therapies [e.g., B-cell-targeted therapies])
Workforce and policy focus	<ul style="list-style-type: none"> Major focus on the provision of dialysis and kidney transplantation services 	<ul style="list-style-type: none"> Early detection, population-based screening, risk-based implementation of guideline-directed therapies

b

Annual rate of eGFR decline in the active arm of diabetic kidney disease trials



Annual rate of eGFR decline in the active arm of IgA nephropathy trial



St.GEORG



Wann auf der ITS dialysieren ?

Und vor allem wann wieder aufhören?

Early vs Late Initiation Of Kidney Replacement Therapy : A Comparison Of RCTs



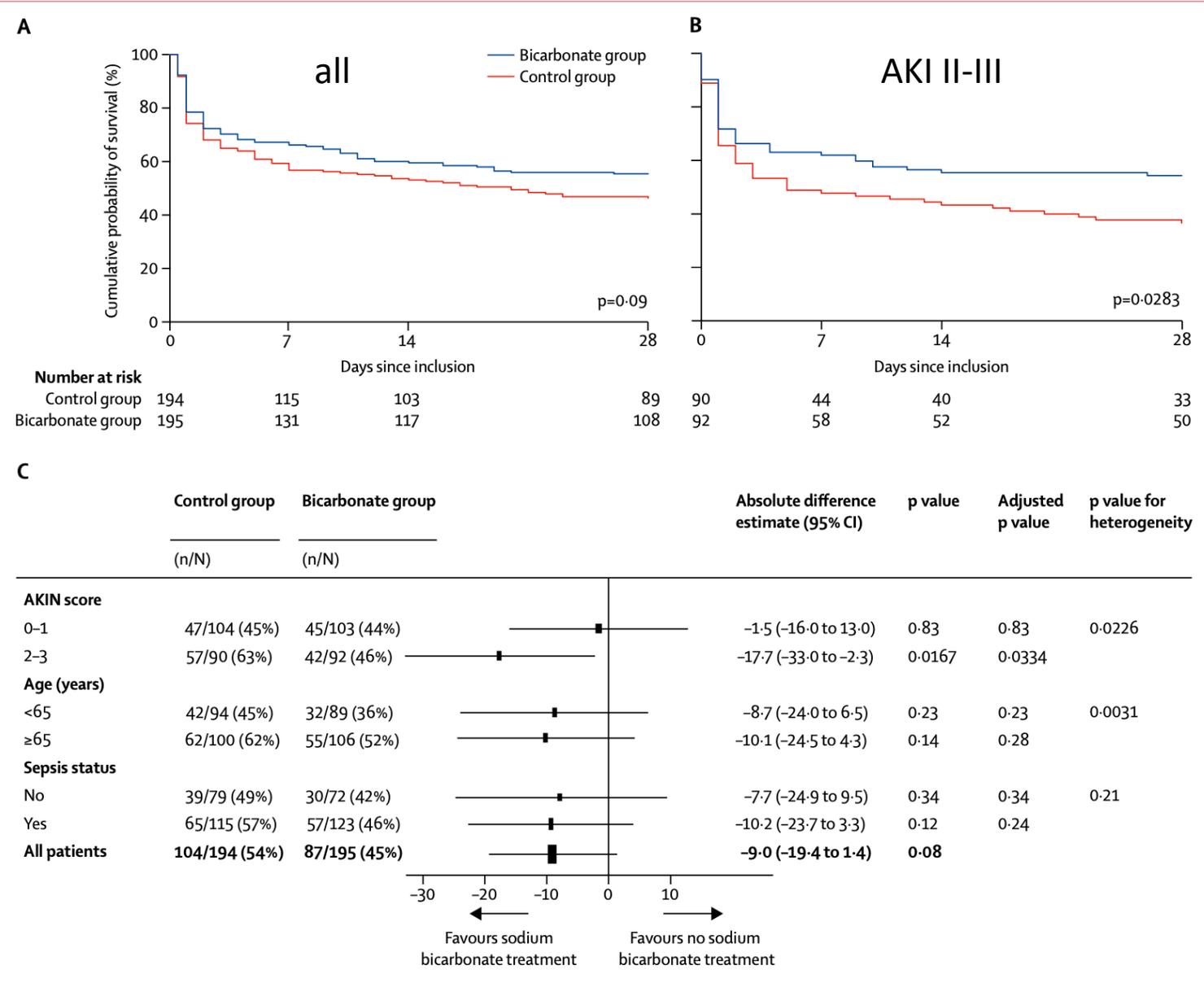
	ELAIN	AKIKI	IDEAL-ICU	STARRT-AKI	AKIKI-2
Study Design	RCT, Single center France	RCT, Multi-Centre France	RCT, Multi-Centre	RCT, Multinational	RCT, Multi-Centre France
Study participants (N)	231	620	488	2927	278
Eligibility criterion	KDIGO stage 2 AKI	KDIGO stage 3 AKI	RIFLE - FAILURE	KDIGO Stage 2 or 3	KDIGO stage 3 with oliguria >72 hrs or BUN 40-50 mmol/l
Early KRT criterion	Within 8 hrs	Within 6 hrs	Within 12 hrs	Within 12 hrs	Within 12 hrs
Delayed KRT criterion	Within 12 hrs or no initiation	<ul style="list-style-type: none"> Life-threatening complications of AKI BUN > 40mmol/l Oliguria persisting >72 hrs 	48 hrs after randomisation in the absence of kidney recovery	<ul style="list-style-type: none"> Life-threatening complications of AKI Persistent AKI for ≥ 72 hrs 	<ul style="list-style-type: none"> BUN >50 mmol/l Life-threatening complication of AKI
Difference in mortality (Early Vs Late)	At 90 d 39.3% vs 54.7% (p=0.03)	At 60 d 48.5% vs 49.7% (p=0.79)	At 90 d 58% vs 54% (p= 0.38)	At 90 d 43.9% vs 43.7% (p=0.92)	At 60 d 44% vs 55% (p=0.07)
Other Key outcomes	Shorter KRT duration and hospital stay in early group	Diuresis occurred earlier in delayed arm	No difference in length of ICU and hospital stay	Higher KRT dependency at 90 d in accelerated arm	KRT free days between D0 and D28 10 vs 12 days (p=0.93)
Complications related to AKI OR KRT (Early Vs delayed)	No difference	CRBSI higher in early group	Hyperkalaemia more in delayed group	More in accelerated arm	No difference
Limitations	Small sample, single centre, mostly surgical patients	Included pts with advanced AKI, 50% pts received IHD	Non blinded, stopped early due to futility	Heterogeneity in groups, Decision of KRT at physician discretion	Small sample size, Debate over BUN levels for KRT initiation
	JAMA 2016	NEJM 2016	NEJM 2018	NEJM 2020	Lancet 2021



Bicarbonat bei Azidose bei ITS Patienten?

Gut, schlecht oder egal?

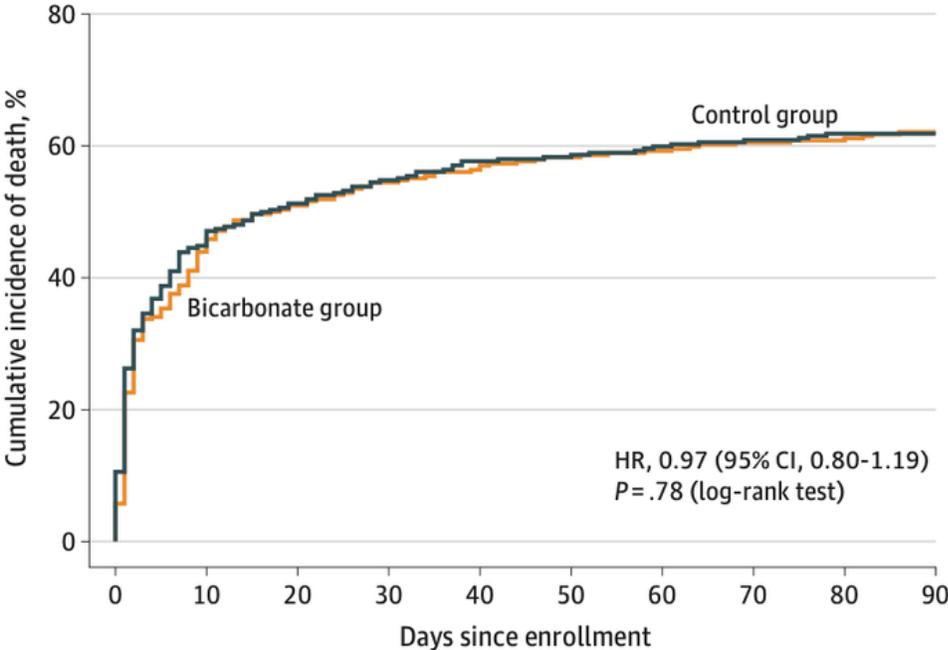
Bicarbonat bei schwerer metabolischer Azidose auf der ITS (BICAR-ICU)



Bicarbonat bei schwerer metabol. Azidose and Akutem Nierenversagen: BICARICU-2

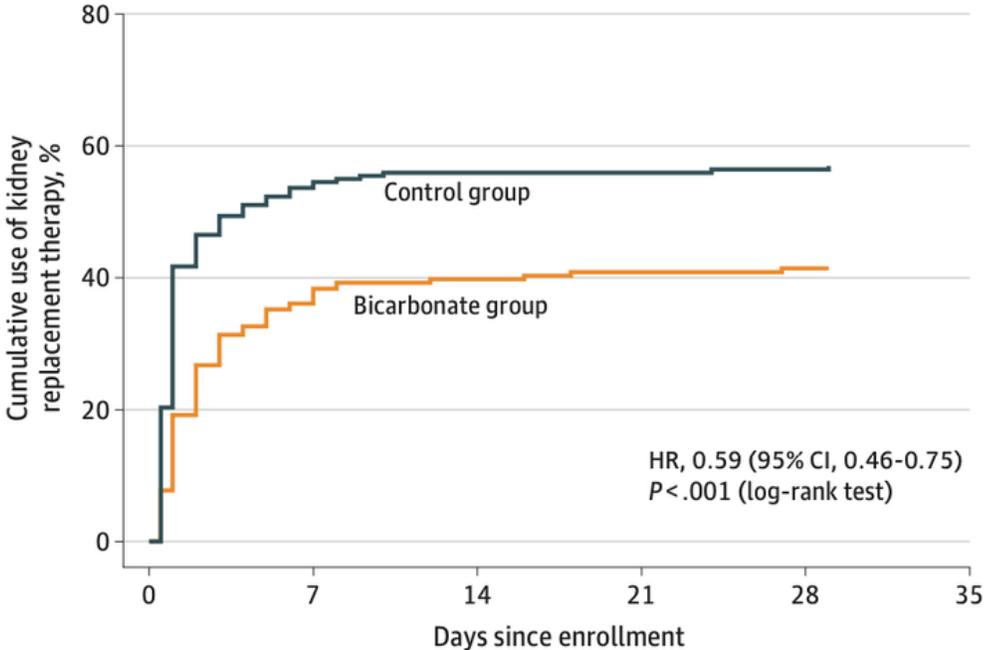
Figure 2. Cumulative Incidence of Death by Day 90 (Primary Outcome) and Cumulative Use of Kidney Replacement Therapy by Day 28 (Secondary Outcome)

A Cumulative incidence of death by day 90



No. at risk		0	7	14	21	28	35	42	49	56	63	70	77	84	91
Bicarbonate group	314	176	154	143	137	131	128	124	123	119					
Control group	313	172	152	141	132	130	125	122	119	118					

B Cumulative use of kidney replacement therapy by day 28



No. at risk		0	7	14	21	28
Bicarbonate group	314	142	114	108	102	
Control group	313	104	91	88	83	

BICAR-ICU2: Does correcting acidemia change dialysis decisions, or outcomes?



Multicentre, open-label, RCT in 33 French ICUs



n=851

Adults with severe metabolic acidemia (pH \leq 7.20, HCO₃⁻ \leq 20 mmol/L)



Randomized within 48 h of ICU admission



Sodium bicarbonate 4.2%



Standard of care

Primary outcome

Day 90 all-cause mortality



62.1%

61.7%

P=.91

Dialysis initiation



35%

50%

Adverse effects

predictable shifts
 \uparrow pH, \uparrow PaCO₂, \uparrow Na⁺, \downarrow K⁺



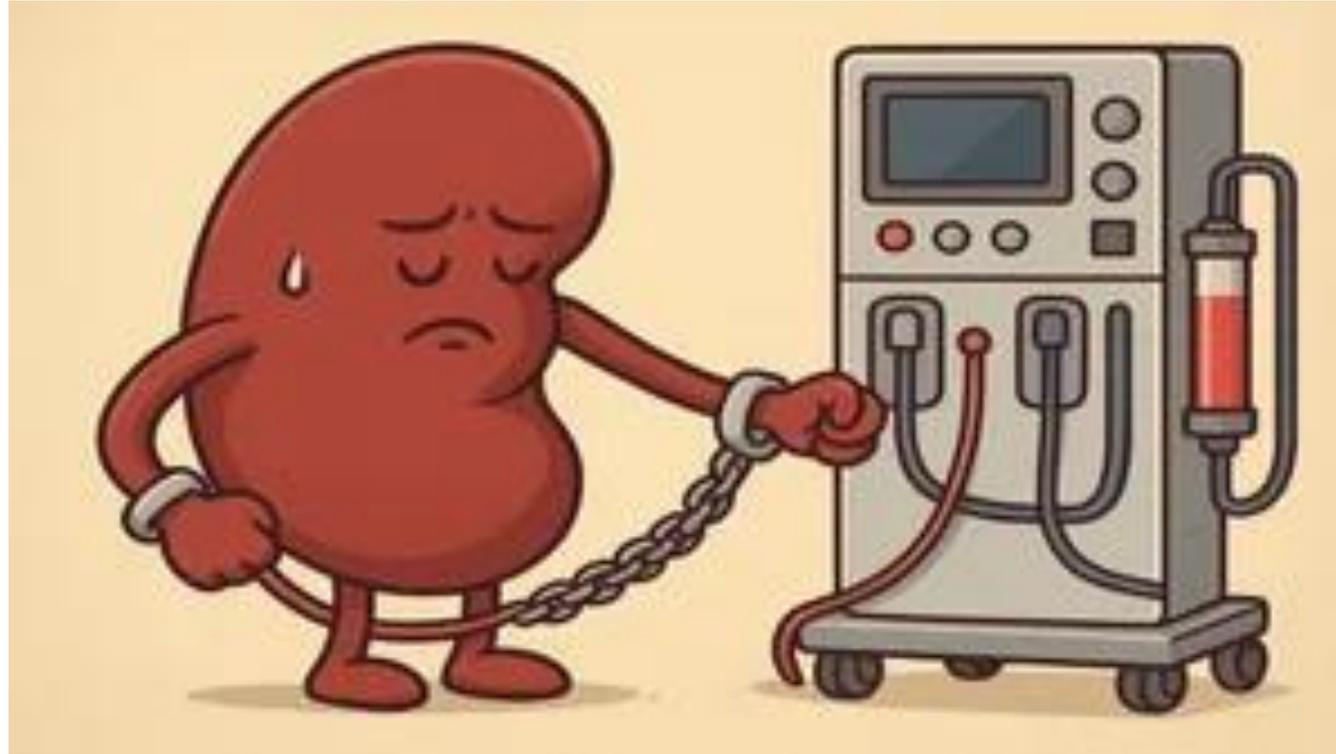
No excess harm

Conclusion: Open-label correction of pH predictably deferred dialysis. The observed reduction in KRT reflected altered thresholds, not renal recovery. The composite endpoint lacked specificity, and a predominantly septic cohort added biological heterogeneity. Bicarbonate improved biochemical metrics without altering clinical outcomes; it rendered acidosis less apparent and dialysis less imminent.

Jung B, et al. Sodium Bicarbonate for Severe Metabolic Acidemia and Acute Kidney Injury- The BICARICU-2 Randomized Clinical Trial. JAMA. 2025

Liberation From Acute Dialysis (LIBERATE-D)

Randomized Clinical Trial



Gibt es eine Art „Dialysis-weaning“?

Klinikum | **St.GEORG**

A Conservative Dialysis Strategy and Kidney Function Recovery in Dialysis-Requiring Acute Kidney Injury

The Liberation From Acute Dialysis (LIBERATE-D) Randomized Clinical Trial

Kathleen D. Liu, MD, PhD, MAS^{1,2}; Edward D. Siew, MD, MSCI^{3,4}; Delphine S. Tuot, MDCM, MAS⁵; [et al](#)

Interventionsgruppe:

Dialyse Stop. Neue Dialyse nur wenn:

- Harnstoff > 40mmol/l
- Hyperkalemia (>6 mmol/L)
- schwere metabolische Azidose (pH <7.15 oder HCO₃ <12 mEq/L)
- Lungenödem mit Hypoxie trotz Diuretika

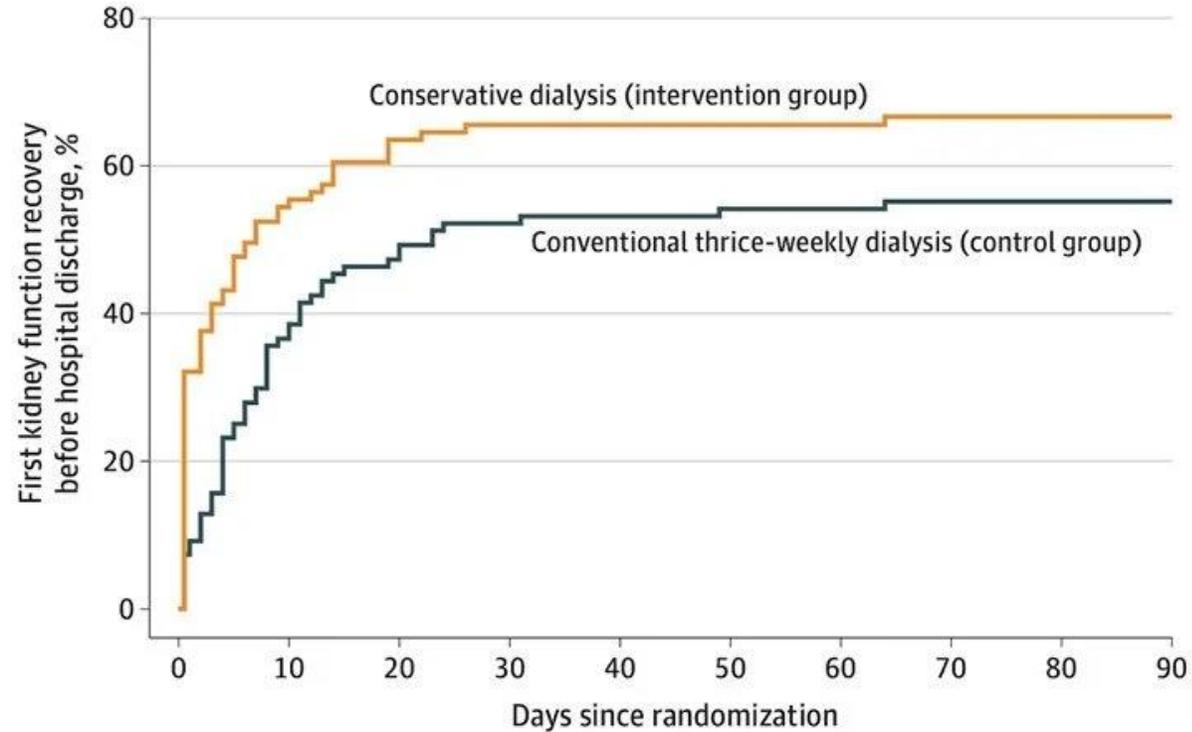
Konventionelle Gruppe:

3x/Wo Dialyse

Stop wenn Diurese >1 L/day ohne Diuretika
Oder >2 L/day mit Diuretika oder
CCL (>20 ml/min), or spontaner Krea-Abfall

Time to first kidney recovery

Figure 2. Time to First Kidney Function Recovery Before Hospital Discharge



No. of patients without kidney function recovery

Conservative dialysis	109	45	36	33	31	31	31	30	30	30
Conventional thrice-weekly dialysis	109	65	54	49	47	46	46	45	45	45

A Conservative Dialysis Strategy and Kidney Function Recovery in Dialysis-Requiring Acute Kidney Injury: The Liberation From Acute Dialysis Trial (LIBERATE-D)



 **Unblinded, randomized, superiority trial**

 **January 2020 through March 2025**

 **4 centers in the US**

 **AKI-D with baseline eGFR >15 mL/min/1.73 m²**

 **909 patients assessed, 221 randomized**

Cohorts and Dialysis Indications

Conservative group (n= 109)

Dialysis IF:
Severe azotemia (BUN >112 mg/dL)
Hyperkalemia (K > 6 mEq/L)
Acidosis (pH <7.15 or bicarb <12 mEq/L)
Acute pulmonary edema w/ hypoxemia (>5L/min, FiO₂ >50%)
Clinician judgment

Conventional group (n=109)

Thrice weekly dialysis UNTIL:
Urine output >1 L/day without diuretics or >2 L/day with diuretics
Creatinine clearance (>20 ml/min)
Spontaneous serum creatinine ↓

Primary Endpoint:

alive & dialysis free for at least 14 consecutive days including discharge



OR 1.76
(95% CI 1.02- 3.03)



Secondary Endpoints:

of dialysis sessions per week & dialysis free days up to day 28

1.8
sessions/wk

-1.4 sessions
(95% CI -1.8 to -1.0)

3.1
sessions/wk

21
consecutive
dialysis free
days

16 days
(95% CI 5 to 27)

5
consecutive
dialysis free
days

Conclusion: A conservative dialysis strategy in AKI-D resulted in a shorter time to and higher rates of recovery of kidney function in the unadjusted analysis. Given uncertainty regarding the estimated effect size, this approach should be tested in a larger study population.

Liu KD, Siew ED, Tuot DS, et al. A Conservative Dialysis Strategy and Kidney Function Recovery in Dialysis-Requiring Acute Kidney Injury: The Liberation From Acute Dialysis (LIBERATE-D) Randomized Clinical Trial. JAMA.2025;doi:10.1001/jama.2025.21530.

✉ Sejal Lakhani (sejalplakhani)

Table 2. Clinical Outcomes by Treatment Groups

	Conservative dialysis intervention group (n = 109)	Conventional thrice-weekly dialysis control group (n = 109)	Estimated difference between proportions (95% CI), % ^a	P value
Primary study end point				
Kidney function recovery at hospital discharge, No. (%)	70 (64.2)	55 (50.5)	13.8 (0.8 to 26.8)	.04
Key secondary study end points				
Dialysis (No./wk) after randomization, all patients, median (IQR) [No.] ^b	1.8 (0 to 2.6) [110]	3.1 (2.6 to 3.5) [110]	-1.4 (-1.8 to -1.0)	<.001
Dialysis-free days to day 28, median (IQR) [No.] ^c	21 (0 to 28) [109]	5 (0 to 21) [109]	16 (5 to 27)	<.001
Other secondary study end points				
Time to kidney function recovery by day 90, median (IQR) [No.]	2 (0 to 9) [79]	8.5 (4 to 22) [76]	-6.5 (-10.2 to -2.8)	<.001
Length of hospital stay after study randomization for patients who survived, median (IQR) [No.]	14 (8 to 26) [100]	15.5 (10 to 28) [102]	-1.5 (-5.8 to 2.8)	.43
In-hospital death, No./total No. (%)	10/110 (9.1)	7/109 (6.4)	2.7 (-4.4 to 9.7)	.46
Kidney function recovery at day 28, No./total No. (%)	71/109 (65.1)	59/109 (54.1)	11.0 (-1.9 to 24.0)	.10
Death by day 28, No./total No. (%)	14/110 (12.7)	7/109 (6.4)	6.3 (-1.4 to 14.0)	.11
Kidney function recovery at day 90, No./total No. (%)	72/108 (66.7)	63/108 (58.3)	8.3 (-4.5 to 21.2)	.21
Death by day 90, No./total No. (%)	16/109 (14.7)	20/108 (18.5)	-3.8 (-13.7 to 6.1)	.45



Phosphatsenkung bei Dialysepatienten

A tale of believe and grief

Phosphate-Binding Agents in Adults With CKD: A Network Meta-analysis of Randomized Trials

Suetonia C Palmer ¹, Sharon Gardner ¹, Marcello Tonelli ², Dimitris Mavridis ³, David W Johnson ⁴, Jonathan C Craig ⁵, Richard French ⁶, Marinella Ruospo ⁷, Giovanni F M Strippoli ⁸

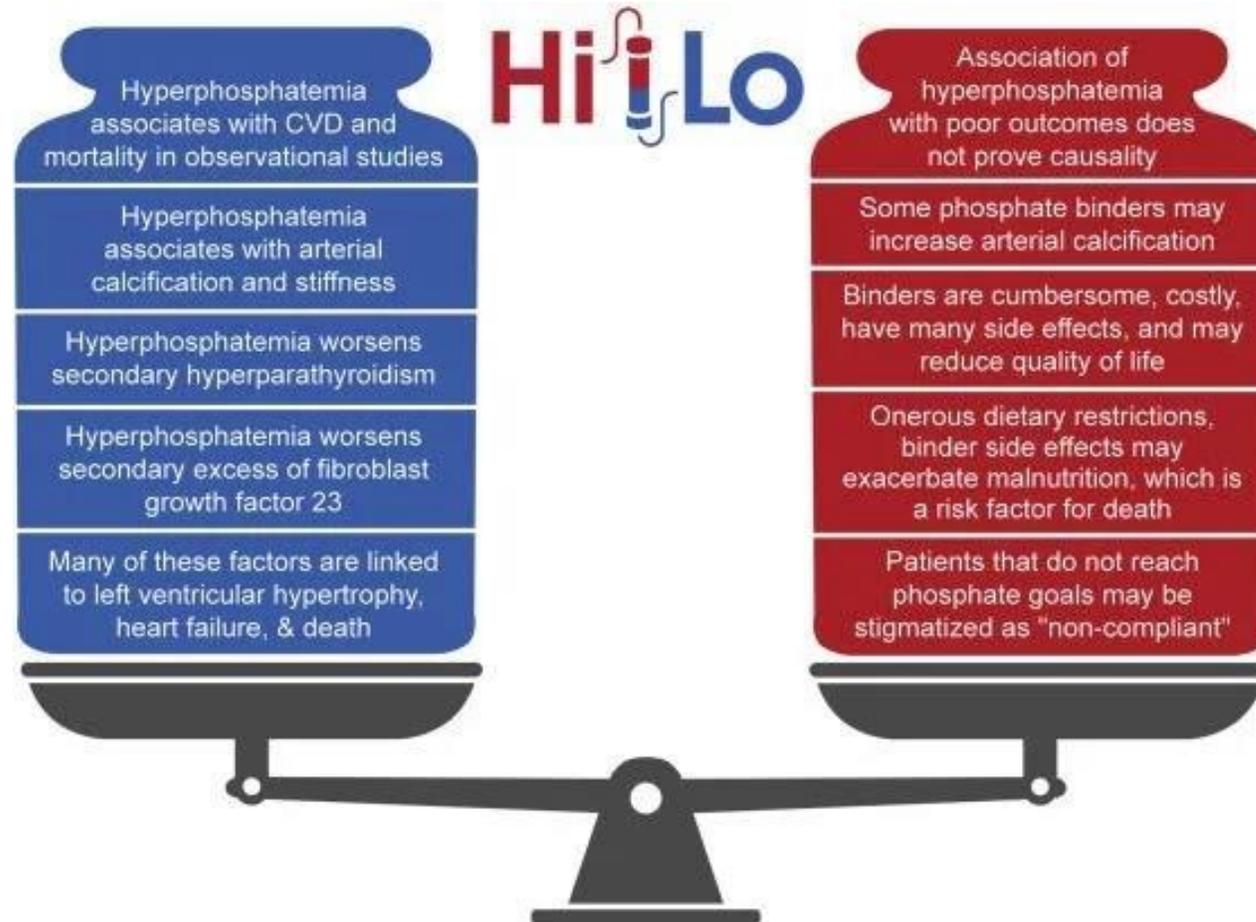
Affiliations + expand

PMID: 27461851 DOI: [10.1053/j.ajkd.2016.05.015](#)

“There is currently no evidence that phosphate-binder treatment reduces mortality compared to placebo in adults with CKD.”

Phosphatsenkung bei Dialysepatienten: *you have to believe*

Factors in favor of and against more aggressive reduction of serum phosphate levels



LANDMARK: Does Lanthanum carbonate reduce cardiovascular events in patients on hemodialysis?



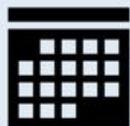
273 dialysis facilities in Japan



2374 pts



≥ 1 risk factors for vascular calcification



Nov 2011 to July 2014



Primary Outcome

Composite cardiovascular event rate



4.80



4.30

Difference 0.5 per 100 person-years
HR 1.11 [95% CI 0.88 to 1.41]

Treatment of hyperphosphatemia with Lanthanum carbonate did not reduce composite cardiovascular events



1:1



Lanthanum carbonate

750 mg/d

n = 1154



Calcium carbonate

3000 mg/d

n = 1155



Serum phosphate level 3.5 - 6.0 mg/dL

Secondary Outcomes



All-cause death rate

Difference 0.43 per 100 person-years
HR 1.10 [95% CI 0.88 to 1.37]



Cardiovascular death rate

Difference 0.61 per 100 person-years
HR 1.51 [95% CI, 1.01 to 2.27]

Effect of Treating Hyperphosphatemia With Lanthanum Carbonate vs Calcium Carbonate on Cardiovascular Events in Patients With Chronic Kidney Disease Undergoing Hemodialysis. JAMA. 2021;325(19):1946-1954.

VA by Missy Hanna
@dr_missyhanna



#NephJC

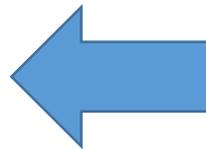
HiLo Studie

- 1. Hi group: Target serum phosphate ≥ 6.5 mg/dl (2.1mmol/L)
- 2. Lo group: Target serum phosphate ≤ 5.5 mg/dl (1.8 mmol/L)

Reached phosphate difference: 0,8mg/dl (0,26mmol/l)

Outcome after a median follow-up of 1.4 years:

57 deaths (16%) in the Hi group
96 deaths (21%) in the Lo group.



Total hospitalizations similar: 720 Hi group vs 685 Lo group.

Is a lower versus higher phosphate target beneficial to patients undergoing hemodialysis? The HiLo Trial



US In-center maintenance hemodialysis patients



Goal to enroll 4400 patients



Transitioned to individual randomization due to baseline imbalances in phosphate



Stopped early due to insufficient enrollment and inadequate separation

Primary hierarchical composite outcome (all-cause death and hospitalization) did not differ:
Win Ratio for Hi versus Lo targets was 0.97 (95% CI, 0.55-1.71)

15
P
Phosphorus

All Cause Mortality 
(per 100 patient years)

All Cause Hospitalization 
(per 100 patient years)



High Phosphate Target (≥ 6.5 mg/dl)
352 patients

11

134

HR .76
(95% CI, 0.48- 1.20)

IR 1.36
(95% CI, 1.11- 1.67)



Low Phosphate Target (< 5.5 mg/dl)
441 patients

13

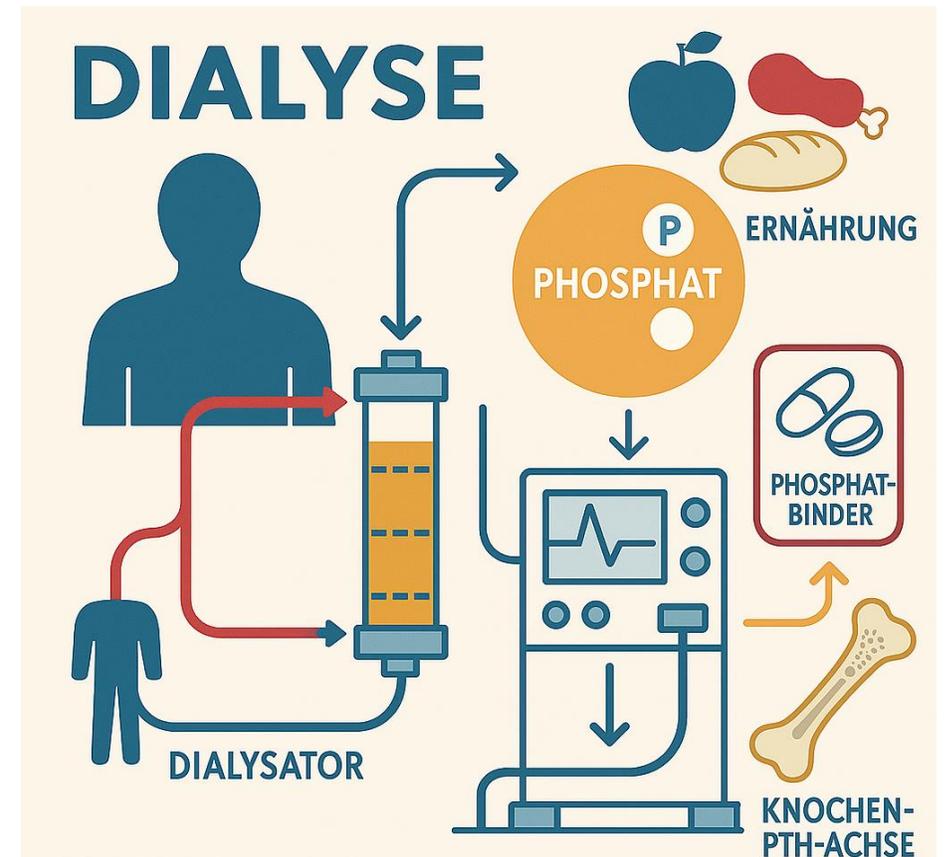
96

Conclusion: Insufficient enrollment and inadequate phosphate separation (<1 mg/dl) between groups precluded inferences about the effects of phosphate targets on clinical outcomes.

Edmonston D, Isakova T, Dember LM, et al. Higher versus lower phosphate targets in hemodialysis: The HiLo trial. *Journal of the American Society of Nephrology*. 2025 Jul 10.

Phosphatsenkung bei Dialysepatienten ist keine evidenzbasierte Intervention.

Es ist „Expert Opinion“ at best....



Spironolacton bei Dialysepatienten

The secret weapon



Spirolacton bei Hämodialysepatienten

Journal of the American College of Cardiology
© 2014 by the American College of Cardiology Foundation
Published by Elsevier Inc.

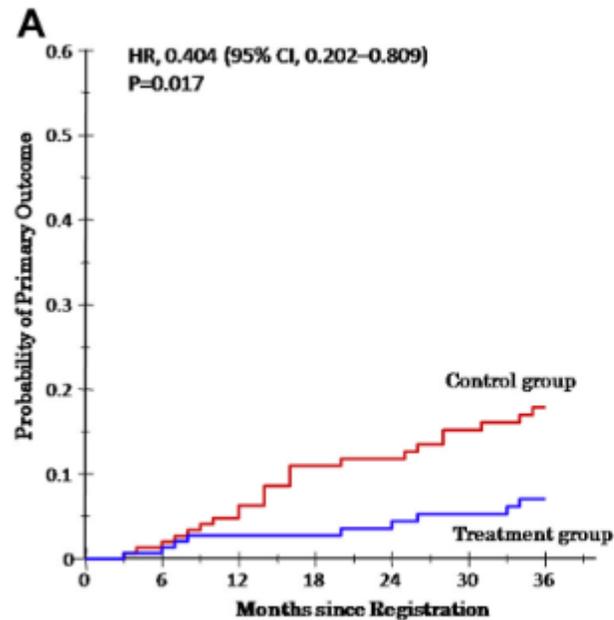
Vol. 63, No. 6, 2014
ISSN 0735-1097/36.00
<http://dx.doi.org/10.1016/j.jacc.2013.09.056>

Spirolactone Reduces Cardiovascular and Cerebrovascular Morbidity and Mortality in Hemodialysis Patients

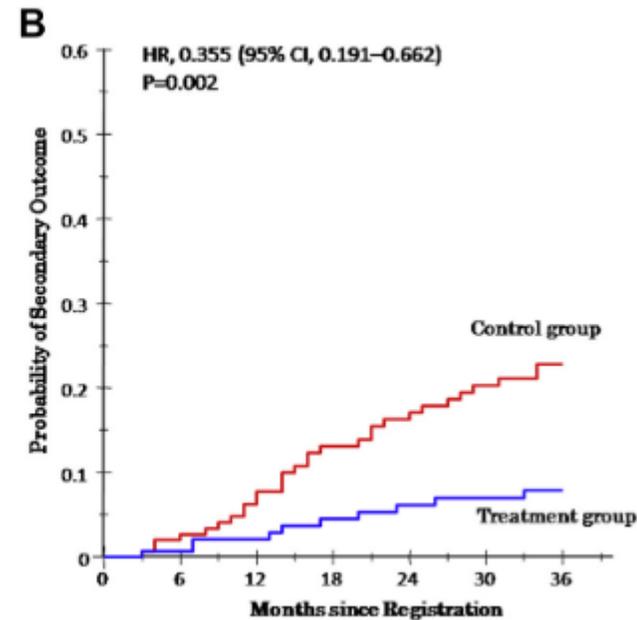
CME

Yoshihiro Matsumoto, MD,* Yasuo Mori, MD,† Shinji Kageyama, MD,‡ Kazuo Arihara, MD,§
Toshikazu Sugiyama, MD,|| Hiromichi Ohmura, MD,¶ Toru Yakushigawa, MD,†
Hatsumi Sugiyama, MD,|| Yasushi Shimada, MD,* Youichi Nojima, MD,* Nobuo Shio, MD‡

A 3-year randomized trial with 309 oligoanuric HD patients



composite of death from CCV events or hospitalization for CCV events



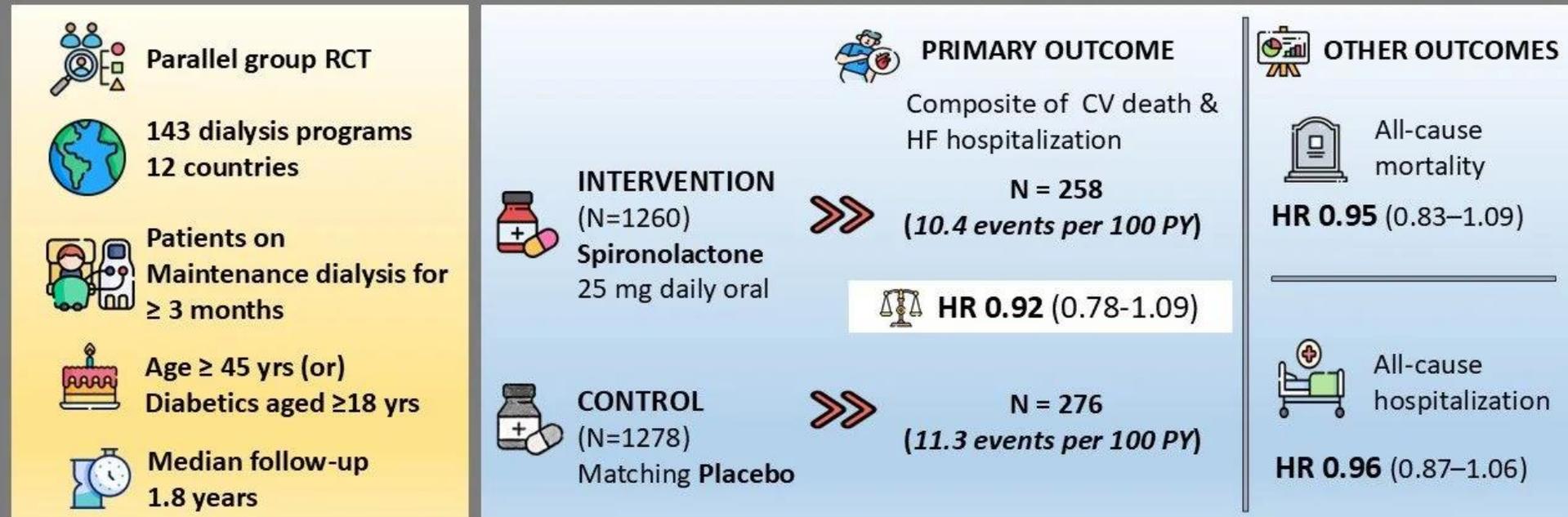
death from all causes

Klinikum | **St.GEORG**

Spironolacton bei Hämodialysepatienten: 2025

(ACHIEVE): an international, parallel-group, randomised controlled trial

Does spironolactone lower the risk of heart failure hospitalization and cardiovascular death in patients on maintenance dialysis?



NOTE: The trial was stopped early for *futility* after a planned interim analysis of 75% of the expected primary outcome events.

Conclusion: Among patients receiving maintenance dialysis, spironolactone 25 mg daily orally did not reduce the composite outcome of cardiovascular mortality and heart failure hospitalization compared with placebo.

Spironolactone versus placebo in patients undergoing maintenance dialysis (ACHIEVE): an international, parallel group, randomised controlled trial.

M Walsh et al., *Lancet* 2025

VA by Akshaya Jayachandran, MD DM



@nephromommyakshu.bsky.social



@DrAkshayaJ

St.GEORG

KFRE: Kidney failure risk equation



THE KIDNEY FAILURE RISK EQUATION

Find out your real risk of kidney failure



KIDNEY FAILURE
RISK CALCULATOR

FACTS &
CHRONIC KIDN

KIDNEY FAILURE RISK CALCULATION

If you don't have the information required below talk to your doctor.

Age (Yrs)	Sex	Region
<input type="text"/>	Select ▾	Select ▾
GFR (mL/Min/1.73M ²)	Urine Albumin: Creatinine Ratio	Units
<input type="text"/>	<input type="text"/>	<input type="text"/>

NEXT

ASSESSMENT

STAGE 3

MODERATE DECREASE IN FUNCTION



11.05 % 36.47 %

Risk thresholds used in health systems include:

- 3-5 % over 5 years for referral to a kidney doctor
- 10 % over 2 years for team based care (Kidney Doctor, Nurse, Dietician, Pharmacist)
- 20-40 % over 2 years for planning a transplant or fistula

Kidney Failure Risk Equation performance according to the etiology of chronic kidney disease in the CKD-CAREMEAU Cohort

ORIGINAL ARTICLE

Kidney Failure Risk Equation performance according to the etiology of chronic kidney disease in the CKD-CAREMEAU cohort

Julien Prouvot^{1,2}, Pedram Ahmadpoor¹, Edouard Clemmer¹, Florian Garo¹, Emilie Pambrun¹, Sylvain Cariou¹, Pascal Reboul¹, Ziyad Messikh¹ and Olivier Moranne^{1,2}

¹Service Néphrologie Dialyse Apherese, Hopitale Universitaire de Nimes, France and ²IDESP Université de Montpellier, France

Objective was to evaluate the performance of the KFRE score, taking chronic kidney disease etiology into account.

Methods



French hospital



CKD of any etiology and eGFR



5-year follow-up

Results



N = 3191



Median age 71 [61–80] years

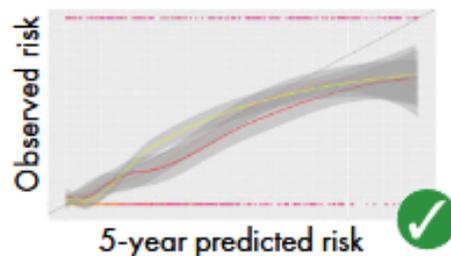


382 RRT

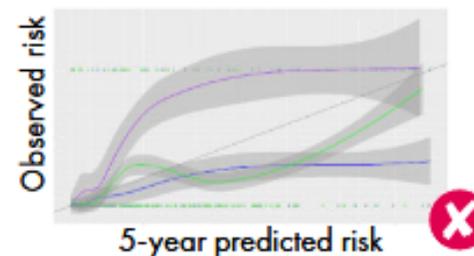
Discrimination: AUC 0.83–0.94, unchanged by etiology

Calibration: 2 groups according to etiology

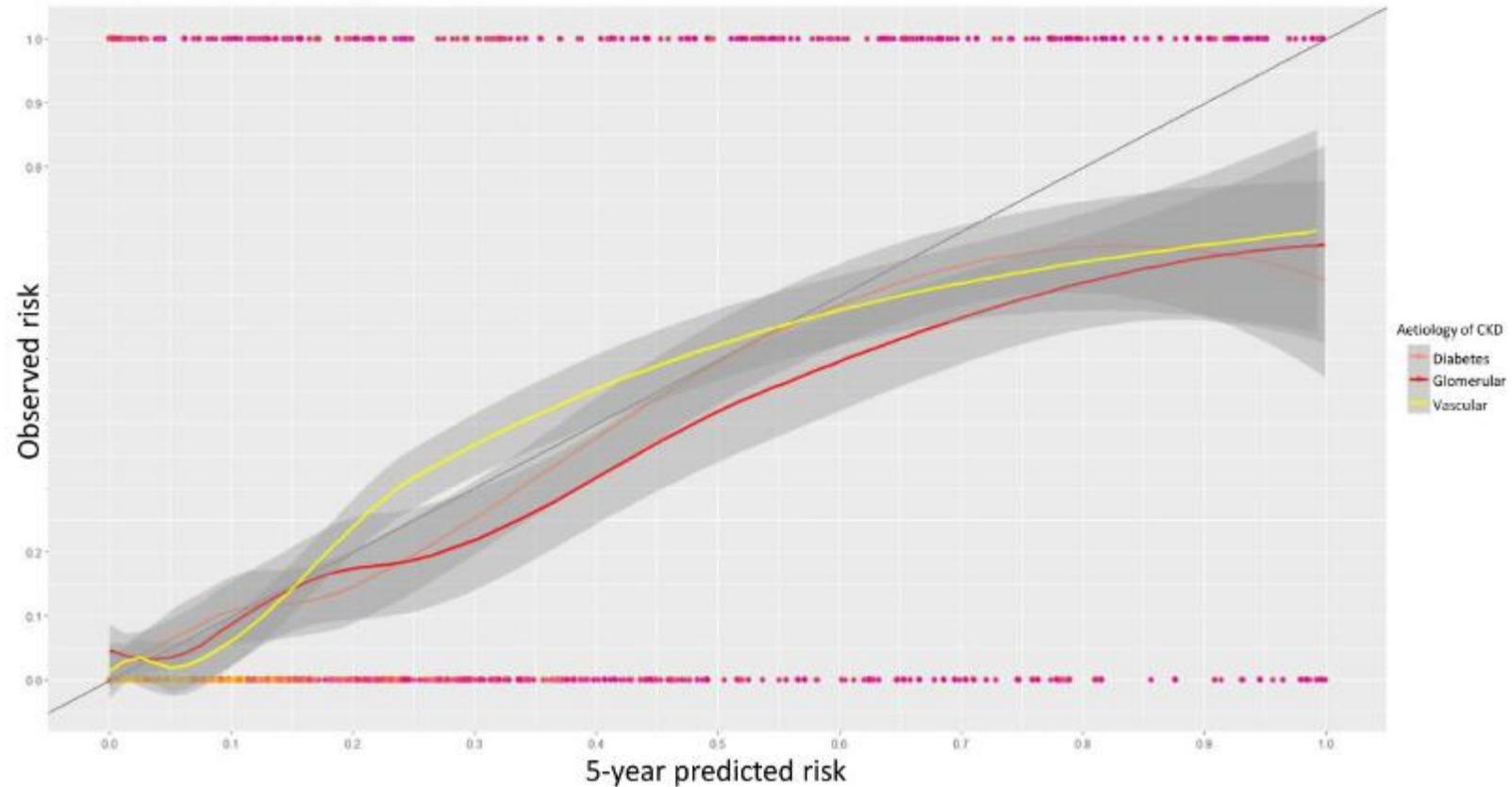
Vascular, glomerular, diabetes



Polycystic, tubulointerstitial, unclassified



Calibration of the KFRE score according to the etiology of CKD



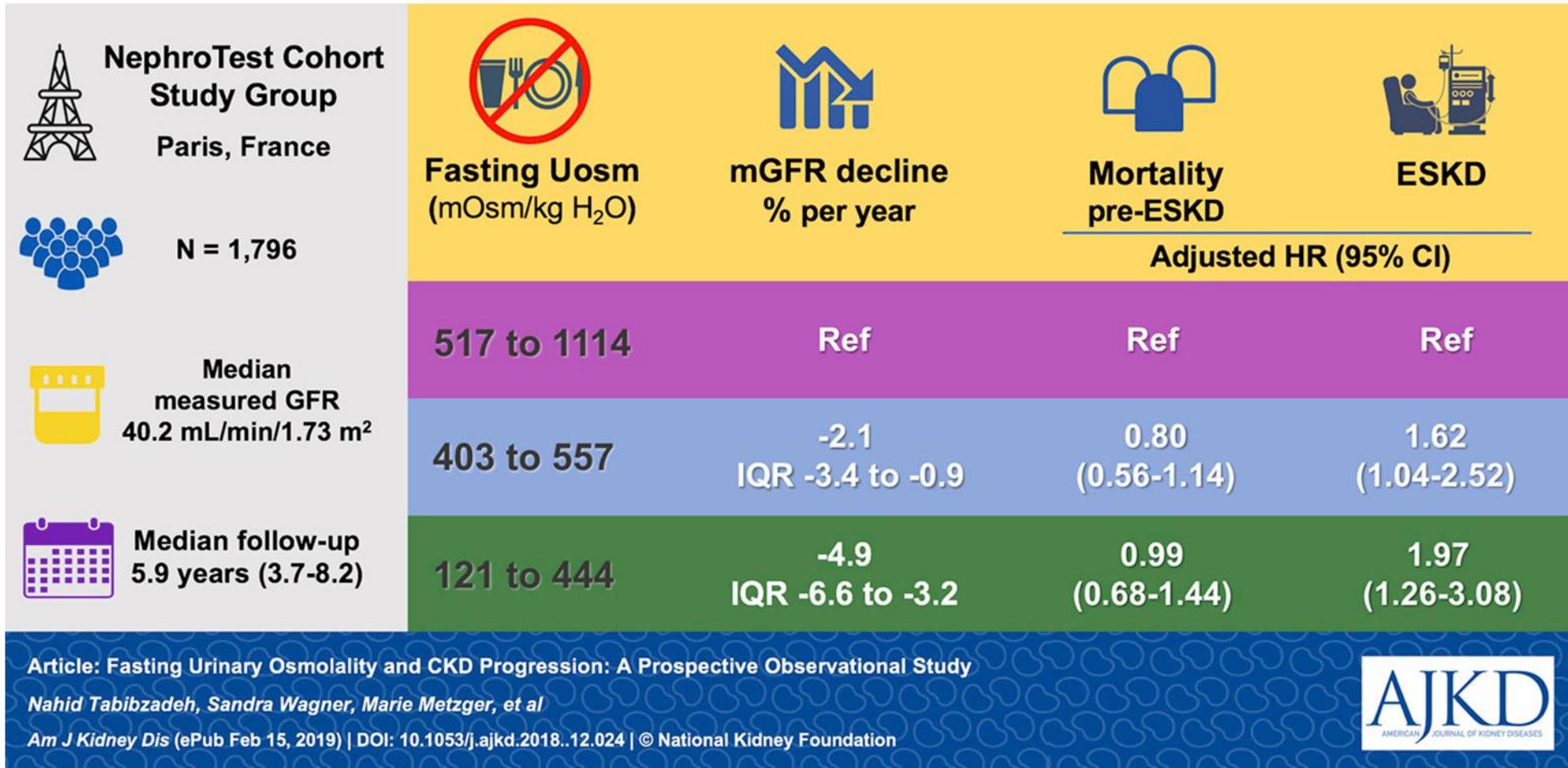
Risikoeinschätzung Progression CKD

UACR, eGFR, DKK3, CKD273, TIMP1.....

Is there more ?

More easy (and cheap) stuff out there?

Urinary Osmolality, CKD Progression, and Mortality



Fasting urine osmolality and risk of kidney disease progression in patients with type 2 diabetes

Does fasting urine osmolality provide prognostic value for kidney disease progression in type 2 diabetes?

Methods

Fasting urine osmolality measured in 2 cohorts with type 2 diabetes



Singapore SMART2D



French SURDIAGENE

Cox regression



Progression to end stage kidney disease (ESKD)



Rapid kidney function decline (RKFD)

Results

Association of fasting urine osmolality:



Singapore SMART2D Cohort (n=1711)



2.94*
(1.12–7.69)



1.47†
(0.95–2.28)



French SURDIAGENE Cohort (n=1097)



1.74*
(0.84–3.58)



1.84†
(1.06–3.19)

*adjusted hazard ratio, †adjusted odds ratio, (95% CI) for low vs. high tertile

Liu, J. J. et al.
NDT (2025)
@NDTSocial

Low level of fasting urine osmolality is associated with increased risk of kidney disease progression independent of conventional risk factors. This readily accessible biomarker may potentially improve risk-stratification for patients with type 2 diabetes.

Icodextrin user vs non_user in CHF



The effect of icodextrin on peritoneal dialysis patients with congestive heart failure: a time-varying exposure design and target trial emulation approach

Focus of study was icodextrin use and clinical outcomes in peritoneal dialysis (PD) patients with congestive heart failure (CHF)

Methods



Retrospective cohort study
1800 PD patients with CHF
2005–2022 in Taiwan



Icodextrin users vs. non-users

Target trial emulation
Cox regression

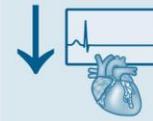
Results

Icodextrin use associated with lower:



All-cause mortality

0.16
(0.13–0.20)



Cardiovascular mortality

0.20
(0.13–0.30)



Sudden death

0.15
(0.11–0.19)



MACE*

0.68
(0.58–0.80)

*cardiovascular death, heart failure hospitalization, myocardial infarction and ischemic stroke
Adjusted hazard ratio (95% CI), vs. non-use

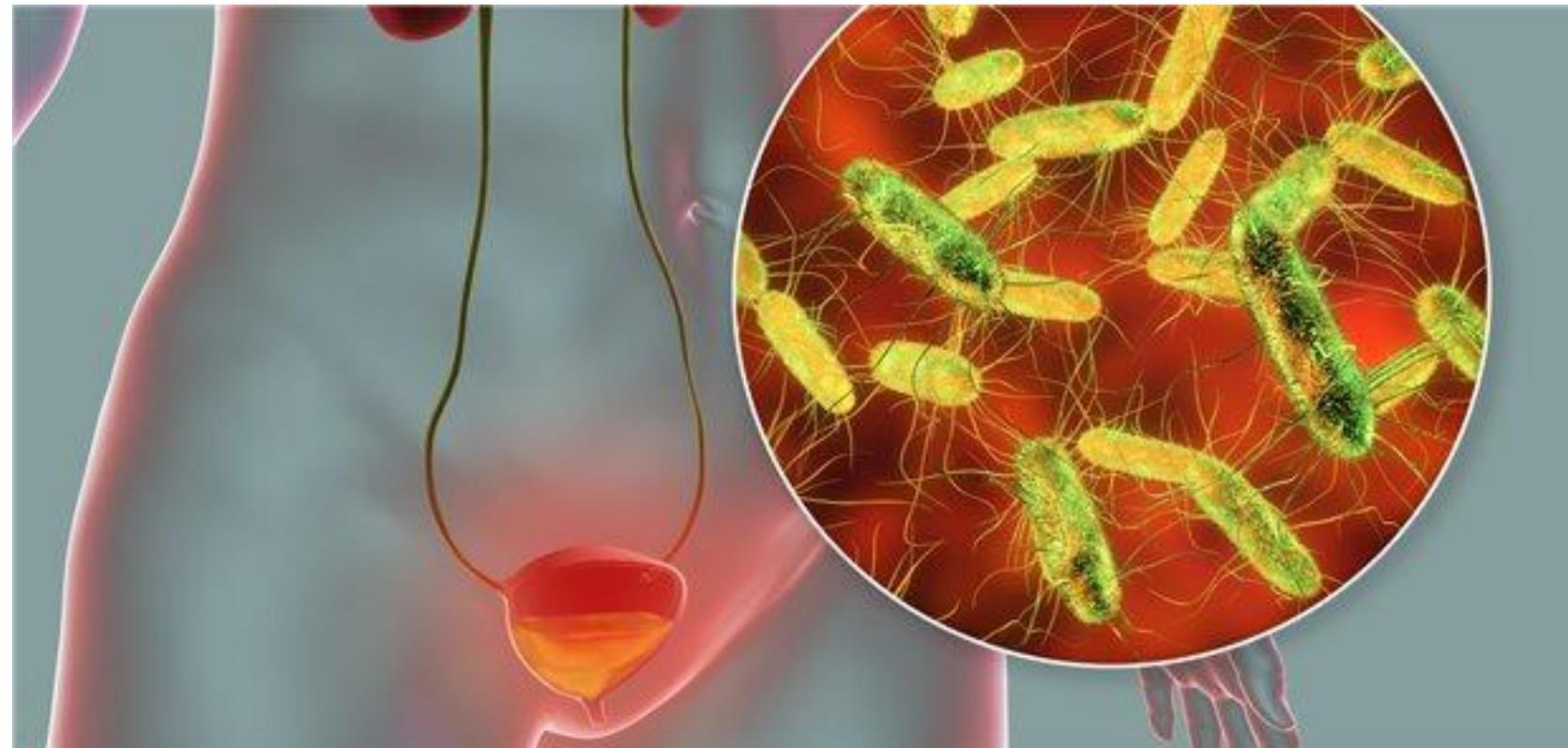
Ma, L. Y. et al.
NDT (2025)
@NDTSocial

In PD patients with CHF, icodextrin use was independently associated with better survival and cardiovascular outcomes. These findings support its preferential use in high-risk PD populations and warrant further prospective investigation.

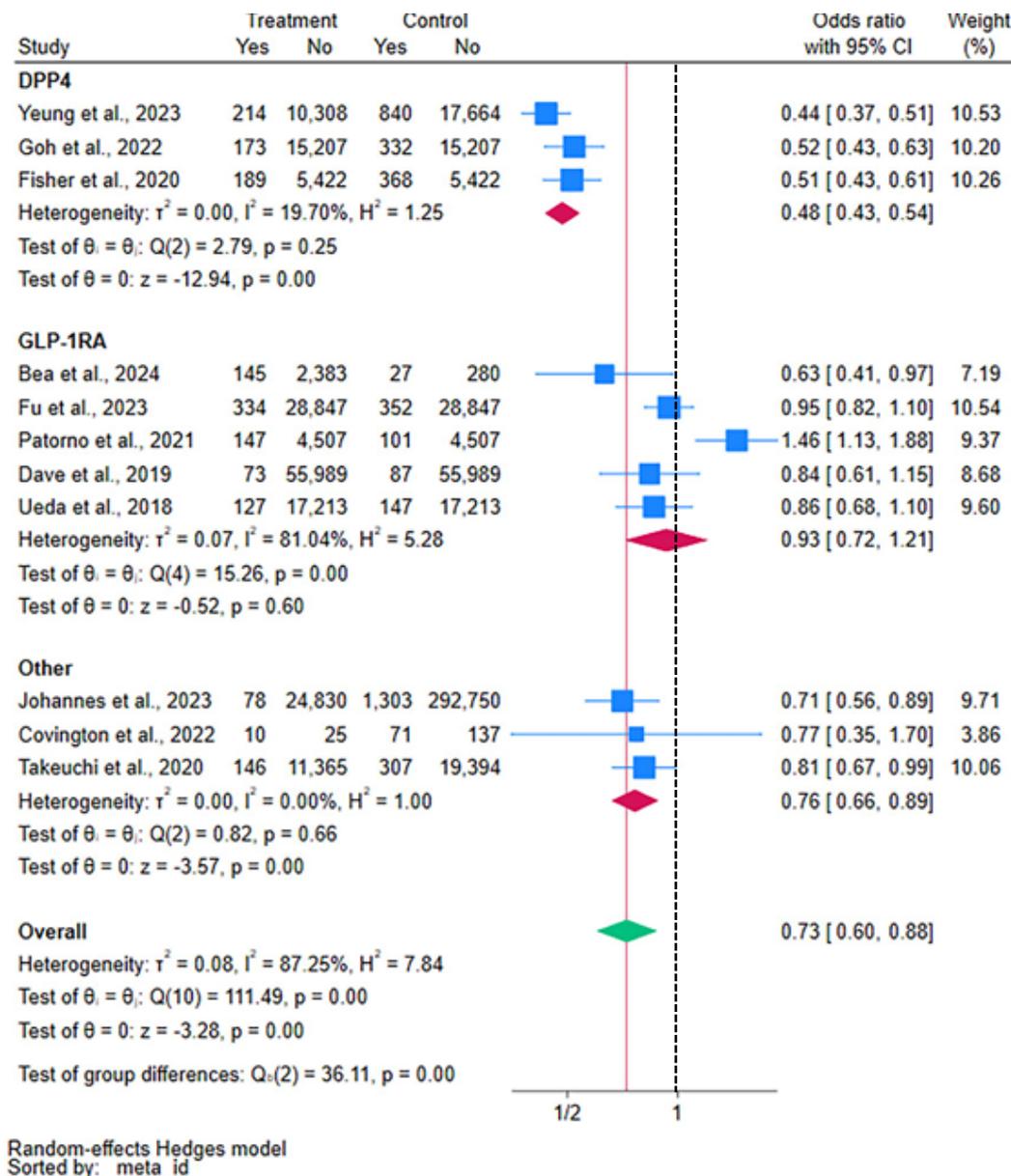
Icodextrin users also showed associations with reduced risks of encapsulating peritoneal sclerosis and transition to hemodialysis

Klinikum | **St.GEORG**

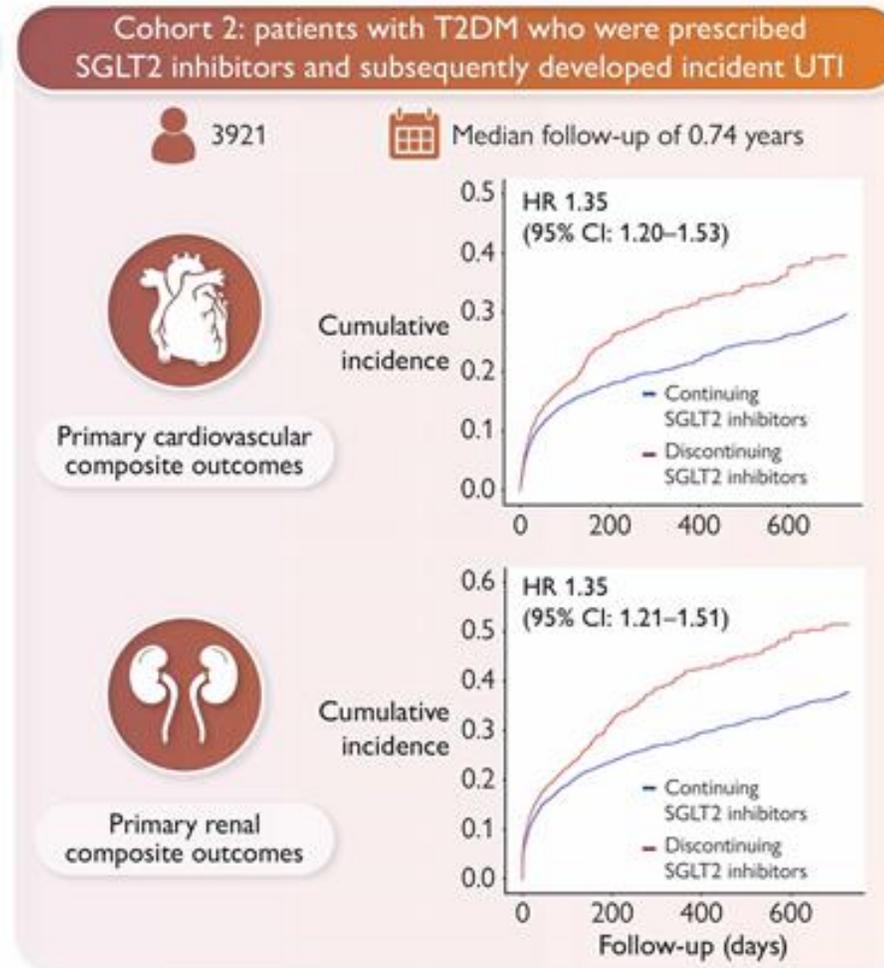
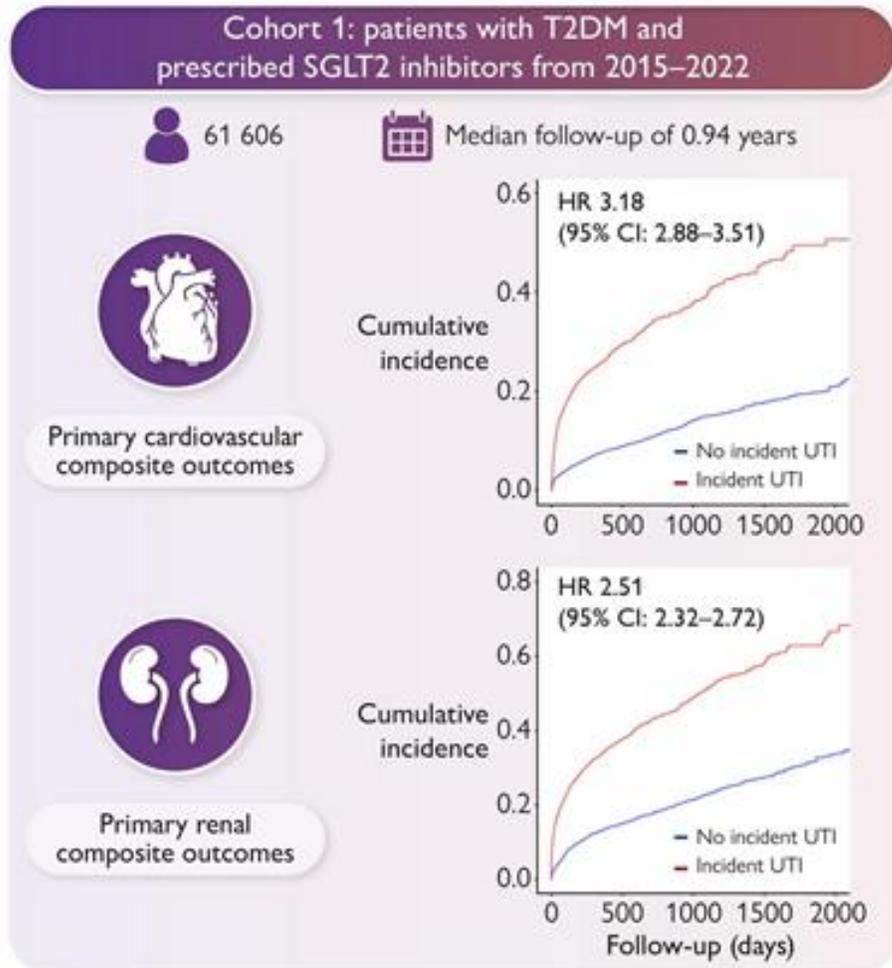
Sorge vor vermehrten Harnwegsinfektionen unter SGLT2i Therapie



Severe UTI risk in SGLT2 inhibitors compared with GLP1-RAs, non-GLP-1RAs, and DPP4 inhibitors.



Urinary tract infection and continuation of SGLT2i

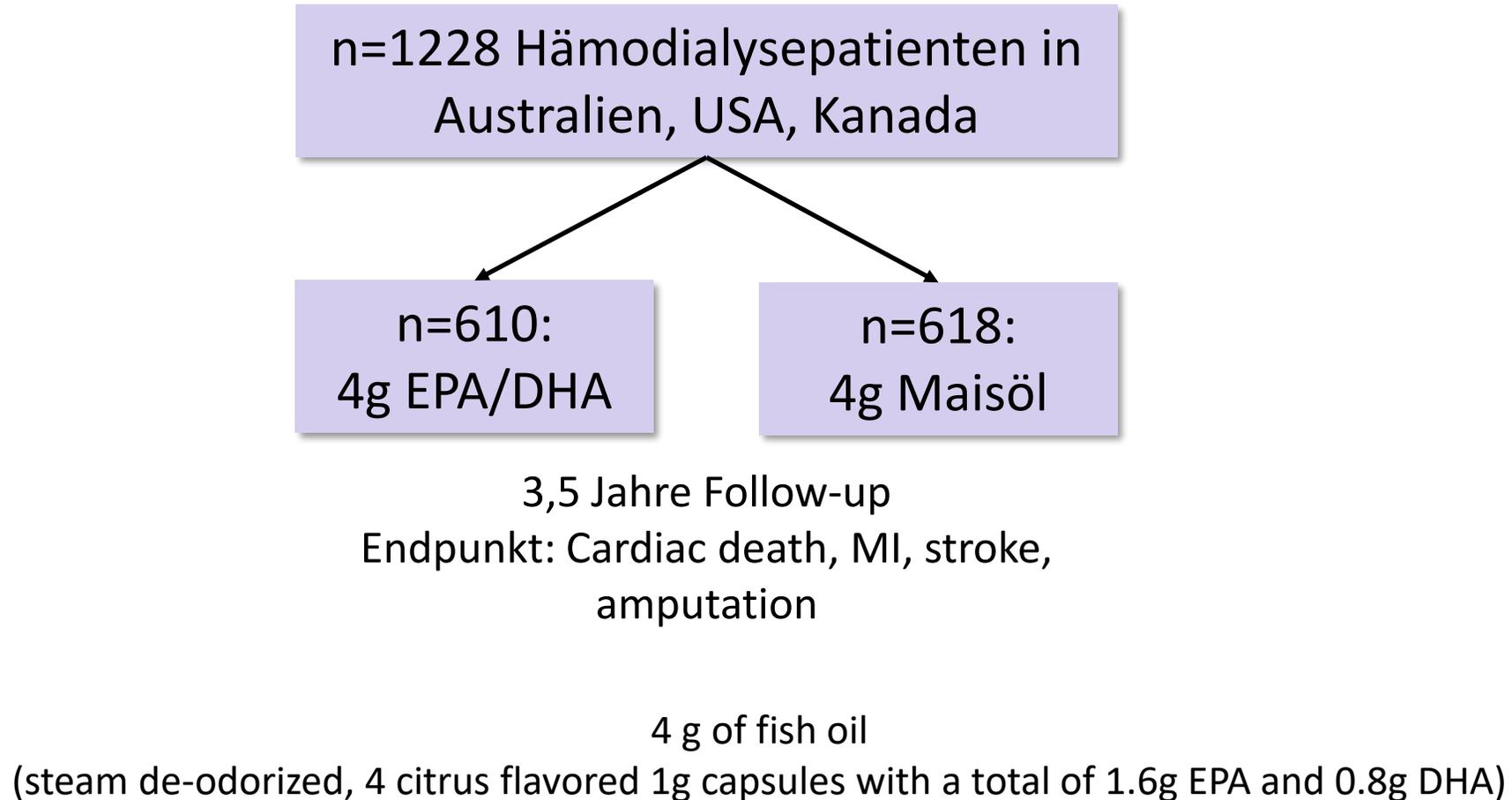


Following a UTI, 32.31% of patients discontinued SGLT2i

Discontinuation was associated with a higher cardiovascular (HR: 1.35) and renal (HR: 1.35) risks compared to continued use

Risk of recurrent UTI was similar (HR: 0.96, 95% CI: 0.22–4.29).

Fischöl (EPA/DHA) bei Dialysepatienten: Die PISCES Studie



Effectiveness of fish oil in controlling inflammation in adult patients undergoing hemodialysis: A systematic review and meta-analysis

Kaneez Fatima¹, Aysal Mahmood¹, Faiza Zafar Sayeed¹, Maryam Raza¹, Rahima Azam¹, Nazish Waris², Muttia Abdul Sattar³, Teesha Rani⁴, Zainab Wahaj⁵, Danisha Kumar¹, Simra Nadeem Siddiqui⁶

Hemodialysis patients, especially those with C-reactive protein > 5 mg/L, responded to fish oil supplementation to reduce their C-reactive protein level

Original Investigation

FREE

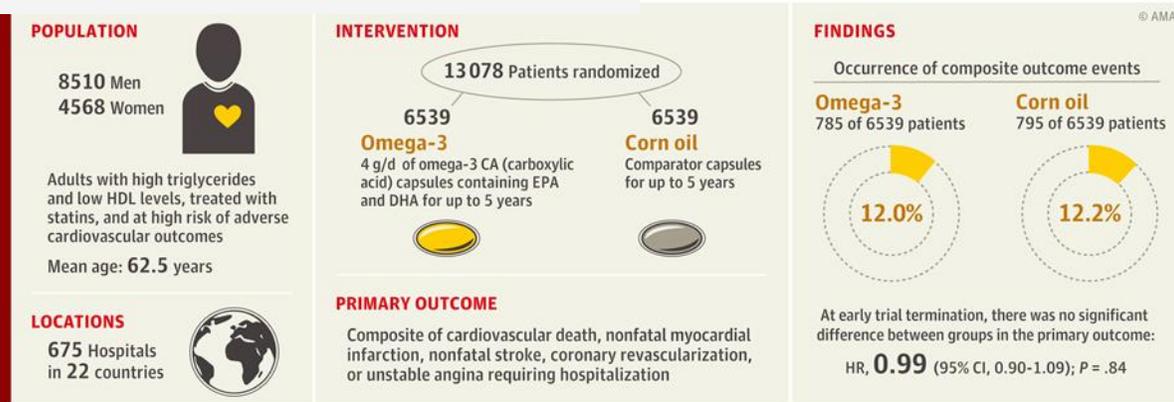
Effect of High-Dose Omega-3 Fatty Acids vs Corn Oil on Major Adverse Cardiovascular Events in Patients at High Cardiovascular Risk

The STRENGTH Randomized Clinical Trial

Stephen J. Nicholls, MBBS, PhD¹; A. Michael Lincoff, MD²; Michelle Garcia, RN, BSN, CCRC²; et al

> Author Affiliations | Article Information

lycerides, and low HDL cholesterol, does adding a carboxylic nt improve cardiovascular outcomes?
this omega-3 fatty acid formulation to reduce major adverse



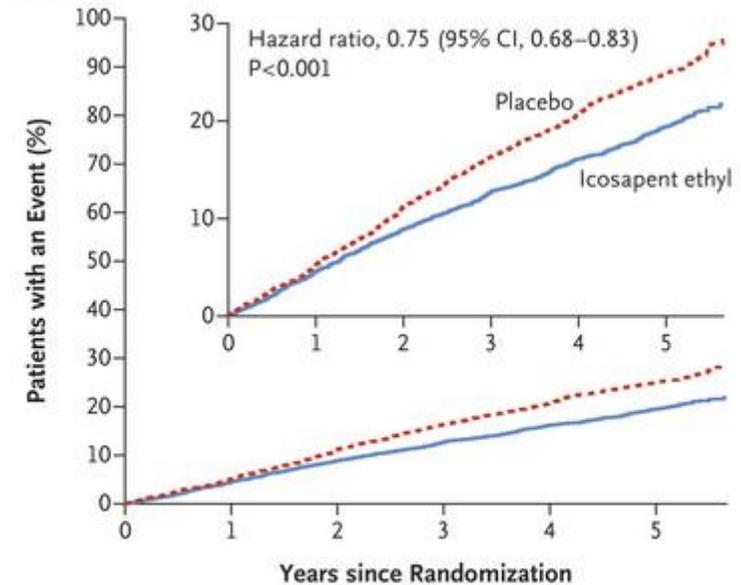
Nicholls SJ, Lincoff AM, Garcia M, et al. Effect of high-dose omega-3 fatty acids vs corn oil on major adverse cardiovascular events in patients at high cardiovascular risk: the STRENGTH randomized clinical trial. JAMA. Published online November 15, 2020. doi:10.1001/jama.2020.22258

Cardiovascular Risk Reduction with Icosapent Ethyl for Hypertriglyceridemia

Authors: Deepak L. Bhatt, M.D., M.P.H., P. Gabriel Steg, M.D., Michael Miller, M.D., Eliot A. Brinton, M.D., Terry A. Jacobson, M.D., Steven B. Ketchum, Ph.D., Ralph T. Doyle, Jr., B.A., +5, for the REDUCE-IT Investigators* Author Info & Affiliations

Published November 10, 2018 | N Engl J Med 2019;380:11-22 | DOI: 10.1056/NEJMoa1812792 | VOL. 380 NO. 1

A Primary End Point

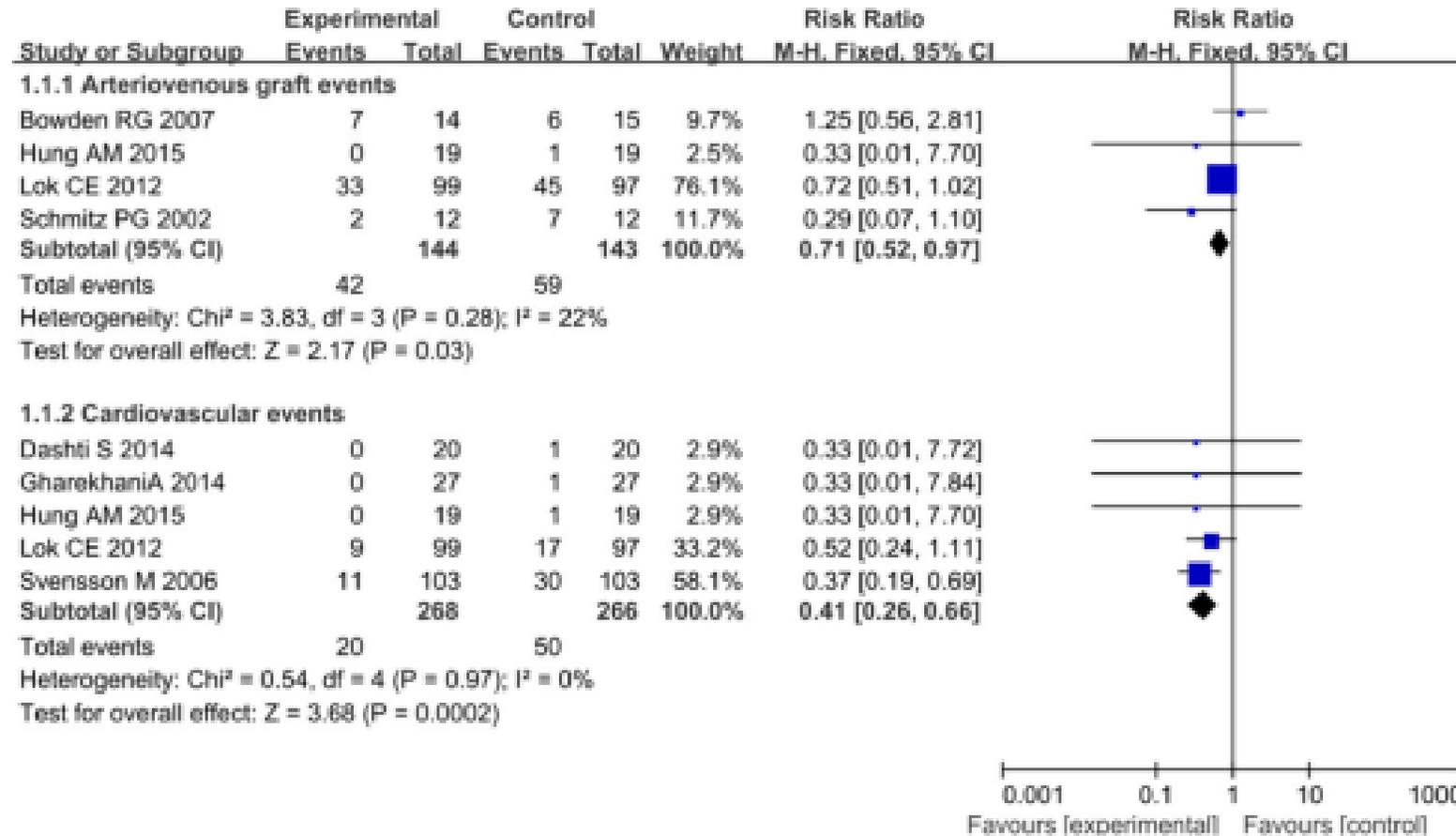


No. at Risk

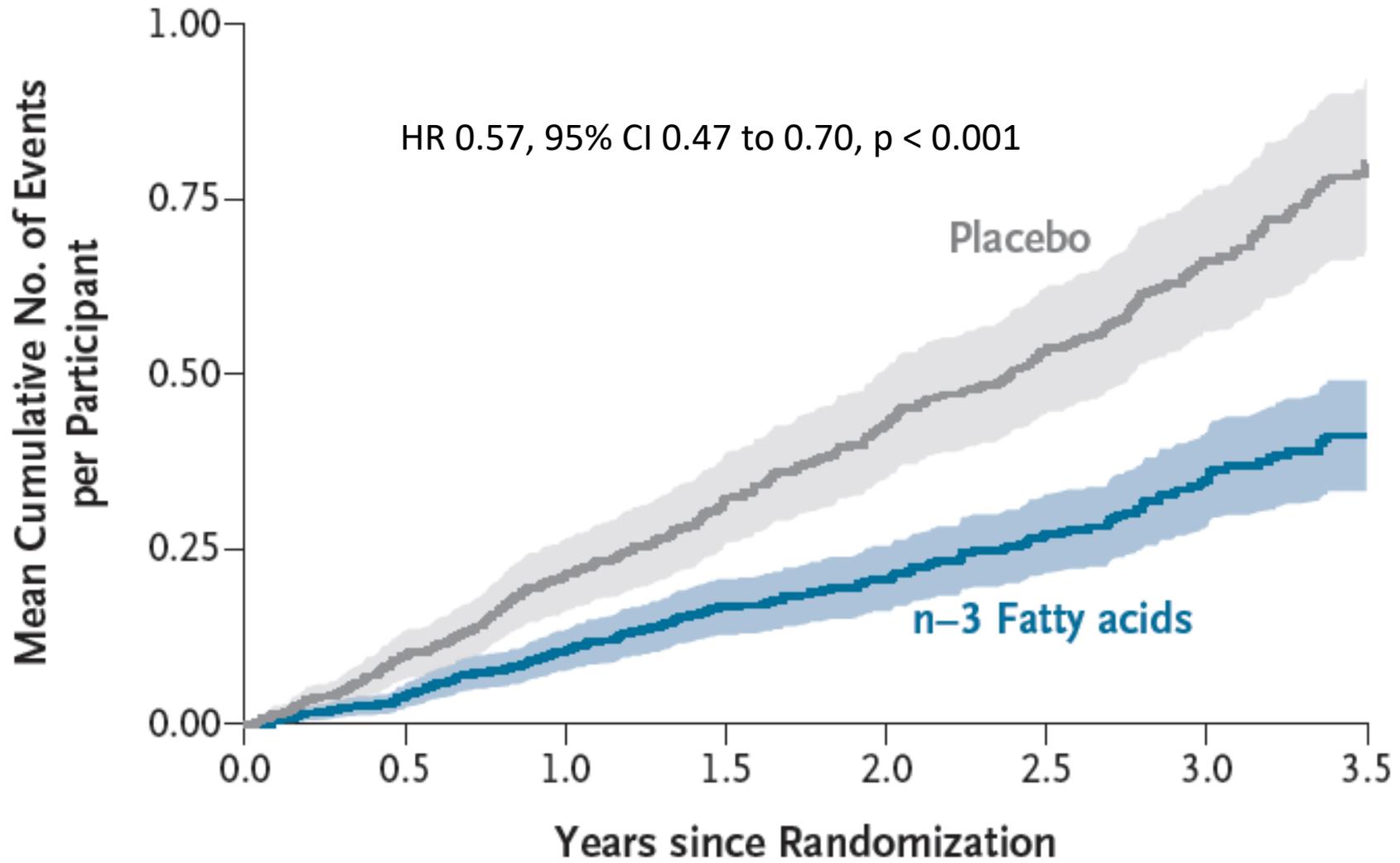
Placebo	4090	3743	3327	2807	2347	1358
Icosapent ethyl	4089	3787	3431	2951	2503	1430

composite end point of cardiovascular death, nonfatal myocardial infarction, nonfatal stroke, coronary revascularization, or unstable angina

effect of fish oil versus control on AV graft events and cardiovascular events in maintenance HD patients



A Serious Cardiovascular Events



St.GEORG

PISCES: Sek. Endpunkte

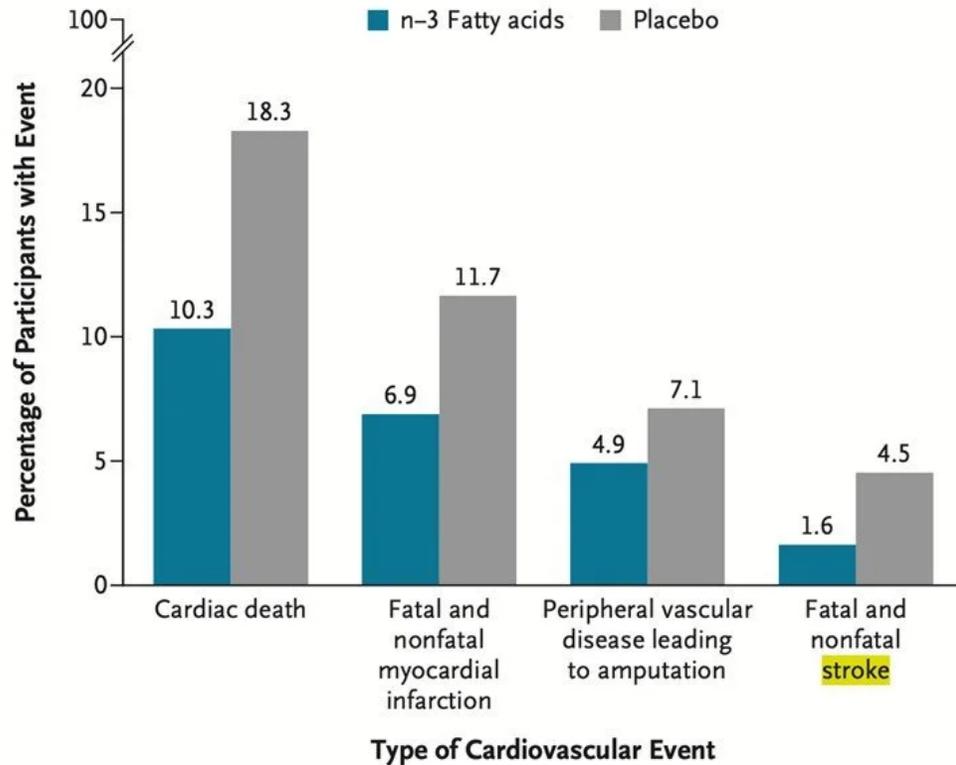


Table 2. Primary and Secondary End Points.*

End Point	Fish Oil		Placebo		Hazard Ratio (95% CI)†
	No. of Events	Rate <i>no. per 1000 patient-days</i>	No. of Events	Rate <i>no. per 1000 patient-days</i>	
Primary end point					
Primary end-point events among all participants	158	0.31	309	0.61	0.57 (0.47–0.70)
Primary end-point events in subgroups based on history of a cardiovascular event at baseline					
Previous cardiovascular event	81	0.43	164	0.91	0.50 (0.37–0.67)
No previous cardiovascular event	77	0.24	145	0.45	0.55 (0.40–0.76)
Secondary end points					
Primary end-point events plus noncardiac death	266	0.52	381	0.76	0.77 (0.65–0.90)
Death from any cause	175	0.34	195	0.39	0.89 (0.73–1.01)
Components of the primary end point					
Cardiac death	63	0.12	113	0.22	0.55 (0.40–0.75)
Fatal and nonfatal myocardial infarction	49	0.10	96	0.19	0.56 (0.40–0.80)
Peripheral vascular disease leading to amputation	35	0.07	66	0.13	0.57 (0.38–0.86)
Fatal and nonfatal stroke	11	0.02	34	0.07	0.37 (0.18–0.76)
First cardiovascular event or death from any cause	215	0.45	270	0.60	0.73 (0.61–0.87)

All cause death (HR 0.89, 95% CI 0.73 to 1.01)

THE “TOO GOOD TO BE TRUE” CAUTION

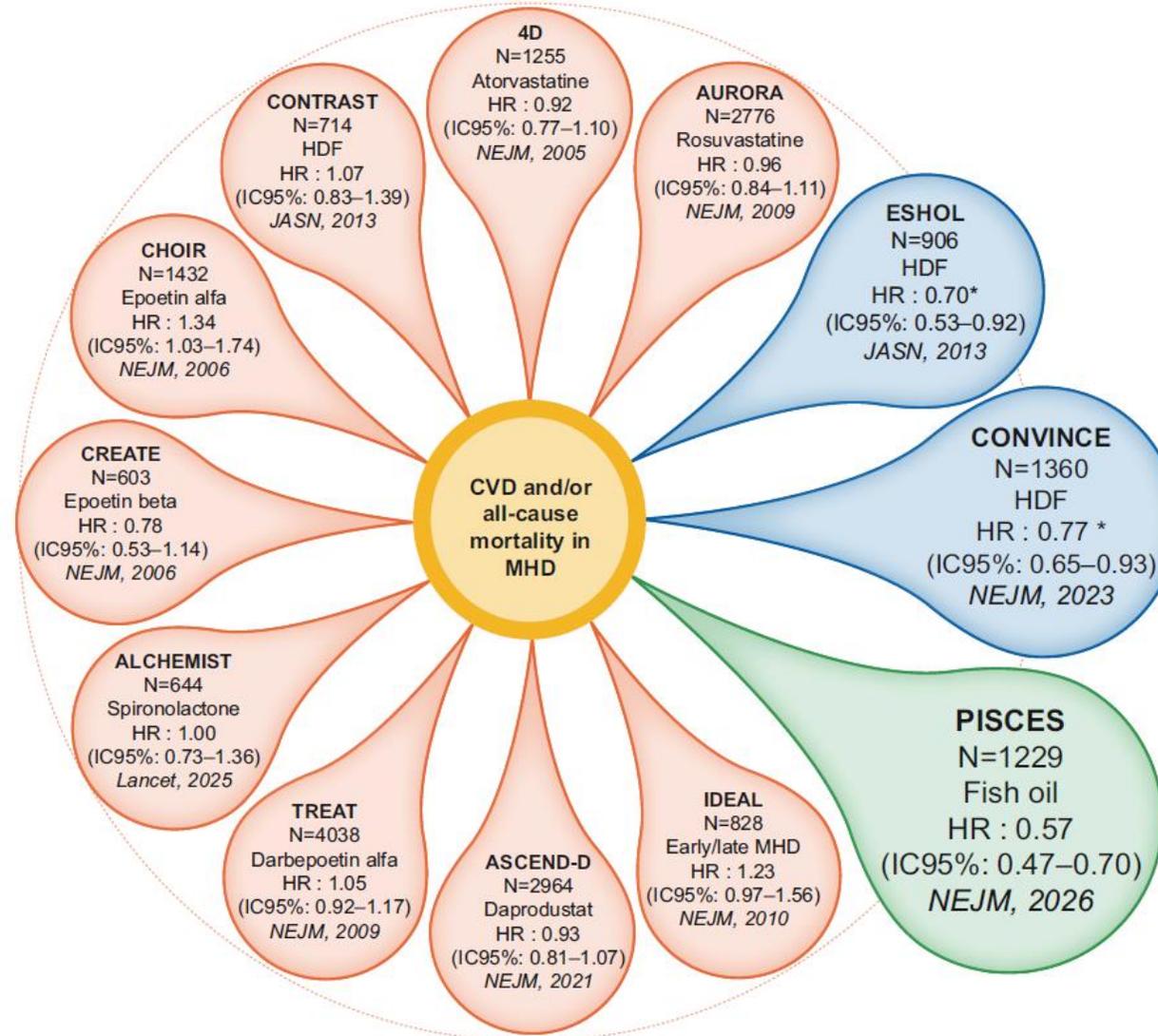


35% mehr Todesfälle (112 vs 82 aus nicht-kardiovaskulärer Ursache)

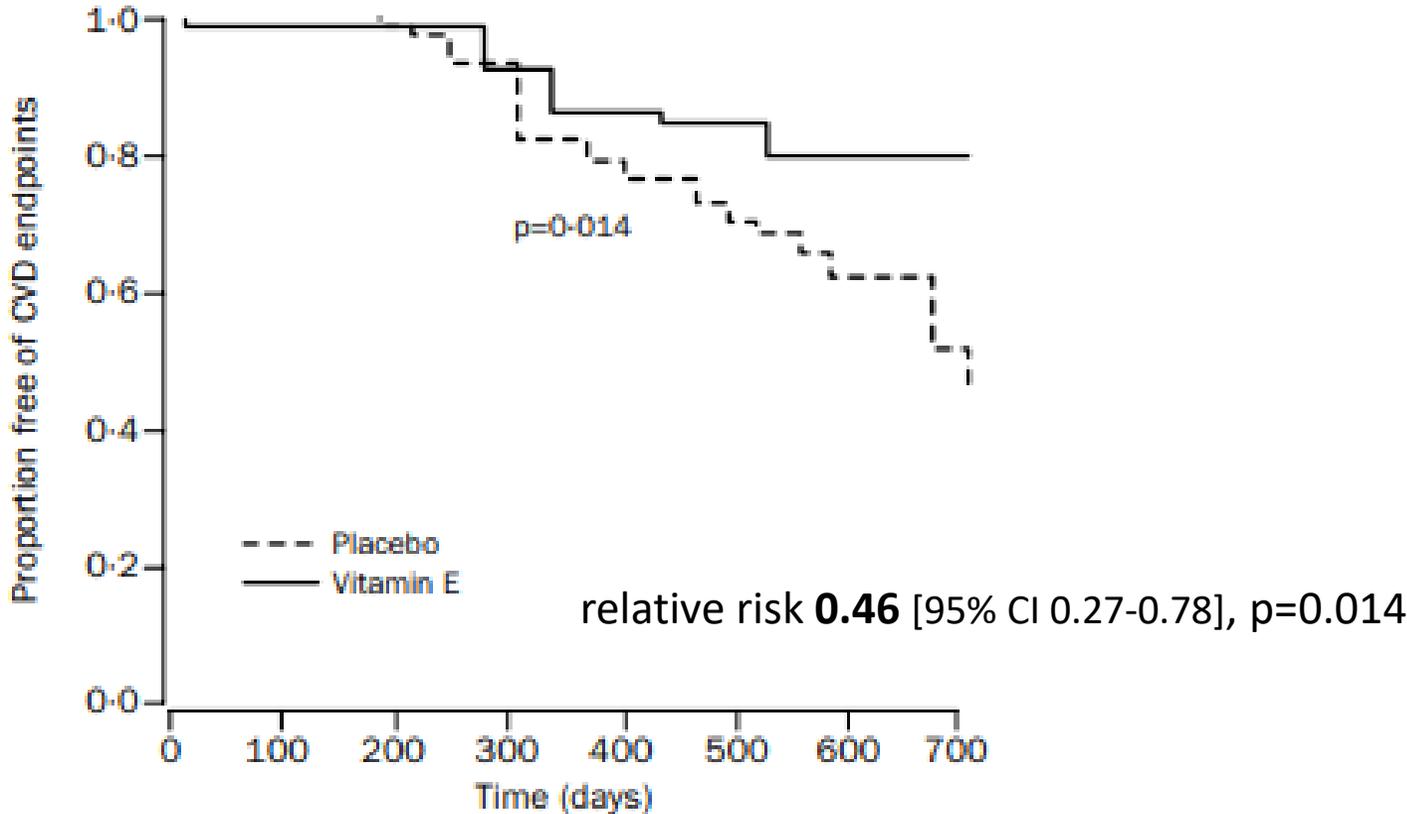
Aber: All cause death (HR 0.89, 95% CI 0.73 to 1.01)

Klinikum | **St.GEORG**

Outcomestudien in Dialysepatienten



**Haemodialysis patients with pre-existing cardiovascular disease:
RCT: 800 IU/day Vitamin E or placebo**



Number at risk

Placebo	99	97	86	74	67	60	59	57
Vitamin E	97	94	91	79	71	67	63	60

primary cardiovascular-disease endpoint
myocardial infarction (fatal and non-fatal), ischaemic stroke, peripheral vascular disease (excluding the arteriovenous fistula), and unstable angina

Wenn ich selbst an der Dialyse wäre....



OMEGA-3 PREMIUM 1500 HERZ + GEHIRN + ABWEHR

Hochdosiert
780 mg EPA / 495 mg DHA
1.500 mg Omega-3*
* Tagesdosis 2 Kapseln

Aus nachhaltigem Fischfang¹
Seefischöl "Friend of the Sea" zertifiziert
Details siehe Rückseite¹

Aus hochgereinigten Seefischölen.
Wertvolle Omega-3-Fettsäuren EPA und
DHA als Beitrag zur normalen Herz-
und DHA zur normalen Gehirnfunktion.

800 I. E. Vitamin D₃*
Vitamin D für den
Erhalt der Knochen
und die Funktion
des Immunsystems.

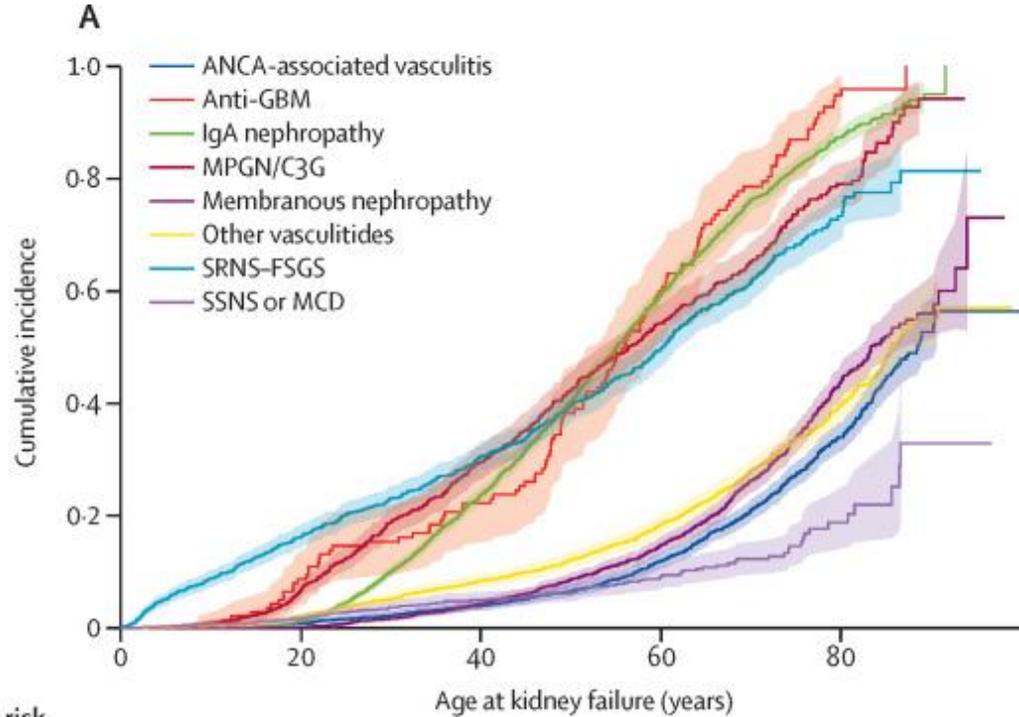
nur 2 Kapseln täglich
120 Kapseln

**PREMIUM
QUALITÄT**

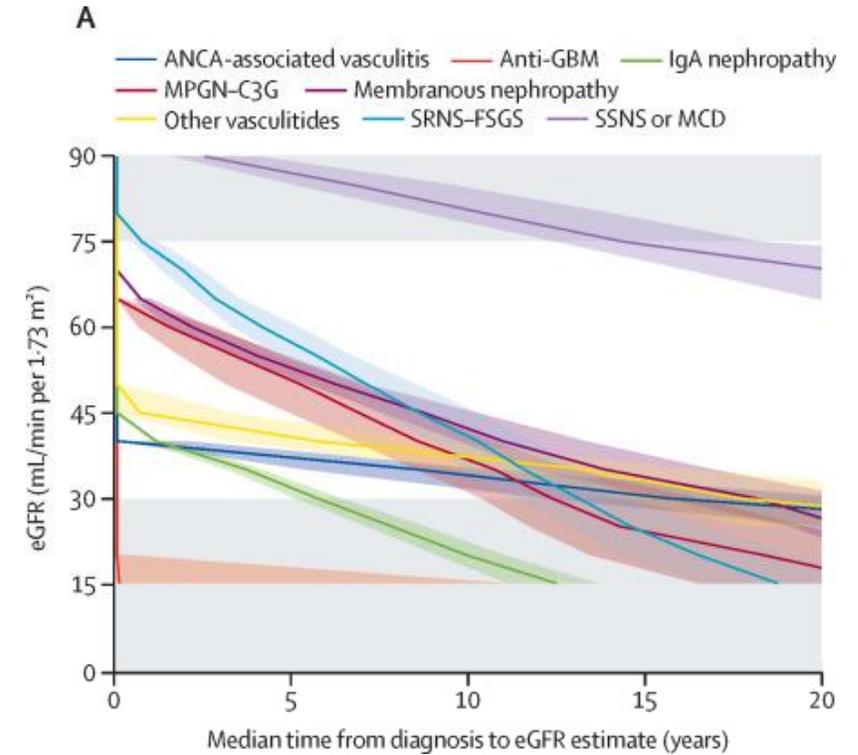
Feature	PISCES Fish Oil (Study)
Formulation	Ethyl esters of omega-3s
Daily total dose	4 g
EPA content (4 capsules)	1.6 g/day
DHA content (4 capsules)	0.8 g/day
EPA:DHA ratio	~2:1

cumulative incidence of kidney failure for glomerular (A) and cystic kidney diseases (B)

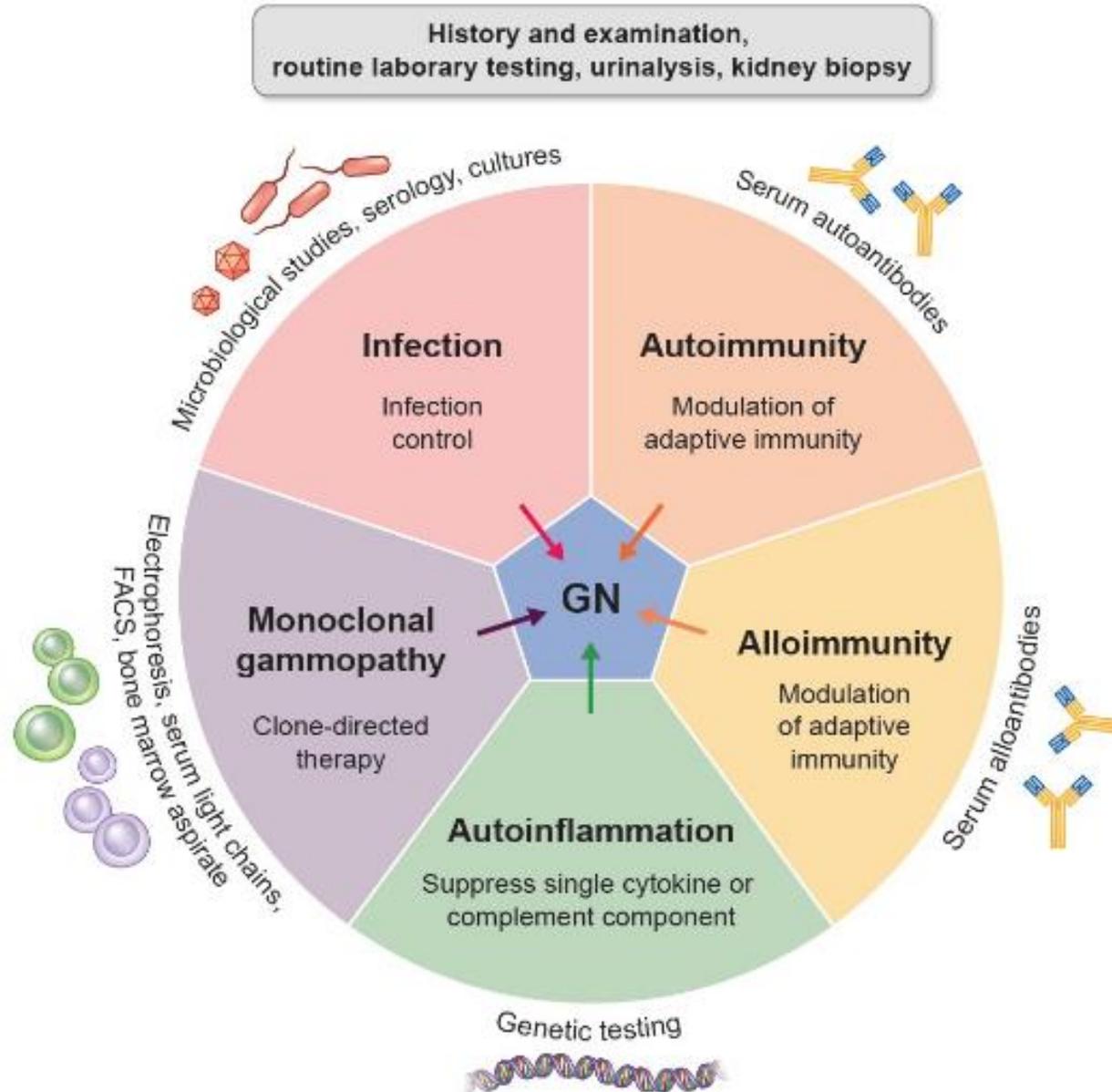
median time from diagnosis to eGFR value for glomerular



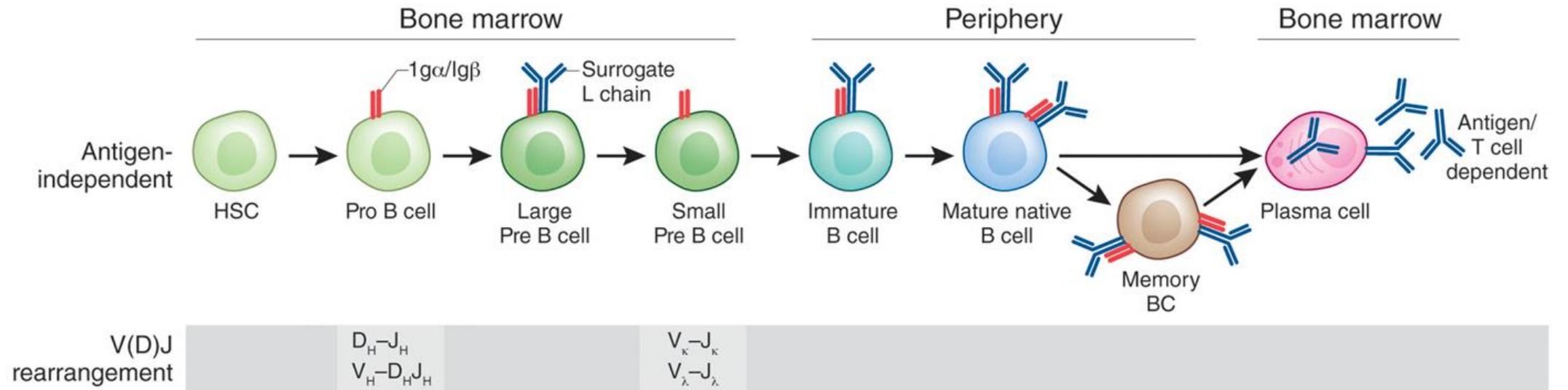
	0	20	40	60	80	100
Number at risk (number censored)						
ANCA-associated vasculitis	2375 (0)	2344 (10)	2183 (98)	1697 (413)	425 (1390)	
Anti-GBM	137 (0)	125 (0)	102 (5)	51 (7)	4 (13)	
IgA nephropathy	4147 (0)	4043 (51)	2741 (481)	959 (1193)	79 (1619)	
MPGN-C3G	1089 (0)	933 (85)	585 (228)	293 (332)	36 (481)	
Membranous nephropathy	2439 (0)	2425 (8)	2209 (126)	1535 (582)	281 (1514)	
Other vasculitides	2331 (0)	2062 (221)	1750 (405)	1286 (694)	319 (1427)	
SRNS-FSGS	1536 (0)	1094 (203)	727 (403)	364 (598)	40 (809)	
SSNS or MCD	1705 (0)	1092 (588)	715 (933)	373 (1252)	60 (1546)	



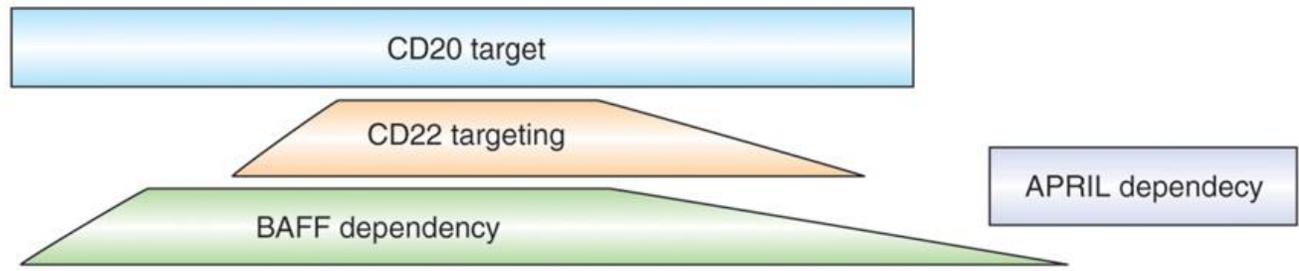
New classification of glomerulonephritis by pathogenesis



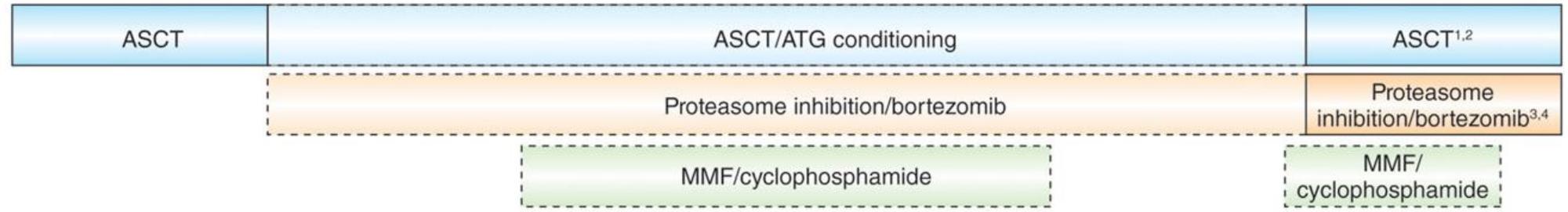
Interventions and their potential to target distinctly B lineage subsets and plasma cells



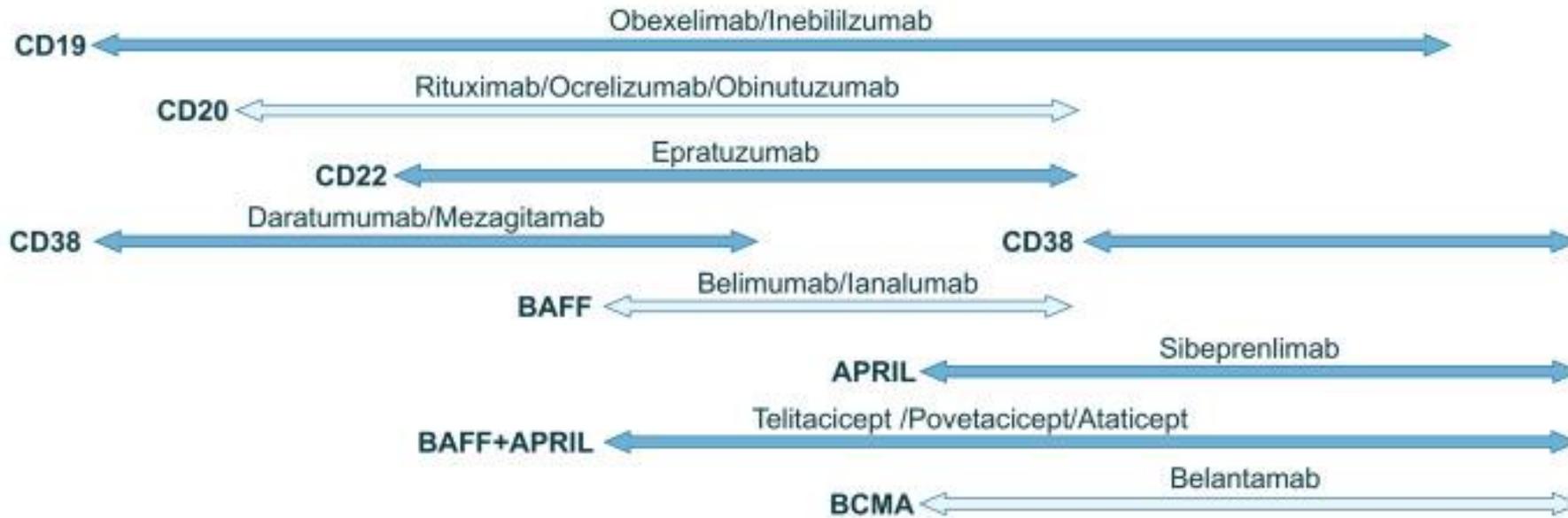
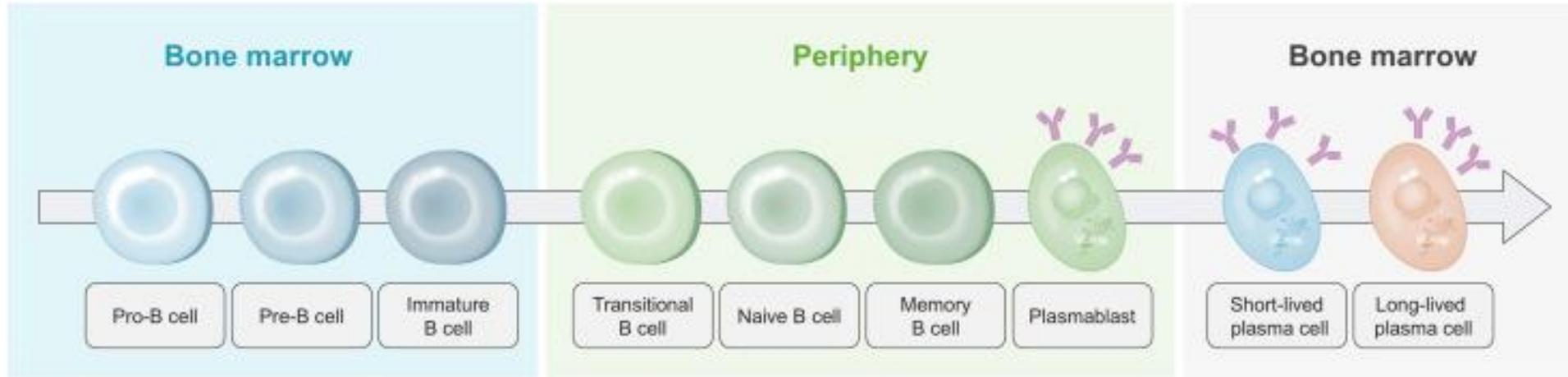
A



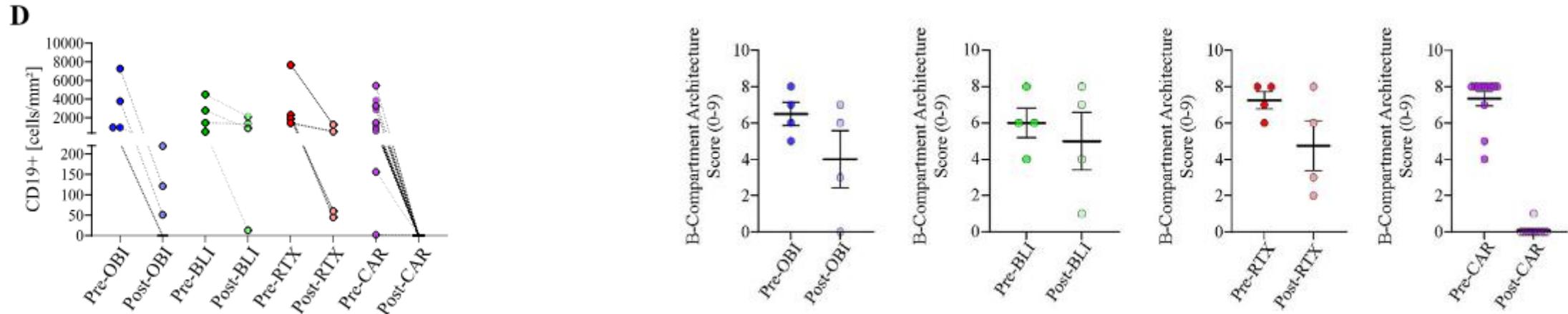
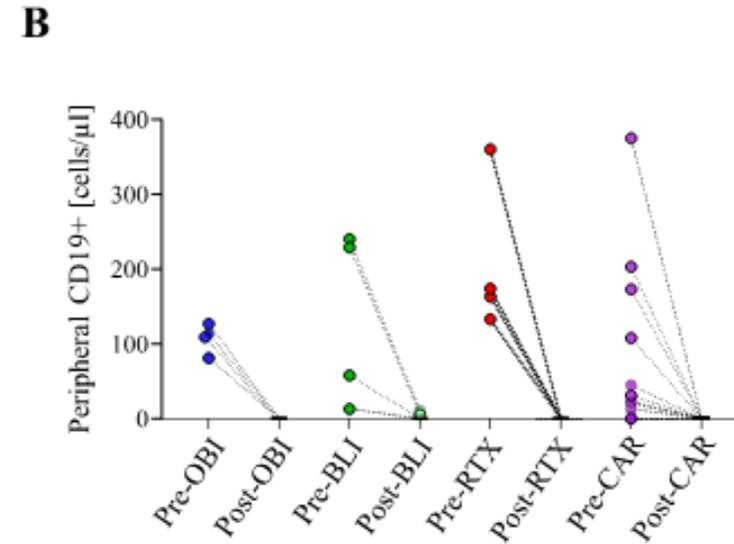
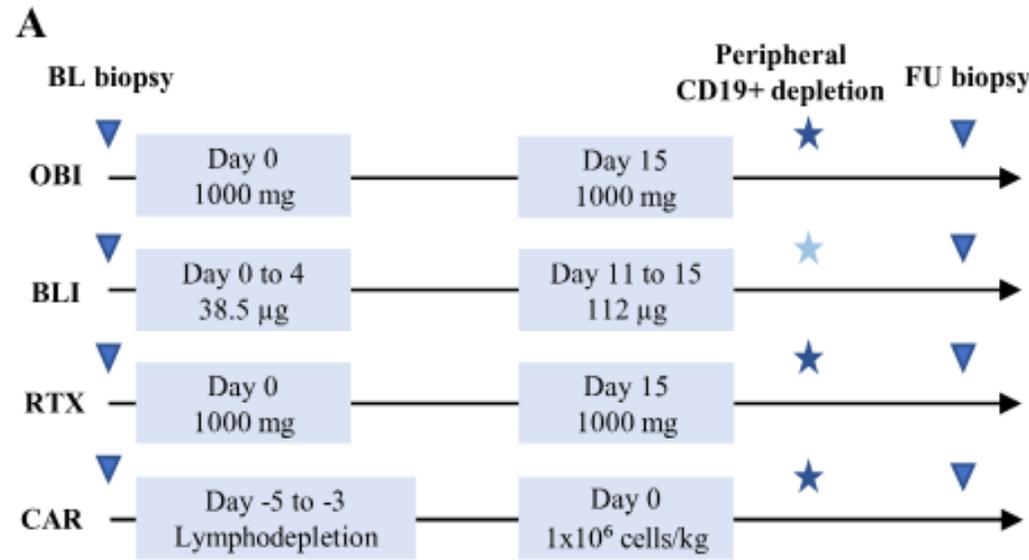
B



B-cell development and therapeutic targets



Effects of different B-cell-depleting strategies on the lymphatic tissue



Laufende CAR Zell Studien bei immun-vermittelten Nierenerkrankungen

Diseases	CAR-expressing cells	Targets	Status		
			Remain to explore	Anticipated	Recruiting
IgAN	CAR T 	CD19	NCT06690359		
MN	CAR T 	CD19	NCT06690359		
		BCMA-CD19	NCT06285279		
AAV	CAR T 	CD19	12 registered studies ^a		
		BCMA	NCT06277427		
		BCMA-CD19	NCT06285279	NCT06350110	
		CD19-CD20	NCT06462144		
	CAR NK 	CD19	NCT06614270		
		CD19	NCT06733935		
LN	CAR T 	CD19	19 registered studies ^b		
		BCMA	NCT06497387	NCT06277427	
		BCMA-CD19	NCT06681337	NCT05085418	
			NCT06497361	NCT06285279	NCT06350110
	CAR NK 	CD19	NCT06518668		
			NCT06377228		
Anti-GBM disease					



ATTENTION: Due to global market conditions, you may experience a delivery delay for your print issue of the New England Journal of Medicine website. We regret any print delays and are working to ensure all issues are delivered as soon as possible.

ORIGINAL ARTICLE



CD19 CAR T-Cell Therapy in Autoimmune Disease — A Case Series with Follow-up

Authors: Fabian Müller, M.D., Jule Taubmann, M.D., Laura Bucci, M.D., Artur Wilhelm, Ph.D., Christina Bergmann, M.D., Simon Völkl, Ph.D., Michael Aigner, Ph.D., ⁺²⁰, and Georg Schett, M.D. [Author Info & Affiliations](#)

Published February 21, 2024 | N Engl J Med 2024;390:687-700 | DOI: 10.1056/NEJMoa2308917 | **VOL. 390 NO. 8**

Copyright © 2024



[nature](#) > [nature medicine](#) > [articles](#) > [article](#)

Article | [Open access](#) | Published: 07 January 2026

CD19 CAR-T cells for treatment-refractory autoimmune diseases: the phase 1/2 CASTLE basket trial

[Fabian Müller](#), [Melanie Hagen](#), [Andreas Wirsching](#), [Soraya Kharboutli](#), [Michael Aigner](#), [Simon Völkl](#), [Sascha Kretschmann](#), [Koray Tascilar](#), [Jule Taubmann](#), [Laura Bucci](#), [Maria Gabriella Raimondo](#), [Christina Bergmann](#), [Tobias Rothe](#), [Giulia Corte](#), [Carlo Tur](#), [Luis Muñoz](#), [Sebastian Böltz](#), [Louis Schuster](#), [Fabian Hartmann](#), [Panagiotis Garantziotis](#), [Silvia Spörl](#), [Ingrid Vasova](#), [Armin Gerbitz](#), [Bernd Spriewald](#), ... [Georg Schett](#)

+ Show authors

MEETING REPORT · [Volume 57, Issue 12](#), P2705-2709, December 10, 2024 · [Open Archive](#)

[Download Full Issue](#)

CAR T cells in autoimmune disease: On the road to remission

[Georg Schett](#) ^{1,2} · [Carl H. June](#) ³

[Affiliations & Notes](#) ▾ [Article Info](#) ▾

Vielen Dank

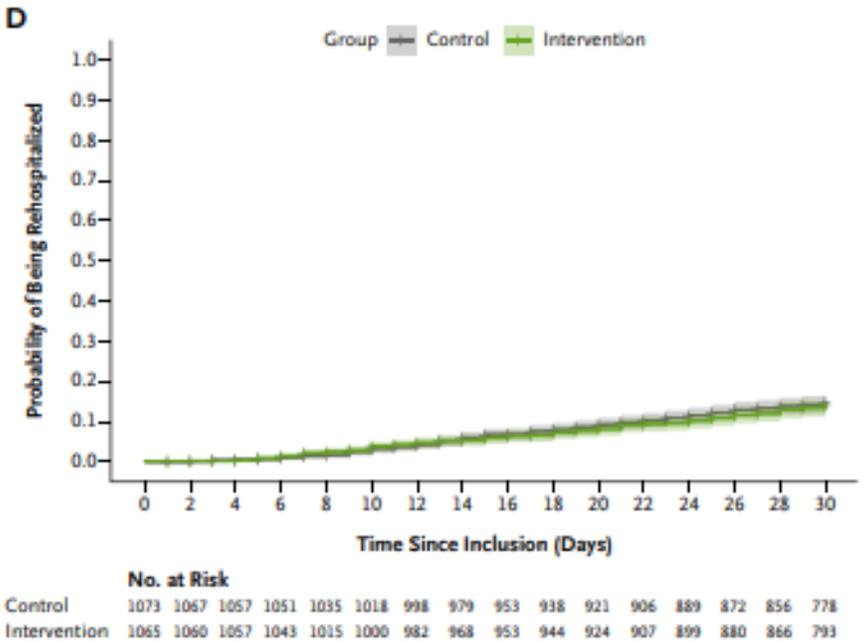
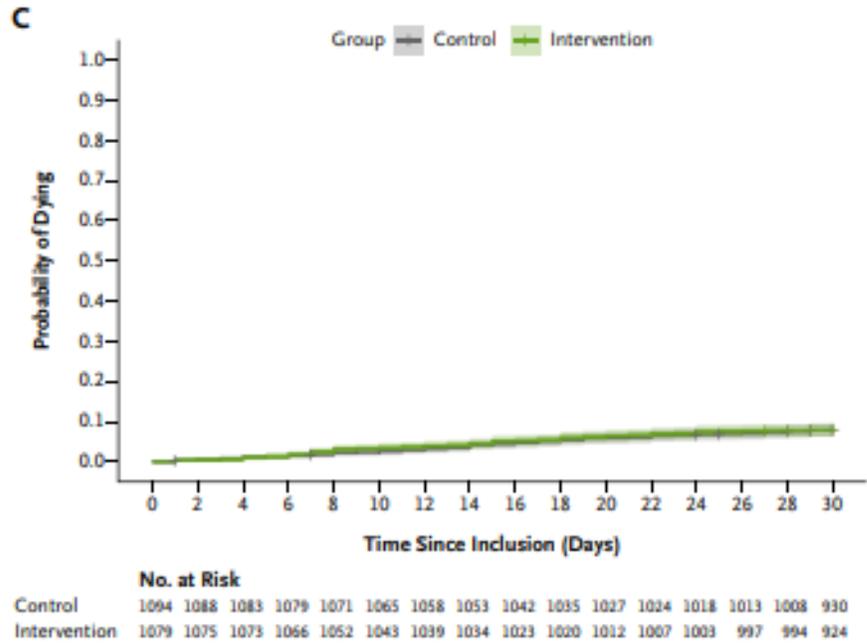
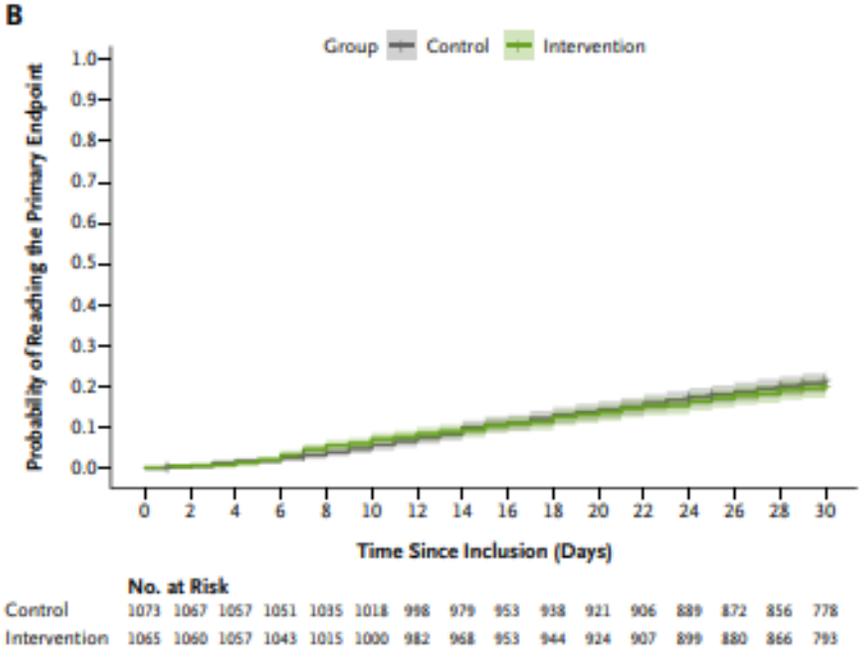
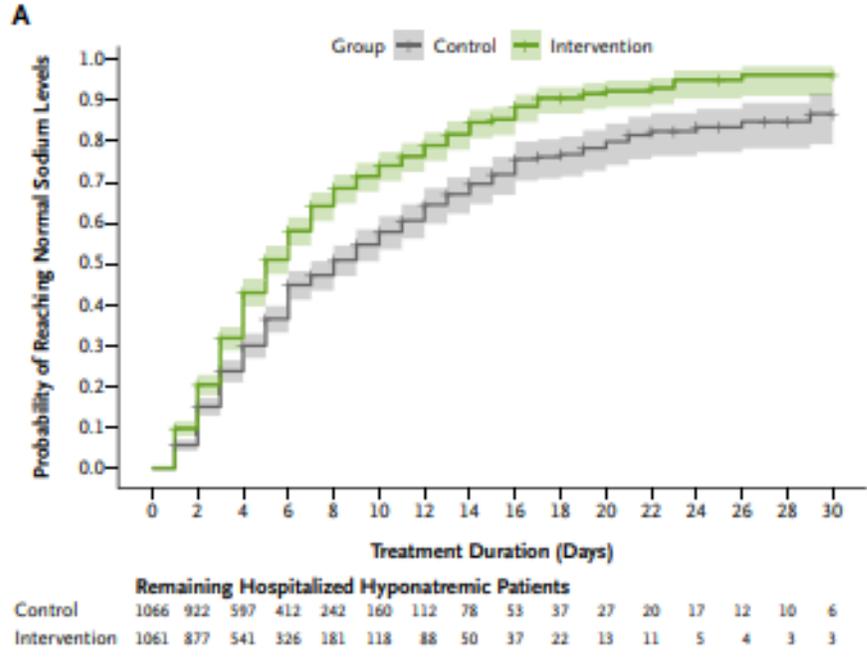


Wir bauen **Zukunft**. Für Ihre **Gesundheit**.

ORIGINAL ARTICLE

A Randomized Trial of Targeted Hyponatremia Correction in Hospitalized Patients

Julie Refardt, M.D., Ph.D.,^{1,2,3} Laura Potasso, M.D., Ph.D.,^{1,2,4} Anissa Pelouto, M.D.,³ Moritz Trappe, M.D.,^{5,6} Claudia Gregoriano, Ph.D.,⁷ Markus Koster, M.D.,⁸ Ivana Dora Vodanovic, M.D.,⁹ Dario Norello, M.D., Ph.D.,¹⁰ Svenja Ravioli, M.D.,^{11,12} Sadrija Cukoski, M.D.,^{5,6} Maria Boesing, M.D., Ph.D.,^{2,4} Basil Ryser, M.D.,¹¹ Lana Sambula, M.D.,⁹ Nikola Rapsch, M.D.,^{5,6} Sophie Monnerat, M.D., Ph.D.,^{1,2} Julia Beck, M.D.,^{1,2} Sven Lustenberger, M.D.,^{1,2} Deborah R. Vogt, Ph.D.,² Laura Werlen, Ph.D.,² Joyce Santos de Jesus, RN,^{1,2} Martina Bontognali, M.D.,⁸ Philipp Schuetz, M.D.,⁷ Adrienne A.M. Zandbergen, M.D., Ph.D.,³ Alessandro Peri, M.D., Ph.D.,^{10,13} Darko Kastelan, M.D., Ph.D.,⁹ Gregor Lindner, M.D.,^{11,12} Joerg Leuppi, M.D., Ph.D.,^{2,4} Stefan Bilz, M.D.,⁸ Beat Mueller, M.D.,⁷ Volker Burst, M.D.,^{5,6} Ewout J. Hoorn, M.D., Ph.D.,³ and Mirjam Christ-Crain, M.D., Ph.D.,^{1,2} for the HIT study investigators*



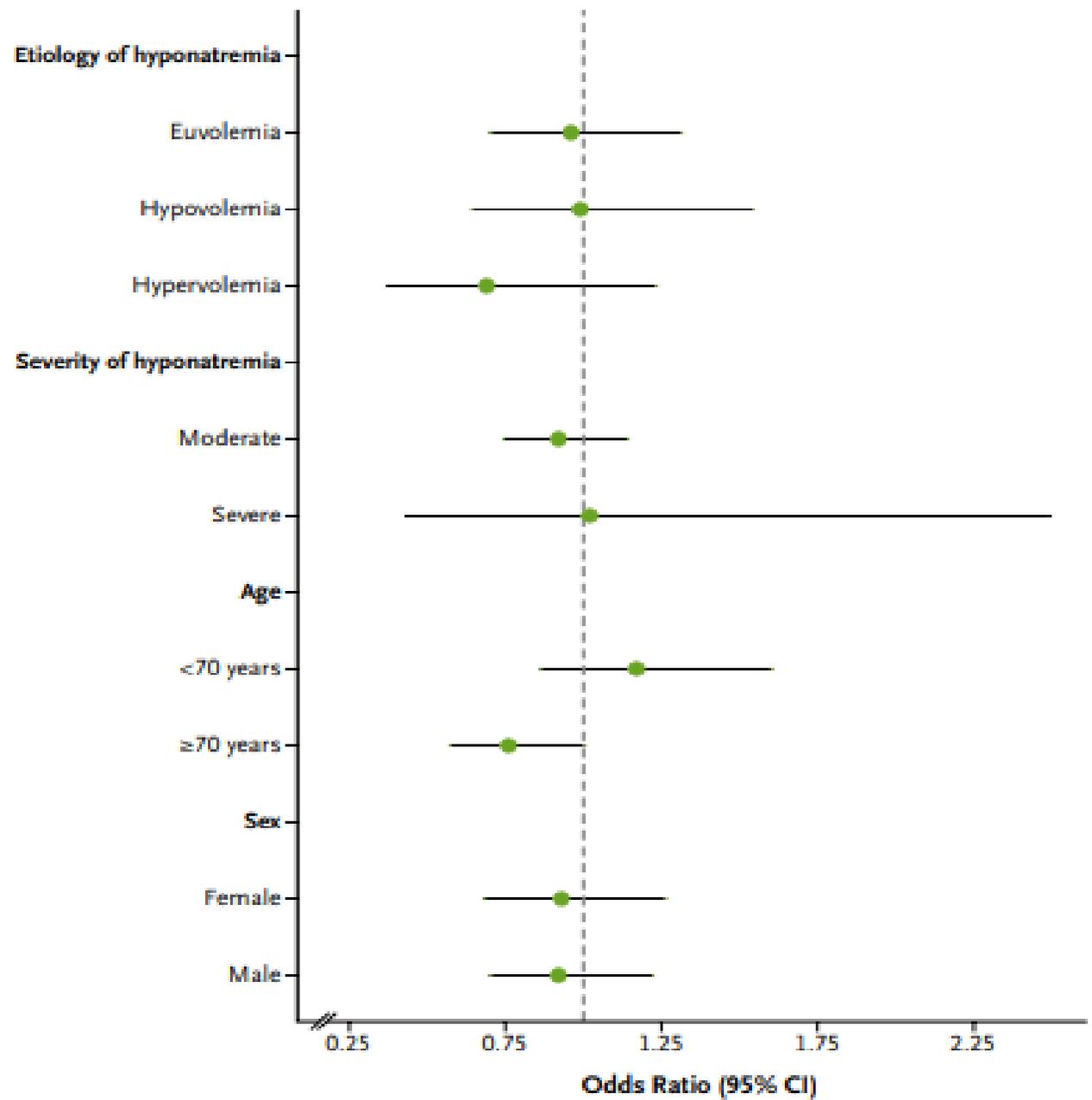


Table 2. Hyponatremia Treatment.*

	Overall (N=2173)	Control (N=1094)	Intervention (N=1079)
Main treatment of hyponatremia† — n (%)			
No treatment	411 (19)	384 (35)	27 (2.5)
Isotonic fluids	526 (24)	253 (23)	273 (25)
Fluid restriction	711 (33)	280 (26)	431 (40)
Oral urea	182 (8)	25 (2.3)	157 (15)
Vaptans	66 (3)	16 (1.5)	50 (4.1)
Other	238 (11)	123 (11)	115 (11)
Specific information missing	39 (2)	13 (1.2)	26 (2.4)
Number of different treatments used — n (%)			
0/missing information	450 (21)	397 (36)	53 (5)
1	781 (36)	363 (33)	418 (39)
2	537 (25)	207 (19)	330 (31)
3	259 (12)	87 (8)	172 (16)
≥4	146 (7)	40 (4)	106 (10)

In addition, our data provide no evidence for a correlation between center-specific rates for reaching normonatremia and the primary outcome (Fig. S4).

POST HOC ANALYSES

No differences in the primary outcome were observed between the main hyponatremia treatments (descriptive evaluation; Table S6). We note a possible association of the effect of the intervention with the Charlson Comorbidity Index (odds ratio, 0.9; 95% CI, 0.81 to 1.00). Reaching a normal plasma sodium level at discharge, regardless of the

treatment group, was associated with decreased odds of the primary outcome of death or rehospitalization within 30 days (odds ratio, 0.74; 95% CI, 0.60 to 0.91).

SAFETY ANALYSES

Overcorrection, defined as an increase in plasma sodium level of greater than 12 mmol/l in any 24-hour period or greater than 18 mmol/l in any 48-hour period, occurred in 25 of 1098 patients (2.3%) randomly assigned to the intervention and 16 of 1114 patients (1.4%) randomly assigned to the control group (P=0.14). Among these, 8 of

UreNa study: Urea vs Tolvaptan bei SIAD

